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Assessment of Public Health Risks Associated with Petrochemical Emissions Surrounding an Oil Refinery

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by

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DEDICATION

This dissertation is dedicated to my family and in loving memory of my mother, Karan L. Pulster, all of whom have made me who I am today. Persistence, perseverance and dedication, all in the name of science.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Refinery operations have been associated with a wide variety of atmospheric emissions consisting of criteria air pollutants, volatile organic components, hazardous air pollutants as well as other pollutants. With approximately 100 oil refineries in the Wider Caribbean region (WCR), hydrocarbons in this region pose significant environmental and human health risks. One of the oldest and largest refineries in the WCR is the Isla Refineriá, which is located on the island of Curaçao, and has been the basis of historical debates and conflict between the public and the local government over the environmental and human health risks. This research aims to establish baseline levels of ambient petrochemical emissions in Curaçao, specifically polycyclic aromatic hydrocarbons (PAHs), inhalable particulate matter (PM_{10}) and sulfur dioxide (SO_2), and to evaluate through comparative literature analysis and recommended public health guidelines the potential health risks in Curaçao. In addition, source elucidation of PAHs was conducted using concentration profiles, distribution profiles, binary diagnostic ratios and factor analysis. Passive air samplers with polyurethane foam collection disks (PAS-PUFs) were deployed in 2011 (n=43) and in 2014 (n=30) to measure ambient PAH concentrations. Ambient PAH concentrations ranged from 1.2 ng/m^3 in 2011 and 27.3 to 660.1 ng/m^3 in 2014, demonstrating no temporal differences. However, there were highly significant spatial differences, with the samples downwind of the refinery having significantly higher ambient PAH concentrations than those upwind in 2014. Source elucidation revealed the ambient PAHs were dominated by petrogenic emission sources (i.e., refinery) in the 2011 and the 2014 downwind samples, whereas the 2014 upwind locations were equally influenced by both petrogenic (i.e., refinery) and

pyrogenic (i.e., vehicle emissions) sources. Available hourly, daily and monthly PM_{10} and SO_2 measurements were downloaded from June 2010 through December 2014 from two local air monitoring stations. Concentrations of both PM_{10} and SO_2 in Curaçao are among the highest reported globally, demonstrating an increasing trend over time and exceed current public health guidelines recommended by local and international agencies. It is plausible that the residents of Curaçao may experience health effects often associated with PM_{10} and SO_2, however the epidemiological evidence is inadequate to infer causality between health effects and long-term exposures. Using the USEPA's risk analysis methodology the resulting cumulative lifetime cancer risk estimates from PAH inhalation were below the level of concern (1.0×10^{-4}). In contrast, by evaluating the potency adjusted concentrations relative to the most toxic compound (benzo[a]pyrene), age class (children and adults) extrapolated and site specific risks indicated levels exceeding the upper bound acceptable risk (1.0×10^{-4}) by almost two orders of magnitude suggesting the need for remediation.

CHAPTER ONE:

INTRODUCTION

1.1 Air Pollution

Air pollution may be considered a major environmental health risk having been associated with a number of acute (e.g., respiratory and cardiovascular events, hospital and emergency room admissions) and chronic (e.g., chronic bronchitis, lung cancer, mortality) effects [1]. Statistics and methods used as part of the Global Burden of Disease Study, including the Institute for Health Metrics and Evaluation and comparable risk assessment methodology, the World Health Organization (WHO) has estimated that 3.7 million premature deaths worldwide were attributable to ambient air pollution in 2012, with almost 90% of those occurring in middle-income countries [2]. Ischemic heart disease (40%) and stroke (40%), were among the highest deaths attributed to ambient air pollution, followed by chronic obstructive pulmonary disease (COPD, 11%), lung cancer (6%), and acute lower respiratory infections (3%). Furthermore, both experimental and epidemiological studies resulted in the 2013 International Agency for Research on Cancer (IARC) conclusion that ambient air pollution is carcinogenic to humans (Group 1) with increased cancer incidences mostly associated with particulate matter (PM)[3].

Air pollution having a similar classification as asbestos, benzene, tobacco smoke and polychlorinated biphenyls is disconcerting, however, it should be emphasized that the individual risk for developing cancer from air pollution is very low, yet the issue should be acknowledged and addressed by the international community. It is important to note, both international and domestic

air quality guidelines and classifications where primarily based on two U.S. cohort studies [4-6] that were intensely scrutinized for their study design limitations, such as confounding factors, biases, exposure characterization and statistical models [7]. Although, reanalyses of the data set replicated and validated the original findings of there being an association between particulate exposure and mortality, when applying new methods for spatial analysis and statistical modelling, modifying effects (i.e., socio-economic covariates) reduced the overall level of mortality risk [7]. However, consistent evidence of an association between ambient air pollution and increased mortality has been provided by the reanalyses of these two US cohort studies, as well as additional meta-analyses and cohort studies [7-14].

In many cases it is difficult to determine direct causality with one particular constituent of air pollution (PM, sulfur dioxide, carbon monoxide, VOCs, etc.) due to its complexity and multiple sources, such as traffic and industrial and refinery operations. More than 98% of air pollution in urban settings are gases or vapor-phase compounds such as carbon monoxide and non-methane hydrocarbons [15]. Due to these complexities, this study primarily focuses on two criteria pollutants (particulate matter and sulfur dioxide) and one hazardous air pollutant (polycyclic aromatic hydrocarbons) as a result of refinery emissions.

1.2 Criteria Air Pollutants: Particulate Matter and Sulfur Dioxide

1.2.1 Particulate Matter (PM)

Particulate matter (PM) is a type of air pollution that generally refers to a mixture of solid particles and liquid droplets that may consist of various sizes and composition which vary both spatially and temporally. Many factors contribute to the chemical composition of PM, including combustion sources, climate, season, and type of urban and/or industrial pollution [16]. Particulate

matter can be emitted from both natural and anthropogenic sources with both primary (directly emitted) and secondary (atmospherically derived) components [17]. Primary sources include wildfires, sea spray, organic matter and the combustion of both fossil fuels and biofuels. Secondary sources of PM include wood smoke, gaseous vegetative emissions and vehicular emissions. The major components of PM consist of semivolatile organic compounds, metals, reactive gases, carbonaceous material mainly from combustion and vehicle exhaust, biological material, and minerals. The components can be expanded further into fine and course particulate matter. Fine or respirable particulate matter may be composed of sulfate, nitrate, ammonium, hydrogen ions, elemental carbon, organic compounds (e.g., hydrocarbons, thiols, ketones, PAHs, etc.), heavy metals (e.g., lead, cadmium, vanadium, copper, zinc, etc.) and particle bound water. Course or inhalable particulate matter may be composed of resuspended dusts, sea salt, mold spores, pollen and miscellaneous airborne debris. The density, concentration, and composition of particulate matter can vary widely across clean air to densely polluted air.

Particle size is directly linked to their potential for causing health problems and can be classified into respirable, inhalable and total dust particles. Respirable particles typically refers to those small enough (<2.5 μm) to easily penetrate the upper airways and respiratory system and deep into the lungs where it is readily absorbed through alveolar membranes. Inhalable (or thoracic) particles (<10 μm) are considered to be able to enter the body but typically get trapped in the upper airways and respiratory tract. Total dust particles include all airborne particles regardless of size and composition. Inhalable particles (2.5 and 10 μm) are capable of penetrating into the lower respiratory tract, with increasing airway penetration with decreasing particle size. Large particle sizes >10 μm are considered to be the least concern as they typically are trapped in the upper airways with little or no penetration into the lungs. In contrast, the smaller inhalable (< 10 μm) and respirable (<2.5 μm) particles pose the greatest problems, since they are able to penetrate deep into the lungs, and some

potentially enter the bloodstream. For particulate monitoring and modelling purposes, respirable (<2.5 μm) and inhalable (< 10 μm) particles are referred to as $PM_{2.5}$ and PM_{10}, respectively. Inhalable particulate matter (< 10 μm) is the principle focus of this study and following discussion.

Recently, inhalation and atmospheric pollution studies have focused on the particulate matter due to the strong correlation of mortality and adverse respiratory health effects compared to any other atmospheric gas [16]. It has been suggested that up to 8% of premature mortalities globally are due to both indoor and outdoor concentrations of particulate matter [2, 7, 18, 19]. A number of studies have illustrated a strong association between long-term exposure to PM and various health effects including accelerated cardiovascular and respiratory mortality, compromised lung function and relative increase of lung cancer risk [9, 15, 20-22]. Inhalable particulate matter (PM_{10}), specifically, has been associated with increases in daily mortality and hospital admissions for respiratory distress (pneumonia, asthma and decreased lung function in children) [21, 23].

Epidemiological studies in Europe and the United States have shown that with each 10 $\mu g/m^3$ increase in PM_{10} all-cause daily mortality increased by 0.5-0.6%, COPD and asthma in people aged 65 or older increased by 1-1.5%, and cardiovascular diseases increased by 0.5 – 1.1% [16]. A recent meta-analysis conducted in China reported that for each 10 $\mu g/m^3$ increase in PM_{10} the mortality risk due to total non-accidental mortality, cardiovascular disease and respiratory disease increased by 0.36%, 0.36% and 0.42%, respectively [24]. An estimated 2.8% increase in mortality and a 6.15% increase in hospital admissions for children in Italian provinces have also been associated with each 10 $\mu g/m^3$ increase in PM_{10} [25]. In addition, global associations were observed between increases in heart failure mortality or hospitalizations (1.63%) and stroke mortality or hospitalizations (0.58%) with each 10 $\mu g/m^3$ increase in PM_{10} [26, 27]. Furthermore, the results from a meta-analysis of 19 cohort studies from around the world demonstrated that PM_{10} was associated with a significant increased risk of lung cancer mortality [9].

Toxicological evidence has shown that PM has several mechanisms (e.g., oxidative stress, mutagenicity, DNA oxidative damage, pro-inflammatory) of adverse cellular effects associated between increasing cellular toxicity with decreasing particle size; thus suggesting associations between chemical composition and particle toxicity are stronger for fine and ultrafine PM [16]. The oxidative potential of fine and ultrafine particles have been shown to ultimately be the result of significant amounts of organic carbon compounds, such as PAHs and quinones [16].

Current guidelines available for ambient outdoor and work place air quality are available from the National Ambient Air Quality Standards (NAAQS), American Conference of Governmental Industrial Hygienists (ACGIH), the National Institute for Occupational Safety and Health (NIOSH) and Occupational Safety and Health Administration (OSHA). Up until 2006, the NAAQS recommended the mean annual ambient air concentrations should not exceed 50 $\mu g/m^3$ for PM_{10}, however this has since been revoked and replaced with a 24-hour maximum that should not exceed 150 $\mu g/m^3$ for PM_{10}, more than once a year, on average over three years. The European Commission recommends the mean annual ambient air concentrations do not exceed 40 $\mu g/m^3$ for PM_{10}. In addition, a 24 hour limit was set at 50 $\mu g/m^3$ with 35 allowable exceedances per year.

1.2.2 Sulfur Dioxide (SO_2).

Sulfur dioxide is one of the six criteria pollutants regulated by the USEPA for which they have developed human health-based and/or environmentally-based criteria for setting permissible limits under the Clean Air Act. Atmospheric SO_2 is primarily the result of anthropogenic activities associated with the burning of fossil fuels and industrial processes (i.e., oil refineries, coal burning, and biomass combustion) but can also be released naturally from volcanic activities, sea-salt emissions and sulfur gas oxidation [28-30]. Due to its high vapor pressure (3,000 mm Hg at 20°C), SO_2 in the atmosphere is primarily found as a colorless gas with a pungent odor and is the main source of acid precipitation

resulting in significant environmental consequences. Sulfur dioxide (SO_2) can be formed from the petroleum refining process and smelting industries, accounting for 15-25% of the estimated 140-350 million tons of sulfur compounds (i.e., sulfur dioxide, sulfuric acids and sulfate) released into the atmosphere annually on a global basis [28, 29, 31]. Typical annual average concentrations of sulfur dioxide, as of 2005, have ranged from 9-35 $\mu g/m^3$ in North America, 8-36 $\mu g/m^3$ in Europe, 40-70 $\mu g/m^3$ in Latin America, 10-100 $\mu g/m^3$ Africa and 20-200 $\mu g/m^3$ in Asia [19].

Sulfur dioxide has been associated with various morbidities since the controlled exposure experiments conducted in the early 1950's [32, 33]. Epidemiological studies have consistently demonstrated that sulfur dioxide causes respiratory irritation, bronchoconstriction and has the potential of causing respiratory and pulmonary changes and cardiovascular abnormalities in both healthy and asthmatic individuals [33-37]. The controlled experiments in the 1950s examined the effects of SO_2 inhalation using controlled exposure experiments on healthy individuals. These studies revealed considerable inter-individual variability among healthy individuals however bronchoconstriction responses in most were induced at levels approaching 5 ppm (10,480-13,100 $\mu g/m^3$). The short term effects from sulfur dioxide exposure at much lower and more plausible episodic concentrations between 0.20 and 1 ppm (524 - 2,620 $\mu g/m^3$) were also evaluated using controlled chamber experiments in normal, atopic and asthmatic volunteers [37, 38]. Normal and atopic volunteers showed little response at these levels, whereas some atopic volunteers and most of the asthmatics developed bronchoconstriction and respiratory symptoms. Even at elevated ventilation during exercise, there has been limited evidence of SO_2 induced respiratory effects in normal, healthy subjects following short-term exposures of $\leq$ 1ppm (2,620 $\mu g/m^3$) [33, 36-38]. Bronchoconstriction and compromised lung function tend to occur at lower concentrations ($\leq$0.4 ppm or 1,048 $\mu g/m^3$) in asthmatic and some atopic individuals, with some reports of symptoms occurring as low as 0.10 ppm (262 $\mu g/m^3$) when combined with exercise or another irritant (*i.e.,* ozone) [35-37, 39-41]. The

respiratory responses in healthy, atopic or asthmatic subjects can also be potentiated by exercise and oral ventilation [36, 37, 41-43].

Significant associations between SO_2 and mortality have also been observed in several studies. Data collected as part of an ongoing morality study by the American Cancer Society, consisting of over a million US adults, reported sulfur oxide pollution (SO_2 and sulfate particulates) at mean standard concentrations between 17.5 – 25.4 $\mu g/m^3$ (6.7 - 9.7 ppb) were significantly associated with all-cause mortality, cardiopulmonary and lung cancer mortality [5]. In Korea, stroke mortalities were reported to be significantly associated (RR = 1.04, 95% CI 1.01 - 1.08) with each increase (17.43 ppb or 45.6 $\mu g/m^3$) of SO_2 [44]. With each 26.2 $\mu g/m^3$ (10 ppb) increase in SO_2, global associations between air pollution and increases in heart failure mortalities or hospitalizations (2.36%; 95% CI 1.35-3.38) and stroke mortalities or hospitalizations (1.53%; 95% CI 0.66-2.41) were observed [26, 27]. With each increase of 50 $\mu g/m^3$ (19 ppb) of SO_2 an association was demonstrated with a 0.8-3% increase in daily mortality during multicity analysis in Europe [45]. Short-term exposures to SO_2 also illustrated an association with increased total mortality (0.75%), cardiovascular mortality (0.83%), and respiratory mortality (1.25%) with each SO_2 increase of 10 $\mu g/m^3$ [46]. Evidence has suggested that excess mortality may occur with 24 hour exposures to mean SO_2 concentrations exceeding 500 $\mu g/m^3$ (~191 ppb)[47].

Both NIOSH and OSHA have established 13 mg/m^3 (5 ppm) as the short term exposure limit (STEL) and the permissible exposure limit (PEL) as an 8-hour time weighted average. The recommended exposure limit (REL-TWA) and the threshold limit value (TLV-TWA) has been set at 5-5.2 mg/m^3 (2 ppm) by both the ACGIH and NIOSH. The SO_2 mean annual (80 $\mu g/m^3$; 30 ppb) and 24-hour (365 $\mu g/m^3$; 139 ppb) ambient air standards previously proposed by the USEPA (NAAQS) have since been revoked. Currently the NAAQS recommends a one hour maximum of 196 $\mu g/m^3$ (75 ppb) averaged over three years and a three hour maximum of 1300 $\mu g/m^3$ (~500 ppb)

not to be exceeded more than once per year. The European Commission has set a 24-hour exposure limit of 125 $\mu g/m^3$ with three permissible exceedances per year. International standards set by the World Health Organization recommends mean annual ambient air concentration do not exceed 40-60 $\mu g/m^3$ (~15-23 ppb) with a 24-hour exposure limit of 100-150 $\mu g/m^3$ (~40-60 ppb).

1.3 Hazardous Air Pollutants: Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) are relatively minor constituents (0.2 - 7%) in crude oil and are among the various hazardous air pollutants emitted by oil refineries, however, they appear to be the most toxic [48]. Although there is limited information available on refinery contributions of ambient PAH concentrations, IARC has classified PAH exposure as a result of petroleum refining as a probable human carcinogenic (Group 2A). In 2011, over 10,000 pounds of PAHs were emitted from petroleum refineries in the United States alone [49]. Global industrial emission rates of PAHs have been estimated to range from 2 μg/kg for benzo[a]pyrene from industrial stacks to over 13,000 μg/kg for low molecular weight PAHs from industrial boilers [50].

Polycyclic aromatic hydrocarbons are a group of non-polar, lipophilic, organic compounds composed of two or more fused aromatic rings that are predominantly anthropogenic but can be found in natural fuel deposits (i.e., crude oil), as well as volcanoes and natural fires. PAHs are formed when organic matter containing carbon and hydrogen is exposed to temperatures 700°C or greater and can occur during pyrolytic processes and incomplete combustion, for instance the incomplete burning of organic substances such as coal, oil, gas, diesel, wood, garbage, or other organic substances (e.g., tobacco and charbroiled meat).

Atmospheric PAHs are primarily from the direct release of natural and anthropogenic sources, with anthropogenic emissions predominating. Natural sources of atmospheric PAHs are primarily from residential wood burning, forest fires and volcanoes. Important anthropogenic (stationary)

sources consist of asphalt, coal tar and coke production, petroleum and aluminum production as well as mobile sources, vehicle and aircraft exhaust. Approximately 80% of the total annual PAH emissions are the result of stationary sources with mobile sources (gasoline and diesel engines) accounting for the remaining 20% [51].

Populations are believed to have the greatest exposure to PAHs through either active or passive inhalation of the compounds in tobacco smoke, wood smoke, wild fires, and air pollution, and ingestion of the compounds in food [52]. In addition, drinking water, grilled or smoked foods, contact with soot and tars, as well as residential areas near hazardous waste sites are also potential exposure routes of higher than background levels of PAHs. The average total daily intake of PAHs by a member of the general population has been estimated to be 0.207 μg from air, 0.027 μg from water, and 0.16-l .6 μg from food [52].

There are more than 100 different PAHs that vary considerably in physical and chemical properties. Table 1.1 illustrates the different physical and chemical characteristics of the PAHs measured in this study. PAHs are often found in complex mixtures whose composition depends on the raw material and the combustion circumstances. Therefore, carcinogenic effects of both individual PAHs and PAH mixtures are difficult to assess [53]. Regulatory agencies typically assess risks posed by mixtures of PAHs by assuming that all carcinogenic PAHs are as potent as benzo[*a*]pyrene (B[*a*]P), one of the most potent PAHs, as well as, one of the most studied. The available information on the toxicity of the PAHs suggests that most are considerably less potent than B[*a*]P and therefore, the EPA approach is likely to overestimate risks [54, 55]. Considering PAHs are typically found in complex mixtures, this approach could also underestimate the risk given the current calculations do not account for possible additive or synergistic effects.

The toxicity of the PAHs is highly structurally dependent, and isomers may therefore vary from being nontoxic to very toxic [56]. The PAHs known for their carcinogenic, mutagenic and

teratogenic properties are benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, benzo[*ghi*]perylene, coronene, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene and ovalene. Benzo[*a*]anthracene, benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, chrysene, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene have been classified as either a possible or probable human carcinogen by a number of agencies [52]. These carcinogenic PAHs are mainly comprised of the 4 to 6 ring compounds.

The history of PAHs date back to 1775 when Sir Percival Pott found the first association between high rates of scrotal cancer and exposure to fireplace soot in London Chimney sweeps [52]. During the 1930's and later, benzo[a]pyrene was identified in domestic soot, urban air pollution, motor vehicle exhausts and cigarette smoke [57]. Since then, other coal tar-related cancers have been induced in laboratory animals and found in humans [58, 59]. In 1947, the relationship between lung cancer and working conditions of gas industry workers and those working with coal tar was established [58]. Twenty years later, a study among gas workers in England and Wales determined the incidence of bronchitis and the lung cancer death rate of coal carbonization processors was found to be 126% and 69% higher than the national rate, respectively [60, 61]. In addition, there have been an increasing number of occupational cohort studies illustrating skin, lung, bladder, kidney and larynx cancers associated with a number of coal tar, pitch, soot and other PAH mixtures [53, 62, 63].

Animal inhalation studies illustrated a significant increase of lung tumors and a dose-dependent incidence of malignant lung tumors in newborn mice exposed to enriched PAH emissions containing 50 and 90 $\mu g/m^3$ of benzo[a]pyrene, 2-3 times the levels observed in older coke plants [64]. A chronic inhalation study using hamsters also demonstrated a dose-response relationship between respirable B[a]P particles and respiratory tract tumorigenesis [65]. A recent panel study of asthmatic children living in an urban industrial area in the proximity to two oil refineries suggested a small decrease in pulmonary functions with mean personal PAH levels of 151 $\mu g/m^3$ [66]. Other

epidemiology studies evaluating lung cancer and respiratory disease have shown geographic gradients with highest mortality in areas closest to petrochemical, steel and other industrial areas [67]. Where human inhalation studies are lacking, molecular epidemiological studies using biomarkers to evaluate genotoxic effects of PAHs are on the rise. Increased lymphocyte PAH-DNA adducts, doubling frequency of *ras* oncogene over expression, sister chromatid exchanges, and chromosomal aberration were noted in individuals residing in highly industrialized regions of Poland with ambient B[a]P concentrations ranging from 15-66 $\mu g/m^3$ [68]. Moreover, the aromatic DNA adducts were reported to be significantly correlated with chromosomal mutations, providing a molecular link between environmental exposure and genetic mutations relevant to cancer and reproductive risk. The detection of genotoxic effects using biomarkers in low level exposures (<20 ng/m^3) is limited. However, a dose-response relationship was reported in foundry workers exposed to B[a]P levels ranging from 2 – 60 ng/m^3 illustrating an increasing trend in PAH-DNA adducts [69].

The permissible exposure limit (PEL) for PAHs in workplace air is 0.2 milligram/cubic meter (mg/m^3) was established by the Occupational Safety and Health Administration (OSHA), however they have not established individual PAH standards for occupational exposure. The OSHA-mandated PAH workroom air standard is an 8–hour time-weighted average (TWA) measured as the benzene-soluble fraction of coal tar pitch volatiles. The OSHA standard for coke oven emissions is 0.15 mg/m^3. The National Institute for Occupational Safety and Health (NIOSH) has recommended that the workplace exposure limit for PAHs be set at the lowest detectable concentration, which is 0.1 mg/m^3 for coal tar pitch volatile agents at the time of the recommendation. In addition, the World Health Organization's risk estimate for ambient air concentrations of PAHs has suggested a lifetime exposure guideline value of 0.1 ng/m^3 B[*a*]P as an indicator and 2 ng/m^3 of fluoranthene as a secondary indicator [70]. Although International and National regulations tend to be in agreement,

for the most part, on maximum allowable levels of PAHs in air and water, state to state regulations and guidelines seem to vary tremendously.

1.4 Passive Air Sampling (PAS)

Passive air sampling has previously been shown to be an appropriate substitute for active sampling techniques in a number of regional and global atmospheric PAH monitoring studies [71-74]. Consequently, PAS using PUFs (PAS-PUF) is the most widely used technique for monitoring PAHs in both environmental (outdoor) and occupational (indoor) settings [71, 73]. PAS-PUFs are particularly attractive samplers as an alternative to active samplers, as they are cost effective, easy to handle and transport (ideal sampler for a developing country), do not require electricity and can yield concurrent time-integrated measurements in locations where active samplers would not be practical over such periods.

Passive sampling is based on the law of diffusion and has been validated as a semi-qualitative method for measuring PAHs in a number of indoor and outdoor studies (regional and global) through the simultaneous deployment of passive and active monitors and chamber calibration studies [73-78]. Calibration experiments have allowed for the generation of sampling rates, albeit not site specific. The sampling rates of both gas and particulate phases PAHs may be influenced and dependent on the physical and chemical properties and environmental variables such as temperature and wind speed [79-81]. Sampling rates are thought to be higher in colder temperatures due to increased wind speeds, however the variability in sampling gas phase compounds was found to be fairly low over typical field conditions.

The PAS-PUF uptake of a chemical from the ambient air is best described by the effective concentration gradient between the air and the sampler and follows the equation:

$$V_S \frac{dC_S}{dt} = k_O A_S (C_A - \frac{C_S}{K_{SA}})$$

Where V_s is the sampler volume, C_s is the analyte concentration in sampler, C_A is the air concentration of the analyte, k_O is the overall mass transfer coefficient, A_s is the sampler surface area, and K_{SA} is the sampler air/partitioning coefficient [82, 83].

Atmospheric PAHs are present in both gaseous and particulate phases, with more than 98% of air pollution in urban settings consisting of the gases or vapor-phase compounds [15]. Previous studies indicate that the low molecular weight (3 -4 and 5 ring) PAHs tend to be more concentrated in the vapor or gaseous phase while the high molecular weight (5 to 6 ring) PAHs are associated with particulates [84, 85]. However, it has been shown that high molecular weight PAHs tend to have increased partitioning to the gas phase in warmer temperatures. He & Balasubramanian [86] found higher fractions of the high molecular weight PAHs in passive samplers in a tropical environment. This study also determined concentrations and patterns of PAHs were not statistically different between actively and passively collected samples. Melymuk et al [83] also provided evidence that PAS-PUFs are sampling a significant portion of the of the particle-phase from calibration and comparison studies. These recent studies support the concept that PAS-PUF samples both the gas and particulate air phases and should be considered representative of bulk air concentrations [83, 86]. In this respect, passive air sampling of PAHs may be more quantitative for both gas and particulate phases in warmer climates.

1.5 Overview of the Oil Refinery Process and Emissions

Geological and geochemical processes generate naturally occurring crude oil which can be processed to derive a variety of petroleum products. Both crude oil and its derived petroleum products are comprised of ~97% hydrocarbons and the remaining 3% of the minor elements nitrogen,

sulfur and oxygen [48]. Generally, the hydrocarbons found in crude oil are characterized by their structure and include saturates, olefins, aromatics and polar compounds. Oil refineries are complex processing plants designed to separate, convert and treat the complex hydrocarbons of crude oil into a variety of products and useable fuels [87].

The refinery process is a multi-step process consisting of distilling, cracking, reforming, blending, and treating resulting in over 2500 refined products such as gasoline, propane, diesel fuel, jet fuel, kerosene, asphalt and fuel and lubricating oils. The first step in the refining process begins with the cleaning, desalting and heating of crude oil until only the waxy residual hydrocarbons remain in liquid form. Distilling essentially separates crude mixtures by either boiling or vaporizing crude oil in fractionating towers. Since each hydrocarbon has different boiling points this allows for the boiling, condensing and collecting of different hydrocarbons at different temperatures controlled within the distilling and fractionating towers. The next step is designed to increase the conversion of hydrocarbons to maximize the amount of gasoline through cracking. Cracking splits long carbon chains of heavy gas oil into shorter hydrocarbon chains (e.g., gasoline) which can then enter the reforming step, geared towards increasing the volume of gasoline produced per barrel of crude. Reforming uses catalytic reactors to rearrange naphtha hydrocarbons into gasoline molecules. The resulting products are further blended and treated to increase their quality by removing impurities. Hydrogen and hydrocarbons are simultaneously heated in a reaction chamber with a catalyst to strip sulfur from hydrocarbons forming hydrogen sulfide which is then removed and neutralized, and the resulting sulfur compounds can be used in other applications such as fertilizers or pharmaceuticals.

Refinery operations have been associated with atmospheric emissions of a wide variety of criteria air pollutants (i.e., sulfur dioxides, nitrogen oxides, carbon monoxide and particulate matter), volatile organic components (i.e., benzene, toluene, ethylbenzene, m-xylene), hazardous air pollutants (i.e., polycyclic aromatic hydrocarbons, carbon monoxide, hydrogen cyanide, mercury), and other

pollutants (i.e., greenhouse gases, hydrogen sulfide). The type and quality of the crude oil, refinery process and refined products all influence the variability, composition and amount of emissions from one refinery to another.

1.6 The Presence of Oil Refineries in the Wider Caribbean

The Wider Caribbean Region (WCR) includes a number of developing countries for which agriculture, oil and gas exploration in conjunction with processing, provide valuable sources of income. However, these factors have created levels of environmental pollution that are of concern regionally, even in the absence of empirical data to document levels and effects of contamination on the health of the environment (e.g., coral reefs), fishery resources and other wildlife, as well as humans. As the WCR is one of the most tourism-dependent regions of the world, factors that affect environmental health and sustainability will have inevitable impacts to the economies and quality of life in many already-needy countries.

The presence of hydrocarbons (i.e., polycyclic aromatic hydrocarbons - PAHs) in this region is one of the most significant threats or potential risk factors to environmental and human health [88, 89]. Industrial point sources contribute 90% of the oil pollution loads entering the WCR coastal areas, mainly from approximately 100 oil refineries operating in this region [90]. One of the largest and oldest refineries in the WCR, Isla Refineriá, opened in 1918 and is located within the densely populated capital of Willemstad, Curaçao on the shores of Schottegat Bay. Although, the refinery was considered obsolete in the mid-1980s, it is still in use today processing ~335,000 barrels per day (www.PDVSA.com) yet it has not been able or required to comply with environmental standards and permit requirements [91].

A legacy of human health and environmental issues is the basis of a historical debate and conflict between the public and the local government of Curaçao. Communities downwind of Isla

Refineriá and the major thoroughfare circling the refinery and the bay, Schottegatweg Ring, are reported to experience higher than average frequencies of headaches, nausea, chronic lung ailments, asthma and cancer. Most of these health complaints originate from local schools. The degree of acute health effects from emissions has been described as fitting the health-scale for disaster response [92]. Sanhueza et al. [93] determined that all areas downwind (≥ 5 km) of the Isla Refineriá in Curaçao were subject to sulfate contamination exceeding levels associated with morbidity (≥ 8-12 $\mu g/m^3$). In addition, the 2007 yearly average SO_2 levels (152 $\mu g/m^3$) measured downwind of the refinery were double the air quality standards for Curaçao (80 $\mu g/m^3$) and exceeded acceptable international guidelines (40-60 $\mu g/m^3$) by almost 2.5 times [47, 94]. In 2009, a court order required the refinery to reduce the excessive SO_2 emissions and particulates starting January 1, 2010.

Anecdotal information from court proceedings inferred that the refinery insists the adverse health effects are caused from heavy motor vehicle traffic emissions from the major thoroughfare alongside the refinery (P. Hoetjes, MINA, personal communication). However, a lack of concrete data makes it difficult to assess the impact that both motor vehicle traffic and refinery emissions have on human health and the marine environment. The major unresolved dispute in Curaçao is whether the petrochemical emissions are solely due to motor vehicle emissions or whether the major contribution comes from one of the largest oil refineries in the WCR, Isla Refineriá [95]. There is no question that motor vehicular emissions (MVE) has become an increasingly dominant contributor to air pollution globally [96]. The adverse health effects associated with elevated exposures to MVEs near busy roadways has emerged as a significant public health concern [96, 97].

The principal air pollutants associated with vehicle combustion engine sources are carbon monoxide, nitrogen oxides, particulate matter, ozone, and black carbon [96, 97]. Motor vehicle emissions have been associated with increased risk for multiple adverse health effects including asthma and allergic diseases, cardiac effects, respiratory symptoms, reduced lung function, growth,

reproductive impairment, premature mortality, and lung cancer [97-101]. Two main questions need to be answered with regard to petrochemical emissions and the public health of Curaçao: First, what is the main source of atmospheric petroleum constituents contributing to the human health problems, motor vehicular emissions (MVE) or refinery emissions? Second, what are the human health risks based on current levels of air pollution?

Given the significance of this current public health risk in Curaçao, there is an urgent need to (a) verify the point source of the petrochemical emissions; (b) establish baseline levels and extent of contamination, (c) conduct a formal human health and environmental risk assessment, and (d) initiate appropriate mitigation measures [102]. To date, these important steps have not been undertaken. The amount of chronic exposure to petrochemical emissions in Curaçao and other parts of the WCR experience and the scarcity of data in this region warrant the imminent development for studies in this region.

1.7 Research Objectives

Exposure assessments are the first critical step for many applications, including compliance with legal standards, disease diagnosis and treatment, risk assessment and management, and occupational and environmental epidemiology. The underlying assumption is that there is a causal relationship between the amount of exposure and the extent of the observed health effect [103]. This project will focus on conducting the first step to a much larger human health and environmental risk assessment that is needed on the island of Curaçao (Figure 1.1). The primary objectives of this study were to:

1) Deploy passive air samplers around the major thoroughfare and along transects that extend radially west of Isla Refineriá to establish baseline levels and the extent of select petrochemicals (i.e., PAHs) in air samples in Willemstad, Curaçao;

2) Evaluate measured levels of inhalable particulate matter (PM_{10}) and sulfur dioxide (SO_2) from refinery operations;
3) Reveal sources of the ambient PAHs in Curaçao (i.e., vehicular vs. refinery emissions) by using concentration profiles, distribution profiles, diagnostic ratios and factor analysis;
4) Determine if spatial trends exist by identifying areas with the highest impact from emissions;
5) Determine if temporal trends exist for PAHs, SO_2 and PM_{10};
6) Verify if levels exceed current public health guidelines for petrochemical emissions.

Specific hypotheses of this research are as follows:

1) Spatial trends in petrochemical emissions will exist with the highest levels being measured downwind of Isla Refineriá;
2) Emission profiles will be indicative of refinery operations as the primary point source;
3) Temporal trends will exist, with levels increasing over time;
4) Ambient concentrations of petrochemical emissions downwind of the refinery will exceed current acceptable guidelines.

Table 1.1 Select physical and chemical characteristics of PAHs measured in this study.

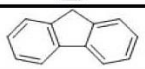
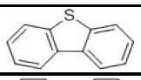
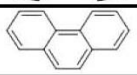
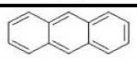
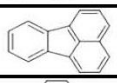
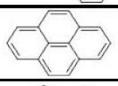
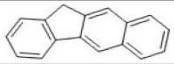
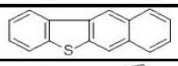
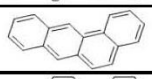
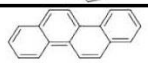
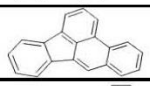
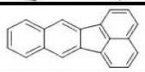
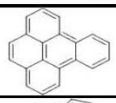
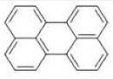
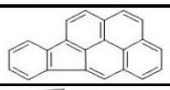
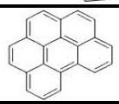
PAH	Chemical formula	Molecular Weight	Boiling Point (°C)	Vapor Pressure (mmHg)	Chemical Structure
Naphthalene	$C_{10}H_8$	128.17	218	0.03 at 25°C	
Acenaphthylene	$C_{12}H_8$	152.19	280	0.029	
Acenaphthene	$C_{12}H_{10}$	154.21	279	$4.47x10^{-3}$	
Fluorene	$C_{13}H_{10}$	166.22	298	$3.2x10^{-4}$ at 25°C	
Dibenzothiophene	$C_{12}H_8S$	184.26	332	0.0±0.7 at 25°C	
Phenanthrene	$C_{14}H_{10}$	178.23	340	$6.8x10^{-4}$ at 25°C	
Anthracene	$C_{14}H_{10}$	178.23	340	1 at 25°C	
Fluoranthene	$C_{16}H_{10}$	202.25	384	$5x10^{-6}$ at 25°C	
Pyrene	$C_{16}H_{10}$	202.25	393	$2.5x10^{-6}$ at 25°C	
Benzo[B]fluorene	$C_{17}H_{12}$	216.28	402	0.0±0.4 at 25°C	
Naphthobenzothiophene	$C_{16}H_{10}S$	234.32	434	0.0±0.1 at 25°C	
Benzo[A]anthracene	$C_{12}H_{12}$	228.29	438	$2.2x10^{-8}$ at 20°C	
Chrysene	$C_{18}H_{12}$	228.29	448	$6.3x10^{-7}$ at 25°C	
Benzo[B]fluoranthene	$C_{20}H_{12}$	252.31	467	$5x10^{-7}$ at 20-25°C	
Benzo[K]fluoranthene	$C_{20}H_{12}$	252.31	480	$9.59x10^{-11}$	
Benzo[E]pyrene	$C_{20}H_{12}$	252.31	312	$5.7x10^{-9}$ at 25°C	
Benzo[A]pyrene	$C_{20}H_{12}$	252.31	495	$5.6x10^{-9}$	
Perylene	$C_{20}H_{12}$	252.31	400	0.0±0.6 at 25°C	
Indeno[1,2,3,-cd]pyrene	$C_{22}H_{12}$	276.33	530	10^{-11}- 10^{-6} at 20°C	
Dibenzo[a,h]anthracene	$C_{22}H_{14}$	278.35	524	$1x10^{-10}$ at 20°C	
Benzo[g,h,i]perylene	$C_{22}H_{12}$	276.33	>500	$1.03x10^{-10}$ at 25°C	

Figure 1.1 Global orientation for the island of Curaçao.

CHAPTER TWO:

RESEARCH METHODS

2.1 Site Selection and Study Design

Curaçao is an island in the southern Caribbean, ~40 miles off the Venezuelan coast. It is currently considered a constituent country of the Kingdom of the Netherlands since its dissolution in 2010 from the Netherland Antilles. Curaçao is located in the Southern Caribbean Dry Zone, which is characterized by a semi-arid to arid climate, with a distinguishable dry and rainy season, and sustained easterlies. The island is approximately 59 kilometers in length, 4 - 11 kilometers wide and a total land mass area of ~443 km^2. The population of ~152,000 consists of greater than 50 nationalities with Dutch and Papiamento as the official languages. The majority of the population (>130,000) resides in Willemstad which is home to the Isla Refineriá.

To address the extent of the emissions, 15 sites were selected in 2011 based on their proximity to Isla Refineriá, Schottegatweg Ring, and along westerly transects from the ring outward to approximately 6 km west (downwind) of the refinery (Table 2.1; Figure 2.1). In addition, the three major communities (Heintje Kool/Buena Vista, Marchena/Wishi, Habaai) involved in the 1999 Environmental Service of Curaçao Health Assessment were included in this study [92]. This 1999 assessment was conducted as a result of health complaints from communities directly exposed to refinery emissions. Passive air samplers containing polyurethane foam collection substrates (PAS-PUFs) were deployed in 13 residential neighborhoods or geozones with a total estimate of 12,000 residents (Table 2.2). In 2014, 30 sites were selected around the entire Schottegatweg Ring to address spatial trends, encompassing approximately 27,000 residents (Table 2.3-2.4; Figure 2.2).

2.2 Passive Air Samplers (PAS-PUF) Polyurethane foam (PUF) collection substrates (P/N TE-1014; 1.27 cm thick x 13.97 cm diameter; density 0.029 g/cm^3) were purchased from Tisch Environmental (Village of Cleves, OH, USA). Prior to deployment, PUFs were individually packed into a 66 mL extraction cell and pre-cleaned with acetone and hexane using an Accelerated Solvent Extractor (ASE 300, Dionex, Sunnyvale, CA, USA). The ASE was programmed for three sequential cycles with the temperature of 100°C, pressure of 1500 psi, static time of 5 min, and a 60% flush volume, and a purge time of 60 seconds.

These pre-cleaned PUF vapor collection substrates were then sealed in solvent rinsed aluminum foil and in air tight containers to avoid contamination during transit to the island and sampling locations. The PUFs were suspended within dome-type passive air samplers (PAS-PUF, Figure 2.3) and deployed in triplicate at each of the 15 sites in 2011, with the exception of one site (Parasasa) deployed in duplicate (n=43). Two of the replicates were lost during processing from the Marie Pampoen site and therefore the concentration is based on the remaining replicate. In 2011, samplers were deployed for ~9 weeks (65 ± 1 day) from 28 February – 6 May, 2011. Based on the 2011 triplicate precision and low variability measured by the mean relative standard deviation (10 ± 5 % RSD) between triplicates, the 2014 PAS-PUFs were deployed individually at 30 sites around Schottegatweg Ring for 9 weeks (63 ± 1 day) from 13 May – 18 July. At the end of each of the deployment periods, the PASs were retrieved and the PUFs were resealed and transported back to Mote Marine Laboratory where they were stored at -20°C until analysis.

2.2.1 PUF Extractions and Analysis

The 2011 triplicate PUFs from each of the 15 sites (n=43) and the 2014 PUFs (n=30) were extracted and analyzed separately. Individual PUFs were placed into a 66 mL stainless steel accelerated solvent extraction (ASE) cell and spiked with deuterated PAH surrogate standards and ortho-

terphenyl (OTP) to monitor recoveries and validate the extraction and clean-up procedures. Each PUF was extracted using 100% methylene chloride under the same ASE conditions as above. The PUF extracts (~80 mL) were reduced to ~1-2 mL using a RapidVap (Labconco Corp., Kansas City, MO, USA) and eluted through an automated GPC system (Fluid Management Systems, Watertown, MA, USA) to remove high molecular weight interferences. For further cleanup and separation of polycyclic aromatic hydrocarbons (PAHs), the extract was then eluted with 80:20 CH_2Cl_2/hexane (v/v) through a neutral silica column (6 g) using a multi-column clean-up system (Automated Power-Prep System, Fluid Management Systems, Watertown, MA, USA). The eluted fraction containing compounds of interest were collected and reduced to 900 uL of methylene chloride.

Prior to instrument analysis, all extracts were spiked with two deuterated PAH internal standards (dibenzothiophene-d_8, benzo[e]pyrene-d_{12}) for quantification of targeted analytes. Extracts (1 μL injection volume) were analyzed for approximately 61 polycyclic aromatic hydrocarbons (21 parent PAHs and homologues; see Chapter 1, Table 1.1), using combined gas chromatography/mass spectrometry (GC/MS; Agilent 7890A/5975C; Agilent Technologies, Inc., Andover, MA, USA). Analyte separation was achieved on a DB-5MS capillary column (30 m x 0.25 μm film thickness x 0.25 mm i.d.; Agilent Technologies, Inc., Andover, MA, USA) with ultrahigh-purity helium as the carrier gas. PAHs were determined in electron impact scan mode (EI) with helium as the carrier gas at 1mL/min. The injector (splitless mode) and transfer line temperatures were set to 300°C and 280°C, respectively. The oven temperature program was as follows: 60°C (0.5 min hold), then 8°C/min to 325°C (3 minute hold) for a total run time of 36.6 minutes. The source and quadrapole temperatures were set to 230°C and 150°C, respectively. All mass spectral data were compared to spectra produced by authentic standards and to previously published library spectra.

2.2.2 Calculated Air Concentrations

Passive air samplers adsorb chemical constituents that can be used to assess ambient concentrations in the atmosphere. The extent to which chemicals are enriched in the sampling substrate relative to air is dependent on the passive sampler medium (PSM), or the air partition coefficient (K_{PSM-A}). The PAS-PUF uptake of a chemical from the ambient air is best described by the effective concentration gradient between the air and the sampler following the equation:

$$V_S \frac{dC_S}{dt} = k_O A_S (C_A - \frac{C_S}{K_{SA}})$$

Where V_s is the sampler volume, C_s is the analyte concentration in sampler, C_A is the air concentration of the analyte, k_O is the overall mass transfer coefficient, A_s is the sampler surface area, and K_{SA} is the sampler air/partitioning coefficient [82, 83]. The K_{PSM-A} and the sampling rates (R) are both necessary to know in order to use PAS semi-quantitatively to assess ambient atmospheric concentrations and both have been previously calculated from field calibration experiments [71, 75]. Compound specific sampling rates were selected from calibration studies performed in a similar tropical environment [86]. Ambient air concentrations (C_{air}; ng/m^3) in Curaçao were then calculated using compound specific sampling rates [86] in the Global Atmospheric Passive Sampling Network's template [75] for calculating air volumes for PAHs as follows:

$$V_{air} = (K'_{PSM-A}) x (V_{PSM}) x (1 - esp \left[\frac{k_A}{K'_{PSM-A}} x \frac{1}{D_{film}} \right] t)$$

$$C_{air} = \frac{m_i}{V_{air}}$$

where K'_{PSM-A} takes into account the passive sampling medium octanol air partitioning coefficient (log K_{OA}) and is calculated by multiplying K_{PSM-A} by the density of the PSM, V_{PSM} is the volume of the passive sampling medium (m^3), k_A is the air-side mass transfer coefficient (m/d), which is equal to the sampling rate (m^3/day) divided by the surface area of the PUF sampler (365 cm^2), D_{film} is the

effective film thickness (m), and t is the deployment time (days), m_i is the mass of the target analyte measured in the passive samples (ng/disk).

2.2.3 Quality Assurance and Quality Control (QA/QC)

A performance-based quality-assurance and quality-control program, including the parallel analysis of procedural blanks and matrix spikes was implemented to ensure data of the highest quality. Quality assurance and quality control guidelines follow TDI-Brooks International, B&B Laboratories Inc., EPA 8270D and NOAA established criteria for PAH analysis. Five custom calibration standards ranging in concentration from 25 to 1000 ng/mL were used. Prior to sample analysis, the initial calibration passed the following established criteria: $R^2 = 0.99 - 1$ for all compounds and surrogates and the % RSD was ≤ 20% for all relative response factors (Table 2.5). The GC response is monitored using a mid-level (250 ng/mL) continuing calibration standard, passing acceptable criteria (% RSD ≤ 25% for 90% of the analytes; ≤ 35% for 10% of the analytes; see Table 2.5). Procedural blanks were checked to confirm they were clear of targeted analytes. Acceptable blanks were considered to contain no more than three times the MDL for two or more target analytes. The method of detection limit (MDL) is defined as three times the standard deviation of the mean concentration of each analyte detected in the blanks. Only two of the 21 parent PAHs were detected in the blanks (naphthalene and phenanthrene). The MDLs were 22 and 196 ng/PUF for naphthalene and phenanthrene, respectively. Instrument detection limits ranged from 0.06 to 0.24 ng/mL (Table 2.6).

Sample analyte concentrations were quantified based on the concentration and response of the internal standards (dibenzothiophene-d_8 and benzo[e]pyrene-d_{12}). All samples and method blanks were spiked with OTP and four (4) deuterated PAH surrogate compounds prior to extraction. All samples passed the acceptable surrogate recovery criteria (% recovery 50-150%). The mean recovery of OTP spiked in the 2011 samples was 104 ± 6%. The 2011 overall recovery for the low and high

molecular weight surrogates were 61 ±11% and 84 ± 9%, respectively. The 2011 overall recovery for the matrix spiked with 18 parent PAHs was 82 ± 25%. The mean recovery of OTP spiked in the 2014 samples was 103 ± 11%. The 2011 overall recovery for the low and high molecular weight surrogates were 91 ±15% and 105 ± 20%, respectively. The 2014 overall recovery for the matrix spiked with 18 parent PAHs was 78 ± 8%. Individual and standard mixtures of PAHs were purchased from AccuStandard (New Haven, CT, USA).

2.3 Source Characterization

2.3.1 Concentration Profiles

Concentration profiles can differentiate between petrogenic and pyrogenic sources by evaluating the distribution of the parent compounds and their homologues. PAHs originating from petrogenic sources primarily consist of the low molecular weight compounds having two or three fused benzene rings and an abundance of substituted PAHs (homologues with 2-3 alkyl carbons), thereby displaying a characteristic bell-shape with respect to the degree of alkylation [104-107]. PAHs originating from pyrogenic sources primarily consist of the high molecular weight compounds having four to six fused benzene rings and dominated by the unsubstituted PAHs or homologues with 1-2 alkyl carbons. Therefore, profiles from petrogenic sources display a characteristic bell-shape with increasing concentrations with increasing degree of alkylation (C0<C1<C2<C3<C4) whereas pyrogenic sources produce a decreasing concentration in the distribution within a homologue series (C0>C1>C2>C3>C4) [105, 106].

2.3.2 Binary Diagnostic Ratios

Binary diagnostic ratios have been useful in identifying emission sources and their contributions to ambient air concentrations in order to distinguish between different sources, such as petrogenic (hydrocarbons associated with petroleum), pyrogenic (hydrocarbons associated with incomplete combustion) or phytogenic (hydrocarbons derived from plants) [50, 108-110]. Diagnostic ratios involve the comparison of PAH pairs that are frequently identified in certain emission sources. Evaluating these binary ratios of PAH pairs, or indicator PAHs, have allowed for the differentiation between vehicular and non-traffic emissions, diesel and gasoline combustion, different crude oil processing and biomass burning. For instance, PAHs with the molecular mass 178 and 202 are frequently used to distinguish between combustion and petroleum sources [108, 109]. Each of the diagnostic approaches has its limitations and uncertainties, therefore it is highly recommended to interpret more than one ratio to identify and confirm a source(s). Table 2.7 describes the 10 parent PAH ratios used in this study to evaluate possible emission sources. Sites with non-detects of both binary ratio compounds were excluded from the analysis. To prevent biases, ratios where one of the compounds was below the detection limit, resulting in a ratio of either 0 or 1, were considered to be less than the instrument detection limit (<IDL).

2.3.3 Factor Analysis and Principle Components Analysis (PCA)

Factor analysis and principle components analysis (PCA) are widely used multivariate statistical techniques primarily used to analyze all sources of variability in a dataset by transforming and reducing the number of correlated variables into *principle components* that account for the majority of the variability. Factor analysis uses PCA to extract the common factors within a dataset. Within the factors, each variable is assigned a factor loading which determines the most representative indicator PAH. Essentially, the grouped variables or factors can then be interpreted as specific emission

sources that are characterized by the most representative indicator PAH(s) [50]. In this study, only detectable levels of the parent PAH compounds from the 2014 downwind sites (18 PAHs), 2014 upwind sites (15 PAHs) and the 2011 sites (18 PAHs) were input into the factor analysis and PCA.

2.4 Criteria Air Pollutant Local Monitoring Station Descriptions

Since mid-2010, two air monitoring stations, Beth Chaim and Kas Chikitu, located in Willemstad, Curaçao (Figure 2.4) have been collecting validated and continuous measurements of air quality parameters (SO_2, PM_{10}, TSP, H_2S). The Beth Chaim station is located at the western edge, downwind of the Schottegat industrial area of the refinery and only measures SO_2 and TSP. Kas Chikitu is located approximately 2-3 km downwind in the Marchena/Wishi residential area and is primarily used to monitor the residential load of SO_2, hydrogen sulfide (H_2S) and PM_{10}. Available hourly and daily measurements of SO_2 and PM_{10} were downloaded from June 1, 2010 through December 31, 2014 for analysis. Twenty-four hour SO_2 daily means were downloaded from the Beth Haim station (n=1605) and the Kas Chikitu station (n=1622), and 24-hr PM_{10} daily means (n=1603) measured at the Kas Chikitu station were also downloaded. Monitoring stations operate in accordance with the ISO/IEC 17025 accreditation (certificate number L 426) of GGD Amsterdam using ultraviolet fluorescence (Thermo 43i-TLE and Thermo 450i Gas Analyzer) and tapered element oscillating balance (TEOM 50C) methodology are used to measure SO_2 and PM_{10}, respectively.

2.5 Calculating Risk

2.5.1 Potency Equivalency Factors (PEFs)

A carcinogenic activity relative to B[a]P can be estimated if potency values have not been calculated for specific PAHs [111]. Estimates of potency relative to B[a]P are known as potency

equivalency factors (PEFs). Therefore, individual chemicals in a mixture can be used to derive an overall cancer potency of a mixture for chemicals that are structurally related and share similar mechanisms of toxicity as B[a]P [112]. Hence, potency equivalency factors (PEFs) can be used as a practical tool for regulatory purposes in predicting toxicity and calculating the relative contribution of individual PAHs to the total carcinogenicity of measured PAHs. Potency equivalent concentrations are obtained by multiplying ambient concentrations of each of the PAHs which have available PEFs and are typically expressed as B[a]P equivalents or potency equivalents (PEQs). The PEFs used in the calculations for this study are from Nisbet and LaGoy [54] and are presented in Table 2.8.

2.5.2 Risk Probability Estimates

A risk analysis was performed using the USEPA Guidelines for Carcinogen Risk Assessment [113]. The USEPA software, ProUCL (Version 5), has the capabilities of producing rigorous decisions making statistics by deriving the upper confidence limit (UCL) of the mean, the upper tolerance limit (UTL) and the upper prediction limit (UPL). The calculated PAH concentrations (ng/m^3) in ambient air and the potency adjusted concentrations (ng-PEQ/m^3) were used to derive 95% upper confidence limits (UCL) using ProUCL. The resulting 95% UCL were then converted to μg/m^3 in order to calculate risk probabilities by multiplying the recommended 95% UCL for each compound by the compound specific inhalation unit risk (IUR, μg/m^3). In order to evaluate potential worse-case scenarios at individual site locations, site specific risk probability estimates were also calculated using the potency adjusted concentrations (μg-PEQ/m^3) without using the 95% UCL. Risk probabilities were only calculated for those compounds with detectable levels and an available IUR and slope factor (Table 2.8).

In addition, cancer risk probability estimates for different age classes (i.e., children, adult residents and adult workers) were calculated to assess the lifetime average daily intake (LADI) from inhalation using the following formulas:

$$\text{Risk} = \text{LADI} \times \text{CSF}$$

$$\text{LADI} = \text{CA} \times \text{IN} \times \text{EF} \times \text{ED} \times (1/\text{BW}) \times (1/\text{AT})$$

Where CSF is the cancer slope factor for each compound (mg/kg/day), CA is the 95% UCL (mg/m^3) or potency equivalents ($mg\text{-}PEQ/m^3$), IN is the inhalation rate (m^3/day), EF is the exposure frequency (days/year), ED is the exposure duration (years), BW is the average body weight (kg), and AT is the averaging time (days per year over a 70 year lifetime). Compound slope factors and variable factors are summarized in Table 2.8-2.9.

2.6 Data and Statistical Analysis

Calculated ambient PAHs are expressed as ng/m^3. Concentrations of SO_2 and PM_{10} are expressed as $\mu g/m^3$. Potency equivalent concentrations are expressed as $ng\text{-}PEQ/m^3$. Non-detectable levels were not substituted with detection limits. In the event a normal distribution was justified, parametric statistics were performed using Student's t-tests and ANOVA to evaluate spatial and temporal differences. If the assumptions of normality were not met, nonparametric Kruskal-Wallis ANOVA and median tests were performed on raw data to reveal any spatial and temporal differences ($\alpha = 0.05$) using Statistica Version 6 (StatSoft Inc., Tulsa, OK, USA).

Table 2.1 Passive air sampling locations in 2011 with distance and direction from the refinery.

Site	MML ID	Neighborhood	Latitude	Longitude	Distance (km)	Direction from Refinery
1	CUR-11-0030 CUR-11-0031 CUR-11-0032	Habaai	12° 07'22.0"	-68°57'00.4"	2.52	West (Downwind)
2	CUR-11-0033 CUR-11-0034 CUR-11-0035	Groot Piscadera	12°08'44.8"	-68°58'02.6"	4.41	West (Downwind)
3	CUR-11-0036 CUR-11-0037 CUR-11-0038	Boca Sami	12°08'26.1"	-68°59'23.5"	6.68	West (Downwind)
4	CUR-11-0039 CUR-11-0040 CUR-11-0041	W. Piscadera Baai	12°08'18.2"	-68°58'21.6"	4.78	West (Downwind)
5	CUR-11-0042 CUR-11-0043 CUR-11-0044	Rooi Catochi	12°07'24.1"	-68°54'10.0"	3.07	East (Upwind)
6	CUR-11-0045 CUR-11-0046 CUR-11-0047	Nieuw Nederland	12° 6'1.96"	-68°55'15.4"	3.68	Southeast (Upwind)
7	CUR-11-0048 CUR-11-0049 CUR-11-0050	Marie Pampoen	12°05'03.8"	-68°55'47.7"	6.5	Southeast (Upwind)
8	CUR-11-0051 CUR-11-0052 CUR-11-0053	E. Buena Vista	12°08'33.5"	-68°55'55.5"	1.14	Northwest (Downwind)
9	CUR-11-0054 CUR-11-0055 CUR-11-0056	W. Buena Vista	12°08'30.2"	-68°56'20.9"	1.46	Northwest (Downwind)
10	CUR-11-0057 CUR-11-0058 CUR-11-0059	Heintje Kool	12°08'23.4"	-68°56'30.1"	1.56	Northwest (Downwind)
11	CUR-11-0060 CUR-11-0061 CUR-11-0062	Roosendaal	12°08'34.3"	-68°57'11.8"	2.86	West (Downwind)
12	CUR-11-0063 CUR-11-0064 CUR-11-0065	Marchena/Wishi	12°07'41.2"	-68°57'13.7"	2.72	West (Downwind)
13	CUR-11-0066 CUR-11-0067 CUR-11-0068	E. Piscadera Baai	12°08'07.6"	-68°57'48.7"	3.75	West (Downwind)
14	CUR-11-0069 CUR-11-0070 CUR-11-0071	Blauw / Curasol	12°08'40.5	-68°58'53.5	5.87	West (Downwind)
15	CUR-11-0072 CUR-11-0073	Parasasa	12°07'21.9"	-68°58'08.2"	4.48	Southwest (Downwind)

Table 2.2 Neighborhood/geozone population sizes located in the 2011 sampling locations.

Neighborhood/Geozone	Population
Blauw (Curasol)	1006
Boca Sami	1108
Buena Vista	3892
Groot Piscadera	749
Habaai	407
Nieuw Nederland	276
Parasasa	171
Rooi Catochi	319
Roosendaal	481
Wishi	841
Marchena	584
Marie Pampoen	1319
Piscadera Baai	787
Total Population in Sampling Zones	11940

Source: Central Bureau of Statistics Curaçao 2011

Table 2.3 Passive air sampling locations in 2014 with distance and direction from the refinery.

Site	Neighborhood	Latitude	Longitude	Distance (km)	Direction
1	Toni Kunchi	12°07'16.9"	-068°53'20.5"	4.62	East (Upwind)
2	Groot Davelaar/Joonchi	12°07'10.2"	-068°54'02.9"	3.47	East (Upwind)
3	Rooi Catochi (East of Schottegatweg Ring)	12°07'23.8"	-068°54'10.8"	3.10	East (Upwind)
4	Emmastad	12°08'36.1"	-068°55'04.4"	1.73	Northeast (Upwind)
5	Beth Haim / Marchena	12°07'57.2"	-068°56'35.4"	1.47	West (Downwind)
6	Habaai	12°07'22.2"	-068°57'00.8"	2.52	West (Downwind)
7	Welgelegen	12°07'18.2"	-068°56'39.7"	2.00	Southwest (Downwind)
8	Punda	12°06'54.9"	-068°55'39.0"	1.97	South (Upwind)
9	Pietermaai / Salina	12°06'18.9"	-068°54'53.8"	3.44	Southeast (Upwind)
10	Steenrijk	12°06'06.5"	-068°54'22.5"	4.29	Southeast (Upwind)
11	Zeelandia	12°06'55.3"	-068°54'22.5"	3.20	Southeast (Upwind)
12	Rooi Catochi (West of Schottegatweg Ring)	12°07'37.7"	-068°54'19.0"	2.73	Southeast (Upwind)
13	Biesheuvel	12°07'55.0"	-068°54'29.8"	2.28	East (Upwind)
14	Emmastad	12°08'23.6"	-068°54'57.4"	1.69	Northeast (Upwind)
15	Brievengat/Groot Kwartier	12°08'37.0"	-068°53'59.2"	3.46	East (Upwind)
16	Suffisant	12°09'05.2"	-068°55'18.9"	2.23	Northeast (Upwind)
17	Buena Vista	12°08'27.0"	-068°56'17.8"	1.30	Northwest (Downwind)
18	Buena Vista	12°08'14.1"	-068°56'45.4"	1.86	Northwest (Downwind)
19	Wanapa	12°08'56.3"	-068°56'41.2"	2.44	Northwest (Downwind)
20	Buena Vista	12°08'45.9"	-068°56'08.5"	1.61	North (Downwind)
21	Buena Vista	12°08'29.7"	-068°55'55.7"	1.00	North (Downwind)
22	Roosendaal	12°08'34.1"	-068°57'11.1"	2.80	Northwest (Downwind)
23	Marchena	12°07'41.4"	-068°57'13.7"	2.69	West (Downwind)
24	Parasasa / Soccor Field	12°07'21.4"	-068°57'43.6"	3.75	Southwest (Downwind)
25	Parasasa	12°07'22.2"	-068°58'08.7"	4.48	Southwest (Downwind)
26	Wishi	12°07'47.6"	-068°57'35.0"	3.28	West (Downwind)
27	Gasparitu / Roosendaal	12°08'19.1"	-068°57'35.2"	3.37	Northwest (Downwind)
28	Suffisant	12°08'19.0"	-068°57'35.2"	2.13	Northeast (Upwind)
29	Domi / Welgelegen	12°08'49.9"	-068°55'00.3"	2.41	Southwest (Downwind)
30	Nieuw Nederland	12° 6'1.96"	68°55'15.46"	3.71	South (Upwind)

Table 2.4 Neighborhood/geozone population sizes in the 2014 sample locations.

Neighborhood/Geozone	Population
Toni Kunchi	379
Groot Davelaar/Joonchi	186
Emmastad	738
Habaai	407
Welgelegen	111
Punda	99
Pietermaai / Salina	2637
Steenrijk	3752
Zeelandia	685
Rooi Catochi (Catootje)	319
Biesheuvel	65
Brievengat/Groot Kwartier	6340
Suffisant	3526
Buena Vista	3892
Wanapa	27
Gasparitu / Roosendaal	481
Marchena	584
Parasasa	171
Wishi	841
Domi / Welgelegen	1358
Nieuw Nederland	276
Total Population in Sampling Zones	26,874

Source: Central Bureau of Statistics Curaçao 2011

Table 2.5 Calibration standards, quantification and QA/QC parameters.

Calibration Compounds	Ref to IS/SS	Quant Ion	Qual Ion	Cal R^2	RRF %RSD (≤20%)	Cont Cal %RSD (≤25% for 90%)
Naphthalene-d_8 (I-1, S-1)	I-1,S-1	136	134	1.00	4	6
Acenaphthene-d_{10} (I-1, S-2)	I-1,S-2	164	162	1.00	4	6
Anthracene-d_{10} (I-1, S-3)	I-1,S-3	188	184	1.00	7	18
Benzo[a]anthracene-d_{12} (I-2,S-4)	I-2,S-4	240	236	1.00	6	24
Perylene-d_{12} (I-2, S-5)	I-2,S-5	264	260	1.00	10	22
Naphthalene	I-1,S-1	128	127	1.00	6	6
Acenaphthylene	I-1,S-2	152	153	1.00	4	26
Acenaphthene	I-1,S-2	154	153	1.00	5	7
Fluorene	I-1,S-2	166	165	1.00	3	8
Dibenzothiophene	I-1,S-3	184	152	1.00	4	1
Phenanthrene	I-1,S-3	178	176	1.00	10	1
Anthracene	I-1,S-3	178	176	1.00	4	13
Fluoranthene	I-2,S-3	202	101	1.00	10	12
Pyrene	I-2,S-3	202	101	1.00	10	18
Benzo[b]fluorene	I-2,S-3	216	nd	1.00	9	31
Napthobenzothiophene	I-2,S-3	234	nd	1.00	6	15
Benzo[a]anthracene	I-2,S-4	228	226	1.00	6	17
Chrysene	I-2,S-4	228	226	1.00	8	6
Benzo[b]fluoranthene	I-2,S-4	252	253	1.00	3	18
Benzo[k]fluoranthene	I-2,S-4	252	253	1.00	11	17
Benzo[e]pyrene	I-2,S-4	252	253	1.00	4	11
Benzo[a]pyrene	I-2,S-4	252	253	1.00	3	19
Perylene	I-2,S-5	252	253	0.99	4	16
Indeno[1,2,3,-cd]pyrene	I-2,S-4	276	277	1.00	7	18
Dibenzo[a,h]anthracene	I-2,S-4	278	279	1.00	17	12
Benzo[g,h,i]perylene	I-2,S-4	276	277	1.00	7	4

Table 2.6 Instrument detection limits (IDL) calculated from seven injections of a 750 pg/mL PAH standard mix.

Analytes	RT	Quant Ion	Conf Ion	IS/SS	Mean Resp.	StDev	% RSD (<20%)	IDL (ng/mL)
Naphthalene	8.970	128	127	I-1,S-1	1266	85	7	0.26
Acenaphthylene	13.445	152	153	I-1,S-2	420	41	10	0.12
Acenaphthene	13.971	154	153	I-1,S-2	353	57	16	0.17
Fluorene	15.500	166	165	I-1,S-2	509	66	13	0.20
Dibenzothiophene	17.937	184	152	I-1,S-3	549	35	6	0.11
Phenanthrene	18.295	178	176	I-1,S-3	689	78	11	0.24
Anthracene	18.447	178	176	I-1,S-3	370	42	11	0.13
Fluoranthene	21.810	202	101	I-2,S-3	490	43	9	0.13
Pyrene	22.449	202	101	I-2,S-3	566	57	10	0.17
Benzo[b]fluorene	23.728	216	226	I-2,S-3	149	20	13	0.06
Napthobenzothiophene	25.130	234	nd	I-2,S-3	281	32	11	0.10
Benzo[a]anthracene	26.050	228	226	I-2,S-4	219	30	14	0.09
Chrysene	26.137	228	226	I-2,S-4	308	58	19	0.17
Benzo[b]fluoranthene	29.020	252	253	I-2,S-4	365	60	16	0.18
Benzo[k]fluoranthene	29.050	252	253	I-2,S-4	197	24	12	0.07
Benzo[e]pyrene	26.697	252	253	I-2,S-4	161	20	12	0.06
Benzo[a]pyrene	29.829	252	253	I-2,S-4	176	30	17	0.09
Perylene	30.032	252	253	I-2,S-5	185	32	17	0.09
Indeno[1,2,3,-cd]pyrene	32.410	276	277	I-2,S-4	162	21	13	0.06
Dibenzo[a,h]anthracene	32.494	278	279	I-2,S-4	174	20	11	0.06
Benzo[g,h,i]perylene	32.941	276	277	I-2,S-4	247	29	12	0.09

Table 2.7 Diagnostic ratios used in this study to elucidate emission sources in Curaçao.

Diagnostic Ratio	Value	Source(s)	Reference(s)
LMW / HMW	>1 <1	Petrogenic Pyrogenic	[114]
PHE /(ANT+PHE)	~0.98 ~-0.78 0.77 ± 12	Petrogenic (crude oil) Pyrogenic (used motor oil) Pyrogenic (vehicle emissions)	[108]
PHE / ANT	>10 <10	Petrogenic Pyrogenic	[109]
FLA / (FLA + PYR)	>0.5 <0.5	Pyrogenic (diesel engines) Petrogenic	[50, 85]
FLA / PYR	<1 >1	Petrogenic Pyrolytic	[108, 109]
B[a]A / (B[a]A + CHR)	<0.2 >0.35-0.5	Pyrogenic Pyrogenic (vehicle combustion)	[114, 115]
B[a]P / (B[a]P + CHR)	0.5 0.73	Pyrogenic (diesel engines) Pyrogenic (gasoline engines)	[50]
IND/ (IND+BghiP)	<0.2 0.2-0.5 >0.5	Petrogenic Pyrogenic (vehicle / crude combustion) Pyrogenic (grass, wood and coal combustion)	[115]
IND / BghiP	0.4 ~1	Pyrogenic (gasoline engines) Pyrogenic (diesel engines)	[50]
B[a]P / BghiP	>0.6	Pyrogenic (traffic emissions)	[116]

ΣLMW/ΣHMW=sum of low molecular weight PAHs (2-3 rings)/sum of high molecular weight PAHs (4-6 rings); ANT=Anthracene, PHE=Phenanthrene; FLA=Fluoranthene; PYR=Pyrene; B[a]A=Benzo[a]anthracene; CHR=Chrysene; B[a]P=Benzo[a]Pyrene; BghiP=Benzo[g,h,i]perylene; Ind=Indeno[1,2,3,-cd]pyrene

Table 2.8 Compound specific inhalation unit risk (IUR) factors, inhalation cancer slope factors (ICSF) and potency equivalency factors (PEFs) used in this study. The benzo[a]pyrene ICSF was substituted for the compounds without a compound specific ICSF and are italicized.

PAH	IUR ($\mu g/m^3$)	ICSF (mg/kg/day)	PEF
Naphthalene	3.40E-05	1.20E-01	0.001
Acenaphthylene	3.40E-05	*3.90E+00*	0.001
Acenaphthene	1.10E-06	*3.90E+00*	0.001
Fluorene	1.10E-06	*3.90E+00*	0.001
Phenanthrene	1.10E-06	*3.90E+00*	0.001
Anthracene	1.10E-05	*3.90E+00*	0.01
Fluoranthene	1.10E-06	*3.90E+00*	0.001
Pyrene	1.10E-06	*3.90E+00*	0.001
Benzo(a)anthracene	1.10E-04	3.90E-01	0.1
Chrysene	1.10E-05	3.90E-02	0.01
Benzo(b)fluoranthene	1.10E-04	3.90E-01	0.1
Benzo(k)fluoranthene	1.10E-04	3.90E-01	0.1
Benzo(a)pyrene	1.10E-03	3.90E+00	1
Indeno(1,2,3-c,d)pyrene	1.10E-04	3.90E-01	0.1
Dibenzo(a,h)anthracene	1.20E-03	4.10E+00	5
Benzo(g,h,i)perylene	1.10E-05	*3.90E+00*	0.01

Table 2.9 Factors used in this study to calculate lifetime average daily intake (LADI) and risk probability estimates for children and adults in Curaçao.

FACTORS	IN	EF	ED	BW	AT
	Inhalation Rate (m^3/day)	Exposure Fx (days/year)	Exposure Duration (years)	Body Weight (kg)	Avg time (days)
Child	10	350	6	15	25550
Adult: Resident	20	350	24	70	25550
Adult: Worker	20	250	25	70	25550

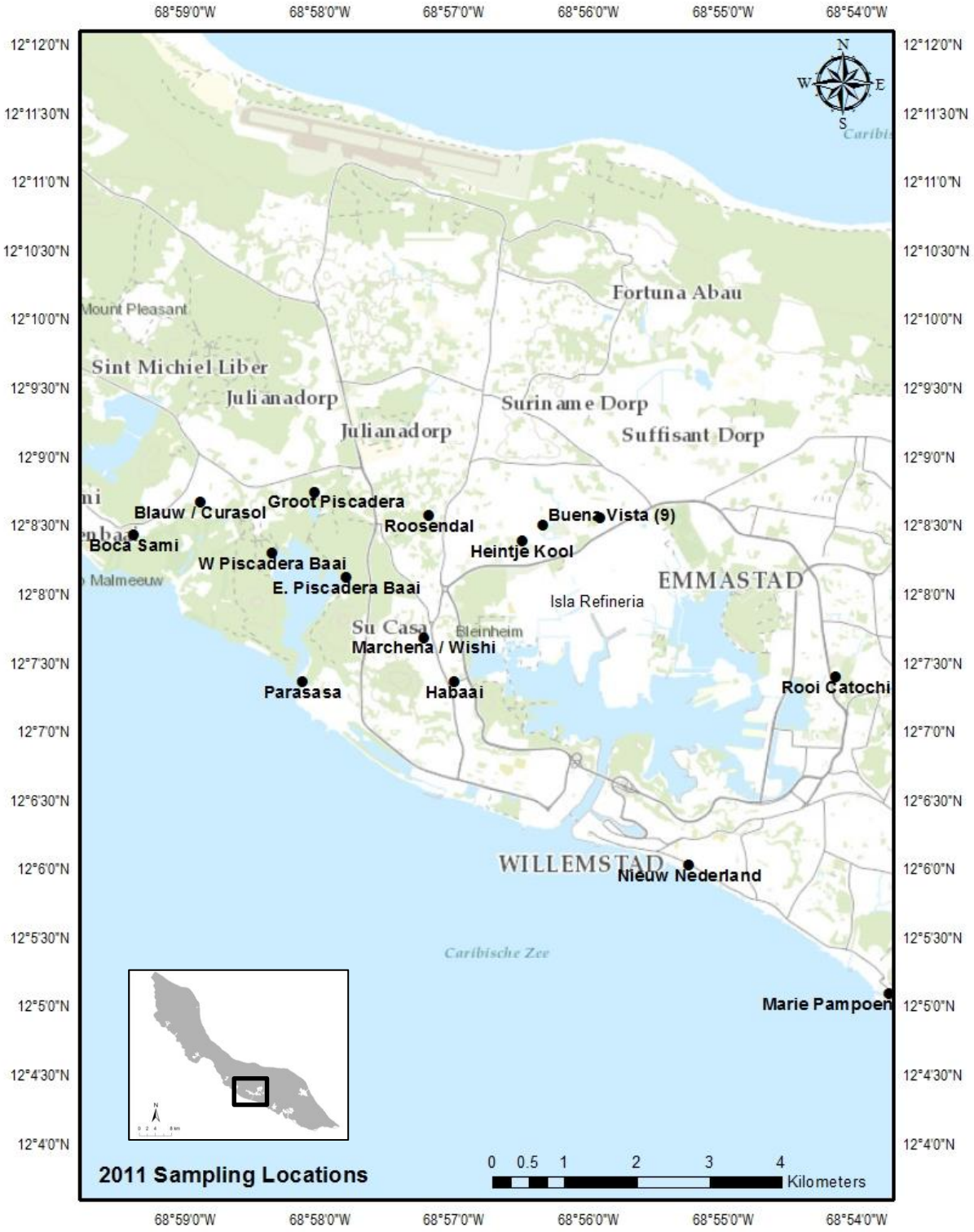


Figure 2.1 Passive air sampling (PAS) locations during the 2011 sampling event in Curaçao.

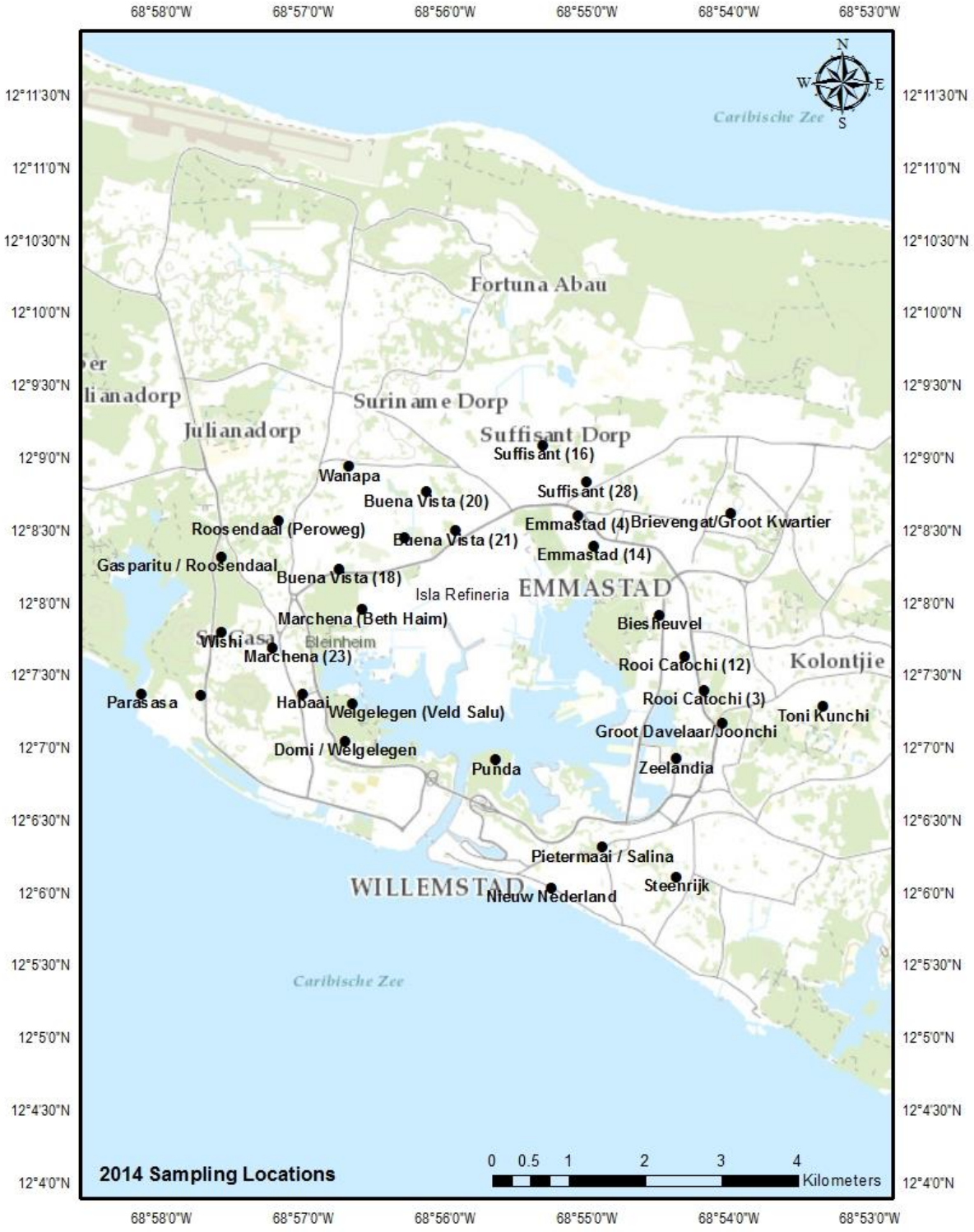


Figure 2.2 Passive air sampling (PAS) locations during the 2014 sampling event in Curaçao.

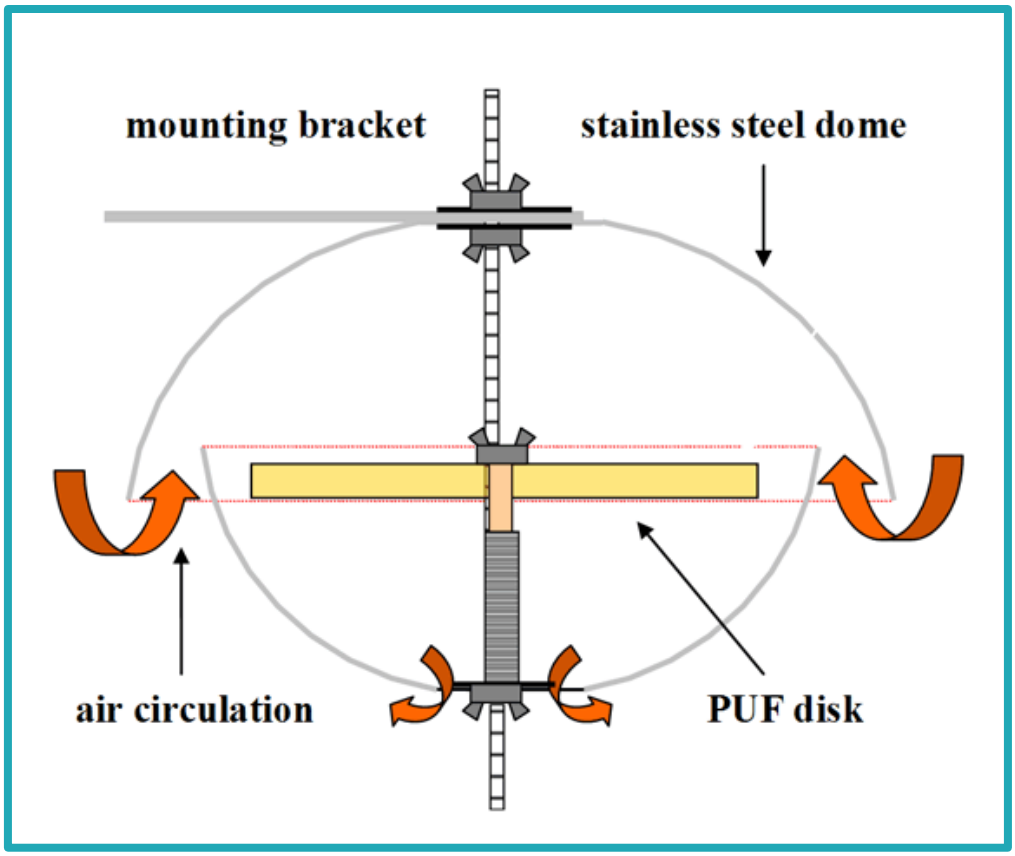


Figure 2.3 Passive sampler schematic.

Figure 2.4 Air monitoring stations, Beth Haim (a) and Kas Chikitu (b), located in Willemstad, Curaçao.

CHAPTER THREE:

INHALABLE PARTICULATE MATTER

3.1 Ambient Concentrations of Inhalable Particulate Matter

Daily PM_{10} concentrations were downloaded from the Kas Chikitu station (n=1,603) from June 1, 2010 through December 31, 2014. The 24-hour daily mean concentrations ranged from 0.37 – 341 $\mu g/m^3$ (Figure 3.1). Mean annual PM_{10} concentrations at the Kas Chikitu station ranged from 31 $\mu g/m^3$ in 2010 to 122 $\mu g/m^3$ in 2014 (Figure 3.2). There were statistically significant temporal trends observed with a strong increasing trend (R^2=0.94) over time. Table 3.1 summarizes the statistical differences between years. In general, the 2014 mean annual PM_{10} concentrations were significantly higher than the previous four years.

3.1.1 Global Comparisons of Ambient PM_{10} Concentrations

The annual average PM_{10} concentrations have increased 74% since 2010. Excluding 2010, since measurements are only for a seven month period, the annual average increased 61% since 2011. In contrast, PM_{10} concentrations in the US have shown a 34% decrease in the 24-hour average concentrations since 1999 and a 31% decrease in annual average ambient concentrations since 1990 (http://www.epa.gov/airtrends). The 2014 annual mean concentrations for PM_{10} in Curaçao (121.5 $\mu g/m^3$) is among some of the highest concentrations reported globally, measuring approximately 13 times higher than those reported in Iceland (9 $\mu g/m^3$), yet were two times lower than levels recorded in Pakistan (282 $\mu g/m^3$) (Figure 3.3).

3.1.2 PM_{10} Compliance with Public Health Guidelines

The maximum annual mean concentrations for PM_{10} that are currently recommended by Curaçao and the European Commission are 75 and 40 $\mu g/m^3$, respectively. The annual mean concentrations for the years 2011 through 2014 exceeded the current PM_{10} guidelines recommended by the European Commission (40 $\mu g/m^3$). Additionally, mean annual PM_{10} concentrations for 2013 (95.9 $\mu g/m^3$) and 2014 (121.5 $\mu g/m^3$) both exceeded the island's guidelines for PM_{10} (75 $\mu g/m^3$). Mean 24-hour maximum concentrations of PM_{10} have also been recommended by NAAQS (150 $\mu g/m^3$), the European Commission (50 $\mu g/m^3$) and Curaçao (150 $\mu g/m^3$). The number of days that have exceeded the 24-hour daily maximum concentrations of PM_{10} has demonstrated strong increasing trends in this study (Figure 3.4). The majority of 2010 (82%), 2011 (64%), and 2012 (60%) were compliant with recommended guidelines for measured PM_{10} concentrations (Figure 3.5). Conversely, a total of 77% of 2013 and 85% of 2014 exceeded all recommended 24-hour guidelines for PM_{10} concentrations. Curaçao allows 5% of the calendar days to exceed 150 $\mu g/m^3$, however, 10%, 22%, and 35% of 2012, 2013, 2014, respectively, exceeded this value [117].

3.1.3 Potential Risks of PM_{10} Inhalation

There are a number of epidemiologic studies on the associations of PM_{10} with various health outcomes, including mortality, morbidity and increased emergency room visits and hospitalization. Within the last twenty years, a number of cohort studies and meta-analyses reported relative risks (RR) and hazard ratios (HR) on various PM_{10} associated mortality and morbidity (Figures 3.6-3.8) [9, 14, 118-144]. This study primarily focused on literature published within the last five years since there are several meta-analyses and reviews covering literature published prior to 2010.

The relative risk reported for various mortality demonstrate positive associations with PM_{10}, however the data is somewhat inconsistent (Figure 3.6). For instance, two studies reported positive associations for respiratory mortality, where only one was statistically significant [121, 130]. Liang et al. [130] reported positive associations, with respiratory mortality (RR: 1.347, 95% CI: 0.990-1.833) during the winter months in Taiwan with a mean PM_{10} concentration of 66.7 $\mu g/m^3$. However this association was not significant since the RR included unity and had a wide confidence interval range suggesting greater uncertainty. In contrast, a meta-analysis consisting of 26 studies in China with annual PM_{10} concentrations ranging from 44-156 $\mu g/m^3$, reported significant positive associations between short-term PM_{10} exposure and respiratory mortality (RR: 1.0057, 95% CI: 1.004-1.0075) [121].

Similarly, relative risks for cardio related mortality also demonstrated both significant and non-significant positive associations with PM_{10} [118, 121, 128, 130]. For each 10 $\mu g/m^3$ of PM_{10}, significant positive associations were found with cardiovascular disease mortality (RR: 1.23, 95% CI: 1.19-1.26), ischemic heart disease mortality (RR: 1.37, 95% CI: 1.28-1.47) and heart failure mortality (RR: 1.11, 95% CI: 1.05-1.17) within a retrospective cohort, containing over 39,000 subjects from northern China [128]. Liang et al. [130] used a time-series regression model to analyze mortality among central Taiwan residents and reported non-significant positive associations during the winter months (mean PM_{10} of 66.7 $\mu g/m^3$) between PM_{10} and cardiovascular mortality for residents less than 65 years of age (RR: 1.12, 95% CI: 0.998-1.258) and borderline, yet significant for residents greater than 65 years old (RR: 1.194; 95%CI: 1.0025-1.425). A meta-analysis in China also reported positive associations with short-term exposures to PM_{10} (annual means ranging from 44-156 $\mu g/m^3$) and cardiopulmonary (RR: 1.0034; 95%CI: 1.0023-1.0046) and cardiovascular mortality (RR: 1.0049; 95%CI: 1.0034-1.0063) [121].

Positive associations were also reported for all-cause and non-accidental mortality [9, 118, 121, 122, 128, 130]. All were significant with the exception of the study by Liang et al. [130] which found a positive yet non-significant association between PM_{10} and all-cause mortality in Taiwan (RR: 1.059; 95%CI: 0.999-1.122). In addition, significant positive associations between lung cancer mortality and long-term PM_{10} exposures (RR: 1.05; 95%CI: 1.03-1.07) were found in a meta-analysis of 19 studies conducted globally [9].

The relative risks reported for various morbidity and hospitalizations is much less convincing of positive associations with PM_{10} since many of the studies report near or include unity (Figure 3.7) [9, 118, 119, 121, 123-127, 129]. Significant positive associations were found in a number of studies between respiratory diseases, respiratory related hospital admissions and lung obstruction and PM_{10} (annual PM_{10} ranged 31-270 $\mu g/m^3$). However, a study in the highly polluted industrial city of Lanzhou, China (PM_{10} daily mean: 197 $\mu g/m^3$) reported positive, non-significant associations between PM_{10} and respiratory diseases (RR: 2.4, 95% CI: 0.5-4.2) and significant positive associations with pneumonia (RR: 5.3, 95% CI: 1.3-9.5) and upper respiratory tract infections in people less than 65 years of age (RR: 13.7, 95% CI: 2.5-26.2) [126]. However, the confidence intervals are relatively wide suggesting increased uncertainty. Relative risks reported for incidences of lung cancer among two meta-analyses were also positive yet were not statistically significant since both included unity [9]. The meta-analysis consisting of 60 studies from 1966-2014 by Wang et al. [118] reported evidence of inconsistent, nonsignificant associations between short-term changes in PM_{10} and hemorrhagic stroke (RR: 1.009; 95% CI: 0.976-1.043), ischemic stroke (RR: 1.0; 95% CI: 0.976-1.024) and cerebrovascular disease (RR: 1.003; 95% CI: 0.999-1.008). A study in Scotland reported positive, nonsignificant associations between PM_{10} (20-22 $\mu g/m^3$ mean annual PM_{10}) and cardiovascular hospital admissions [129]. In contrast, a study in China (44-156 $\mu g/m^3$ mean annual PM_{10}) and Iran (111.3 $\mu g/m^3$ mean

annual PM_{10}) both reported significant positive associations with cardiovascular related hospital admissions [121, 127].

Hazard ratios were also reported in a number of studies for various mortality and risks associated with PM_{10} [14, 131-144] (Figure 3.8). In 2008, Puett et al. [133] reported significant positive associations between PM_{10} and all-cause mortality (HR: 1.11; 95% CI: 1.01-1.23) and cardiovascular disease mortality (HR: 1.35; 95% CI: 1.03-1.77) in the Nurses' Health Study consisting of 66,250 women with a mean age of 62 years. In 2009, Puett et al. [132] reported nonsignificant positive associations between $PM_{10-2.5}$ and all-cause mortality (HR: 1.03; 95% CI: 0.89-1.18) and cardiovascular disease mortality (HR: 1.14; 95% CI: 0.73-1.77) also for the Nurses' Health Study. It is important to note in the latter study $PM_{2.5}$ was subtracted from PM_{10} concentrations suggesting the associations found with PM_{10} in the earlier study were potentially influenced by $PM_{2.5}$. Nonsignificant associations (HR: 1.22, 95% CI: 0.91-1.63) were found between long-term PM_{10} (13.5-48.1 $\mu g/m^3$ annual PM_{10}) exposure and cardiovascular disease mortality in twenty cohorts across 13 countries in Europe (ESCAPE Project) [136]. In contrast, several studies found significant positive associations with cardio-related events. A prospective cohort consisting of 4800 women (mean age 55 years old) in Germany found significant positive associations between long-term PM_{10} (34.8-52.5 $\mu g/m^3$ annual mean PM_{10}) exposure and cardiopulmonary mortality (HR: 1.39, 95% CI: 1.17-1.64)[131]. In addition, a study of 11 cohorts in the ESCAPE project reported a positive associations (HR: 1.12, 95% CI: 1.01-1.25) with long-term PM_{10} (14-48 $\mu g/m^3$ annual mean) exposure and coronary events [139].

A large cohort study in England consisting of over 800,000 patients, aged 40-89 years, reported positive associations (HR: 1.16, 95% CI: 1.12-1.21) between PM_{10} (19.7 $\mu g/m^3$ annual mean) and respiratory mortality [140]. Positive associations (HR: 1.023, 95% CI: 1.005-1.042) were also reported for a cohort of 71,000 middle aged Chinese men exposed to much higher concentrations of PM_{10} (104

$\mu g/m^3$ annual mean) than those measured in the English cohort study [137]. In contrast, the prospective cohort study in Germany reported nonsignificant associations (HR: 0.96, 95% CI: 0.6-1.53) with respiratory mortality in middle aged women [131].

A number of cohort studies in the USA, Germany, England, Norway and China have also reported inconsistent associations (HRs) between PM_{10} and lung cancer mortality [131, 137, 140, 142, 143]. Significant positive associations were found between PM_{10} and lung cancer mortality in Norwegian women between the ages of 51 and 70 (HR: 1.22; 95% CI: 1.1-1.37) and between 71 and 90 (HR: 1.18; 95% CI: 1.04-1.33)[142]. A German cohort study also reported significant positive associations (HR: 1.84; 95% CI: 1.23-2.74) in women with a mean age of 55, however the confidence interval range is relative wide [131]. In contrast, the USA, Norwegian and Chinese cohorts reported nonsignificant positive associations of PM_{10} and lung cancer mortality in men [137, 142, 143].

In summary, the epidemiological studies presented largely demonstrate positive associations between health effects and PM_{10} although some lack statistical significance. Cardiovascular and respiratory effects and mortality were observed in locations with annual mean concentrations ranging from 7.7-270 $\mu g/m^3$. Potential inconsistencies between studies and results could be due to different PM_{10} constituents between geographical regions as well as various study designs and methodology. Nonetheless, the published literature presented herein is consistent with previous (<2010) epidemiological studies of which the USEPA based their conclusions regarding the associations between health effects and PM_{10}. The USEPA concluded that the evidence provided in the literature and the biological plausibility was *suggestive* of a causal relationship between short-term exposures to $PM_{10-2.5}$ and cardiovascular effects, respiratory effects, mortality, yet there was inadequate evidence to suggest causative relationships with long-term exposures [120]. In the epidemiological studies valuated in the USEPA review, associations between short-term $PM_{10-2.5}$ exposures and cardiovascular

and respiratory effects and mortality were observed in studies with mean 24-hour average $PM_{10-2.5}$ ranging from 5.6-33.2 $\mu g/m^3$ and maximum concentrations ranging from 24.6-418 $\mu g/m^3$.

Table 3.1 Multiple comparisons *p* values (2-tailed) for Kas Chikitu PM_{10} ($\mu g/m^3$) concentrations by year. Kruskal-Wallis ANOVA and median test: H(4, *n*=1603)=533.4777, *p*=0.000. Statistically significant *p*-values are shown in red.

	2010	2011	2012	2013	2014
2010		0.000091	0.000000	0.000000	0.000000
2011	0.000091		0.315295	0.000000	0.000000
2012	0.000000	0.315295		0.000000	0.000000
2013	0.000000	0.000000	0.000000		0.000025
2014	0.000000	0.000000	0.000000	0.000025	

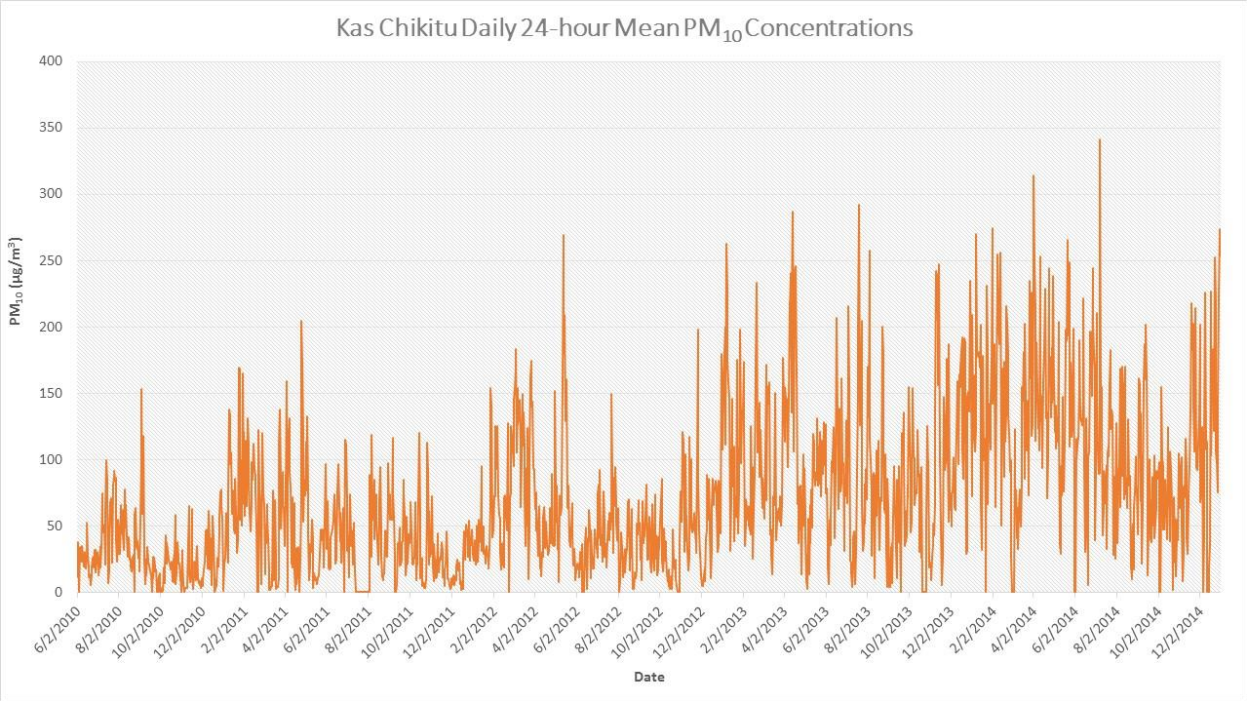


Figure 3.1 Daily 24-hour mean PM_{10} concentrations collected at the Kas Chikitu air monitoring station in Curaçao from June 1, 2010 through December 31, 2014.

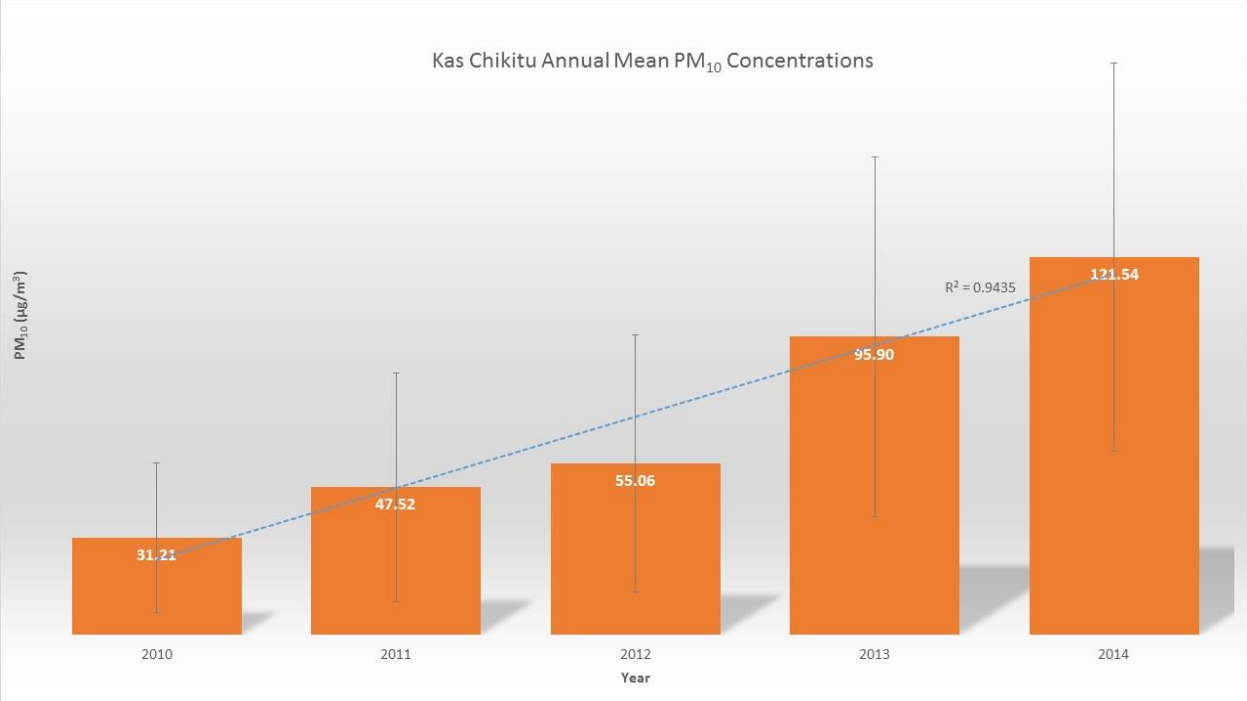


Figure 3.2 Annual mean concentrations of PM_{10} measured at the Kas Chikitu air monitoring station in Curaçao for the years 2010 through 2014. Levels illustrate a strong increasing trend (R^2=0.94) over time.

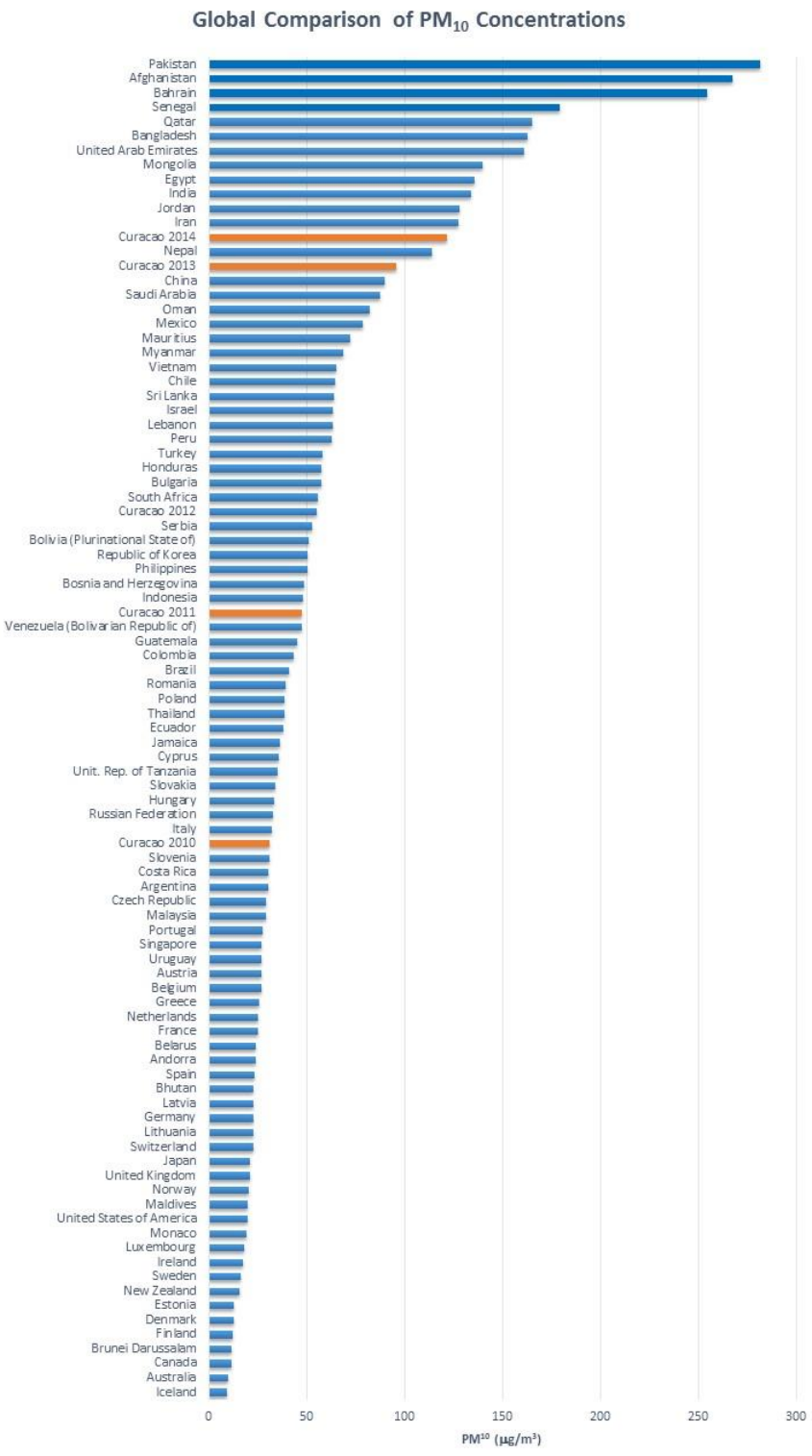


Figure 3.3 Global comparison of mean annual PM_{10} concentrations ($\mu g/m^3$). Concentrations from other countries courtesy of WHO ambient air pollution database.

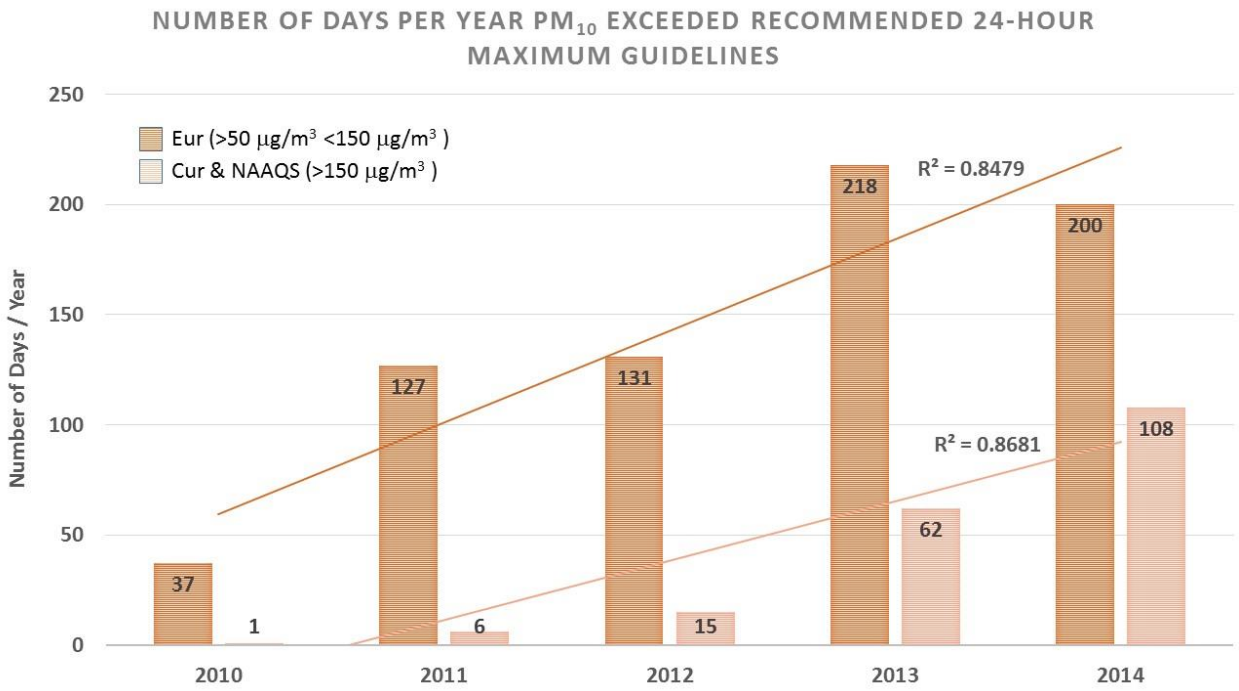


Figure 3.4 Number of days per year PM_{10} concentrations exceeded recommended guidelines. The current 24-hour maximum concentrations recommended by the European Commission (Eur), NAAQS and Curaçao (Cur) are 50 μg/m^3, 150 μg/m^3 and 150 μg/m^3, respectively.

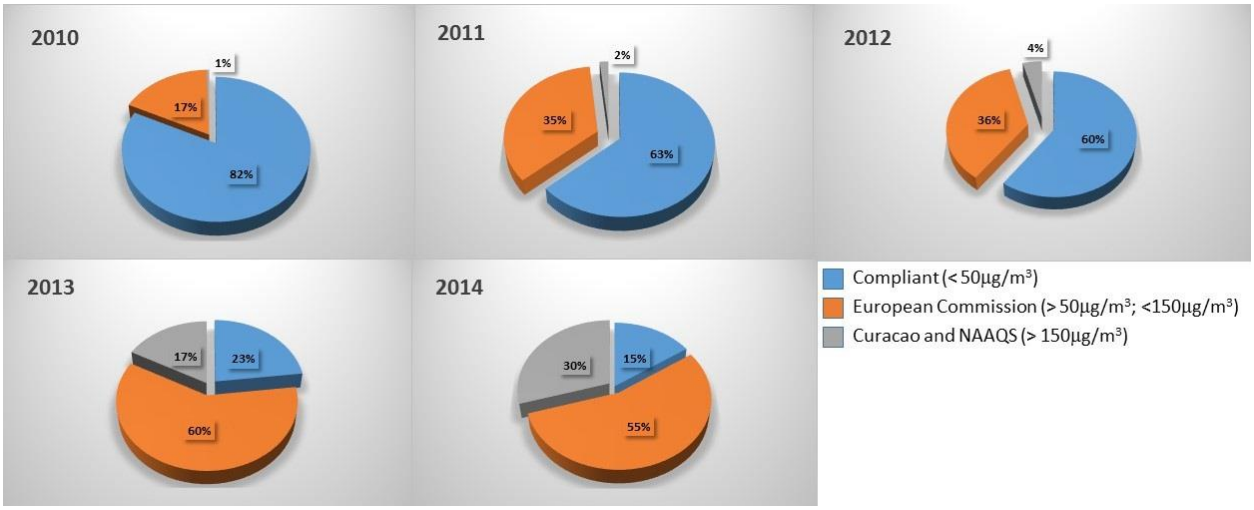


Figure 3.5 Percentages of each year that were either in compliance or exceeded current maximum 24-hour guidelines for PM_{10} concentrations.

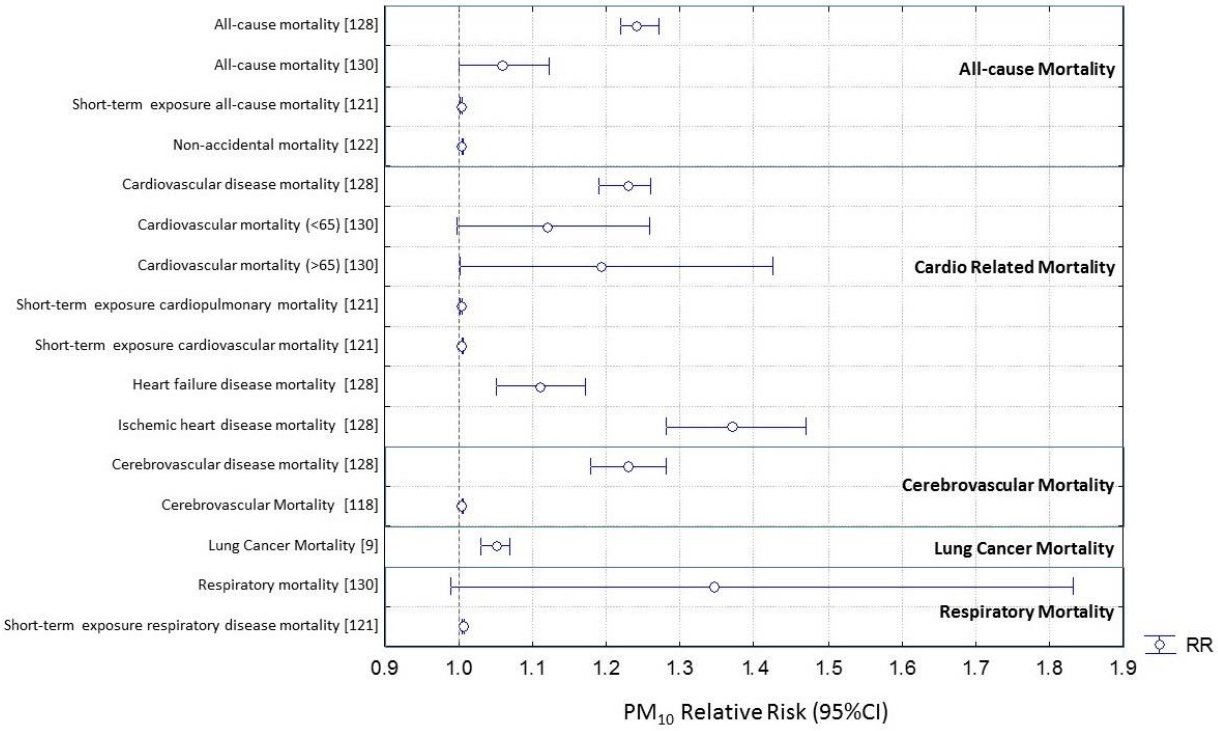


Figure 3.6 Relative risk estimates (95% CI) for PM_{10} associated mortality from published literature.

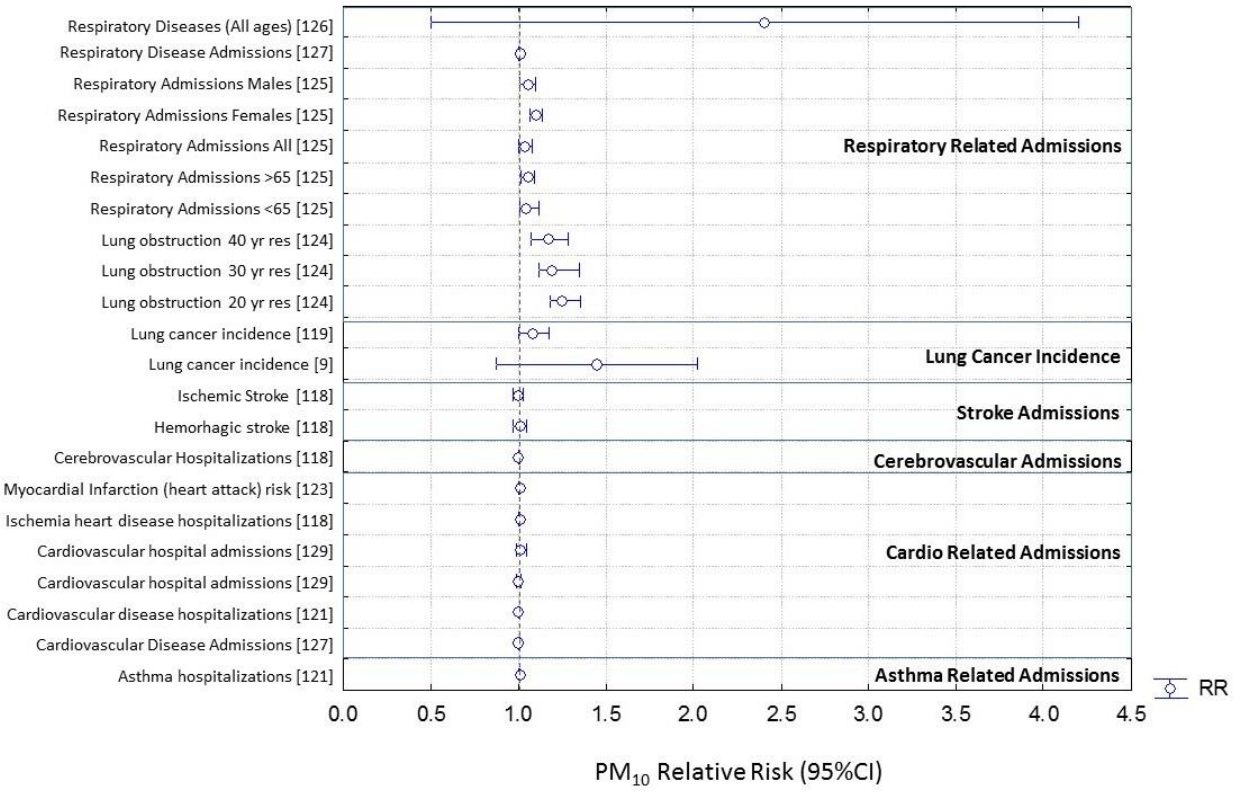


Figure 3.7 Relative risk estimates (95% CI) for PM_{10} associated morbidity and hospital admissions from published literature.

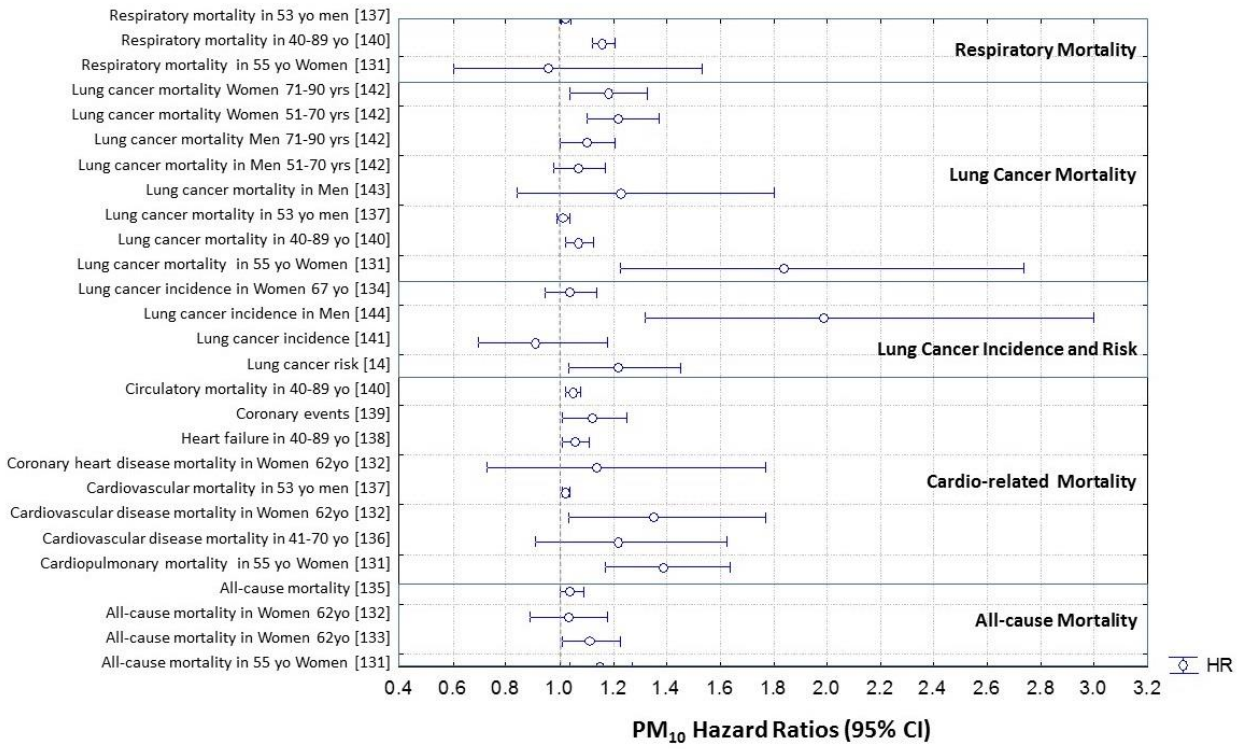


Figure 3.8 Hazard ratios (95% CI) for PM_{10} associated mortality and morbidity from published literature.

CHAPTER FOUR:

SULFUR DIOXIDE

4.1 Ambient Concentrations of Sulfur Dioxide (SO_2)

Daily SO_2 mean concentrations were downloaded from two local air stations in Curaçao, Beth Haim (*n*=1,605) and Kas Chikitu (*n*=1,622). The 24-hour daily mean concentrations ranged from 0.2 to 449 $\mu g/m^3$ and 6.8 to 139 $\mu g/m^3$ at the Beth Haim and Kas Chikitu stations, respectively (Figure 4.1). Mean annual concentrations at the Beth Haim station ranged from 38.9 $\mu g/m^3$ in 2010 to 170.4 $\mu g/m^3$ in 2014 (Figure 4.2). Statistically significant temporal trends were observed at the Beth Haim station with a moderately strong increasing trend (R^2=0.86) over time (Figure 4.3). Table 4.1 summarizes the statistical differences in annual SO_2 concentrations between years at the Beth Haim air monitoring station. Mean annual SO_2 concentrations at the Kas Chikitu station ranged from 35.6 $\mu g/m^3$ in 2010 to 55.5 $\mu g/m^3$ in 2014, also illustrating a moderately strong increasing trend (R^2=0.86) over time (Table 4.2; Figure 4.2).

4.1.1 Global Comparison of SO_2 Concentrations

Annual concentrations measured at the Beth Haim station increased 338% since 2010 and 36% since 2011. Similarly, annual concentrations increased at the Kas Chikitu station 36% since 2010 and 17% since 2011. Conversely, global trends for SO_2 have illustrated decreases. For instance, a 50% decrease in annual average SO_2 concentrations was reported in the Yangtze Delta region of eastern China (2005-2010) and in Europe (2001-2010) [145, 146]. The US also reported an 81%

decrease in the daily one hour maximum average for SO_2 concentrations from 1980-2013 [147]. Decreases were also observed in annual SO_2 concentrations in Mexico City with an 84% decrease between 1986 and 2003 [148]. The annual SO_2 concentrations measured at both the Beth Haim and Kas Chikitu station for the years 2010-2014 are among the highest reported globally (Figure 4.3). The 2014 concentrations measured at Beth Haim were more than 200 times greater than those measured in Nuraminis, Italy (0.72 $\mu g/m^3$) in 2012 (http://www.eea.europa.eu). In addition, the 2013 annual SO_2 concentrations (155.9 $\mu g/m^3$) at the Beth Haim station were over 5 times higher than the 2013 US annual average (29.4 $\mu g/m^3$).

4.1.2 SO_2 Compliance with Public Health Guidelines

The maximum annual mean concentrations for SO_2 that are currently recommended by Curaçao and WHO are 80 $\mu g/m^3$ and between 40-60 $\mu g/m^3$, respectively. The SO_2 annual mean concentrations in 2010 at both the Beth Haim (38.9 $\mu g/m^3$) and Kas Chikitu (35.6 $\mu g/m^3$) air stations was the only year concentrations were less than the recommended guidelines, however, it is important to note the measurements were only for a 7 month period (July-December). The 24-hour guidelines issued by the European Commission and Curaçao both recommend 125 $\mu g/m^3$ with three permissible excursions. Curaçao also mean 24-hour maximum concentrations do not exceed 365 $\mu g/m^3$ more than once per year. In addition, WHO recommends 24-hour maximum concentrations of 100-150 $\mu g/m^3$. Similar to the trends observed for PM_{10}, the number of days that exceed the 24-hour guidelines at both stations are also increasing over time, although the trends are not as strong as the trends observed for PM_{10} (Figures 4.4-4.5). The 24-hour recommended SO_2 guidelines were within compliance for the majority of 2010 (95%), 2011 (78%) and 2012 (84%) were within compliance at the Beth Haim Station (Figure 4.6). However, 2013 and 2014 exceeded the recommended SO_2

guidelines 74% and 81% of the year, respectively. Curaçao allows three excursions per year above the 24-hour maximum recommendation (125 μg/m^3). In 2011, 2012, 2013, and 2014 this value was exceeded 30, 15, 40 and 54 times. In contrast, the daily SO_2 concentrations measured at the Kas Chikitu station were compliant for the majority of each of the years measured in this study [2010 (99%). 2011 (98%), 2012 (96%), 2013 (98%) and 2014 (95%)]. The only year that exceeded the number of permissible of excursions was 2012, with a total of 4 days exceeding 125 μg/m^3.

4.1.3 Potential Risks of SO_2 Inhalation

Many epidemiological studies have also reported relative risk (RR) estimates, hazard ratios (HR) and odds ratios (OR) for associations between mortality and morbidity and SO_2 with many of the same inconsistencies observed with PM_{10} (Figures 4.7-4.9) [121, 123, 126, 127, 130, 149-158]. This study primarily focused on literature published within the last five years since there are several meta-analyses and reviews covering literature published prior to 2010.

Many of the studies reported relative risk estimates that were either close to or included unity or had large confidence intervals (Figure 4.7). A study evaluating air pollution effects on residents of central Taiwan, reported positive relative risk estimates (RR: 1.043, 95% CI: 1.018-1.098) for associations between SO_2 (12.6 μg/m^3 winter mean concentration) and all-cause mortality during the winter months[130]. Similarly, the health impacts were assessed in the megacity of Iran and also reported positive associations (RR: 1.004, 95% CI: 1.003-1.0048) between all-cause mortality and SO_2 concentrations (89.2 μg/m^3 annual mean)[150]. Lai et al. [121] conducted a meta-analysis of research between 1989 and 2010 reporting health effects on Chinese populations in China, Taiwan and Hong Kong, which also resulted in positive associations (RR: 1.007, 95% CI: 1.0045-1.0097) between SO_2 (14-213 μg/m^3 annual mean) and all-cause mortality.

Several studies reported positive associations between SO_2 and cardio related mortality and risk [121, 123, 130, 149, 150]. A Canadian study reported positive associations (RR: 1.061, 95% CI: 1.018-1.105) between SO_2 (15.72 $\mu g/m^3$ spring mean) and cardiovascular mortality during the spring when the weather was described as dry and tropical [149]. The meta-analysis evaluating air pollution effects on Chinese populations also reported positive associations between short-term SO_2 exposure (14-213 $\mu g/m^3$ annual mean) and cardiopulmonary (RR: 1.01, 95% CI: 1.009-1.015) and cardiovascular mortality (RR: 1.007, 95% CI: 1.004-1.01) [121]. A meta-analysis of 34 studies concluded statistically significant positive associations (RR: 1.01, 95% CI: 1.003-1.017) with SO_2 and increased risks of myocardial infarction [123].

The relative risks reported for chronic obstructive pulmonary disease (COPD) and respiratory mortality or morbidity appear to be much less conclusive. Significant positive associations between SO_2) and COPD were reported in Shiraz (674.9 $\mu g/m^3$ annual mean; RR: 1.095, 95% CI: 1.07-1.11) and Tabriz, Iran (19 $\mu g/m^3$ annual mean; RR: 1.0044, 95% CI: 1.0-1.011) [127, 151]. In contrast, a study reported nonsignificant associations (RR: 1.9, 95% CI: -3.9-8) with COPD in the heavily polluted city of Lanzhou, China where the mean SO_2 concentrations (79 $\mu g/m^3$ annual mean) are four times higher than those reported in Tabriz, Iran and almost an order of magnitude lower than those reported in Shiraz, Iran [126]. Nonsignificant positive associations were found between respiratory mortality and SO_2 in Canada (RR: 1.11, 95% CI: 0.996-1.238) and Taiwan (RR: 1.176, 95% CI: 0.998-1.384) [130, 149]. In contrast, significant positive associations were reported in Iran (RR: 1.01, 95% CI: 1.006-1.014) and in a 26 study meta-analysis study (RR: 1.012, 95% CI: 1.0058-1.0199) [121, 150].

A number of studies also reported odds ratios for various morbidities (Figure 4.8). Amster et al. [152] reported positive associations between asthma (OR: 1.89, 95% CI: 1.1-3.25) and shortness of breath (OR: 1.09, 95% CI: 1.1-3.27) with total ambient SO_2 concentrations of 6.6 $\mu g/m^3$, yet

interestingly, nonsignificant associations were observed with higher SO_2 (43 $\mu g/m^3$) concentrations related to specific coal-fired power plant events. A meta-analysis evaluating effects from long-term air pollution exposure reported nonsignificant associations (OR: 1.02, 95% CI: 0.97-1.08) between SO_2 and asthma prevalence and wheezing in children [155]. Conversely, in a cross-sectional study consisting of over 23,000 Chinese children, significant positive associations (OR: 1.23, 95% CI: 1.14-1.32) were reported between SO_2 (50.3 $\mu g/m^3$ annual mean) and asthma [159].

The hazard ratios reported for mortality and various respiratory morbidities also illustrated positive associations with SO_2 and various endpoints (Figure 4.9). The National English Cohort study, consisting of over 800,000 participants, reported significant positive associations (HR: 1.05, 95% CI: 1.01-1.08) between lung cancer mortality and SO_2 [140]. Similarly, a Japanese cohort study, consisting of over 63,000 study participants, also reported significant positive associations (HR: 1.26, 95% CI: 1.07-1.48) between lung cancer mortality and SO_2, although the confidence interval range is relatively wide [158]. Hazard ratios were also reported for respiratory mortality and COPD. Both a National English Cohort (HR: 1.09, 95% CI: 1.06-1.12) and the Japanese Cohort (HR: 1.43, 95% CI: 1.33-1.54) studies reported significant positive associations between SO_2 and respiratory mortality [140, 158]. For COPD, significant associations were reported in a National English Cohort study (HR: 1.07, 95% CI: 1.03-1.11) yet nonsignificant associations (HR: 1.32, 95% CI: 0.88-1.98) were reported in the Japanese cohort study [157, 158].

In summary, the epidemiological studies presented largely demonstrate positive associations between health effects and SO_2 although many lack statistical significance. Potential inconsistencies between studies and results could be due to confounding factors with copollutants and various study designs and methodology. In general, cardiovascular and respiratory effects and mortality were observed in locations with a wide range in annual mean concentrations ranging from 4675 $\mu g/m^3$.

The results from these studies were similar to those the USEPA based their conclusions in addition to animal and toxicological studies providing biological plausibility. Short-term exposures to SO_2 were determined to have a *causal* relationship between respiratory morbidity yet the evidence was inadequate to infer a causality with cardiovascular health [160]. In addition, the epidemiological evidence on the effect of short-term SO_2 exposures on all-cause and cardiopulmonary mortality is also *suggestive* of a causal relationship at ambient concentrations. In contrast, the available evidence was inadequate to infer causal relationships between long-term SO_2 exposures and respiratory effects (including asthma), cardiovascular effects and mortality. In the epidemiological studies evaluated in the USEPA review, associations between short-term SO_2 exposures and respiratory effects were observed in locations with mean 24-hour average SO_2 concentrations ranging from 2.62 to 78.6 $\mu g/m^3$, with maximum values ranging from 31.4 to 196.5 $\mu g/m^3$. In addition, associations with mortality were observed with mean 24-hour average SO_2 concentrations less than 26.2 $\mu g/m^3$.

Table 4.1 Multiple comparisons *p* values (2-tailed) for Beth Haim SO_2 ($\mu g/m^3$) concentrations by year. Kruskal-Wallis ANOVA and median test: H(4, *n*=1605)=717.3665, *p*=0.000. Statistically significant *p*-values are shown in red.

	2010	2011	2012	2013	2014
2010		0.000354	0.001280	0.000000	0.000000
2011	0.000354		1.000000	0.000000	0.000000
2012	0.001280	1.000000		0.000000	0.000000
2013	0.000000	0.000000	0.000000		0.401721
2014	0.000000	0.000000	0.000000	0.401721	

Table 4.2 Multiple comparisons *p* values (2-tailed) for Kas Chikitu SO_2 ($\mu g/m^3$) concentrations by year. Kruskal-Wallis ANOVA and median test: H(4, *n*=1622)=148.9794, *p*=0.000. Statistically significant *p*-values are shown in red.

	2010	2011	2012	2013	2014
2010		0.000000	0.000000	0.000000	0.000000
2011	0.000000		1.000000	0.125708	0.000000
2012	0.000000	1.000000		0.042149	0.000000
2013	0.000000	0.125708	0.042149		0.003736
2014	0.000000	0.000000	0.000000	0.003736	

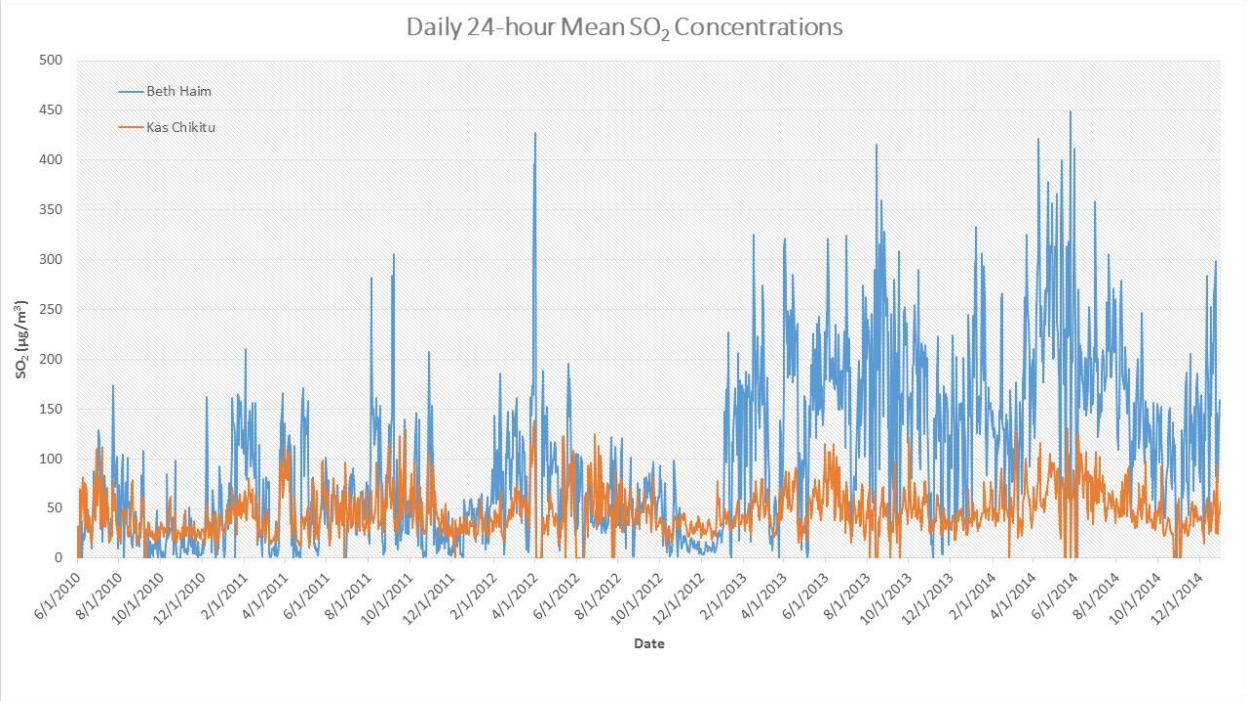


Figure 4.1 Daily 24-hour mean concentrations of SO_2 (µg/m^3) measured at the Beth Haim and Kas Chikitu air monitoring stations in Curaçao from June 1, 2010 through December 31, 2014.

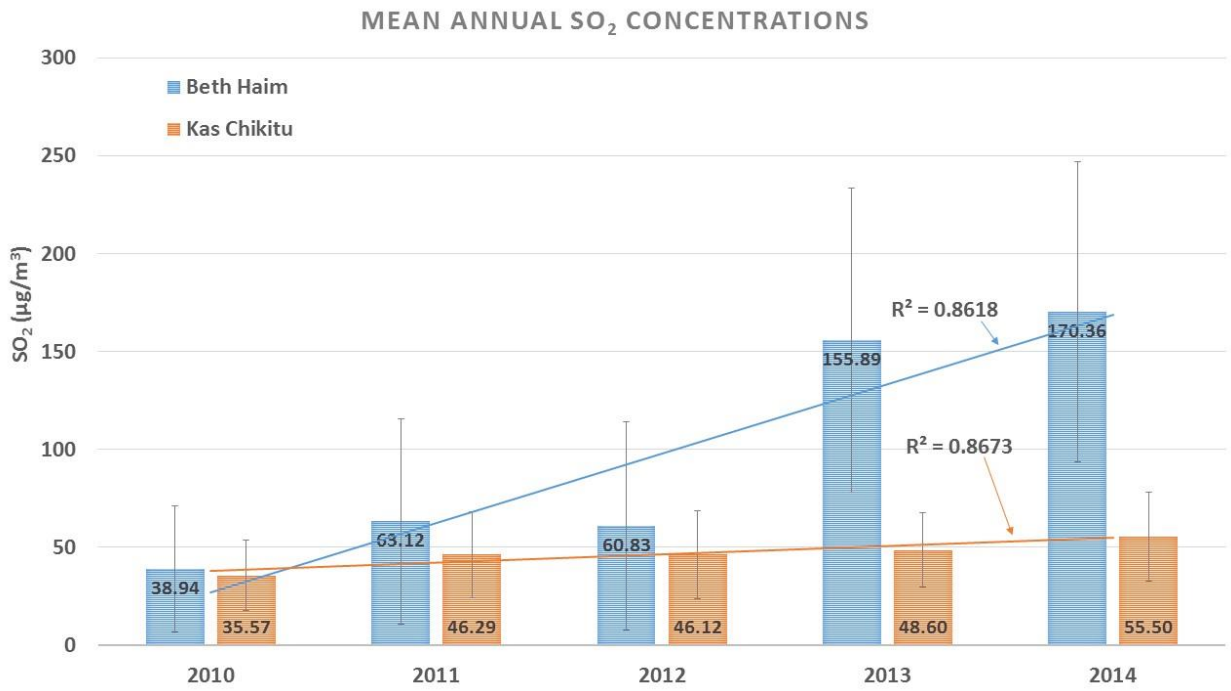


Figure 4.2 Mean annual SO_2 concentrations ($\mu g/m^3$) measured at Beth Haim (■)and Kas Chikitu (■) demonstrate significant temporal trends for the years 2010 through 2014.

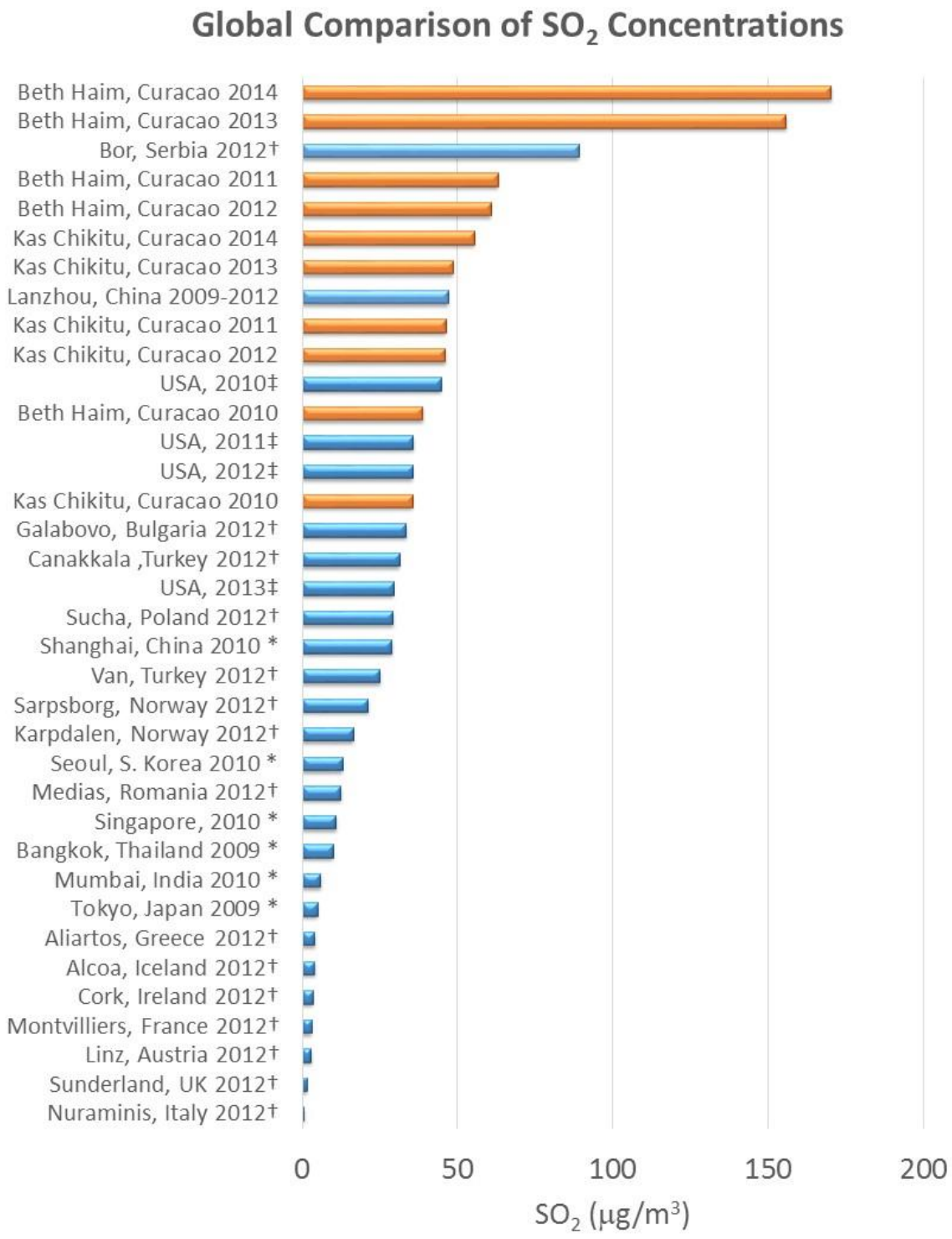


Figure 4.3 Global comparison of mean annual SO_2 concentrations (µg/m^3). Sources for the concentrations from other countries are as follows †European Environment Agency, ‡USEPA and *Clean Air Asia Citites ACT.

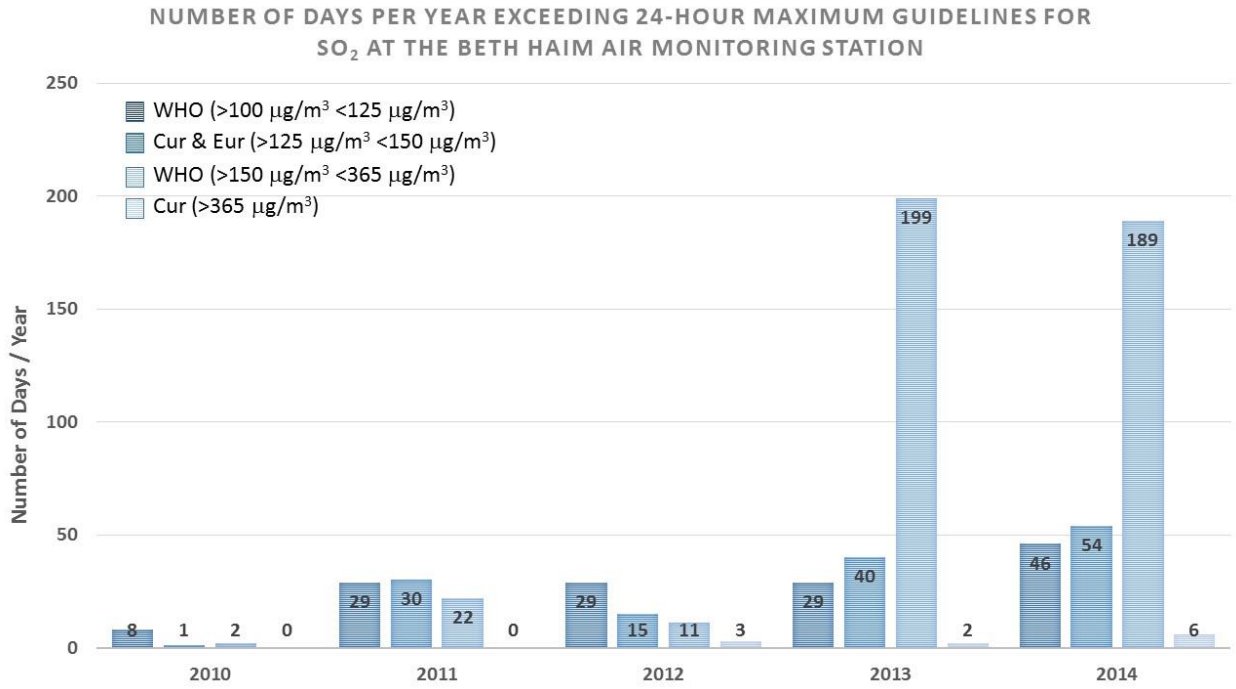


Figure 4.4 Number of days per year the 24-hour maximum guidelines for SO_2 concentrations were exceeded at the Beth Haim air station. Current recommended 24-hour maximum concentrations recommended by the World Health Organization (WHO) and the European Commission (Eur) are 100-150 μg/m^3 and 125 μg/m^3, respectively. Curaçao (Cur) recommends 125 μg/m^3 with three excursions per year and 365 μg/m^3 with one permissible excursion per year.

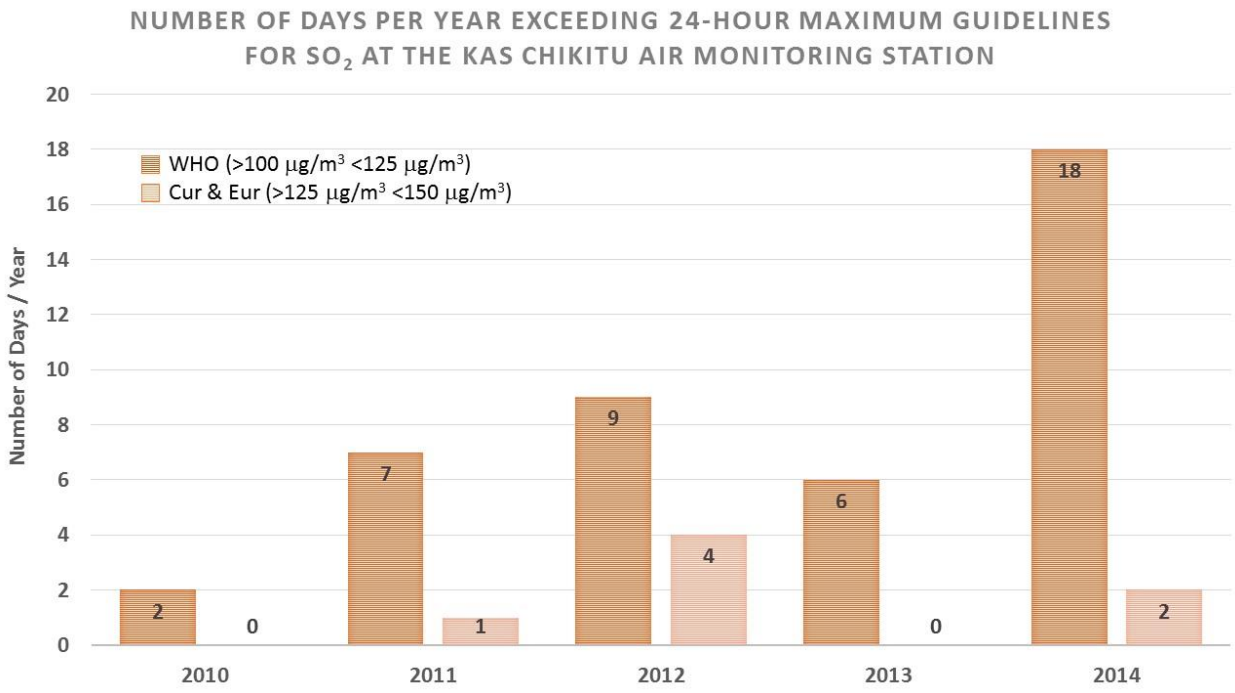


Figure 4.5 The number of days per year SO_2 concentrations exceeded available recommended 24-hour maximum guidelines at the Kas Chikitu station. The current 24-hour maximum concentration guidelines recommended by the World Health Organization (WHO), the European Commission (Eur) and Curaçao are 100-150 μg/m^3, 125 μg/m^3 and 125 μg/m^3, respectively.

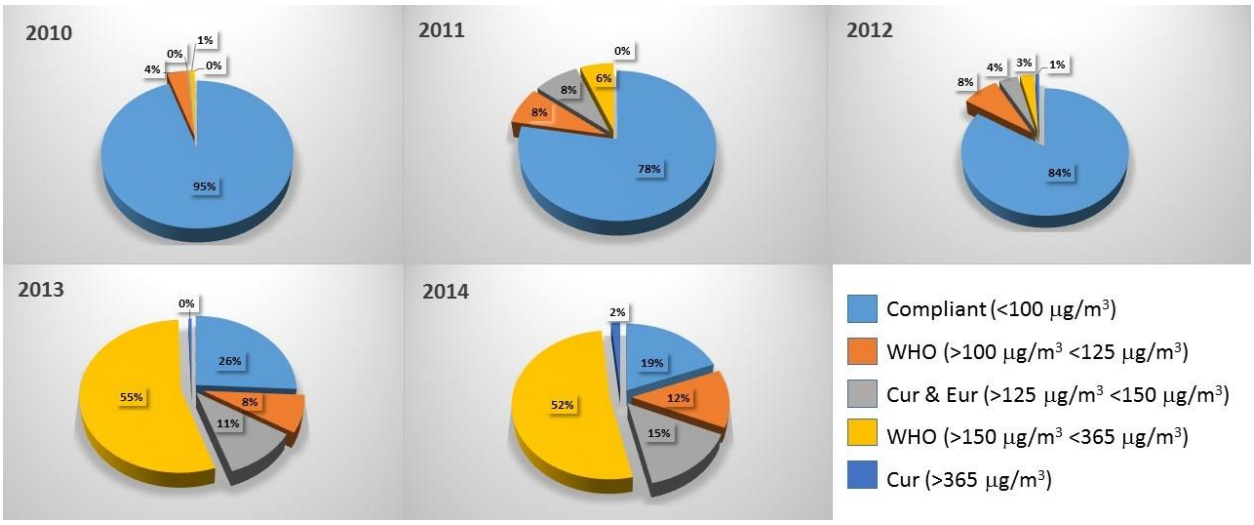


Figure 4.6 Percentages of each year that were either in compliance or exceeded current maximum 24-hour guidelines for SO_2 concentrations at the Beth Haim air monitoring station.

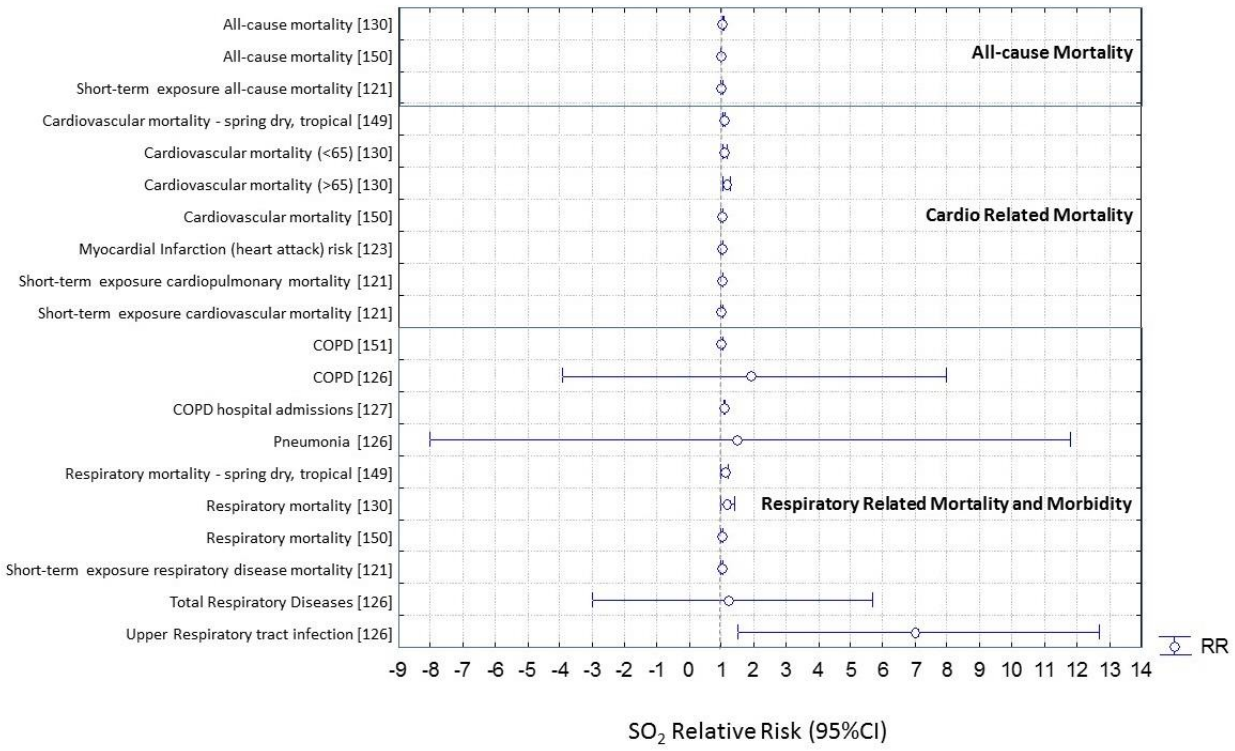


Figure 4.7 Relative risk estimates (95% CI) for SO_2 associated mortality and morbidity from published literature.

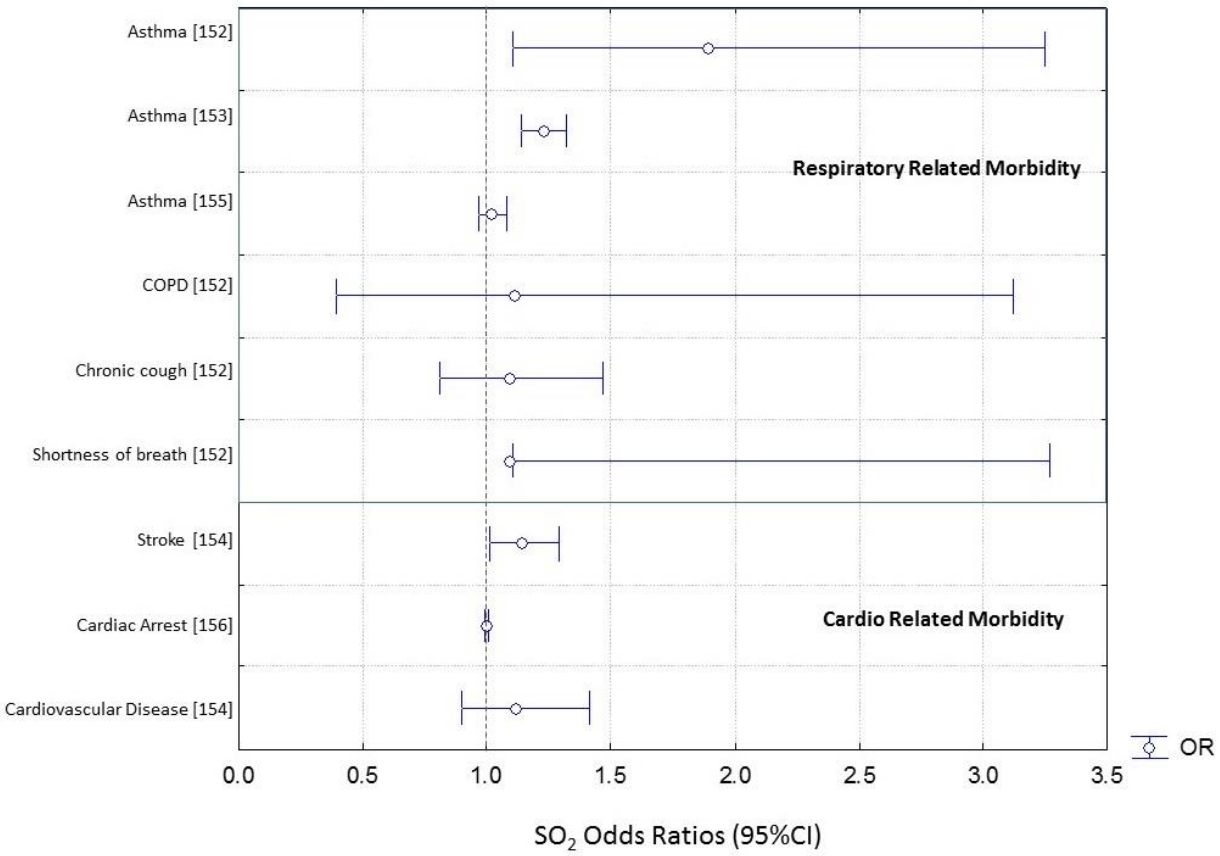


Figure 4.8 Odds ratios (95% CI) for SO_2 associated morbidity from published literature.

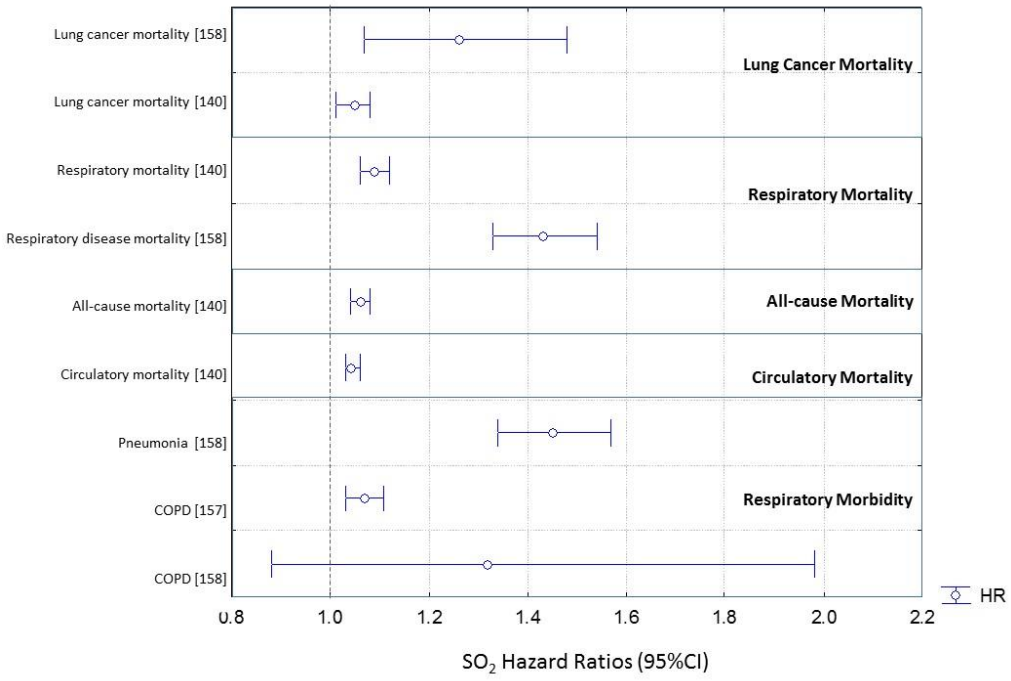


Figure 4.9 Hazard ratios (95% CI) for SO_2 associated morbidity from published literature.

CHAPTER FIVE:

POLYCYCLIC AROMATIC HYDROCARBONS

5.1 Ambient Concentrations of Polycyclic Aromatic Hydrocarbons in Curaçao

5.1.1 Levels of Ambient PAHs Collected in 2011

Calculated ambient concentrations of PAHs during the 2011 sampling event ranged from 1.2 to 790.9 ng/m^3 (Figure 5.1). Table 5.1 summarizes the site specific polyurethane foam disk concentrations (ng/disk) and the calculated ambient PAHs (ng/m^3) in Curaçao during 2011. The highest levels of PAHs were measured at the two sites directly downwind of Isla Refineriá, Habaai (790.9 ng/m^3) and Marchena (454.4 ng/m^3). The lowest levels were measured in Blauw/Curasol (1.2 ng/m^3) which was one of the furthest sites west of the refinery (~6 km). The PAH concentrations measured at Habaai were over 600 times higher than those measured at Blauw/Curasol and were almost 15 times higher than the most eastern or upwind site, Marie Pampoen (54.6 ng/m^3). However, there were no clear trends from east (upwind) to west (downwind) or with increasing distance from the refinery. This was not surprising considering the majority of the 2011 passive samplers were deployed downwind of the refinery to assess the extent of PAH detection. Nevertheless, the two sites directly downwind (Habaai & Marchena) of the refinery had concentrations significantly higher than all other sites equaling one to two orders of magnitude higher than the eastern and western most sites, respectively. In order of decreasing mean concentrations of targeted PAHs, Habaai had the highest levels followed by Marchena > W. Buena Vista > Heintje Kool > Boka Sami > W. Piscadera Baai > Groot Piscadera > Nieuw Nederland > Rooi Catochi > E. Piscadera Baai > E. Buena Vista > Roosendaal > Marie Pampoen > Parasasa > Blauw/ Curasol.

The 2011 sampling event was primarily to determine the extent of the plume based on the consistent direction of the Trade Winds, which is a critical factor in ambient levels and distribution of PAHs. PAS-PUFs were deployed in the general path determined by yearly average wind distribution plots in Curaçao (Figure 5.2). Atmospheric PAHs were detected ~6.68 km west of the refinery in Boca Sami (163.8 ng/m^3 ΣPAH), which was the western most site sampled during 2011. Levels were moderately elevated considering the distance however, this could be attributed to the terrain elevation and physical geography. Boca Sami was the highest point sampled with an elevation of approximately 120 feet above sea level whereas the refinery sits at approximately 22 feet above sea level.

5.1.2 Levels of Ambient PAHs Collected in 2014

Atmospheric PAH concentrations during the 2014 sampling event ranged from 27.3 to 660.1 ng/m^3 (Figure 5.3). Table 5.2 summarizes the site specific polyurethane foam disk concentrations (ng/disk) and the calculated ambient PAHs (ng/m^3) in Curaçao during 2014. There were no observable trends or statistical differences (p=0.25) between the mean ambient PAHs measured in Curaçao during 2011 (183.6 ± 201.4 ng/m^3) and 2014 (145.2 ± 164.0 ng/m^3; Figure 5.4). The calculated 2014 PAH levels ranged from 31.4 to 660.1 ng/m^3 and 27.3 to 69.8 ng/m^3 for the downwind (west of the refinery) sites and upwind (east of the refinery) sites, respectively. The sites downwind (mean ± sd: 248.1 ± 181.4 ng/m^3) of Isla Refineriá and Schottegat Harbor had statistically higher (p=0.0006) atmospheric PAH levels compared to the upwind sites (42.3 ± 11.9 ng/m^3; Figure 5.5). In order of decreasing mean concentrations of targeted PAHs measured in the upwind sites, Punda had the highest concentrations of PAHs (69.8 ng/m^3) followed by Steenrijk > Emmastad (Site #4) > Zeelandia > Suffisant (Site #28) > Biesheuvel > Brievengat / Groot Kwartier > Toni Kunchi > Pietermaai / Salina > Rooi Catochi (Site #12) > Emmastad (Site #14) > Rooi Catochi (Site #3) > Suffisant (Site #16) > Nieuw Nederland > Groot Davelaar / Joonchi. For the 2014 sites downwind,

Welgelegen (Veld Salu) had the highest measured concentrations of targeted PAHs (660.1 ng/m^3), followed by Marchena (Beth Haim) >Habaai >Marchena (Site #23) > Wishi > Buena Vista (Site #18) > Buena Vista (Site #17) > Buena Vista (Site #21) > Domi / Welgelegen > Parasasa > Roosendaal / Gasparitu > Parasasa (Site #24) > Gasparitu > Buena Vista (Site #20).

5.1.3 Global Comparisons of Ambient PAH Concentrations

Calculated ambient concentrations of PAHs in Curaçao ranged from 1.2 to 790.9 ng/m^3 in 2011, with the highest levels of PAHs measured at the two sites directly downwind of Isla Refineriá, Habaai (790.9 ng/m^3) and Marchena (454.4 ng/m^3). Ambient PAH concentrations in 2014 ranged from 27.3 to 660.1 ng/m^3. Although there were no temporal differences between the 2011 and 2014 ambient concentrations there were significant spatial differences between the 2014 upwind and downwind sites in relation to Isla Refineriá. The consistent direction of the Trade Winds is a critical factor in ambient levels of PAHs downwind of the refinery. These results are supported by a previous study evaluating other emission constituents (i.e., total suspended particulate, sulfate, chlorides, lead) which concluded the refinery affects a substantial portion of the western side of the island, and more so, those sites directly downwind of the refinery [161].

In general, the ambient concentration of PAH levels in this study were consistent with other urban and industrial regions found globally, but up to three orders of magnitude higher than some remote and rural areas (Figure 5.6)[74, 77, 162-173]. The mean concentration of PAHs from the 2014 sites located downwind (248.1 ng/m^3) of Isla Refineriá were among some of the highest reported ambient PAHs globally, and were almost 200 times higher than those reported in some areas of Europe and Spain [74, 162]. The PAH concentrations measured at the 2011 sites and the sites located downwind of Isla Refineriá were on the same order of magnitude as China, Turkey, and India [166,

167, 169]. In comparison, the sites located upwind of Isla Refineriá were 33 times higher than remote areas of Europe, yet almost 6 times lower than those measured downwind in this study and almost 24 times lower than the levels reported in Saudi Arabia [74, 171]. Although the ambient PAHs measured upwind (42.3 ± 11.9 ng/m^3) of Isla Refineriá were substantially lower than those measured near industrial areas in Saudi Arabia, they were similar to those measured in other residential areas surrounding an oil refinery in Saudi Arabia [173].

5.1.4 Ambient PAH Concentrations and Compliance

Most of the recommended guidelines for PAH concentrations are specific to occupational exposures. However, the World Health Organization's risk estimate for ambient air concentrations of PAHs has suggested a lifetime exposure guideline value of 0.1 ng/m^3 B[*a*]P as an indicator and 2 ng/m^3 of fluoranthene as a secondary indicator, theoretically leading to one excess cancer case in 100,000 (1E-5) individuals [70]. Although International and National regulations tend to be in agreement for the most part, on maximum allowable levels of PAHs in air and water, state to state regulations and guidelines seem to vary tremendously.

The only site in 2011 that had detectable levels of B[a]P was Habaai with a calculated ambient air concentration of 0.13 ng/m^3. This is slightly higher than the WHO recommended lifetime exposure guideline of 0.1 ng/m^3 B[*a*]P, which theoretically would lead to one extra cancer case in 100,000 exposed individuals [70]. The B[a]P concentrations in the 2014 ambient air samples (upwind and downwind) ranged from non-detect to 0.27 ng/m^3 in Buena Vista (site #18). The mean concentration from all sites in 2014 was 0.04 ± 0.06 ng/m^3 for B[a]P. The 2014 B[a]P mean concentration for the sites located downwind and upwind of the refinery were 0.05 ± 0.07 ng/m^3 and 0.03 ± 0.06 ng/m^3, respectively and were not statistically different (p=0.36). Two sites, one site upwind (Emmastad, site #4, 0.23 ng/m^3) and one site downwind (Buena Vista, site #18, 0.27 ng/m^3),

exceeded the WHO recommended lifetime exposure guideline of 0.1 ng/m^3 B[*a*]P, yet a few sites were approaching this level (i.e., Buena Vista (site #21), Domi/Welgelegen, and Nieuw Nederland). It is noteworthy to mention, that the two sites exceeding the B[a]P guideline were located along Schottegat Ring, suggesting vehicle emissions as the primary source of B[a]P.

Mean fluoranthene concentrations in the 2011 ambient air was 2.97 ± 2.73 ng/m^3, with concentrations ranging from non-detect at the Blauw/Curasol site to 11.4 ng/m^3 at the Habaai site. Habaai had concentrations almost six times higher than the recommended value of 2 ng/m^3. The mean fluoranthene concentrations were 3.15 ± 3.04 ng/m^3 and 2.28 ± 0.55 ng/m^3 for the sites located downwind (west) and upwind (east) of the refinery, respectively. Eleven of the 15 sites sampled in 2011 were above the WHO recommended lifetime exposure guideline of 2 ng/m^3 of fluoranthene, which theoretically would lead to one extra cancer case in 100,000 (1E-5) exposed individuals. The sites below this level were Blauw/Curasol, west Piscadera Baai, Marie Pampoen, and Parasasa.

The 2014 fluoranthene levels in ambient air ranged from 0.58 ng/m^3 in Suffisant (site #16) to 7.74 ng/m^3 in Rooi Catochi (site #3). The mean fluoranthene concentration at all sites was 2.74 ± 1.74 ng/m^3. The 2014 mean concentrations of fluoranthene downwind (2.76 ± 1.34 ng/m^3) and upwind (2.71 ± 2.11 ng/m^3) of the refinery were not statistically different (p=0.94). Sixteen of the 30 locations sampled exceeded the WHO recommended lifetime exposure guideline of 2 ng/m^3 of fluoranthene in 2014.

5.2 Source Characterization

Identifying and understanding the impact of emission sources is critical for proper risk assessment and management [110]. The emission of PAHs are from a variety of anthropogenic sources which can be categorized as domestic sources, mobile sources, industrial and agricultural sources. Binary diagnostic ratios and PAH concentration profiles have been useful in identifying

emission sources and their contributions to ambient air concentrations in order to distinguish between different sources, such as petrogenic, pyrogenic or phytogenic [50, 174-176]. PAHs originating from petrogenic sources primarily consist of the low molecular weight compounds in contrast to the pyrogenic sources which primarily consist of the high molecular weight compounds [104-107].

5.2.1 Concentration Profiles

Ambient air samples collected in 2011 were dominated by the 2-3 ring compounds, accounting for 97% of the measured PAHs, followed by the 4-ring (3%), and the 5-6 ring (0.05%) compounds (Figure 5.7). Similarly, the air samples collected downwind of the refinery in 2014 were also dominated by the 2-3 ring compounds, accounting for 96% of the measured PAHs in the samples, followed by the 4-ring (4%) and the 5-6 ring (0.1%) compounds (Figure 5.8). The air samples collected upwind of the refinery were dominated by the 2-3 ring compounds (84%) but to a lesser extent, followed by the 4-5 ring (15%) and 5-6 ring compounds (0.5%; Figure 5.9).

The 2011 concentration distribution profiles illustrate both a petrogenic and pyrogenic signal (Figure 5.10). The profile is dominated by the low molecular weight compounds (2-3 rings) and the fluorenes, dibenzothiophenes and phenanthrene and anthracene series all demonstrate a classic bell-shape curve indicative of petrogenic sources. The naphthalene and fluoranthene and pyrene series, however, are dominated by the parent compound (C0) with decreasing concentration with increasing alkylation, indicative of a pyrogenic source. The 2014 downwind and upwind PAH profiles are also dominated by the low molecular weight compounds (2-3 rings) and all the homologue series illustrate the classic bell shape characteristic of petrogenic sources, with the exception of the fluoranthene and pyrene series (Figure 5.11-5.12).

Overall, the concentration and distribution profiles in this study from the 2011 sites and the 2014 upwind and downwind sites, were all dominated by low molecular weight compounds, suggesting

a petrogenic source. In petroleum refining, the 2-3 ring compounds account for approximately 94% of the PAHs [50]. In this study, the 2-3 ring PAH compounds accounted for 97%, 96% and 84% at the 2011 sites, the 2014 downwind sites and the 2014 upwind sites, respectively. This would also suggest a strong petrogenic source and that the 2011 and the 2014 downwind locations may be highly influenced by Isla Refineriá. In contrast, the profiles from the 2014 upwind sites are indicative of both pyrogenic and petrogenic sources.

5.2.2 Binary Diagnostic Ratios

Using binary diagnostic ratios, studies have been able to differentiate between vehicular and non-traffic emissions, diesel and gasoline combustion, different crude oil processing and biomass burning [50, 104, 114-116]. However, each of the diagnostic approaches has its limitations and uncertainties. For instance, the ratios of anthracene/(anthracene+phenanthrene) [ANT/(ANT+PHE)] may be strongly influenced by photoreactions resulting in ratios close to 1, whereas photoreactions can result in higher values for the fluoranthene/(fluoranthene+pyrene) [FLA/(FLA=PYR)] ratio [110, 177]. Consequently, more than one diagnostic ratio should be used to confirm the indicated source(s), therefore 10 different ratios were analyzed in this study.

The PAH binary diagnostic ratios for the 2011 and 2014 air samples indicated a combination of both petrogenic and pyrogenic sources depending on the ratio (Table 5.3-5.4). The low molecular weight to the high molecular weight compounds (LMW/HMW >1), phenanthrene to phenanthrene plus anthracene (PHN/PHN+ANT=~0.98) and the phenanthrene to anthracene (PHN/ANT >10) from all the 2011 sites suggested a dominant petrogenic (i.e., petroleum, refinery) source. Fluoranthene to fluoranthene plus pyrene (FLA/FLA+PYR >0.5) suggested a pyrogenic source of grass, wood or coal combustion (>0.5) (Table 5.3). Fluoranthene to pyrene (FLA/PYR >1) also suggested a pyrogenic source mainly of coal combustion. The benzo[a]chrysene to benzo[a]chrysene

plus chrysene (BaA/BaA+CHR>0.35) indicated combustion as a source. The additional four ratios (BaP/BaP+CHR, IND/IND+BghiP, IND/BghiP, BaP/BghiP) are primarily used to discriminate between pyrogenic sources (i.e., gasoline vs diesel) however either one or both of the parent compounds were not detected at 14 of the 15 sites collected in 2011 and therefore, were not included in the diagnostic ratio analysis.

The PAH diagnostic ratios for the 2014 air samples also indicated a combination of both petrogenic and pyrogenic sources depending on the ratio (Table 5.4). Similar to the 2011 samples, the low molecular weight to the high molecular weight compounds (LMW/HMW >1) at all of the 2014 sites indicated a dominant petrogenic source. Phenanthrene to phenanthrene plus anthracene (PHN/PHN+ANT) suggested a combination of petrogenic and pyrogenic sources with potential influences from crude oil (PHN/PHN+ANT=~0.98), used motor oil (PHN/PHN+ANT= ~0.78) and gasoline vehicle emissions (PHN/PHN+ANT= ~0.77±12). Similar to the 2011 air samples, the fluoranthene to fluoranthene plus pyrene (FLA/FLA+PYR >0.5) ratios suggested a pyrogenic source of grass, wood or coal combustion (>0.5), fluoranthene to pyrene (FLA/PYR >1) suggested a pyrogenic source mainly of coal combustion and benzo[a]chrysene to benzo[a]chrysene plus chrysene (BaA/BaA+CHR>0.35) indicated combustion as a source.

As a result, the diagnostic ratios used in this study, suggested both petrogenic and pyrogenic sources. The LMW/HMW, PHN/(PHN+ANT), and the PHN/ANT ratios all indicated a dominance of a petrogenic source for all of the 2011 sites in this study. The PHN/(PHN+ANT) ratio for all the 2011 sites were between 0.96 and 0.98 which are close to the values found for crude oil (0.98)[108]. In addition, the ratios for FLA/(FLA+PYR), FLA/PYR and BaA/(BaA+CHR) indicated the presence of pyrogenic (i.e., coal) and combustion sources. Similarly, the diagnostic ratios calculated for all the 2014 sites indicated a combination of petrogenic and pyrogenic sources. It is

also important to note, that interpreting diagnostic ratios should be done with caution as values may change with environmental fate as well as with vapor and particulate phases.

5.2.3 Factor Analysis and Principle Components Analysis

In addition to concentration profiles, distribution profiles and binary diagnostic ratios, factor analysis and principle components analysis was also conducted to assist in elucidating potential sources in Curaçao. Factor loadings for each of the parent PAHs help explain the principle components associated with sites and sources in order to differentiate and identify specific emission sources.

The factor analysis and principle components revealed three factors or potential emission sources for the 2011 air samples, explaining 89% of the variance in the dataset, with the major results and factor loadings summarized in Table 5.5. Factor 1 accounted for 73% of the variance, followed by factor 2 accounting for 10% and factor 3 accounting for 6% of the variance. The indicatory PAHs identified in factor 1 accounting for 73% of the total variance in the dataset were acenaphthylene, acenaphthene, fluorene, dibenzothiophene, phenanthrene, anthracene and pyrene. A previous study investigating PAH emissions from various industrial stacks in Taiwan had identified acenaphthylene, acenaphthene and anthracene as the indicatory PAHs of a cement plant [178]. Additionally, fluorene, phenanthrene, anthracene and pyrene are also considered predominant coal combustion tracers [114, 179]. Factor 2 accounts for 10% of the variance and is heavily weighted with benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene and benzo[g,h,i]perylene, all of which have been associated with vehicle emissions. Factor 3, which accounts for 6% had only one dominate PAH, dibenzo[a,h]anthracene. Dibenzo[a]anthracene has been previously associated with vehicle emissions, however by itself, it may not have a specific source meaning [114].

Although there is no known source of coal combustion on the island, the refinery not only produces asphalt (<1%) but it's utilities are mainly fueled by asphalt, more commonly referred to as

bitumen [180]. The differences between bitumen and coal can be characterized by not only there PAH concentrations but also there compositional signature. Greater proportions of alkylated phenanthrene homologues, as well as the more stable isomers, chrysene, benzo[e]pyrene or benzo[g,h,i]perylene are commonly found in petrogenic PAH sources such as crude oil, bitumen and asphalt. In contrast, pyrogenic PAHs, such as coal tar, tend to be dominated by phenanthrene, fluoranthene, pyrene and a greater proportion of the less stable isomers, benzo[a]anthracene, benzo[a]pyrene and indeno[1,2,3-cd]pyrene [115, 181]. Therefore, the refinery's asphalt fuel source may help to explain the factor loadings seen in the 2011 samples since they were dominated by indicatory PAH of petrogenic sources for crude oil, bitumen and asphalt (i.e., acenaphthylene, acenaphthene, anthracene, and pyrene). This is also supported by the 2011 BaA/BaA+CHR) ratios observed (0.21-0.59). Asphalt ratios have been reported to be between 0.16-0.39 whereas coal tar has ratios between 0.51-0.56.

The major results and factor loadings from the factor analysis and principle components analysis for the 2014 downwind samples are summarized in Tables 5.6. The principle components analysis was able to discriminate between the downwind and upwind samples, explaining 69% of the variability in the dataset (Figure 5.13). The 2014 downwind and upwind sites revealed different factor loadings suggesting the influence of different emission sources. The factor analysis for the 2014 locations downwind of the refinery revealed four major factors or potential emission sources, explaining 89% of the variance (Table 5.6). Factor 1 accounted for 44% of the contribution and was dominated by the low molecular weight compounds (fluorene, dibenzothiophene, phenanthrene and anthracene), suggesting petrogenic sources. Similar to the 2011 factor loadings, asphalt, cement, and coal indicator PAHs are also observed albeit to a lesser degree. Additional influences are seen in factors 2-4 with the signals from petrogenic (i.e., fluoranthene) as well as the high molecular weight PAHs associated with pyrogenic sources. Factor 3, accounting for 15%, is dominated by indeno[1,2,3-

cd]pyrene and dibenzo[a,h]anthracene. Factor 4, accounting for 12%, is dominated by benzo[a]pyrene and perylene. Indeno[1,2,3-cd]pyrene and dibenzo[a,h]anthracene are typically associated with vehicle emissions and benzo[a]pyrene has been associated with both vehicle emissions and oil combustion [114].

In contrast, the upwind sites sampled in 2014 revealed only 2 factors explaining 77% of the variance (Table 5.7). The two factors 1 and 2, accounted for 39% and 37%, respectively, suggesting a relatively homogeneous mixture of emission sources. Factor 1 was dominated by the low molecular weight compounds (fluorene, dibenzothiophene, phenanthrene) indicative of petrogenic sources as well as pyrogenic sources (i.e., indeno[1,2,3-cd]pyrene). Benzo[g,h,i]perylene was also a dominant indicator PAH in factor 1 and has been associated with both pyrogenic (e.g., vehicle emissions) as well as petrogenic sources (e.g., crude oil, bitumen, asphalt). Factor 2 is dominated by fluoranthene, pyrene, benzo[a]anthracene and chrysene. Fluoranthene, pyrene and chrysene are indicative of coal combustion and benzo[a]anthracene is indicative of oil combustion.

5.3 Risk

5.3.1 Toxic Equivalency Factors

In the 2011 ambient air samples collected in Curaçao, the mean potency equivalent concentrations ranged from 0.0001 ng-PEQ/m^3 for benzo[g,h,i]perylene and acenaphthylene to 0.1582 ng-PEQ/m^3 for dibenzo[a,h]anthracene (Table 5.8). Dibenzo[a,h]anthracene and phenanthrene contributed 55 and 14% respectively, to the total carcinogenicity of quantified PAHs in 2011. In the 2014 downwind samples, the mean B[a]P equivalents ranged from 0.0002 ng-PEQ/m^3 for acenaphthylene to 0.0469 ng-PEQ/m^3 for benzo[a]pyrene (Table 5.9). In the 2014 downwind samples benzo[a]pyrene and benzo[a]anthracene accounted for 29 and 14% respectively, to the total

carcinogenicity of quantified PAHs. In the 2014 upwind samples, the mean potency equivalents ranged from 0.0001 ng-PEQ/m^3 for benzo[g,h,i]perylene to 0.0255 ng-PEQ/m^3 for benzo[a]pyrene (Table 5.10). Benzo[a]pyrene and benzo[a]anthracene accounted for 33 and 21% respectively, to the carcinogenicity of quantified PAHs in the 2014 upwind samples.

5.3.2 Risk Probability Estimates

The 95% UCLs calculated by the USEPA's ProUCL software and the estimated risk probabilities for the PAHs ($\mu g/m^3$) sampled in 2011 and in 2014 are summarized in Table 5.11 and Figure 5.14. The estimated risk probabilities using the 95% UCL, produced using the USEPA ProUCL software, resulted in risk probability estimates ranged from 6.03E-09 to 1.93E-06, 1.25E-09 to 7.94E-07, and 2.54E-09 to 1.74E-07 for the 2011, 2014 downwind and 2014 upwind samples, respectively.. The total risk probability estimate for the 2014 downwind samples and the 2011 samples were 1.1E-06 and 2.7E-06, theoretically leading to one and approximately three excess cancer cases in 1,000,000 exposed individuals, respectively. All risk probability estimates using the 95% UCL for the carcinogenic PAHs were below 1.0E-06 and are considered negligible. However, when extrapolating for children and adults, these levels all exceeded 1.0E-06 yet were still less than the priority level warranting remediation (1.0E-04)(Figure 5.15).

The risk analysis for cancer risk probability estimates based on the 95% UCLs calculated from the ambient PAH concentrations ($\mu g/m^3$) in 2011 ranged from 6.03E-09 for chrysene to 1.93E-06 for naphthalene. The total risk probability estimate for the $\sum_{10}$PAHs (2.69E-06) were almost an order of magnitude higher than the risk from the carcinogenic PAHs ($\sum_3$PAHs) (5.95E-07). Naphthalene and dibenzo[a,h]anthracene contributed the highest risk in 2011. The cumulative lifetime risks using the 95% UCLs of the ambient PAH concentrations (mg/m^3) were then extrapolated to estimate risks for

children, adult residents and adult workers in 2011 (Figure 5.15). Extrapolated lifetime risk probability estimates based on the 95% UCLs for children adult residents and adult workers were 2.6E-05, 4.4E-05, 3.3E-05, respectively and were approaching the upper bound acceptable risk (1.0E-04). Fluorene, phenanthrene and fluoranthene all posed the highest risk, although they were still below the upper bound acceptable risk (1.0E-4) level for children, adult residents and workers (Figure 5.16).

The cancer risk probability estimates based on the 95% UCLs calculated from the 2014 downwind ambient PAHs ($\mu g/m^3$) ranged from 1.25E-09 for benzo[g,h,i]perylene to 7.94E-07 for naphthalene, with a total risk of 1.07E-06 for the $\sum_{14}$PAHs (Table 5.11 and Figure 5.14). The risk from the carcinogenic PAHs ($\sum_{6}$PAHs; 1.97E-07) at the downwind sites, however, was below the upper bound risk (1.0E-04). The total cancer risk probability estimates for the $\sum_{16}$PAHs (3.69E-07) and the carcinogenic PAHs ($\sum_{6}$PAHs; 2.32E-07) from the 2014 upwind ambient PAHs were both below the USEPAs upper bound acceptable risk (1.0E-06). The cumulative lifetime probability estimates extrapolated for children, adult residents and adult workers based on the 2014 downwind and upwind 95% UCLs were all above the USEPA's acceptable risk (Figure 5.15). The risk probability estimates for children and adult residents were almost three times higher downwind of the refinery than for those upwind. The highest risk probability estimates were associated with fluorene, phenanthrene and fluoranthene in the 2014 sites, exceeding the upper bound risk level recommended by the USEPA (Figure 5.16).

Site specific risk estimates were also calculated by multiplying the potency equivalent concentrations ($\mu g\text{-}PEQ/m^3$) by the IUR ($\mu g/m^3$). The cancer probability estimates for all sites based on the 2011 B[a]P equivalent concentrations were all below the USEPA's acceptable risk level, although the total risk for the 2011 mean (2.03E-07), Habaai (5.92E-07), Rooi Catochi (4.25E-07) and Heintje Kool (3.12E-07) are approaching the upper bound acceptable risk (Figure 5.17). Naphthalene,

benzo[a]anthracene, benzo[a]pyrene, dibenzo[a,h]anthracene were associated with the highest risk probability estimates (Figure 5.18). The cumulative lifetime extrapolated risks were for the 2011 potency equivalent concentrations (mg-PEQ/m^3) were at least two orders of magnitude below the upper bound risk level (1.0E-04) for children, adult residents and adult workers with phenanthrene and dibenzo[a,h]anthracene being associated with the highest risk (Figures 5.19-5.20).

The cancer probability estimates for all sites based on the 2014 downwind and upwind potency equivalent concentrations were all at least two orders of magnitude below the USEPA's upper bound acceptable risk level, however the overall total risk estimate for the downwind sites (mean: 6.84E-08) were twice as high as the upwind sites (mean: 3.19E-08) (Figure 5.21). Benzo[a]pyrene was associated with the highest risk in the 2014 samples (Figure 5.22). The cumulative lifetime extrapolated risks using the potency equivalent concentrations were all at least three orders of magnitude below the upper bound acceptable level (1.0E-04) for children, adult residents and adult workers located upwind from the refinery in 2014 with benzo[a]pyrene being associated with the highest risk at most sites (Figure 5.23-5.25). In contrast, the total cumulative lifetime extrapolated risk probabilities for children, adult residents and adult workers for the 2014 mean for the downwind sites, Habaai and Wishi were more than two orders of magnitude higher than the upper bound acceptable risk of 1.0E-04, with up to 11 PAHs exceeding this level (Figure 5.23 and 5.24).

Adjusting the 2011 and the 2014 upwind PAH concentrations by the potency equivalent factors revealed site specific and cumulative lifetime risk probability estimates below 1.0E-06 and are considered negligible. However, after adjusting the 2014 downwind PAH concentrations by the potency equivalent factors the site specific and cumulative lifetime risk probability estimates for the overall 2014 downwind mean concentrations, Habaai and Wishi all exceeded levels considered sufficiently large to warrant remediation (1.0E-4) by up to two orders of magnitude. The cumulative lifetime risk probability estimates based on the potency equivalents for the 2014 downwind mean

(2.07E-3 – 3.55E-3), Habaai (9.34E-3 – 1.6E-2) and Wishi (2.17E-2 – 3.72E-2) were up to three orders of magnitude higher than those in Taiyuan, China (9.8E-7 – 1.03E-5) [182] and up to five orders of magnitude than those in Shenzhen, China (1.96E-7 – 1.33E-6) [183]. Calculating risks without using the 95% upper confidence limits presents uncertainties associated with estimating the true average concentrations, therefore it is imperative to be cognizant these concentrations may represent potential worse case scenarios and may not be representative of the true average.

Hence, risk probability estimates were also calculated for the 95% UCLs for the potency equivalent concentrations. Assuming additivity, the cumulative lifetime cancer risk estimates using the 95% UCLs for the potency equivalent concentrations were up to three orders of magnitude lower than the upper bound acceptable risk level (1.0E-4) and were 3.21E-07, 1.28E-07 and 2.25E-07 for the 2011, 2014 downwind and 2014 upwind sites, respectively (Figure 5.26). Dibenzo[a,h]anthracene accounted for the highest risk (2.59E-07) in 2011, whereas benzo[a]pyrene accounted for the highest risk in the 2014 downwind (1.21E-07) and upwind (2.19E-07) sites.

It is important to note that if all PAHs are considered carcinogenic and as carcinogenic as B[a]P, the theoretical lifetime cancer risk from inhalation may both be overestimated or underestimated depending on the adequacy of the cancer bioassays for B[a]P, whether PAHs may be synergistic, additive or antagonistic, if not all carcinogenic PAHs are included, if noncarcinogenic PAHs potentiate the activity of carcinogenic PAHs and by potentially forcing a linear term at low doses in the linearized multistage model [111]. Moreover, B[a]P is generally relatively low in ambient air leading to its efficacy as an indicator PAH for ambient air.

5.3.3 Proximity to Petrochemical Complexes and Potential Risks

Epidemiological studies have suggested that refinery workers are subjected to increased risks for the development of kidney, stomach, brain, pancreatic, prostate, hematopoietic and lymphatic cancers, leukemia, and other lung and skin diseases than the general population [184-188]. Various mortalities and morbidities have also been associated with residential proximity to refineries, including asthma, wheezing, altered blood profiles, compromised lung function, lung cancer and allergic response. Rusconi et al. [189] detected an increase in wheezing and markers of inflammation and oxidative stress and decreased lung function in children living in close proximity to a high complexity refinery in Sardinia, Italy. Significant associations were also found between wheezing and exposed communities living next to petrochemical plants in Brazil [190]. Children and adolescents near a petrochemical site in Spain with an annual mean B[a]P concentration of 0.11 ng/m^3, also had higher prevalence of respiratory hospitalizations and nocturnal cough, yet, reduced lung function and higher prevalence of asthma were not observed [191]. In contrast, Smargiassi et al. [66] previously reported a small decrease in pulmonary functions in asthmatic children living near an industrial complex and refineries with personal PAH levels (151 ±99 μg/m^3). However, this was a panel study evaluating 72 children over 10 consecutive days, which resulted in difficulty in detecting effects. Interestingly, out of 3,230 children (ages 0-14) in Curaçao, 60% had asthma or chronic bronchitis, which is four times the global average for children (14%) (Table 5.12)[192, 193].

Increased white blood cells, platelet counts, creatinine and liver enzymes were observed in exposed residents surrounding a British Petroleum plant following a flaring incident [194]. A recent study in Saudi Arabia reported proximities to an oil refinery were associated with prehypertension in boys (mean age of 12 years) and increases of PAH (36.8 ng/m^3) and total suspended particulate matter (TSP, 444 μg/m^3) exposures [173]. In people aged 0-64, lung cancer and respiratory disease

demonstrated gradients with the highest mortality in areas closest to petrochemical and steel complexes in northeast England [67]. However, another study in Sweden found no evidence of increased cancer risks with proximity to petrochemical industries [195]. A case-control study in Italy found statistically non-significant moderate increases in risk for lung, bladder and lymphohematopoietic neoplasms in populations residing within 2km from the center of a petrochemical plant [196].

Although the data presented herein is generally inconsistent in terms of proximity to petrochemical complexes and health effects, qualitative evidence through epidemiological studies have demonstrated increased mortality in humans due to lung cancers as a result of exposures to coke-oven emissions, roofing-tar emissions and cigarette smoke, all of which contain benzo[a]pyrene, chrysene, benzo[a]anthracene, benzo[b]fluorene and dibenzo[a,h]anthracene as well as other carcinogenic PAHs [188]. The lungs are a major target organ of PAH carcinogenicity and therefore heavy exposures entails a substantial increased risk of lung cancer as well as, skin and bladder cancers [53]. Yet is nearly impossible for these studies to determine the risk posed by each individual PAH or the interaction between PAHs and particles in the induction of lung cancer [53]. Hence, PAH mixtures as a whole are considered carcinogenic with B[a]P serving as a surrogate indicator of toxicity, with fluoranthene as a secondary indicator compound considering its relatively high levels in ambient air. Therefore, representative concentrations for PAH mixtures of 0.1 ng/m^3 of B[a]P or 2 ng/m^3 fluoranthene would theoretically lead to an estimated upper-bound lifetime cancer risk of 1.0E-05.

Table 5.1 The 2011 site specific polyurethane foam disk concentrations (ng/disk) and calculated ambient PAHs (ng/m^3) in Curaçao.

Sample ID	Habaai		W. Piscadera Baai		Nieuw Nederland	
Air Volume/Conc	(ng/disk)	(ng/m^3)	(ng/disk)	(ng/m^3)	(ng/disk)	(ng/m^3)
Naphthalene	229.30	67.31	163.30	47.94	104.90	30.79
C1 Naphthalenes	212.30	25.55	109.60	13.19	99.90	12.02
C2 Naphthalenes	611.30	36.63	170.70	10.22	154.90	9.28
C3 Naphthalenes	1165.40	38.79	260.70	8.64	145.20	4.84
C4 Naphthalenes	1745.60	33.22	279.20	5.22	129.20	2.47
Acenaphthylene	30.80	0.72	<IDL	<IDL	<IDL	<IDL
Acenaphthene	61.10	2.96	31.30	1.51	8.40	0.41
Fluorene	289.20	11.94	116.30	4.80	87.80	3.63
C1 Fluorenes	1268.90	22.28	317.00	5.54	215.40	3.79
C2 Fluorenes	3051.60	44.00	420.50	6.01	391.70	5.66
C3 Fluorenes	4002.30	44.17	411.90	4.48	409.00	4.54
C4 Fluorenes	2799.90	14.94	<IDL	<IDL	259.40	1.40
Dibenzothiophene	645.30	6.80	91.20	0.95	46.70	0.49
C1 DBT	1066.40	7.44	86.70	0.59	54.50	0.38
C2 DBT	5138.90	26.76	301.10	1.52	118.00	0.62
C3 DBT	3789.10	16.92	141.80	0.61	54.60	0.25
C4 DBT	1677.30	6.94	22.10	0.09	14.20	0.06
Phenanthrene	9637.00	155.42	1786.00	28.22	1992.90	32.37
Anthracene	260.60	4.62	44.40	0.77	52.40	0.94
C1 Phen_Anthr	5892.00	82.27	697.80	9.43	559.70	7.90
C2 Phen_Anthr	4923.80	58.97	384.40	4.43	317.40	3.85
C3 Phen_Anthr	3744.60	42.00	248.50	2.67	176.20	2.01
C4 Phen_Anthr	1027.10	11.16	43.00	0.45	<IDL	<IDL
Fluoranthene	1130.90	11.44	123.80	1.20	244.50	2.51
Pyrene	837.10	5.85	85.90	0.58	149.80	1.06
C1 Flu/Pyrene	717.70	4.68	56.90	0.36	59.50	0.39
C2 Flu/Pyrene	296.70	1.87	10.00	0.06	<IDL	<IDL
C3 Flu/Pyrene	130.40	0.81	<IDL	<IDL	<IDL	<IDL
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	129.00	1.03	<IDL	<IDL	8.40	0.07
Chrysene	178.10	1.07	<IDL	<IDL	19.50	0.12
C1 B(a)A/Chrys/Triph	137.40	0.81	<IDL	<IDL	<IDL	<IDL
C2 B(a)A/Chrys/Triph	84.70	0.49	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	52.00	0.35	<IDL	<IDL	<IDL	<IDL
Benzo(k)fluoranthene	33.60	0.12	<IDL	<IDL	<IDL	<IDL
Benzo(a)pyrene	20.70	0.13	<IDL	<IDL	<IDL	<IDL
Perylene	12.30	0.05	<IDL	<IDL	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	19.30	0.10	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	15.20	0.07	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	42.10	0.18	<IDL	<IDL	<IDL	<IDL
∑PAHs	57107.00	790.88	6404.10	159.49	5874.10	131.81

Table 5.1 (Continued)

Sample ID	Groot Piscadera		Boca Sami		Rooi Catochi	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	94.04	27.61	93.87	27.56	146.24	42.93
C1 Naphthalenes	51.49	6.20	68.25	8.22	29.33	3.53
C2 Naphthalenes	80.70	4.84	118.17	7.08	9.47	0.57
C3 Naphthalenes	125.47	4.18	189.32	6.30	52.30	1.75
C4 Naphthalenes	133.82	2.55	291.18	5.54	60.30	1.17
Acenaphthylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Acenaphthene	18.66	0.90	<IDL	<IDL	8.41	0.41
Fluorene	90.68	3.74	115.29	4.76	95.89	3.96
C1 Fluorenes	274.74	4.82	431.09	7.57	174.31	3.08
C2 Fluorenes	598.43	8.63	722.97	10.42	289.41	4.21
C3 Fluorenes	583.57	6.44	881.45	9.73	232.62	2.61
C4 Fluorenes	304.22	1.62	357.22	1.91	139.97	0.78
Dibenzothiophene	47.00	0.49	77.65	0.82	52.78	0.56
C1 DBT	21.80	0.15	110.18	0.77	<IDL	<IDL
C2 DBT	98.01	0.51	277.86	1.45	<IDL	<IDL
C3 DBT	24.48	0.11	158.76	0.71	<IDL	<IDL
C4 DBT	<IDL	<IDL	79.45	0.33	<IDL	<IDL
Phenanthrene	2285.88	36.86	2334.15	37.64	2534.52	41.79
Anthracene	59.31	1.05	62.04	1.10	42.76	0.78
C1 Phen_Anthr	890.17	12.43	1000.91	13.98	399.93	5.78
C2 Phen_Anthr	460.32	5.51	595.91	7.14	200.05	2.50
C3 Phen_Anthr	205.43	2.30	370.43	4.15	75.54	0.89
C4 Phen_Anthr	61.86	0.67	147.02	1.60	<IDL	<IDL
Fluoranthene	270.91	2.74	215.40	2.18	252.32	2.67
Pyrene	192.71	1.35	150.35	1.05	136.50	0.99
C1 Flu/Pyrene	130.27	0.85	99.94	0.65	39.60	0.27
C2 Flu/Pyrene	101.88	0.64	54.77	0.35	<IDL	<IDL
C3 Flu/Pyrene	<IDL	<IDL	11.83	0.07	<IDL	<IDL
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	69.49	0.56	44.48	0.36	9.79	0.08
Chrysene	72.21	0.43	40.46	0.24	<IDL	<IDL
C1 B(a)A/Chrys/Triph	36.49	0.21	9.01	0.05	<IDL	<IDL
C2 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(k)fluoranthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Perylene	<IDL	<IDL	11.06	0.04	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	71.64	0.35
Benzo(g,h,i)perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAHs	7384.05	138.41	9120.47	163.76	5053.68	121.65

Table 5.1 (Continued)

Sample ID	Marie Pampoen		E. Buena Vista (#8)		W. Buena Vista (#9)	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	74.38	21.84	44.01	12.92	479.48	140.76
C1 Naphthalenes	<IDL	<IDL	17.46	2.10	37.76	4.54
C2 Naphthalenes	<IDL	<IDL	29.60	1.77	67.10	4.02
C3 Naphthalenes	<IDL	<IDL	70.79	2.36	143.71	4.78
C4 Naphthalenes	<IDL	<IDL	109.47	2.08	231.92	4.41
Acenaphthylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Fluorene	39.51	1.63	63.70	2.63	92.92	3.84
C1 Fluorenes	42.37	0.74	228.14	4.01	354.60	6.23
C2 Fluorenes	174.20	2.50	403.49	5.82	684.63	9.87
C3 Fluorenes	223.93	2.46	565.42	6.24	873.42	9.64
C4 Fluorenes	124.53	0.66	256.34	1.37	459.49	2.45
Dibenzothiophene	26.96	0.28	34.72	0.37	76.01	0.80
C1 DBT	37.92	0.26	33.40	0.23	61.57	0.43
C2 DBT	116.70	0.60	126.68	0.66	218.49	1.14
C3 DBT	52.86	0.23	60.83	0.27	137.39	0.61
C4 DBT	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Phenanthrene	727.79	11.66	1389.61	22.41	2558.44	41.26
Anthracene	<IDL	<IDL	54.77	0.97	73.77	1.31
C1 Phen_Anthr	328.07	4.53	663.99	9.27	1142.89	15.96
C2 Phen_Anthr	225.75	2.67	552.81	6.62	1031.33	12.35
C3 Phen_Anthr	163.17	1.80	239.74	2.69	573.02	6.43
C4 Phen_Anthr	44.65	0.48	44.24	0.48	230.96	2.51
Fluoranthene	165.39	1.65	265.51	2.69	323.24	3.27
Pyrene	70.90	0.49	195.36	1.37	263.75	1.84
C1 Flu/Pyrene	25.65	0.17	68.64	0.45	122.83	0.80
C2 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	51.76	0.33
C3 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	<IDL	<IDL	<IDL	<IDL	30.05	0.24
Chrysene	<IDL	<IDL	19.46	0.12	51.19	0.31
C1 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C2 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(k)fluoranthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAHs	2664.75	54.65	5538.20	89.89	10371.72	280.13

Table 5.1 (Continued)

Sample ID	Heintje Kool		Roosendaal		Marchena / Wishi	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	73.38	21.54	13.30	3.90	8.57	2.52
C1 Naphthalenes	94.18	11.34	24.04	2.89	140.71	16.94
C2 Naphthalenes	150.16	9.00	41.80	2.51	373.94	22.41
C3 Naphthalenes	228.11	7.61	82.66	2.77	521.77	17.40
C4 Naphthalenes	258.78	4.96	90.45	1.75	460.70	8.82
Acenaphthylene	<IDL	<IDL	<IDL	<IDL	38.23	0.90
Acenaphthene	<IDL	<IDL	<IDL	<IDL	51.23	2.48
Fluorene	89.34	3.69	79.24	3.27	285.95	11.81
C1 Fluorenes	318.01	5.59	231.80	4.09	1076.06	18.93
C2 Fluorenes	634.30	9.17	312.55	4.55	2065.03	29.86
C3 Fluorenes	949.37	10.53	484.01	5.43	3008.74	33.38
C4 Fluorenes	403.71	2.18	<IDL	<IDL	1003.27	5.43
Dibenzothiophene	92.69	0.98	53.84	0.57	466.89	4.93
C1 DBT	95.16	0.67	40.24	0.29	688.38	4.84
C2 DBT	383.34	2.02	126.66	0.68	2555.83	13.46
C3 DBT	258.17	1.17	<IDL	<IDL	1593.62	7.21
C4 DBT	98.72	0.41	<IDL	<IDL	607.42	2.55
Phenanthrene	2326.62	37.79	1643.23	27.09	6991.75	113.57
Anthracene	72.12	1.29	47.81	0.87	241.99	4.33
C1 Phen_Anthr	1110.68	15.69	664.93	9.61	3603.52	50.89
C2 Phen_Anthr	1094.82	13.29	528.45	6.60	3477.01	42.21
C3 Phen_Anthr	612.65	6.97	243.96	2.86	1663.26	18.93
C4 Phen_Anthr	275.04	3.03	18.23	0.21	632.10	6.97
Fluoranthene	360.60	3.70	189.94	2.01	576.32	5.91
Pyrene	287.85	2.04	140.12	1.02	519.37	3.68
C1 Flu/Pyrene	143.78	0.95	55.31	0.38	<IDL	<IDL
C2 Flu/Pyrene	68.58	0.44	<IDL	<IDL	162.14	1.04
C3 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	29.57	0.24	<IDL	<IDL	53.92	0.44
Chrysene	71.36	0.43	9.04	0.06	107.86	0.66
C1 B(a)A/Chrys/Triph	10.01	0.06	<IDL	<IDL	64.63	0.38
C2 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	8.38	0.05
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	10.56	0.07	<IDL	<IDL	<IDL	<IDL
Benzo(k)fluoranthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	10.79	0.05	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAHs	10612.47	176.92	5121.62	83.41	33048.58	452.93

Table 5.1 (Continued)

Sample ID	E. Piscadera Baai		Blauw / Curasol		Parasasa	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C1 Naphthalenes	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C2 Naphthalenes	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C3 Naphthalenes	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 Naphthalenes	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Acenaphthylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Fluorene	105.92	4.37	<IDL	<IDL	<IDL	<IDL
C1 Fluorenes	358.97	6.33	<IDL	<IDL	43.40	0.77
C2 Fluorenes	462.28	6.71	<IDL	<IDL	71.84	1.05
C3 Fluorenes	308.07	3.44	<IDL	<IDL	76.00	0.85
C4 Fluorenes	345.59	1.90	<IDL	<IDL	42.37	0.24
Dibenzothiophene	122.94	1.30	<IDL	<IDL	<IDL	<IDL
C1 DBT	126.94	0.90	<IDL	<IDL	<IDL	<IDL
C2 DBT	440.79	2.35	<IDL	<IDL	52.20	0.28
C3 DBT	233.91	1.07	<IDL	<IDL	30.45	0.14
C4 DBT	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Phenanthrene	2029.71	33.21	<IDL	<IDL	<IDL	<IDL
Anthracene	59.39	1.07	<IDL	<IDL	<IDL	<IDL
C1 Phen_Anthr	966.24	13.81	48.26	0.69	125.01	1.81
C2 Phen_Anthr	842.21	10.37	40.49	0.50	131.45	1.64
C3 Phen_Anthr	443.74	5.13	<IDL	<IDL	61.69	0.72
C4 Phen_Anthr	160.54	1.80	<IDL	<IDL	27.14	0.31
Fluoranthene	220.88	2.30	<IDL	<IDL	32.78	0.35
Pyrene	174.54	1.25	<IDL	<IDL	<IDL	<IDL
C1 Flu/Pyrene	94.23	0.63	<IDL	<IDL	<IDL	<IDL
C2 Flu/Pyrene	40.65	0.26	<IDL	<IDL	<IDL	<IDL
C3 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	9.20	0.08	<IDL	<IDL	<IDL	<IDL
Chrysene	46.03	0.28	<IDL	<IDL	<IDL	<IDL
C1 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C2 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(k)fluoranthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAHs	7592.76	98.56	88.74	1.19	694.35	8.15

Table 5.2 The 2014 site specific polyurethane foam disk concentrations (ng/disk) and calculated ambient PAHs (ng/m^3) in Curaçao.

Sample ID	Habaai		Parasasa (#25)		Nieuw Nederland	
Air Volume/Conc	(ng/disk)	(ng/m^3)	(ng/disk)	(ng/m^3)	(ng/disk)	(ng/m^3)
Naphthalene	6.39	1.98	10.42	3.24	2.69	0.84
C1 Naphthalenes	75.65	9.74	57.98	7.46	27.98	3.60
C2 Naphthalenes	217.18	14.03	86.39	5.58	36.63	2.37
C3 Naphthalenes	438.74	15.76	95.23	3.42	<IDL	<IDL
C4 Naphthalenes	375.91	7.62	223.12	4.53	23.74	0.48
Acenaphthylene	<IDL	<IDL	7.35	0.19	<IDL	<IDL
Acenaphthene	<IDL	<IDL	12.65	0.66	<IDL	<IDL
Fluorene	137.75	6.16	51.73	2.31	39.88	1.78
C1 Fluorenes	587.05	11.20	290.71	5.55	124.17	2.36
C2 Fluorenes	2196.86	34.32	815.91	12.75	106.65	1.66
C3 Fluorenes	3485.46	41.35	1387.04	16.45	<IDL	<IDL
C4 Fluorenes	2317.15	12.68	1059.44	5.80	<IDL	<IDL
Dibenzothiophene	287.42	3.28	84.18	0.96	20.04	0.23
C1 DBT	615.96	4.58	358.31	2.67	<IDL	<IDL
C2 DBT	3412.54	18.59	942.73	5.14	<IDL	<IDL
C3 DBT	2956.59	13.63	694.75	3.20	<IDL	<IDL
C4 DBT	1429.85	6.06	557.46	2.36	<IDL	<IDL
Phenanthrene	1295.38	22.17	468.99	8.03	266.00	4.49
Anthracene	127.35	2.38	42.43	0.79	<IDL	<IDL
C1 Phen_Anthr	4387.71	63.75	1425.61	20.71	220.27	3.13
C2 Phen_Anthr	5884.58	72.50	1851.93	22.82	201.40	2.42
C3 Phen_Anthr	4110.94	47.16	1211.66	13.90	99.10	1.10
C4 Phen_Anthr	1153.40	12.77	446.76	4.95	<IDL	<IDL
Fluoranthene	485.99	5.04	301.73	3.13	139.92	1.41
Pyrene	537.45	3.87	338.89	2.44	111.16	0.78
C1 Flu/Pyrene	574.89	3.84	190.41	1.27	54.82	0.36
C2 Flu/Pyrene	427.31	2.75	217.43	1.40	38.58	0.24
C3 Flu/Pyrene	152.96	0.97	65.85	0.42	<IDL	<IDL
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	50.27	0.41	32.71	0.27	20.83	0.16
Chrysene	112.72	0.69	79.29	0.49	27.76	0.17
C1 B(a)A/Chrys/Triph	104.92	0.63	77.67	0.46	<IDL	<IDL
C2 B(a)A/Chrys/Triph	64.26	0.38	46.21	0.27	<IDL	<IDL
C3 B(a)A/Chrys/Triph	20.17	0.12	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	39.01	0.27	38.41	0.26	22.42	0.15
Benzo(k)fluoranthene	12.39	0.04	11.82	0.04	19.15	0.07
Benzo(a)pyrene	6.60	0.04	4.95	0.03	9.99	0.06
Perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	<IDL	<IDL	16.58	0.07	<IDL	<IDL
∑PAHs	38088.80	440.78	13604.73	164.02	1613.18	27.86

Table 5.2 (Continued)

Sample ID	Buena Vista (#21)		Biesheuvel		Buena Vista (#17)	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	129.69	40.28	9.02	2.80	146.02	45.35
C1 Naphthalenes	297.46	38.29	34.83	4.48	410.40	52.83
C2 Naphthalenes	409.51	26.46	45.46	2.94	615.76	39.78
C3 Naphthalenes	334.37	12.05	69.38	2.50	527.67	19.01
C4 Naphthalenes	238.86	4.90	29.09	0.60	285.99	5.87
Acenaphthylene	<IDL	<IDL	<IDL	<IDL	15.75	0.40
Acenaphthene	<IDL	<IDL	<IDL	<IDL	27.91	1.46
Fluorene	86.89	3.89	62.85	2.81	88.11	3.94
C1 Fluorenes	312.97	5.99	114.92	2.20	<IDL	<IDL
C2 Fluorenes	425.78	6.69	201.05	3.16	452.97	7.11
C3 Fluorenes	501.00	6.00	292.41	3.50	732.33	8.77
C4 Fluorenes	<IDL	<IDL	99.09	0.56	390.58	2.20
Dibenzothiophene	39.31	0.45	18.66	0.21	96.27	1.11
C1 DBT	145.00	1.09	11.77	0.09	329.35	2.49
C2 DBT	154.85	0.86	<IDL	<IDL	578.63	3.22
C3 DBT	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 DBT	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Phenanthrene	335.33	5.82	278.00	4.82	405.43	7.04
Anthracene	50.14	0.95	38.69	0.74	40.44	0.77
C1 Phen_Anthr	574.75	8.54	326.92	4.86	986.44	14.66
C2 Phen_Anthr	513.86	6.51	279.24	3.54	1108.55	14.04
C3 Phen_Anthr	282.15	3.33	117.80	1.39	798.05	9.43
C4 Phen_Anthr	76.40	0.87	<IDL	<IDL	387.79	4.43
Fluoranthene	168.31	1.80	167.00	1.78	334.62	3.57
Pyrene	172.81	1.28	153.74	1.14	470.11	3.48
C1 Flu/Pyrene	99.95	0.69	89.20	0.61	95.37	0.66
C2 Flu/Pyrene	52.67	0.35	48.16	0.32	59.78	0.40
C3 Flu/Pyrene	30.21	0.20	<IDL	<IDL	27.67	0.18
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	12.09	0.10	12.97	0.11	21.91	0.18
Chrysene	22.02	0.14	16.31	0.10	49.43	0.31
C1 B(a)A/Chrys/Triph	9.05	0.06	20.26	0.12	31.41	0.19
C2 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	14.84	0.11	12.81	0.09	24.98	0.18
Benzo(k)fluoranthene	5.97	0.02	5.77	0.02	8.56	0.03
Benzo(a)pyrene	11.93	0.08	5.90	0.04	3.83	0.03
Perylene	12.41	0.05	1.82	0.01	5.16	0.02
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	1.00	0.01	9.82	0.05
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	7.49	0.04
Benzo(g,h,i)perylene	9.80	0.04	5.46	0.02	11.13	0.05
∑PAHs	5530.38	177.88	2569.58	45.58	9585.71	253.27

Table 5.2 (Continued)

Sample ID	Brievengat/Groot Kwartier		Wanapa		Domi/Welgelegen 2014	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	6.35	1.97	<IDL	<IDL	53.86	16.73
C1 Naphthalenes	45.33	5.84	17.91	2.31	73.14	9.41
C2 Naphthalenes	38.56	2.49	51.30	3.31	130.62	8.44
C3 Naphthalenes	28.61	1.03	49.66	1.79	166.63	6.00
C4 Naphthalenes	<IDL	<IDL	31.44	0.65	143.14	2.94
Acenaphthylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Fluorene	53.71	2.40	40.30	1.80	70.32	3.15
C1 Fluorenes	105.36	2.01	83.09	1.59	231.82	4.44
C2 Fluorenes	213.84	3.34	198.04	3.11	828.35	13.01
C3 Fluorenes	434.04	5.15	<IDL	<IDL	1159.68	13.89
C4 Fluorenes	<IDL	<IDL	<IDL	<IDL	782.07	4.40
Dibenzothiophene	16.72	0.19	13.12	0.15	83.56	0.96
C1 DBT	<IDL	<IDL	18.78	0.14	445.19	3.36
C2 DBT	<IDL	<IDL	<IDL	<IDL	910.11	5.07
C3 DBT	<IDL	<IDL	<IDL	<IDL	561.40	2.66
C4 DBT	<IDL	<IDL	<IDL	<IDL	316.10	1.38
Phenanthrene	176.78	3.03	137.65	2.39	452.79	7.86
Anthracene	<IDL	<IDL	30.15	0.57	51.35	0.98
C1 Phen_Anthr	223.55	3.25	275.78	4.10	1156.33	17.19
C2 Phen_Anthr	221.13	2.72	283.11	3.59	1545.15	19.57
C3 Phen_Anthr	131.47	1.51	197.63	2.34	1179.26	13.93
C4 Phen_Anthr	<IDL	<IDL	49.87	0.57	403.72	4.61
Fluoranthene	274.24	2.84	118.84	1.27	204.90	2.19
Pyrene	215.28	1.55	104.70	0.77	199.64	1.48
C1 Flu/Pyrene	160.62	1.07	63.40	0.44	135.32	0.93
C2 Flu/Pyrene	178.14	1.15	35.42	0.24	117.23	0.78
C3 Flu/Pyrene	87.18	0.55	<IDL	<IDL	35.72	0.23
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	17.02	0.14	9.07	0.08	21.07	0.18
Chrysene	48.16	0.30	14.70	0.09	48.24	0.30
C1 B(a)A/Chrys/Triph	31.83	0.19	<IDL	<IDL	19.49	0.12
C2 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	<IDL	<IDL	8.38	0.06	13.14	0.09
Benzo(k)fluoranthene	<IDL	<IDL	5.23	0.02	8.00	0.03
Benzo(a)pyrene	<IDL	<IDL	2.08	0.01	9.74	0.07
Perylene	<IDL	<IDL	7.86	0.03	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAHs	2707.92	42.72	1847.51	31.41	11557.08	166.36

Table 5.2 (Continued)

Sample ID	Emmastad (#4)		Emmastad (#14)		Punda	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	4.52	1.40	1.68	0.52	14.97	4.65
C1 Naphthalenes	28.78	3.70	27.95	3.60	59.41	7.65
C2 Naphthalenes	33.37	2.16	34.73	2.24	64.38	4.16
C3 Naphthalenes	40.44	1.46	48.30	1.74	101.51	3.66
C4 Naphthalenes	50.23	1.03	39.51	0.81	83.50	1.71
Acenaphthylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Fluorene	46.63	2.09	43.16	1.93	115.46	5.16
C1 Fluorenes	102.70	1.97	90.32	1.73	149.83	2.87
C2 Fluorenes	205.73	3.23	134.91	2.12	233.48	3.67
C3 Fluorenes	295.47	3.54	247.94	2.97	333.51	3.99
C4 Fluorenes	<IDL	<IDL	165.83	0.93	155.15	0.87
Dibenzothiophene	11.18	0.13	12.37	0.14	58.57	0.67
C1 DBT	14.50	0.11	<IDL	<IDL	82.57	0.62
C2 DBT	56.82	0.32	<IDL	<IDL	145.02	0.81
C3 DBT	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 DBT	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Phenanthrene	229.31	3.98	131.79	2.29	542.18	9.41
Anthracene	26.22	0.50	17.86	0.34	23.52	0.45
C1 Phen_Anthr	362.54	5.39	251.61	3.74	402.88	5.99
C2 Phen_Anthr	328.14	4.16	227.03	2.87	176.28	2.23
C3 Phen_Anthr	211.61	2.50	131.30	1.55	116.86	1.38
C4 Phen_Anthr	<IDL	<IDL	49.06	0.56	<IDL	<IDL
Fluoranthene	569.57	6.08	191.18	2.04	395.15	4.22
Pyrene	604.74	4.48	180.76	1.34	306.90	2.27
C1 Flu/Pyrene	238.79	1.64	93.86	0.65	130.88	0.90
C2 Flu/Pyrene	130.55	0.87	69.93	0.46	135.56	0.90
C3 Flu/Pyrene	71.24	0.47	46.78	0.31	<IDL	<IDL
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	49.84	0.42	17.96	0.15	27.38	0.23
Chrysene	81.17	0.51	29.09	0.18	72.19	0.46
C1 B(a)A/Chrys/Triph	59.39	0.37	<IDL	<IDL	43.83	0.27
C2 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	55.73	0.40	14.72	0.10	45.45	0.32
Benzo(k)fluoranthene	19.01	0.07	6.93	0.03	25.88	0.09
Benzo(a)pyrene	34.04	0.23	<IDL	<IDL	<IDL	<IDL
Perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	19.80	0.10
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	<IDL	<IDL	<IDL	<IDL	21.38	0.09
∑PAHs	3962.26	53.17	2306.56	35.35	4083.48	69.82

Table 5.2 (Continued)

Sample ID	Gasparitu		Toni Kunchi		Suffisant (#28)	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	33.50	10.40	9.90	3.07	6.17	1.92
C1 Naphthalenes	114.69	14.76	48.09	6.19	41.54	5.35
C2 Naphthalenes	185.71	11.99	39.97	2.58	41.68	2.69
C3 Naphthalenes	203.96	7.33	38.47	1.38	47.62	1.71
C4 Naphthalenes	114.65	2.33	<IDL	<IDL	<IDL	<IDL
Acenaphthylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Fluorene	80.83	3.62	83.52	3.74	61.38	2.75
C1 Fluorenes	240.05	4.58	66.51	1.27	131.69	2.51
C2 Fluorenes	238.62	3.73	199.70	3.12	196.29	3.07
C3 Fluorenes	304.86	3.62	304.12	3.61	385.57	4.57
C4 Fluorenes	157.38	0.86	<IDL	<IDL	<IDL	<IDL
Dibenzothiophene	71.27	0.81	21.62	0.25	17.29	0.20
C1 DBT	74.73	0.56	23.14	0.17	<IDL	<IDL
C2 DBT	263.79	1.44	69.59	0.38	<IDL	<IDL
C3 DBT	120.64	0.56	<IDL	<IDL	<IDL	<IDL
C4 DBT	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Phenanthrene	341.72	5.85	272.77	4.67	228.85	3.92
Anthracene	33.29	0.62	20.73	0.39	29.32	0.55
C1 Phen_Anthr	543.37	7.89	249.80	3.63	370.96	5.39
C2 Phen_Anthr	463.41	5.71	210.81	2.60	318.85	3.93
C3 Phen_Anthr	179.38	2.06	91.37	1.05	271.64	3.12
C4 Phen_Anthr	<IDL	<IDL	42.74	0.47	78.20	0.87
Fluoranthene	187.86	1.95	132.20	1.37	122.68	1.27
Pyrene	184.24	1.33	76.78	0.55	107.90	0.78
C1 Flu/Pyrene	77.63	0.52	51.39	0.34	69.28	0.46
C2 Flu/Pyrene	41.99	0.27	35.91	0.23	61.98	0.40
C3 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	38.77	0.25
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	17.17	0.14	7.79	0.06	12.24	0.10
Chrysene	31.56	0.19	16.79	0.10	22.16	0.14
C1 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C2 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	18.66	0.13	6.38	0.04	9.87	0.07
Benzo(k)fluoranthene	10.43	0.04	5.24	0.02	5.41	0.02
Benzo(a)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAHs	4335.39	93.28	2125.33	41.29	2677.34	46.01

Table 5.2 (Continued)

Sample ID	Marchena/ Beth Haim		Groot Davelaar/Joonchi		Pietermaai / Salina	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	121.30	37.67	10.23	3.18	<IDL	<IDL
C1 Naphthalenes	293.53	37.78	43.82	5.64	22.09	2.84
C2 Naphthalenes	632.25	40.85	39.07	2.52	28.22	1.82
C3 Naphthalenes	901.12	32.47	27.75	1.00	36.73	1.32
C4 Naphthalenes	425.90	8.74	<IDL	<IDL	34.41	0.71
Acenaphthylene	24.46	0.62	<IDL	<IDL	<IDL	<IDL
Acenaphthene	39.96	2.09	<IDL	<IDL	<IDL	<IDL
Fluorene	200.81	8.98	59.33	2.65	41.70	1.87
C1 Fluorenes	700.87	13.42	68.13	1.30	73.33	1.40
C2 Fluorenes	1595.14	25.05	90.31	1.41	178.07	2.80
C3 Fluorenes	1753.86	21.00	<IDL	<IDL	140.18	1.68
C4 Fluorenes	1467.64	8.26	<IDL	<IDL	85.56	0.48
Dibenzothiophene	443.65	5.10	19.21	0.22	13.15	0.15
C1 DBT	646.85	4.88	9.32	0.07	18.37	0.14
C2 DBT	2559.84	14.26	29.78	0.16	<IDL	<IDL
C3 DBT	1552.16	7.35	<IDL	<IDL	<IDL	<IDL
C4 DBT	672.02	2.93	<IDL	<IDL	<IDL	<IDL
Phenanthrene	1339.07	23.24	189.96	3.25	175.40	3.04
Anthracene	134.94	2.57	22.83	0.43	22.15	0.42
C1 Phen_Anthr	4084.66	60.71	159.20	2.31	308.58	4.59
C2 Phen_Anthr	4369.42	55.33	117.79	1.45	296.51	3.75
C3 Phen_Anthr	2567.92	30.34	46.37	0.53	149.29	1.76
C4 Phen_Anthr	820.99	9.37	<IDL	<IDL	<IDL	<IDL
Fluoranthene	283.27	3.02	59.13	0.61	406.88	4.34
Pyrene	383.19	2.84	39.30	0.28	408.78	3.03
C1 Flu/Pyrene	205.72	1.42	16.37	0.11	223.92	1.54
C2 Flu/Pyrene	179.79	1.19	13.17	0.08	139.92	0.93
C3 Flu/Pyrene	66.55	0.43	<IDL	<IDL	<IDL	<IDL
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	44.31	0.37	4.73	0.04	28.93	0.24
Chrysene	101.08	0.64	8.59	0.05	57.31	0.36
C1 B(a)A/Chrys/Triph	57.32	0.35	<IDL	<IDL	30.36	0.19
C2 B(a)A/Chrys/Triph	36.32	0.22	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	37.54	0.27	<IDL	<IDL	34.90	0.25
Benzo(k)fluoranthene	12.56	0.05	<IDL	<IDL	16.63	0.06
Benzo(a)pyrene	7.74	0.05	<IDL	<IDL	<IDL	<IDL
Perylene	12.08	0.05	<IDL	<IDL	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAHs	28775.83	463.93	1074.39	27.31	2971.37	39.72

Table 5.2 (Continued)

Sample ID	Buena Vista (#18)		Marchena		Rooi Catochi (#3)	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	71.31	22.15	36.39	11.30	<IDL	<IDL
C1 Naphthalenes	182.93	23.55	154.50	19.89	15.72	2.02
C2 Naphthalenes	362.00	23.39	440.49	28.46	20.55	1.33
C3 Naphthalenes	514.51	18.54	797.19	28.72	24.91	0.90
C4 Naphthalenes	314.18	6.45	542.25	11.12	23.99	0.49
Acenaphthylene	14.56	0.37	<IDL	<IDL	<IDL	<IDL
Acenaphthene	33.97	1.77	33.11	1.73	<IDL	<IDL
Fluorene	195.31	8.74	172.96	7.74	25.13	1.12
C1 Fluorenes	704.44	13.49	785.83	15.04	50.72	0.97
C2 Fluorenes	993.26	15.60	2086.92	32.77	83.66	1.31
C3 Fluorenes	1554.04	18.61	2762.86	33.09	<IDL	<IDL
C4 Fluorenes	740.27	4.17	1894.22	10.67	<IDL	<IDL
Dibenzothiophene	226.34	2.60	414.13	4.76	11.26	0.13
C1 DBT	246.52	1.86	1481.95	11.19	<IDL	<IDL
C2 DBT	823.39	4.59	3087.34	17.19	<IDL	<IDL
C3 DBT	501.21	2.37	1999.89	9.47	<IDL	<IDL
C4 DBT	223.62	0.98	736.78	3.22	<IDL	<IDL
Phenanthrene	1051.66	18.25	1326.35	23.02	63.47	1.10
Anthracene	113.52	2.16	152.97	2.91	<IDL	<IDL
C1 Phen_Anthr	1718.48	25.54	4154.67	61.75	158.09	2.35
C2 Phen_Anthr	1564.27	19.81	4360.33	55.22	139.83	1.77
C3 Phen_Anthr	835.26	9.87	2168.03	25.62	68.72	0.81
C4 Phen_Anthr	205.02	2.34	781.41	8.92	55.01	0.63
Fluoranthene	343.18	3.66	286.14	3.06	724.75	7.74
Pyrene	364.26	2.70	383.46	2.84	543.52	4.02
C1 Flu/Pyrene	213.32	1.47	317.06	2.18	334.42	2.30
C2 Flu/Pyrene	150.16	1.00	212.50	1.41	356.78	2.37
C3 Flu/Pyrene	92.02	0.60	65.38	0.43	234.90	1.53
C4 Flu/Pyrene	22.39	0.15	35.80	0.23	<IDL	<IDL
Benzo(a)anthracene	49.54	0.42	38.76	0.33	38.68	0.32
Chrysene	81.26	0.51	79.27	0.50	77.36	0.49
C1 B(a)A/Chrys/Triph	51.82	0.32	61.02	0.38	<IDL	<IDL
C2 B(a)A/Chrys/Triph	<IDL	<IDL	54.25	0.33	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	55.97	0.40	34.67	0.25	<IDL	<IDL
Benzo(k)fluoranthene	36.92	0.13	11.45	0.04	<IDL	<IDL
Benzo(a)pyrene	40.93	0.27	4.71	0.03	<IDL	<IDL
Perylene	19.89	0.08	<IDL	<IDL	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	36.89	0.16	13.29	0.06	<IDL	<IDL
∑PAHs	14748.62	259.05	31968.33	435.85	3051.47	33.72

Table 5.2 (Continued)

Sample ID	Roosendaal / Gasparitu		Parasasa (#24)		Rooi Catochi (#12)	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	34.86	10.83	6.90	2.14	7.20	2.24
C1 Naphthalenes	119.91	15.44	36.82	4.74	33.78	4.35
C2 Naphthalenes	171.70	11.09	78.15	5.05	43.15	2.79
C3 Naphthalenes	137.06	4.92	117.82	4.24	46.36	1.67
C4 Naphthalenes	98.18	1.99	209.40	4.30	43.97	0.90
Acenaphthylene	6.99	0.18	8.74	0.22	<IDL	<IDL
Acenaphthene	16.94	0.88	13.42	0.70	<IDL	<IDL
Fluorene	88.34	3.95	54.57	2.44	45.06	2.02
C1 Fluorenes	290.71	5.55	288.20	5.52	157.34	3.01
C2 Fluorenes	527.58	8.24	494.43	7.76	195.39	3.07
C3 Fluorenes	670.03	7.95	851.68	10.20	266.51	3.19
C4 Fluorenes	<IDL	<IDL	471.04	2.65	<IDL	<IDL
Dibenzothiophene	55.80	0.64	69.24	0.80	14.45	0.17
C1 DBT	168.69	1.26	236.05	1.78	14.19	0.11
C2 DBT	205.56	1.12	554.22	3.09	73.61	0.41
C3 DBT	<IDL	<IDL	348.73	1.65	<IDL	<IDL
C4 DBT	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Phenanthrene	450.03	7.70	272.44	4.73	157.48	2.73
Anthracene	28.09	0.53	29.96	0.57	21.83	0.42
C1 Phen_Anthr	672.85	9.78	787.34	11.70	215.21	3.20
C2 Phen_Anthr	686.18	8.45	868.95	11.00	196.09	2.48
C3 Phen_Anthr	421.64	4.84	653.25	7.72	101.43	1.20
C4 Phen_Anthr	<IDL	<IDL	264.78	3.02	13.13	0.15
Fluoranthene	561.44	5.82	107.61	1.15	103.48	1.10
Pyrene	483.95	3.49	111.32	0.82	<IDL	<IDL
C1 Flu/Pyrene	254.73	1.70	89.68	0.62	46.84	0.32
C2 Flu/Pyrene	203.63	1.31	64.03	0.43	32.48	0.22
C3 Flu/Pyrene	42.29	0.27	14.58	0.10	<IDL	<IDL
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	51.57	0.42	12.53	0.11	10.64	0.09
Chrysene	102.49	0.63	24.21	0.15	16.03	0.10
C1 B(a)A/Chrys/Triph	76.89	0.46	22.06	0.14	<IDL	<IDL
C2 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	64.16	0.44	13.09	0.09	12.33	0.09
Benzo(k)fluoranthene	20.95	0.07	5.77	0.02	10.34	0.04
Benzo(a)pyrene	<IDL	<IDL	<IDL	<IDL	3.96	0.03
Perylene	<IDL	<IDL	<IDL	<IDL	5.67	0.02
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	31.29	0.13	3.37	0.01	4.71	0.02
∑PAHs	6744.53	120.07	7184.38	99.67	1892.66	36.12

Table 5.2 (Continued)

Sample ID	Steenrijk		Buena Vista (#20)		Suffisant (#16)	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	16.55	5.14	17.92	5.57	<IDL	<IDL
C1 Naphthalenes	48.92	6.30	37.80	4.87	26.20	3.37
C2 Naphthalenes	44.58	2.88	60.41	3.90	39.91	2.58
C3 Naphthalenes	41.70	1.50	64.11	2.31	44.67	1.61
C4 Naphthalenes	46.25	0.95	63.86	1.31	47.77	0.98
Acenaphthylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Fluorene	51.71	2.31	54.22	2.43	38.39	1.72
C1 Fluorenes	187.50	3.59	179.07	3.43	161.11	3.08
C2 Fluorenes	329.87	5.18	163.85	2.57	209.16	3.28
C3 Fluorenes	362.15	4.34	321.79	3.85	188.74	2.26
C4 Fluorenes	253.68	1.43	<IDL	<IDL	<IDL	<IDL
Dibenzothiophene	16.52	0.19	17.59	0.20	11.78	0.14
C1 DBT	18.31	0.14	52.85	0.40	38.14	0.29
C2 DBT	84.21	0.47	111.45	0.62	52.78	0.29
C3 DBT	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 DBT	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Phenanthrene	203.09	3.52	198.78	3.45	96.32	1.67
Anthracene	17.16	0.33	29.40	0.56	18.43	0.35
C1 Phen_Anthr	254.84	3.79	258.33	3.84	157.52	2.34
C2 Phen_Anthr	251.77	3.19	236.75	3.00	137.21	1.74
C3 Phen_Anthr	188.03	2.22	120.00	1.42	102.76	1.21
C4 Phen_Anthr	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Fluoranthene	350.70	3.74	147.32	1.57	54.05	0.58
Pyrene	272.66	2.02	119.89	0.89	41.46	0.31
C1 Flu/Pyrene	190.79	1.31	79.56	0.55	48.33	0.33
C2 Flu/Pyrene	129.26	0.86	42.19	0.28	27.34	0.18
C3 Flu/Pyrene	60.79	0.40	14.41	0.09	<IDL	<IDL
C4 Flu/Pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	24.01	0.20	13.02	0.11	7.53	0.06
Chrysene	52.90	0.33	23.36	0.15	14.93	0.09
C1 B(a)A/Chrys/Triph	26.88	0.17	<IDL	<IDL	<IDL	<IDL
C2 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	36.38	0.26	13.36	0.10	4.88	0.03
Benzo(k)fluoranthene	17.96	0.07	5.69	0.02	3.81	0.01
Benzo(a)pyrene	<IDL	<IDL	6.84	0.05	<IDL	<IDL
Perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAHs	3579.17	56.82	2453.82	47.52	1573.22	28.52

Table 5.2 (Continued)

Sample ID	Welgelegen (Veld Salu)		Wishi		Zeelandia	
Air Volume/Conc	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)	(ng/disk)	(ng/m³)
Naphthalene	25.99	8.07	47.20	14.66	12.98	4.03
C1 Naphthalenes	118.58	15.26	145.43	18.72	39.24	5.05
C2 Naphthalenes	375.94	24.29	333.72	21.55	45.67	2.95
C3 Naphthalenes	802.57	28.92	589.02	21.16	40.05	1.44
C4 Naphthalenes	1158.56	23.77	799.84	16.22	70.22	1.44
Acenaphthylene	<IDL	<IDL	14.19	0.36	<IDL	<IDL
Acenaphthene	32.49	1.70	31.00	1.62	<IDL	<IDL
Fluorene	180.00	8.05	176.85	7.91	51.19	2.29
C1 Fluorenes	1111.73	21.28	576.50	11.00	63.25	1.21
C2 Fluorenes	3175.45	49.87	1402.80	21.91	358.74	5.63
C3 Fluorenes	5183.78	62.08	2250.91	26.70	375.27	4.49
C4 Fluorenes	3497.27	19.69	1044.18	5.71	360.12	2.03
Dibenzothiophene	396.83	4.56	284.65	3.25	22.89	0.26
C1 DBT	1861.08	14.05	799.02	5.95	17.57	0.13
C2 DBT	4798.94	26.72	1639.70	8.93	94.09	0.52
C3 DBT	4142.27	19.61	1000.20	4.61	<IDL	<IDL
C4 DBT	2045.33	8.93	724.95	3.07	<IDL	<IDL
Phenanthrene	1419.12	24.63	1073.07	18.37	233.30	4.05
Anthracene	167.50	3.19	117.20	2.19	37.13	0.71
C1 Phen_Anthr	5997.50	89.14	2427.71	35.27	277.99	4.13
C2 Phen_Anthr	8122.76	102.86	2387.03	29.41	272.83	3.45
C3 Phen_Anthr	5364.10	63.38	1412.20	16.20	212.93	2.52
C4 Phen_Anthr	2237.22	25.55	515.51	5.71	38.37	0.44
Fluoranthene	158.11	1.69	233.62	2.42	146.43	1.56
Pyrene	350.60	2.59	279.80	2.02	132.68	0.98
C1 Flu/Pyrene	604.86	4.16	194.97	1.30	77.55	0.53
C2 Flu/Pyrene	455.54	3.03	140.77	0.91	41.64	0.28
C3 Flu/Pyrene	155.66	1.02	40.65	0.26	11.17	0.07
C4 Flu/Pyrene	93.88	0.61	<IDL	<IDL	<IDL	<IDL
Benzo(a)anthracene	17.83	0.15	27.25	0.22	15.83	0.13
Chrysene	70.96	0.45	52.90	0.32	23.37	0.15
C1 B(a)A/Chrys/Triph	71.90	0.44	20.74	0.12	<IDL	<IDL
C2 B(a)A/Chrys/Triph	44.25	0.27	<IDL	<IDL	<IDL	<IDL
C3 B(a)A/Chrys/Triph	5.21	0.03	<IDL	<IDL	<IDL	<IDL
C4 B(a)A/Chrys/Triph	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(b)fluoranthene	9.25	0.07	26.27	0.18	17.87	0.13
Benzo(k)fluoranthene	6.37	0.02	12.02	0.04	8.60	0.03
Benzo(a)pyrene	<IDL	<IDL	6.53	0.04	4.08	0.03
Perylene	<IDL	<IDL	<IDL	<IDL	8.66	0.04
Indeno(1,2,3-c,d)pyrene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo(g,h,i)perylene	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAHs	54259.43	660.14	20828.40	308.34	3111.71	50.72

Table 5.3 The 2011 site specific binary diagnostic ratios used in this study to elucidate emission sources.

	LMW/HMW	PHN / (PHN+ANT)	PHN/ANT	FLA / (FLA+PYR)	FLA / PYR	BaA/ (BaA+CHR)
Habaai	12	0.97	34	0.7	2	0.5
W. Piscadera Baai	47	0.97	37	0.7	2	<IDL
Nieuw Nederland	18	0.97	35	0.7	2	0.4
Groot Piscadera	14	0.97	35	0.7	2	0.6
Boca Sami	19	0.97	34	0.7	2	0.6
Rooi Catochi	22	0.98	54	0.7	3	1.0
Marie Pampoen	17	<IDL	<IDL	0.8	3	<IDL
Buena Vista (#8)	9	0.96	23	0.7	2	<IDL
Buena Vista (#9)	33	0.97	32	0.6	2	0.4
Heintje Kool	10	0.97	29	0.6	2	0.4
Roosendaal	12	0.97	31	0.7	2	<IDL
Marchena	13	0.96	26	0.6	2	0.4
E. Piscadera	10	0.97	31	0.6	2	0.2
2011 Mean ratio	17	0.97	33	0.7	2	0.5
Dominant source	Petro	Petro	Petro	Pyro	Pyro	Combustion

Table 5.4 The 2014 site specific binary diagnostic ratios used in this study to elucidate emission sources.

	LMW/ HMW	PHN/ (PHN+ANT)	PHN/ ANT	FLA / (FLA+PYR)	FLA / PYR	BaA/ (BaA+CHR)
Habaai	3.5	0.90	9.3	0.6	1.3	0.4
Parasasa (#25)	2.4	0.91	10	0.6	1.3	0.4
Buena Vista (#21)	14	0.86	6.1	0.6	1.4	0.4
Buena Vista (#17)	7.6	0.90	9.1	0.5	1.0	0.4
Wanapa	2.1	0.81	4.2	0.6	1.6	0.5
Domi/Welgelegen	6.8	0.89	8.0	0.6	1.5	0.4
Gasparitu	5.6	0.90	9.4	0.6	1.5	0.4
Marchena (Beth Haim)	11	0.90	9.1	0.5	1.1	0.4
Buena Vista (#18)	6.7	0.89	8.4	0.6	1.4	0.4
Marchena	7.3	0.89	7.9	0.5	1.1	0.4
Roosendaal / Gasparitu	2.2	0.94	14.6	0.6	1.7	0.4
Parasasa (#24)	4.9	0.89	8.3	0.6	1.4	0.4
Welgelegen (Veld Salu)	10	0.89	7.7	0.4	0.7	0.3
Wishi	9.2	0.89	8.4	0.5	1.2	0.4
Biesheuvel	3.4	0.87	6.6	0.6	1.6	0.5
Toni Kunchi	5.6	0.92	12.0	0.7	2.5	0.4
Suffisant (#28)	3.9	0.88	7.1	0.6	1.6	0.4
Nieuw Nederland	2.6	<IDL	<IDL	0.6	1.8	0.5
Groot Davelaar/Joonchi	9.9	0.88	7.6	0.7	2.2	0.4
Pietermaai / Salina	0.7	0.88	7.2	0.6	1.4	0.4
Brievengat/Groot Kwartier	1.6	<IDL	<IDL	0.6	1.8	0.3
Emmastad (#4)	0.7	0.89	8.0	0.6	1.4	0.4
Rooi Catochi (#3)	0.2	<IDL	<IDL	0.7	1.9	0.4
Emmastad (#14)	1.4	0.87	6.7	0.6	1.5	0.5
Punda	2.6	0.95	21.0	0.7	1.9	0.3
Rooi Catochi (#12)	5.1	0.87	6.6	1.0	<IDL	0.5
Steenrijk	1.7	0.92	10.8	0.6	1.9	0.4
Buena Vista (#20)	4.2	0.86	6.2	0.6	1.8	0.4
Suffisant (#16)	3.6	0.83	4.8	0.7	1.9	0.4
Zeelandia	3.7	0.85	5.7	0.6	1.6	0.5
2014 Mean Downwind Ratios	6.7	0.82	8.6	0.6	1.3	0.4
2014 Mean Upwind Ratios	3.2	0.83	8.5	0.7	1.8	0.4
Dominant source	Petro	Petro/Pyro	Petro/Pyro	Pyro	Pyro	Combustion

Table 5.5 Factor loadings (varimax raw) for the 2011 ambient air PAH concentrations using principle components extraction method. Factor loadings >0.80 are in marked red and were grouped together indicating the major PAHs for each factor.

	Factor 1	Factor 2	Factor 3
Naphthalene	0.069092	0.291203	0.610572
Acenaphthylene	0.928838	0.223230	-0.096831
Acenaphthene	0.840067	0.334748	0.016594
Fluorene	0.933055	0.274561	0.080976
Dibenzothiophene	0.864688	0.478324	-0.014071
Phenanthrene	0.874449	0.459011	0.135915
Anthracene	0.933681	0.336468	0.020383
Fluoranthene	0.750875	0.612102	0.152977
Pyrene	0.818118	0.527827	0.096016
Benzo[a]anthracene	0.651210	0.624531	0.053527
Chrysene	0.778861	0.520710	-0.036876
Benzo[b]fluoranthene	0.401618	0.888082	0.072340
Benzo[k]fluoranthene	0.404704	0.898834	0.065241
Benzo[a]pyrene	0.404704	0.898834	0.065241
Perylene	0.234045	0.770073	-0.049868
Indeno[1,2,3-c,d]pyrene	0.404704	0.898834	0.065241
Dibenzo[a,h]anthracene	0.028624	0.027703	0.848257
Benzo[g,h,i]perylene	0.404704	0.898834	0.065241
Eigenvalue	13.06391	1.804325	1.088077
% Total Variance	73%	10%	6%

Table 5.6 Factor loadings (varimax raw) for the 2014 downwind ambient air PAH concentrations using principle components extraction method. Factor loadings >0.80 are in marked red and were grouped together indicating the major PAHs for each factor.

	Factor 1	Factor 2	Factor 3	Factor 4
Naphthalene	0.170147	-0.057020	-0.723645	0.441199
Acenaphthylene	0.332802	0.214705	-0.507602	0.434378
Acenaphthene	0.736120	0.215076	-0.334698	0.277825
Fluorene	0.911737	0.213036	0.000335	0.291474
Dibenzothiophene	0.983953	0.139247	0.028202	-0.028601
Phenanthrene	0.949233	0.265649	0.096467	0.007521
Anthracene	0.961866	0.103725	0.122068	0.031631
Fluoranthene	0.071405	0.963968	-0.136215	-0.033400
Pyrene	0.435011	0.799886	-0.340439	-0.111841
Benzo[a]anthracene	0.386444	0.877104	0.078285	0.193375
Chrysene	0.539071	0.805248	0.040083	-0.042217
Benzo[b]fluoranthene	0.119729	0.920121	0.019094	0.321559
Benzo[k]fluoranthene	0.128996	0.651872	0.119794	0.684424
Benzo[a]pyrene	0.111026	0.186178	0.099856	0.885108
Perylene	0.107341	-0.025067	-0.149898	0.903521
Indeno[1,2,3-c,d]pyrene	-0.123765	0.055115	-0.957469	-0.066918
Dibenzo[a,h]anthracene	-0.123765	0.055115	-0.957469	-0.066918
Benzo[g,h,i]perylene	-0.175079	0.683762	-0.029908	0.566979
Eigenvalue	7.877513	3.283947	2.713859	2.235517
% Total Variance	44%	18%	15%	12%

Table 5.7 Factor loadings (varimax raw) for the 2014 upwind ambient air PAH concentrations using principle components extraction method. Factor loadings >0.80 are in marked red and were grouped together indicating the major PAHs for each factor.

	Factor 1	Factor 2
Naphthalene	0.695454	-0.228918
Fluorene	0.910446	-0.153661
Dibenzothiophene	0.952249	0.000250
Phenanthrene	0.957727	0.067877
Anthracene	0.380751	-0.250903
Fluoranthene	-0.043536	0.916133
Pyrene	-0.057878	0.959316
Benzo[a]anthracene	-0.010211	0.987936
Chrysene	0.151275	0.944552
Benzo[b]fluoranthene	0.489824	0.685589
Benzo[k]fluoranthene	0.612779	0.518031
Benzo[a]pyrene	-0.056780	0.576498
Perylene	0.074205	-0.332510
Indeno[1,2,3-c,d]pyrene	0.899960	0.193801
Benzo[g,h,i]perylene	0.896546	0.106610
Eigenvalue	5.910118	4.639134
% Total Variance	39%	37%

Table 5.8 Potency equivalents (ng-PEQ/m^3) and the percent contribution for each of the 2011 sites.

Potency Equivalent Concentrations (ng-PEQ/m^3)					
	2011 Mean	**Habaai**	**W. Piscadera Baai**	**Nieuw Nederland**	**Groot Piscadera**
Naphthalene	0.0298	0.0673	0.0479	0.0308	0.0276
Acenaphthylene	0.0001	0.0007	<IDL	<IDL	<IDL
Acenaphthene	0.0006	0.0030	0.0015	0.0004	0.0009
Fluorene	0.0043	0.0119	0.0036	0.0037	0.0048
Phenanthrene	0.0413	0.1554	0.0324	0.0369	0.0376
Anthracene	0.0127	0.0462	0.0077	0.0094	0.0105
Fluoranthene	0.0030	0.0114	0.0012	0.0025	0.0027
Pyrene	0.0015	0.0059	0.0006	0.0011	0.0013
Benzo[a]anthracene	0.0206	0.1030	<IDL	0.0068	0.0555
Chrysene	0.0025	0.0107	<IDL	0.0012	0.0043
Benzo[b]fluoranthene	0.0028	0.0353	<IDL	<IDL	<IDL
Benzo[k]fluoranthene	0.0008	0.0116	<IDL	<IDL	<IDL
Benzo[a]pyrene	0.0088	0.1318	<IDL	<IDL	<IDL
Indeno[1,2,3-c,d]pyrene	0.0006	0.0132	<IDL	<IDL	<IDL
Dibenzo[a,h]anthracene	0.1582	0.4840	<IDL	<IDL	<IDL
Benzo[g,h,i]perylene	0.0001	0.0007	<IDL	<IDL	<IDL
∑PAH	0.2877	1.0922	0.0949	0.0928	0.1454
% Potency Equivalent Contributions					
Naphthalene	10%	6%	51%	33%	19%
Acenaphthylene	0%	0%	0%	0%	0%
Acenaphthene	0%	0%	2%	0%	1%
Fluorene	1%	1%	4%	4%	3%
Phenanthrene	14%	14%	34%	40%	26%
Anthracene	4%	4%	8%	10%	7%
Fluoranthene	1%	1%	1%	3%	2%
Pyrene	1%	1%	1%	1%	1%
Benzo[a]anthracene	7%	9%	0%	7%	38%
Chrysene	1%	1%	0%	1%	3%
Benzo[b]fluoranthene	1%	3%	0%	0%	0%
Benzo[k]fluoranthene	0%	1%	0%	0%	0%
Benzo[a]pyrene	3%	12%	0%	0%	0%
Indeno[1,2,3-c,d]pyrene	0%	1%	0%	0%	0%
Dibenzo[a,h]anthracene	55%	44%	0%	0%	0%
Benzo[g,h,i]perylene	0%	0%	0%	0%	0%
% Total	100%	100%	100%	100%	100%

Table 5.8 (Continued)

Potency Equivalent Concentrations (ng-PEQ/m^3)					
	Boca Sami	**Rooi Catochi**	**Marie Pampoen**	**Buena Vista (#8)**	**Buena Vista (#9)**
Naphthalene	0.0276	0.0429	0.0218	0.0129	0.1408
Acenaphthylene	<IDL	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	0.0004	<IDL	<IDL	<IDL
Fluorene	0.0040	0.0016	0.0026	0.0038	0.0037
Phenanthrene	0.0418	0.0117	0.0224	0.0413	0.0378
Anthracene	0.0110	0.0078	<IDL	0.0097	0.0131
Fluoranthene	0.0022	0.0027	0.0017	0.0027	0.0033
Pyrene	0.0011	0.0010	0.0005	0.0014	0.0018
Benzo[a]anthracene	0.0355	0.0082	<IDL	<IDL	0.0240
Chrysene	0.0024	<IDL	<IDL	0.0012	0.0031
Benzo[b]fluoranthene	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo[k]fluoranthene	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo[a]pyrene	<IDL	<IDL	<IDL	<IDL	<IDL
Indeno[1,2,3-c,d]pyrene	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo[a,h]anthracene	1.7598	<IDL	<IDL	<IDL	0.2567
Benzo[g,h,i]perylene	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAH	1.8853	0.0763	0.0490	0.0730	0.4843
% Potency Equivalent Contributions					
Naphthalene	1%	56%	45%	18%	29%
Acenaphthylene	0%	0%	0%	0%	0%
Acenaphthene	0%	1%	0%	0%	0%
Fluorene	0%	2%	5%	5%	1%
Phenanthrene	2%	15%	46%	57%	8%
Anthracene	1%	10%	0%	13%	3%
Fluoranthene	0%	3%	3%	4%	1%
Pyrene	0%	1%	1%	2%	0%
Benzo[a]anthracene	2%	11%	0%	0%	5%
Chrysene	0%	0%	0%	2%	1%
Benzo[b]fluoranthene	0%	0%	0%	0%	0%
Benzo[k]fluoranthene	0%	0%	0%	0%	0%
Benzo[a]pyrene	0%	0%	0%	0%	0%
Indeno[1,2,3-c,d]pyrene	0%	0%	0%	0%	0%
Dibenzo[a,h]anthracene	93%	0%	0%	0%	53%
Benzo[g,h,i]perylene	0%	0%	0%	0%	0%
% Total	100%	100%	100%	100%	100%

Table 5.8 (Continued)

Potency Equivalent Concentrations (ng-PEQ/m^3)					
	Heintje Kool	Roosendaal	Marchena	E. Piscadera	Parasasa
Naphthalene	0.0215	0.0039	0.0025	<IDL	<IDL
Acenaphthylene	<IDL	<IDL	0.0009	<IDL	<IDL
Acenaphthene	<IDL	<IDL	0.0025	<IDL	<IDL
Fluorene	0.0033	0.0118	0.0044	<IDL	0.0043
Phenanthrene	0.0271	0.1136	0.0332	<IDL	0.0413
Anthracene	0.0129	0.0087	0.0433	0.0107	<IDL
Fluoranthene	0.0037	0.0020	0.0059	0.0023	0.0003
Pyrene	0.0020	0.0010	0.0037	0.0013	<IDL
Benzo[a]anthracene	0.0240	<IDL	0.0437	0.0076	<IDL
Chrysene	0.0043	0.0006	0.0066	0.0028	<IDL
Benzo[b]fluoranthene	0.0073	<IDL	<IDL	<IDL	<IDL
Benzo[k]fluoranthene	<IDL	<IDL	<IDL	<IDL	<IDL
Benzo[a]pyrene	<IDL	<IDL	<IDL	<IDL	<IDL
Indeno[1,2,3-c,d]pyrene	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo[a,h]anthracene	<IDL	<IDL	<IDL	<IDL	0.1582
Benzo[g,h,i]perylene	<IDL	<IDL	<IDL	<IDL	<IDL
∑PAH	0.1062	0.1416	0.1467	0.0247	0.2041
% Potency Equivalent Contributions					
Naphthalene	20%	3%	2%	0%	0%
Acenaphthylene	0%	0%	1%	0%	0%
Acenaphthene	0%	0%	2%	0%	0%
Fluorene	3%	8%	3%	0%	2%
Phenanthrene	26%	80%	23%	0%	20%
Anthracene	12%	6%	30%	43%	0%
Fluoranthene	3%	1%	4%	9%	0%
Pyrene	2%	1%	3%	5%	0%
Benzo[a]anthracene	23%	0%	30%	31%	0%
Chrysene	4%	0%	4%	12%	0%
Benzo[b]fluoranthene	7%	0%	0%	0%	0%
Benzo[k]fluoranthene	0%	0%	0%	0%	0%
Benzo[a]pyrene	0%	0%	0%	0%	0%
Indeno[1,2,3-c,d]pyrene	0%	0%	0%	0%	0%
Dibenzo[a,h]anthracene	0%	0%	0%	0%	78%
Benzo[g,h,i]perylene	0%	0%	0%	0%	0%
% Total	100%	100%	100%	100%	100%

Table 5.9 Potency equivalents (ng-PEQ/m^3) and the percent contribution for the 2014 downwind sites.

Potency Equivalent Concentrations (ng-PEQ/m^3)				
	2014 Downwind Mean	**Habaai**	**Parasasa (#25)**	**Buena Vista (#21)**
Naphthalene	0.0154	0.0020	0.0032	0.0403
Acenaphthylene	0.0002	<IDL	0.0002	<IDL
Acenaphthene	0.0008	<IDL	0.0007	<IDL
Fluorene	0.0050	0.0062	0.0023	0.0039
Phenanthrene	0.0122	0.0222	0.0080	0.0058
Anthracene	0.0145	0.0238	0.0079	0.0095
Fluoranthene	0.0028	0.0050	0.0031	0.0018
Pyrene	0.0022	0.0039	0.0024	0.0013
Benzo[a]anthracene	0.0232	0.0410	0.0266	0.0102
Chrysene	0.0037	0.0069	0.0049	0.0014
Benzo[b]fluoranthene	0.0193	0.0269	0.0265	0.0106
Benzo[k]fluoranthene	0.0042	0.0044	0.0042	0.0022
Benzo[a]pyrene	0.0469	0.0427	0.0320	0.0797
Indeno[1,2,3-c,d]pyrene	0.0003	<IDL	<IDL	<IDL
Dibenzo[a,h]anthracene	0.0123	<IDL	<IDL	<IDL
Benzo[g,h,i]perylene	0.0004	0.0000	0.0007	0.0004
∑PAH	0.1631	0.1849	0.1228	0.1670
% Potency Equivalent Contributions				
Naphthalene	9%	1%	3%	24%
Acenaphthylene	0%	0%	0%	0%
Acenaphthene	1%	0%	1%	0%
Fluorene	3%	3%	2%	2%
Phenanthrene	7%	12%	7%	3%
Anthracene	9%	13%	6%	6%
Fluoranthene	2%	3%	3%	1%
Pyrene	1%	2%	2%	1%
Benzo[a]anthracene	14%	22%	22%	6%
Chrysene	2%	4%	4%	1%
Benzo[b]fluoranthene	12%	15%	22%	6%
Benzo[k]fluoranthene	3%	2%	3%	1%
Benzo[a]pyrene	29%	23%	26%	48%
Indeno[1,2,3-c,d]pyrene	0%	0%	0%	0%
Dibenzo(a,h)anthracene	8%	0%	0%	0%
Benzo(g,h,i)perylene	0%	0%	1%	0%
% Total	100%	100%	100%	100%

Table 5.9 (Continued)

Potency Equivalent Concentrations (ng-PEQ/m^3)				
	Buena Vista (#17)	Wanapa	Domi/Welgelegen	Gasparito
Naphthalene	0.0453	<IDL	0.0167	0.0104
Acenaphthylene	0.0004	<IDL	<IDL	<IDL
Acenaphthene	0.0015	<IDL	<IDL	<IDL
Fluorene	0.0039	0.0018	0.0031	0.0036
Phenanthrene	0.0070	0.0024	0.0079	0.0058
Anthracene	0.0077	0.0057	0.0098	0.0062
Fluoranthene	0.0036	0.0013	0.0022	0.0019
Pyrene	0.0035	0.0008	0.0015	0.0013
Benzo[a]anthracene	0.0184	0.0076	0.0177	0.0140
Chrysene	0.0031	0.0009	0.0030	0.0019
Benzo[b]fluoranthene	0.0178	0.0060	0.0093	0.0129
Benzo[k]fluoranthene	0.0031	0.0019	0.0029	0.0037
Benzo[a]pyrene	0.0256	0.0139	0.0650	<IDL
Indeno[1,2,3-c,d]pyrene	0.0052	<IDL	<IDL	<IDL
Dibenzo[a,h]anthracene	0.1841	<IDL	<IDL	<IDL
Benzo[g,h,i]perylene	0.0005	<IDL	<IDL	<IDL
∑PAH	0.3306	0.0423	0.1392	0.0618
% Potency Equivalent Contributions				
Naphthalene	14%	0%	12%	17%
Acenaphthylene	0%	0%	0%	0%
Acenaphthene	0%	0%	0%	0%
Fluorene	1%	4%	2%	6%
Phenanthrene	2%	6%	6%	9%
Anthracene	2%	14%	7%	10%
Fluoranthene	1%	3%	2%	3%
Pyrene	1%	2%	1%	2%
Benzo[a]anthracene	6%	18%	13%	23%
Chrysene	1%	2%	2%	3%
Benzo[b]fluoranthene	5%	14%	7%	21%
Benzo[k]fluoranthene	1%	4%	2%	6%
Benzo[a]pyrene	8%	33%	47%	0%
Indeno[1,2,3-c,d]pyrene	2%	0%	0%	0%
Dibenzo(a,h)anthracene	56%	0%	0%	0%
Benzo(g,h,i)perylene	0%	0%	0%	0%
% Total	100%	100%	100%	100%

Table 5.9 (Continued)

Potency Equivalent Concentrations (ng-PEQ/m^3)				
	Marchena/Beth Haim	Buena Vista (#18)	Marchena	Roosendaal / Gasparitu
Naphthalene	0.0377	0.0221	0.0113	0.0108
Acenaphthylene	0.0006	0.0004	<IDL	0.0002
Acenaphthene	0.0021	0.0018	0.0017	0.0009
Fluorene	0.0090	0.0087	0.0077	0.0040
Phenanthrene	0.0232	0.0183	0.0230	0.0077
Anthracene	0.0257	0.0216	0.0291	0.0053
Fluoranthene	0.0030	0.0037	0.0031	0.0058
Pyrene	0.0028	0.0027	0.0028	0.0035
Benzo[a]anthracene	0.0372	0.0416	0.0326	0.0420
Chrysene	0.0064	0.0051	0.0050	0.0063
Benzo[b]fluoranthene	0.0267	0.0398	0.0247	0.0442
Benzo[k]fluoranthene	0.0046	0.0134	0.0041	0.0074
Benzo[a]pyrene	0.0517	0.2733	0.0315	<IDL
Indeno[1,2,3-c,d]pyrene	<IDL	<IDL	<IDL	<IDL
Dibenzo[a,h]anthracene	<IDL	<IDL	<IDL	<IDL
Benzo[g,h,i]perylene	<IDL	0.0016	0.0006	0.0013
∑PAH	0.2307	0.4541	0.1772	0.1393
% Potency Equivalent Contributions				
Naphthalene	16%	5%	6%	8%
Acenaphthylene	0%	0%	0%	0%
Acenaphthene	1%	0%	1%	1%
Fluorene	4%	2%	4%	3%
Phenanthrene	10%	4%	13%	6%
Anthracene	11%	5%	16%	4%
Fluoranthene	1%	1%	2%	4%
Pyrene	1%	1%	2%	3%
Benzo[a]anthracene	16%	9%	18%	30%
Chrysene	3%	1%	3%	5%
Benzo[b]fluoranthene	12%	9%	14%	32%
Benzo[k]fluoranthene	2%	3%	2%	5%
Benzo[a]pyrene	22%	60%	18%	0%
Indeno[1,2,3-c,d]pyrene	0%	0%	0%	0%
Dibenzo[a,h]anthracene	0%	0%	0%	0%
Benzo[g,h,i]perylene	0%	0%	0%	1%
% Total	100%	100%	100%	100%

Table 5.9 (Continued)

Potency Equivalent Concentrations (ng-PEQ/m^3)				
	Parasasa (#24)	Buena Vista (#20)	Welgelegen (Veld Salu)	Wishi
Naphthalene	0.0021	0.0056	0.0081	0.0147
Acenaphthylene	0.0002	<IDL	<IDL	0.0004
Acenaphthene	0.0007	<IDL	0.0017	0.0016
Fluorene	0.0024	0.0024	0.0081	0.0079
Phenanthrene	0.0047	0.0035	0.0246	0.0184
Anthracene	0.0057	0.0056	0.0319	0.0219
Fluoranthene	0.0011	0.0016	0.0017	0.0024
Pyrene	0.0008	0.0009	0.0026	0.0020
Benzo[a]anthracene	0.0105	0.0109	0.0150	0.0222
Chrysene	0.0015	0.0015	0.0045	0.0032
Benzo[b]fluoranthene	0.0093	0.0095	0.0066	0.0181
Benzo[k]fluoranthene	0.0021	0.0021	0.0023	0.0042
Benzo[a]pyrene	<IDL	0.0457	<IDL	0.0422
Indeno[1,2,3-c,d]pyrene	<IDL	<IDL	<IDL	<IDL
Dibenzo[a,h]anthracene	<IDL	<IDL	<IDL	<IDL
Benzo[g,h,i]perylene	0.0001	<IDL	<IDL	<IDL
∑PAH	0.0415	0.0891	0.1070	0.1593
% Potency Equivalent Contributions				
Naphthalene	5%	6%	8%	9%
Acenaphthylene	1%	0%	0%	0%
Acenaphthene	2%	0%	2%	1%
Fluorene	6%	3%	8%	5%
Phenanthrene	11%	4%	23%	12%
Anthracene	14%	6%	30%	14%
Fluoranthene	3%	2%	2%	2%
Pyrene	2%	1%	2%	1%
Benzo[a]anthracene	25%	12%	14%	14%
Chrysene	4%	2%	4%	2%
Benzo[b]fluoranthene	22%	11%	6%	11%
Benzo[k]fluoranthene	5%	2%	2%	3%
Benzo[a]pyrene	0%	51%	0%	27%
Indeno[1,2,3-c,d]pyrene	0%	0%	0%	0%
Dibenzo[a,h]anthracene	0%	0%	0%	0%
Benzo[g,h,i]perylene	0%	0%	0%	0%
% Total	100%	100%	100%	100%

Table 5.10 Potency equivalents (ng-PEQ/m^3) and the percent contribution for the 2014 upwind sites.

Potency Equivalent Concentrations (ng-PEQ/m^3)				
	2014 Upwind Mean	**Punda**	**Nieuw Nederland**	**Biesheuvel**
	0.0021	0.0046	0.0008	0.0028
Acenaphthylene	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	<IDL	<IDL	<IDL
Fluorene	0.0024	0.0052	0.0018	0.0028
Phenanthrene	0.0037	0.0094	0.0045	0.0048
Anthracene	0.0037	0.0045	<IDL	0.0074
Fluoranthene	0.0027	0.0042	0.0014	0.0018
Pyrene	0.0016	0.0023	0.0008	0.0011
Benzo[a]anthracene	0.0165	0.0230	0.0165	0.0109
Chrysene	0.0024	0.0046	0.0017	0.0010
Benzo[b]fluoranthene	0.0129	0.0323	0.0150	0.0091
Benzo[k]fluoranthene	0.0035	0.0094	0.0065	0.0021
Benzo[a]pyrene	0.0255	<IDL	0.0627	0.0394
Indeno[1,2,3-c,d]pyrene	0.0007	0.0104	<IDL	0.0005
Dibenzo[a,h]anthracene	<IDL	<IDL	<IDL	<IDL
Benzo[g,h,i]perylene	0.0001	0.0009	<IDL	0.0002
∑PAH	0.07784	0.11081	0.11163	0.08401
% Potency Equivalent Contributions				
Naphthalene	3%	4%	1%	3%
Acenaphthylene	0%	0%	0%	0%
Acenaphthene	0%	0%	0%	0%
Fluorene	3%	5%	2%	3%
Phenanthrene	5%	8%	4%	6%
Anthracene	5%	4%	0%	9%
Fluoranthene	3%	4%	1%	2%
Pyrene	2%	2%	1%	1%
Benzo[a]anthracene	21%	21%	15%	13%
Chrysene	3%	4%	1%	1%
Benzo[b]fluoranthene	17%	29%	13%	11%
Benzo[k]fluoranthene	4%	8%	6%	2%
Benzo[a]pyrene	33%	0%	56%	47%
Indeno[1,2,3-c,d]pyrene	1%	9%	0%	1%
Dibenzo[a,h]anthracene	0%	0%	0%	0%
Benzo[g,h,i]perylene	0%	1%	0%	0%
% Total	100%	100%	100%	100%

Table 5.10 (Continued)

Potency Equivalent Concentrations (ng-PEQ/m^3)				
	Brievengat / Groot Kwartier	Emmastad (#4)	Emmastad (#14)	Toni Kunchi
Naphthalene	0.0020	0.0014	0.0005	0.0031
Acenaphthylene	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	<IDL	<IDL	<IDL
Fluorene	0.0024	0.0021	0.0019	0.0037
Phenanthrene	0.0030	0.0040	0.0023	0.0047
Anthracene	<IDL	0.0050	0.0034	0.0039
Fluoranthene	0.0028	0.0061	0.0020	0.0014
Pyrene	0.0016	0.0045	0.0013	0.0006
Benzo[a]anthracene	0.0139	0.0419	0.0151	0.0063
Chrysene	0.0030	0.0051	0.0018	0.0010
Benzo[b]fluoranthene	<IDL	0.0396	0.0105	0.0044
Benzo[k]fluoranthene	<IDL	0.0069	0.0025	0.0018
Benzo[a]pyrene	<IDL	0.2273	<IDL	<IDL
Indeno[1,2,3-c,d]pyrene	<IDL	<IDL	<IDL	<IDL
Dibenzo[a,h]anthracene	<IDL	<IDL	<IDL	<IDL
Benzo[g,h,i]perylene	<IDL	<IDL	<IDL	<IDL
∑PAH	0.0286	0.3438	0.0414	0.0309
% Potency Equivalent Contributions				
Naphthalene	7%	0%	1%	10%
Acenaphthylene	0%	0%	0%	0%
Acenaphthene	0%	0%	0%	0%
Fluorene	8%	1%	5%	12%
Phenanthrene	11%	1%	6%	15%
Anthracene	0%	1%	8%	13%
Fluoranthene	10%	2%	5%	4%
Pyrene	5%	1%	3%	2%
Benzo[a]anthracene	48%	12%	36%	21%
Chrysene	10%	1%	4%	3%
Benzo[b]fluoranthene	0%	12%	25%	14%
Benzo[k]fluoranthene	0%	2%	6%	6%
Benzo[a]pyrene	0%	66%	0%	0%
Indeno[1,2,3-c,d]pyrene	0%	0%	0%	0%
Dibenzo[a,h]anthracene	0%	0%	0%	0%
Benzo[g,h,i]perylene	0%	0%	0%	0%
% Total	100%	100%	100%	100%

Table 5.10 (Continued)

Potency Equivalent Concentrations (ng-PEQ/m^3)				
	Suffisant (#28)	Groot Davelaar/Joonchi	Pietermaai / Salina	Rooi Catochi (#12)
Naphthalene	0.0019	0.0032	<IDL	<IDL
Acenaphthylene	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	<IDL	<IDL	<IDL
Fluorene	0.0027	0.0027	0.0019	0.0011
Phenanthrene	0.0039	0.0033	0.0030	0.0011
Anthracene	0.0055	0.0043	0.0042	0.0000
Fluoranthene	0.0013	0.0006	0.0043	0.0077
Pyrene	0.0008	0.0003	0.0030	0.0040
Benzo[a]anthracene	0.0100	0.0039	0.0243	0.0325
Chrysene	0.0014	0.0005	0.0036	0.0049
Benzo[b]fluoranthene	0.0068	<IDL	0.0248	<IDL
Benzo[k]fluoranthene	0.0019	<IDL	0.0060	<IDL
Benzo[a]pyrene	<IDL	<IDL	<IDL	<IDL
Indeno[1,2,3-c,d]pyrene	<IDL	<IDL	<IDL	<IDL
Dibenzo[a,h]anthracene	<IDL	<IDL	<IDL	<IDL
Benzo[g,h,i]perylene	<IDL	<IDL	<IDL	<IDL
∑PAH	0.0362	0.0186	0.0753	0.0514
% Potency Equivalent Contributions				
Naphthalene	5%	17%	0%	0%
Acenaphthylene	0%	0%	0%	0%
Acenaphthene	0%	0%	0%	0%
Fluorene	8%	14%	2%	2%
Phenanthrene	11%	17%	4%	2%
Anthracene	15%	23%	6%	0%
Fluoranthene	4%	3%	6%	15%
Pyrene	2%	2%	4%	8%
Benzo[a]anthracene	28%	21%	32%	63%
Chrysene	4%	3%	5%	10%
Benzo[b]fluoranthene	19%	0%	33%	0%
Benzo[k]fluoranthene	5%	0%	8%	0%
Benzo[a]pyrene	0%	0%	0%	0%
Indeno[1,2,3-c,d]pyrene	0%	0%	0%	0%
Dibenzo[a,h]anthracene	0%	0%	0%	0%
Benzo[g,h,i]perylene	0%	0%	0%	0%
% Total	100%	100%	100%	100%

Table 5.10 (Continued)

Potency Equivalent Concentrations (ng-PEQ/m^3)				
	Rooi Catochi (#3)	Steenrijk	Suffisant (#16)	Zeelandia
Naphthalene	0.0022	0.0051	<IDL	<IDL
Acenaphthylene	<IDL	<IDL	<IDL	<IDL
Acenaphthene	<IDL	<IDL	<IDL	<IDL
Fluorene	0.0020	0.0023	0.0017	0.0023
Phenanthrene	0.0027	0.0035	0.0017	0.0040
Anthracene	0.0042	0.0033	0.0035	0.0071
Fluoranthene	0.0011	0.0037	0.0006	0.0016
Pyrene	<IDL	0.0020	0.0003	0.0010
Benzo[a]anthracene	0.0089	0.0202	0.0063	0.0133
Chrysene	0.0010	0.0033	0.0009	0.0015
Benzo[b]fluoranthene	0.0088	0.0259	0.0035	0.0127
Benzo[k]fluoranthene	0.0037	0.0065	0.0014	0.0031
Benzo[a]pyrene	0.0264	<IDL	<IDL	0.0272
Indeno[1,2,3-c,d]pyrene	<IDL	<IDL	<IDL	<IDL
Dibenzo[a,h]anthracene	<IDL	<IDL	<IDL	<IDL
Benzo[g,h,i]perylene	<IDL	<IDL	<IDL	<IDL
∑PAH	0.0614	0.0759	0.0199	0.0778
% Potency Equivalent Contributions				
Naphthalene	4%	7%	0%	5%
Acenaphthylene	0%	0%	0%	0%
Acenaphthene	0%	0%	0%	0%
Fluorene	3%	3%	9%	3%
Phenanthrene	4%	5%	8%	5%
Anthracene	7%	4%	18%	9%
Fluoranthene	2%	5%	3%	2%
Pyrene	0%	3%	2%	1%
Benzo[a]anthracene	15%	27%	32%	17%
Chrysene	2%	4%	5%	2%
Benzo[b]fluoranthene	14%	34%	17%	16%
Benzo[k]fluoranthene	6%	9%	7%	4%
Benzo[a]pyrene	43%	0%	0%	35%
Indeno[1,2,3-c,d]pyrene	0%	0%	0%	0%
Dibenzo[a,h]anthracene	0%	0%	0%	0%
Benzo[g,h,i]perylene	0%	0%	0%	0%
% Total	100%	100%	100%	100%

Table 5.11 The inhalation unit risk (IUR), 95% upper confidence limits (UCL, µg/m^3) and the risk probability estimates for select PAHs calculated from USEPA's software, ProUCL.

		PAH 95% UCL (µg/m^3)			Cancer Risk Probability Estimates		
PAH	**IUR (µg/m3)**	**2011**	**2014 Downwind**	**2014 Upwind**	**2011**	**2014 Downwind**	**2014 Upwind**
Naphthalene	3.40E-05	0.0567	0.0234	0.0034	1.93E-06	7.94E-07	1.16E-07
Acenaphthylene	3.40E-05	<IDL	0.0005	<IDL	<IDL	1.53E-08	<IDL
Acenaphthene	1.10E-06	0.0023	0.0017	<IDL	2.56E-09	1.90E-09	<IDL
Fluorene	1.10E-06	0.0088	0.0067	0.0029	9.67E-09	7.37E-09	3.17E-09
Phenanthrene	1.10E-06	0.0964	0.0207	0.0046	1.06E-07	2.27E-08	5.06E-09
Anthracene	1.10E-05	0.0033	0.0026	0.0005	3.63E-08	2.81E-08	5.91E-09
Fluoranthene	1.10E-06	0.0049	0.0034	0.0037	5.39E-09	3.71E-09	4.04E-09
Pyrene	1.10E-06	0.0027	0.0027	0.0023	2.95E-09	2.93E-09	2.54E-09
Benzo(a)anthracene	1.10E-04	0.0005	0.0003	0.0002	5.87E-08	3.18E-08	2.33E-08
Chrysene	1.10E-05	0.0005	0.0005	0.0003	6.03E-09	5.09E-09	3.39E-09
Benzo(b)fluoranthene	1.10E-04	<IDL	0.0002	0.0002	<IDL	2.72E-08	2.44E-08
Benzo(k)fluoranthene	1.10E-04	<IDL	0.0001	0.0001	<IDL	6.07E-09	6.25E-09
Benzo(a)pyrene	1.10E-03	<IDL	0.0001	0.0002	<IDL	1.25E-07	1.74E-07
Indeno(1,2,3-c,d)pyrene	1.10E-04	<IDL	<IDL	<IDL	<IDL	<IDL	<IDL
Dibenzo(a,h)anthracene	1.20E-03	0.0004	<IDL	<IDL	5.30E-07	<IDL	<IDL
Benzo(g,h,i)perylene	1.10E-05	<IDL	0.0001	0.0001	<IDL	1.25E-09	1.27E-09
∑PAH		0.1766	0.0628	0.0185	2.69E-06	1.07E-06	3.69E-07
∑Carcinogenic PAHs		0.0015	0.0013	0.0011	5.95E-07	1.97E-07	2.32E-07

Table 5.12 Incidences of common diseases by age group in Curaçao.

Population by most common diseases	Age Groups											
	0-14				15-24				25-44			
	Male	Female	Total	%	Male	Female	Total	%	Male	Female	Total	%
High blood pressure	50	48	98	3	65	114	179	7	959	1615	2574	32
Diabetes	17	35	52	2	37	46	83	3	314	415	729	9
Glaucoma/pressure in the eyes	16	18	34	1	34	30	64	3	138	238	376	5
Asthma/chronic bronchitis/CARA	1157	768	1925	60	550	582	1132	46	492	1178	1670	21
Cancer	10	11	21	1	13	5	18	1	15	54	69	1
Sickle cell	72	87	159	5	76	101	177	7	120	257	377	5
Heart problems	114	106	220	7	92	109	201	8	187	313	500	6
Consequences of heart attack	6	6	12	0	8	9	17	1	26	25	51	1
Consequences of brain hemorrhage	8	6	14	0	8	4	12	0	17	33	50	1
Serious kidney problems	16	10	26	1	9	10	19	1	74	83	157	2
Dementia/Alzheimer	3	2	5	0	5	0	5	0	17	7	24	0
Other	371	293	664	21	224	338	562	23	528	833	1361	17
Total	1840	1390	3230	100	1121	1348	2469	100	2887	5051	7938	100

Population by most common diseases	45-64				65+				Total			
	Male	Female	Total	%	Male	Female	Total	%	Male	Female	Total	%
High blood pressure	3863	6583	10446	43	2770	5095	7865	36	7707	13455	21162	35
Diabetes	1747	2697	4444	18	1627	2701	4328	20	3742	5894	9636	16
Glaucoma/pressure in the eyes	656	991	1647	7	911	1406	2317	10	1755	2683	4438	7
Asthma/chronic bronchitis/CARA	457	1068	1525	6	231	376	607	3	2887	3972	6859	11
Cancer	110	278	388	2	197	182	379	2	345	530	875	1
Sickle cell	139	327	466	2	54	86	140	1	461	858	1319	2
Heart problems	780	1125	1905	8	1041	1430	2471	11	2214	3083	5297	9
Consequences of heart attack	223	189	412	2	325	275	600	3	588	504	1092	2
Consequences of brain hemorrhage	225	169	394	2	341	364	705	3	599	576	1175	2
Serious kidney problems	221	238	459	2	199	225	424	2	519	566	1085	2
Dementia/Alzheimer	60	59	119	0	323	603	926	4	408	671	1079	2
Other	823	1308	2131	9	474	844	1318	6	2420	3616	6036	10
Total	9304	15032	24336	100	8493	13587	22080	100	23645	36408	60053	100

Source: Central Bureau of Statistics Curaçao (Census 2011)[192].

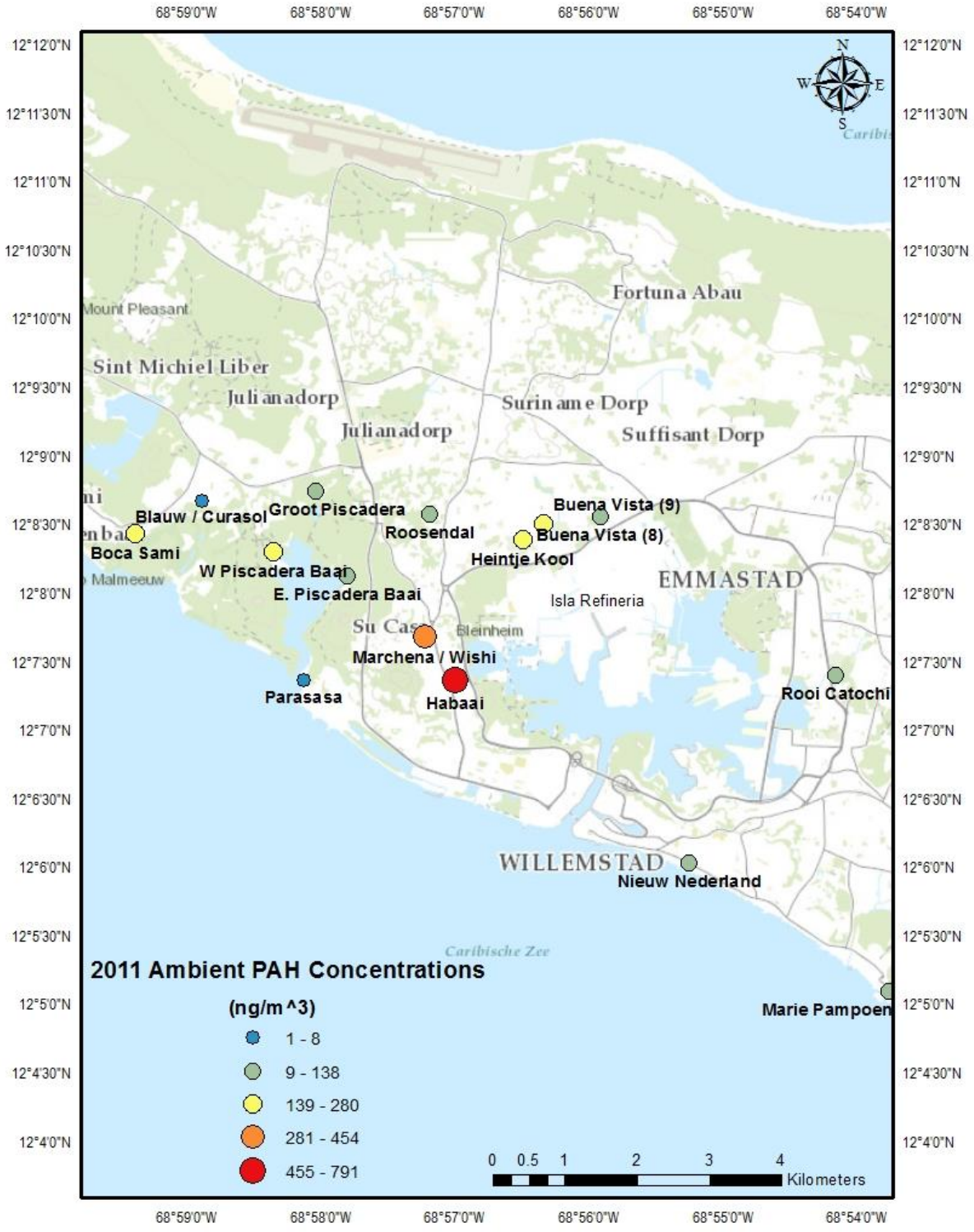


Figure 5.1 Calculated ambient PAH concentrations (ng/m^3) in Curaçao from the 2011 sampling event.

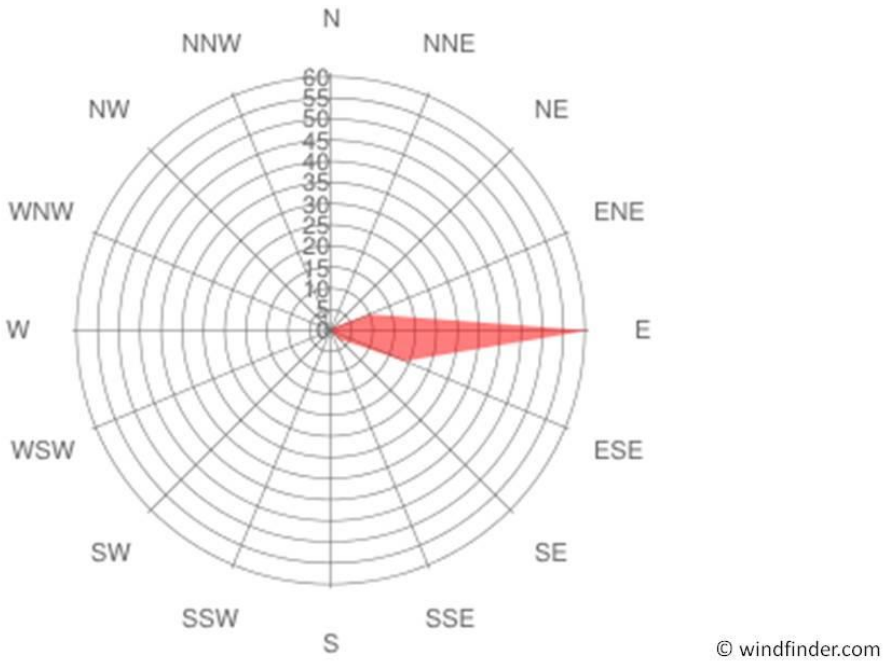


Figure 5.2 Wind rose illustrating the percent wind direction distribution in Curaçao based on observations from 2000 through 2014 (http://www.windfinder.com/windstatistics/Curaçao).

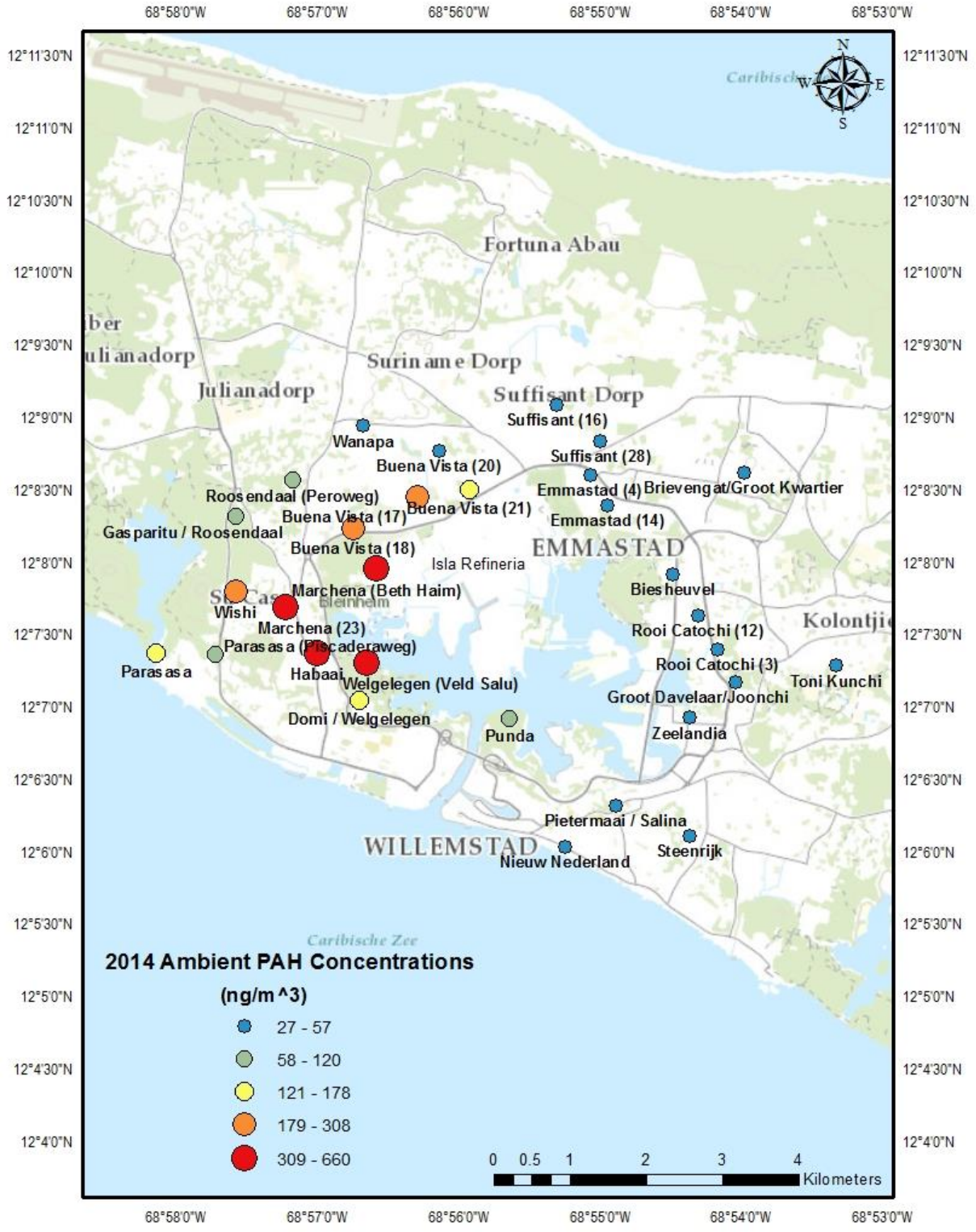


Figure 5.3 Calculated ambient PAH concentrations (ng/m^3) in Curaçao during the 2014 sampling event.

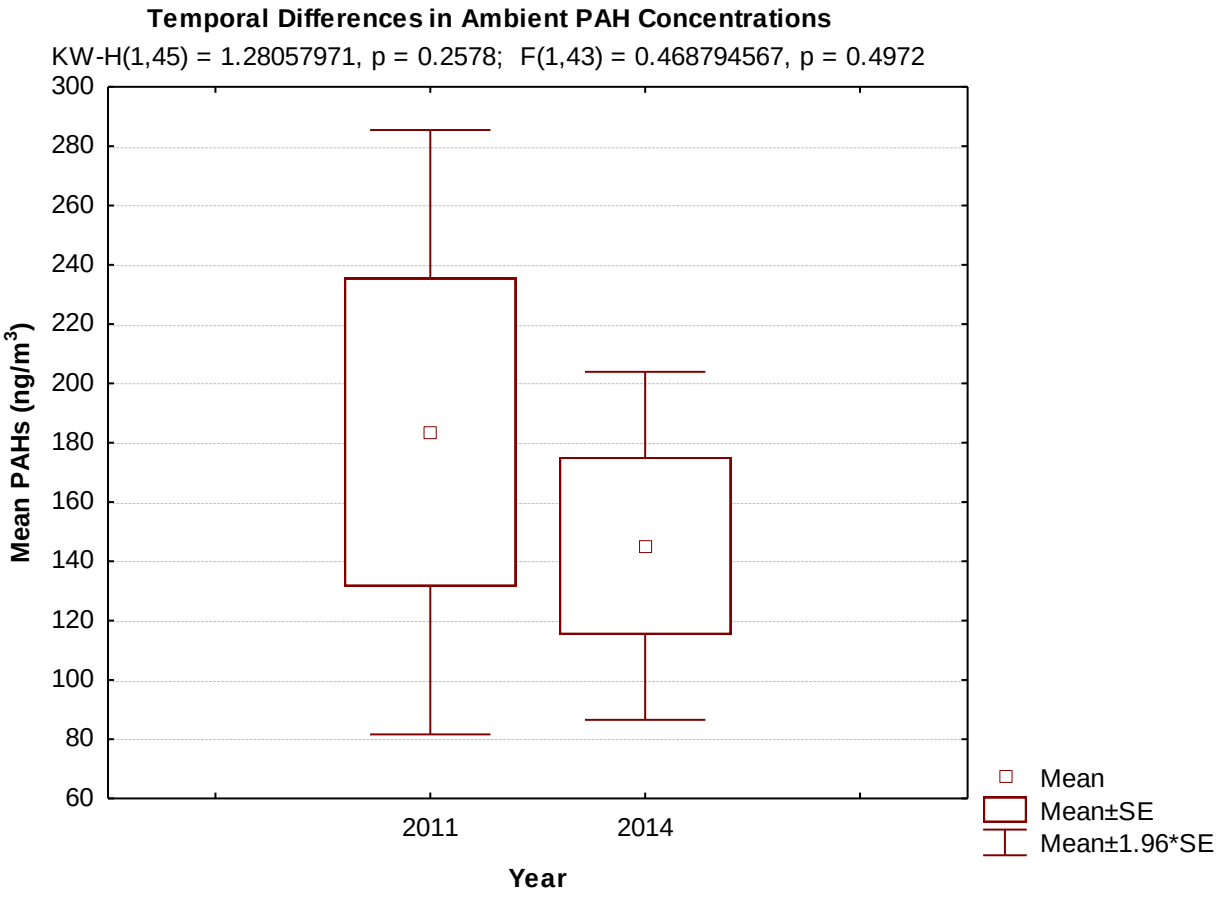


Figure 5.4 Box plot of the mean ambient PAH concentrations (ng/m^3) in Curaçao during 2011 and 2014. No statistical differences (p=0.49) were observed between the two years.

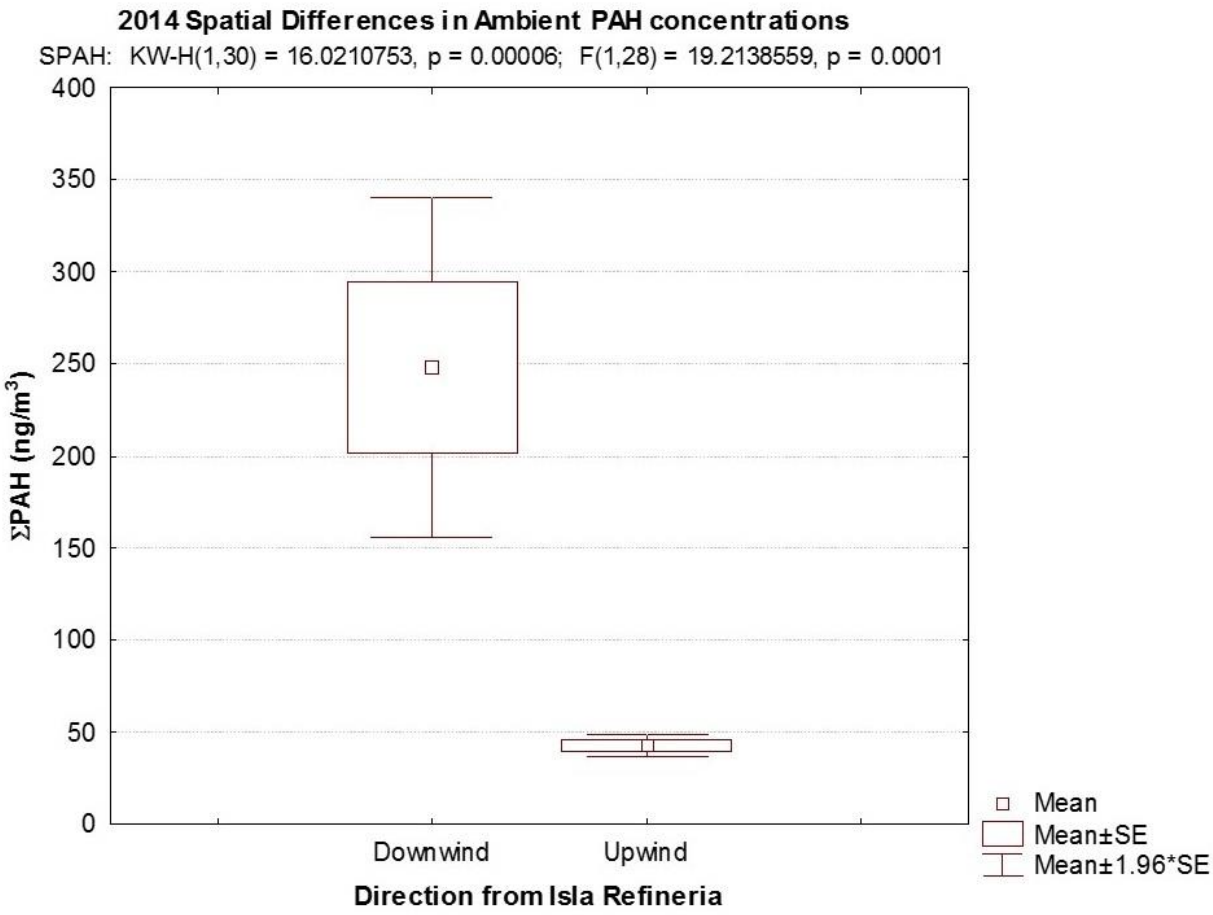


Figure 5.5 Box plot of the spatial differences observed between ambient PAH concentrations (ng/m^3) at sites located downwind and upwind of the refinery in Curaçao during 2014. Ambient PAH concentrations were significantly higher (p=0.00006) at the sites downwind of the refinery compared to those upwind.

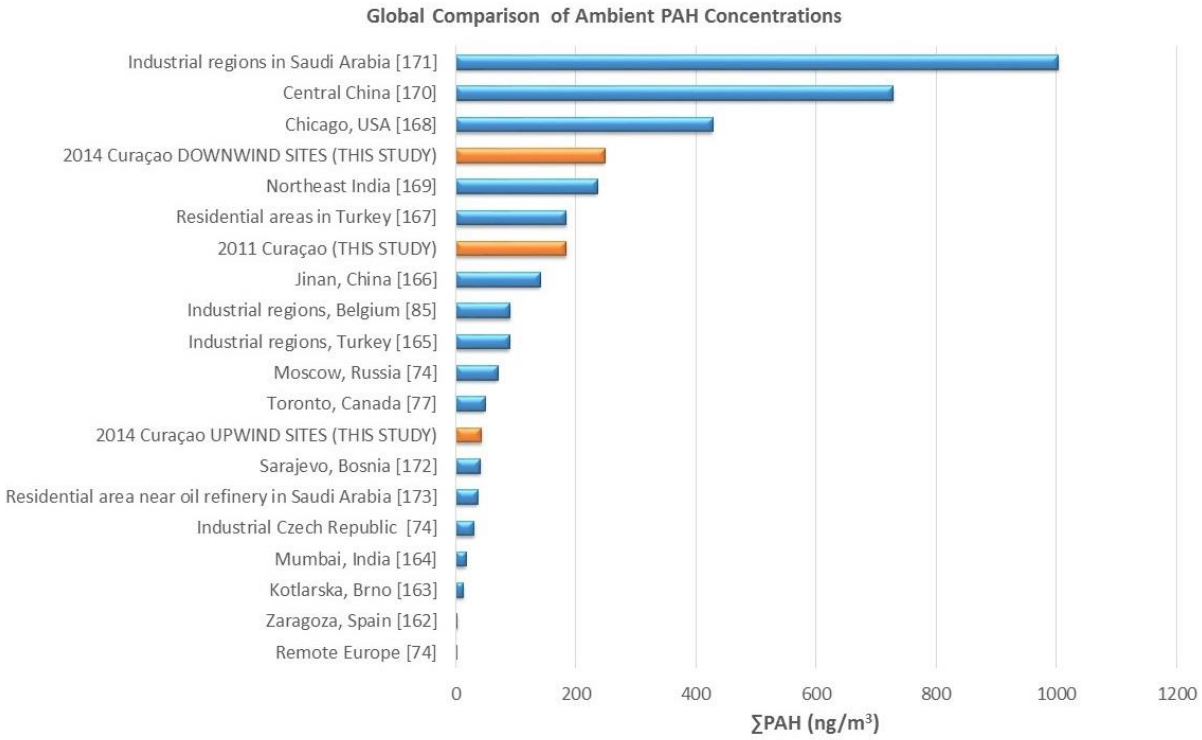


Figure 5.6 Global comparison of ambient PAH concentrations. Concentrations measured in this study are shown in orange.

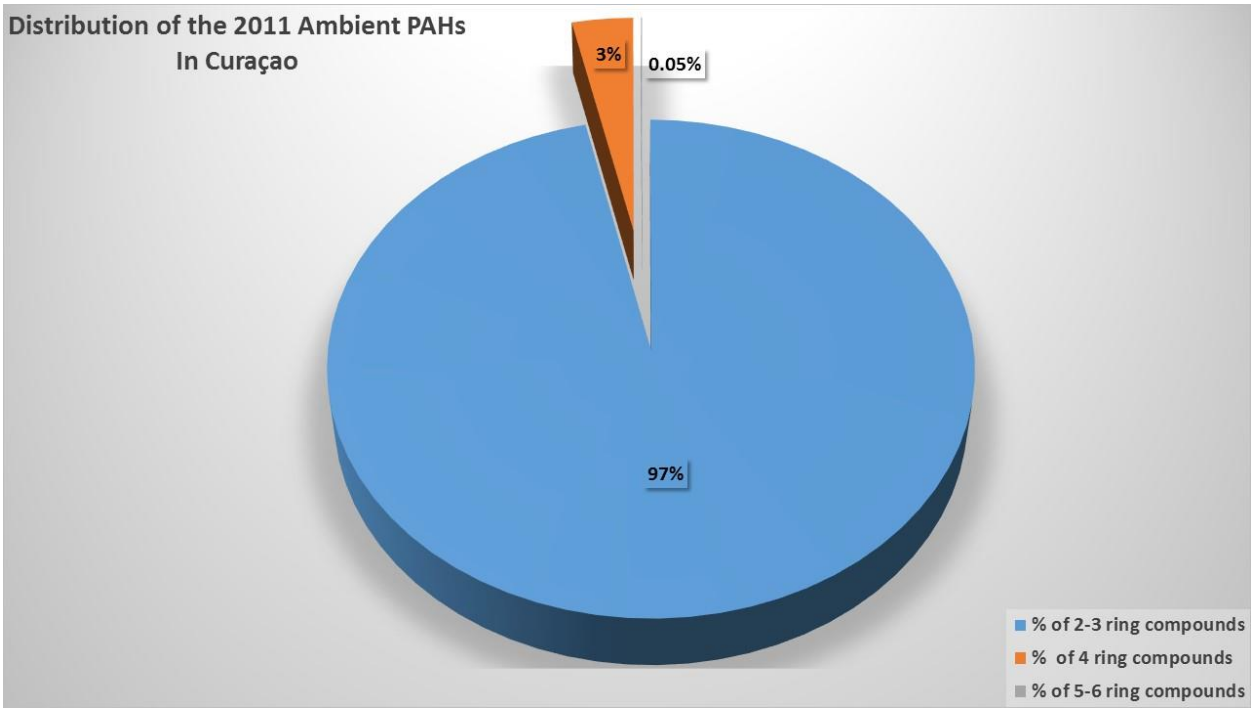


Figure 5.7 Percentages of mass concentrations for the parent PAH compounds for the 2011 sites sampled in Curaçao.

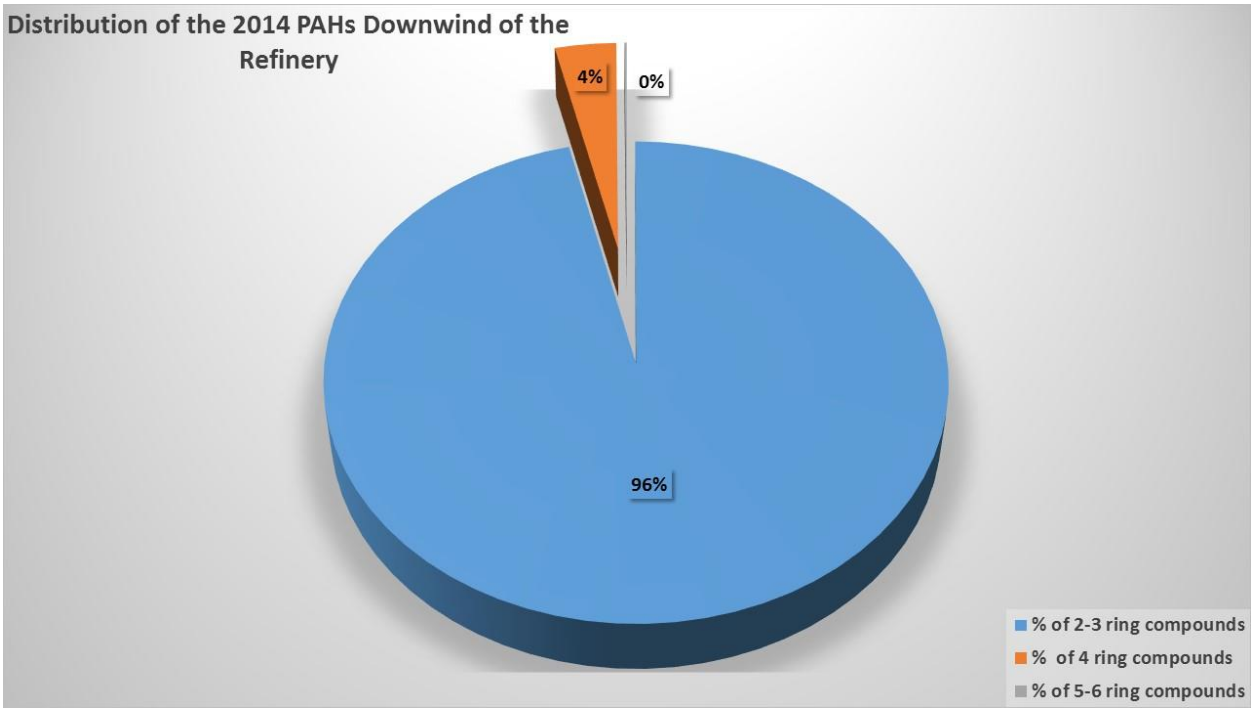


Figure 5.8 Percentage of mass concentrations for the parent PAH compounds in the 2014 samples downwind from Isla Refineriá in Willemstad, Curaçao.

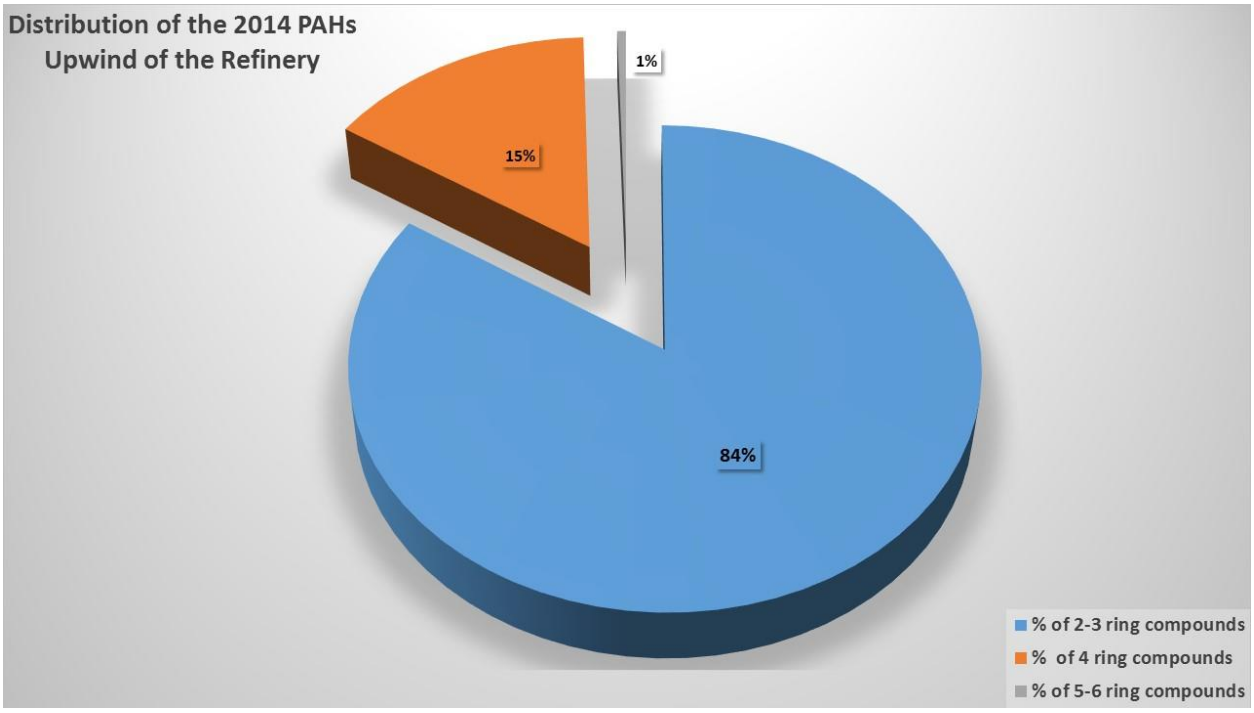


Figure 5.9 Percentages of the mass concentrations for the parent PAH compounds in the 2014 samples upwind of Isla Refineriá in Willemstad, Curaçao.

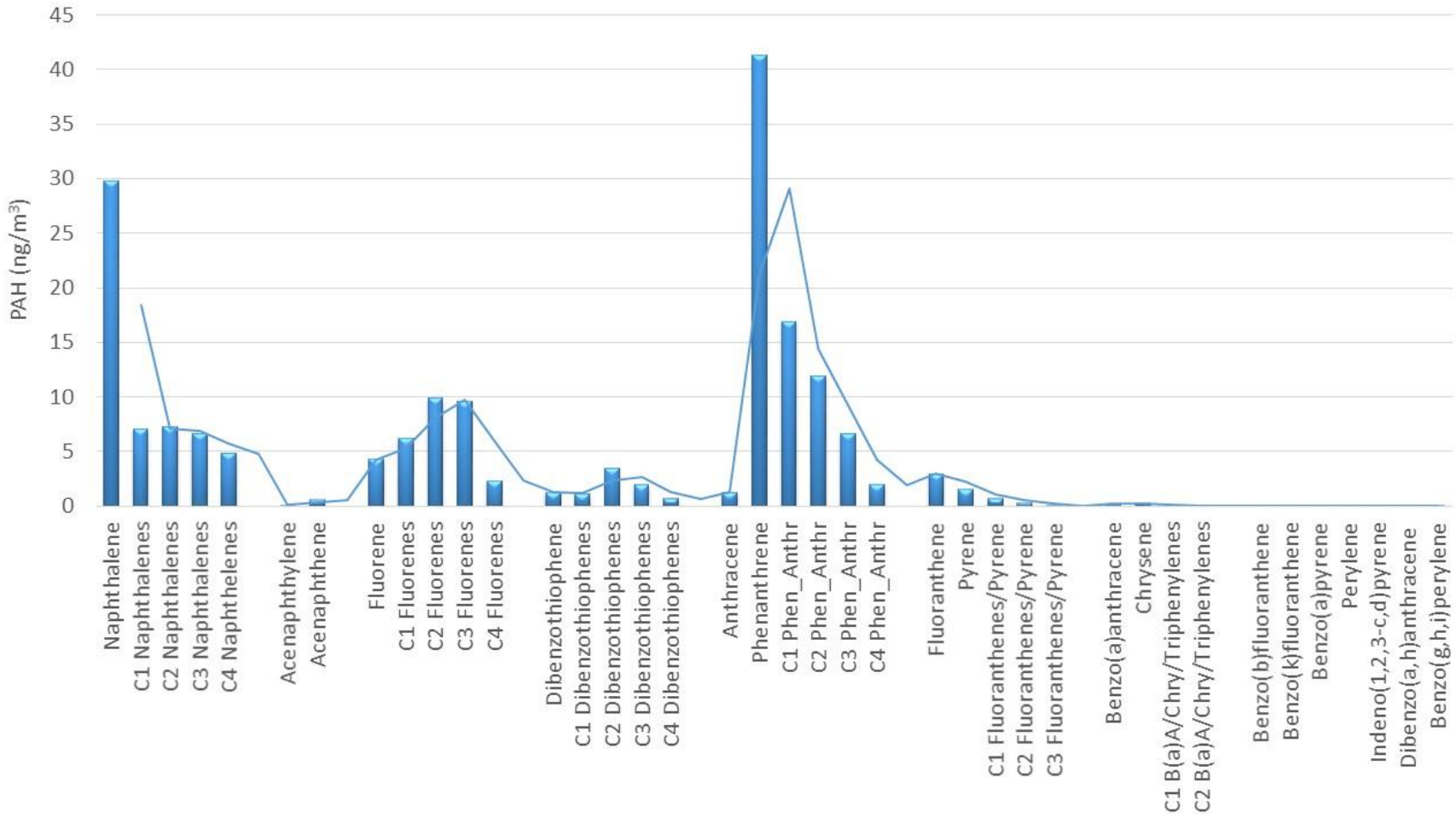


Figure 5.10 The 2011 PAH distribution profiles for the samples collected in Curaçao.

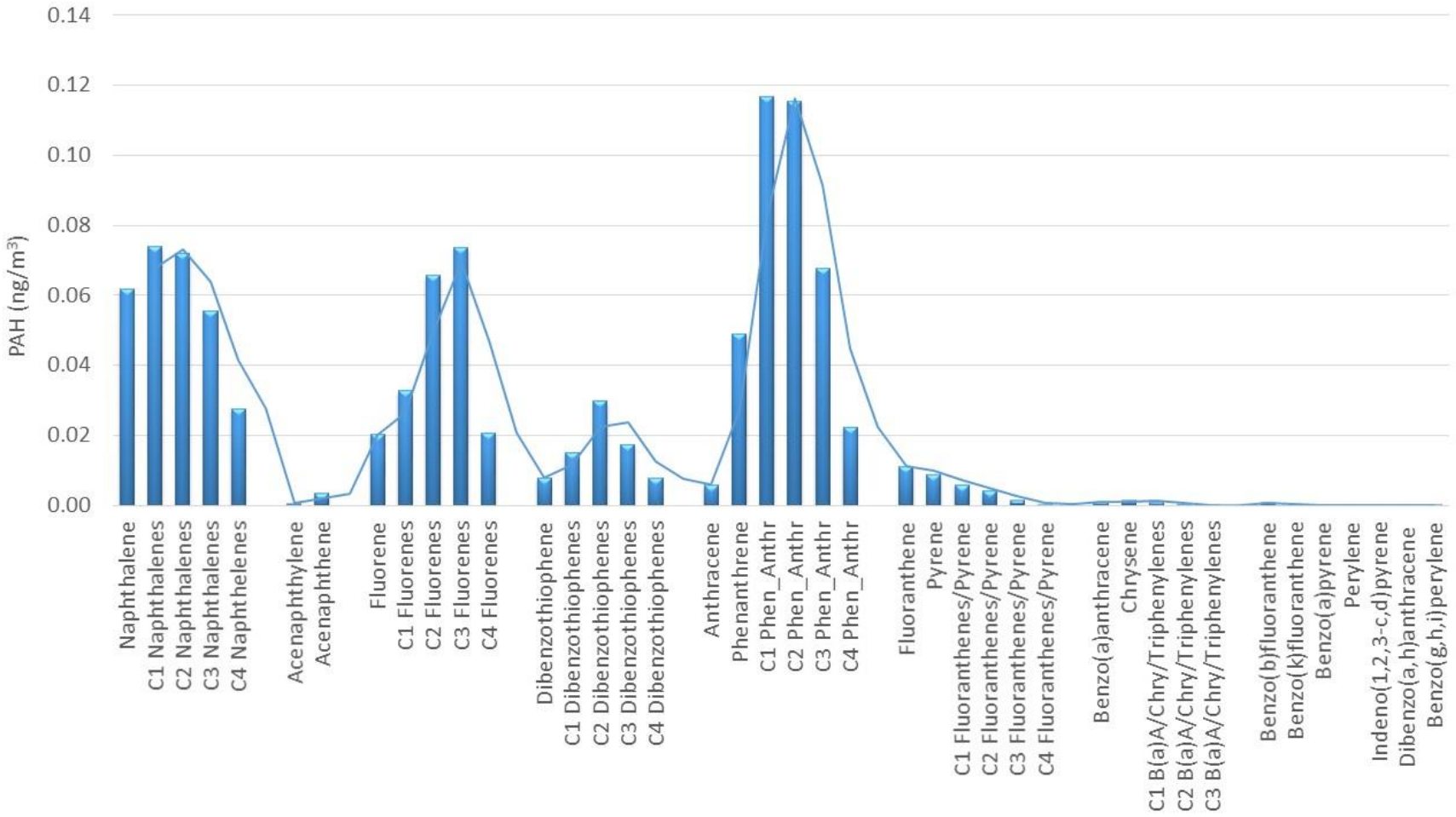


Figure 5.11 The 2014 PAH distribution profiles for the samples collected downwind of Isla Refineriá in Curaçao.

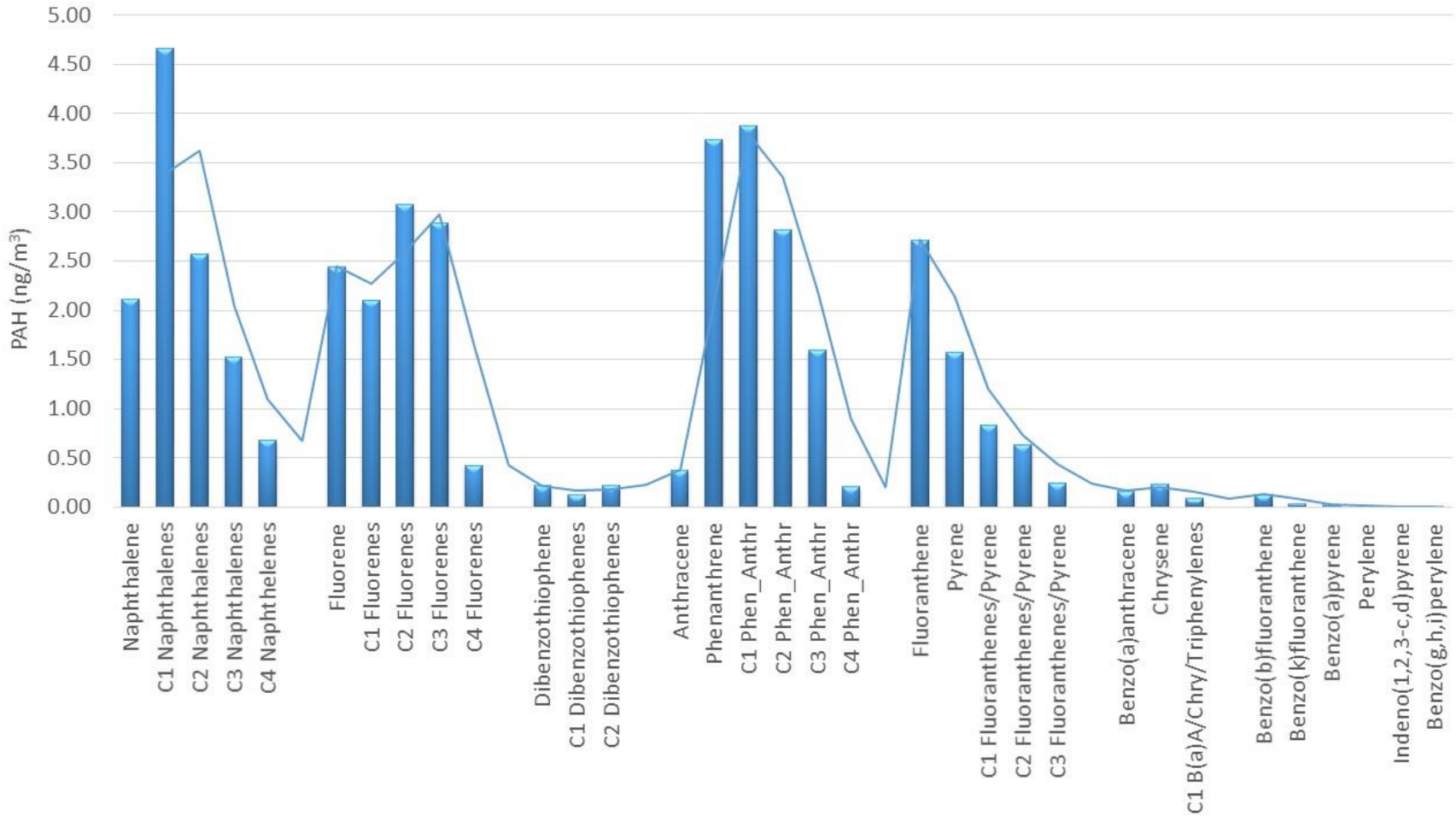


Figure 5.12 The 2014 PAH distribution profiles for the samples collected upwind of Isla Refineriá in Curaçao.

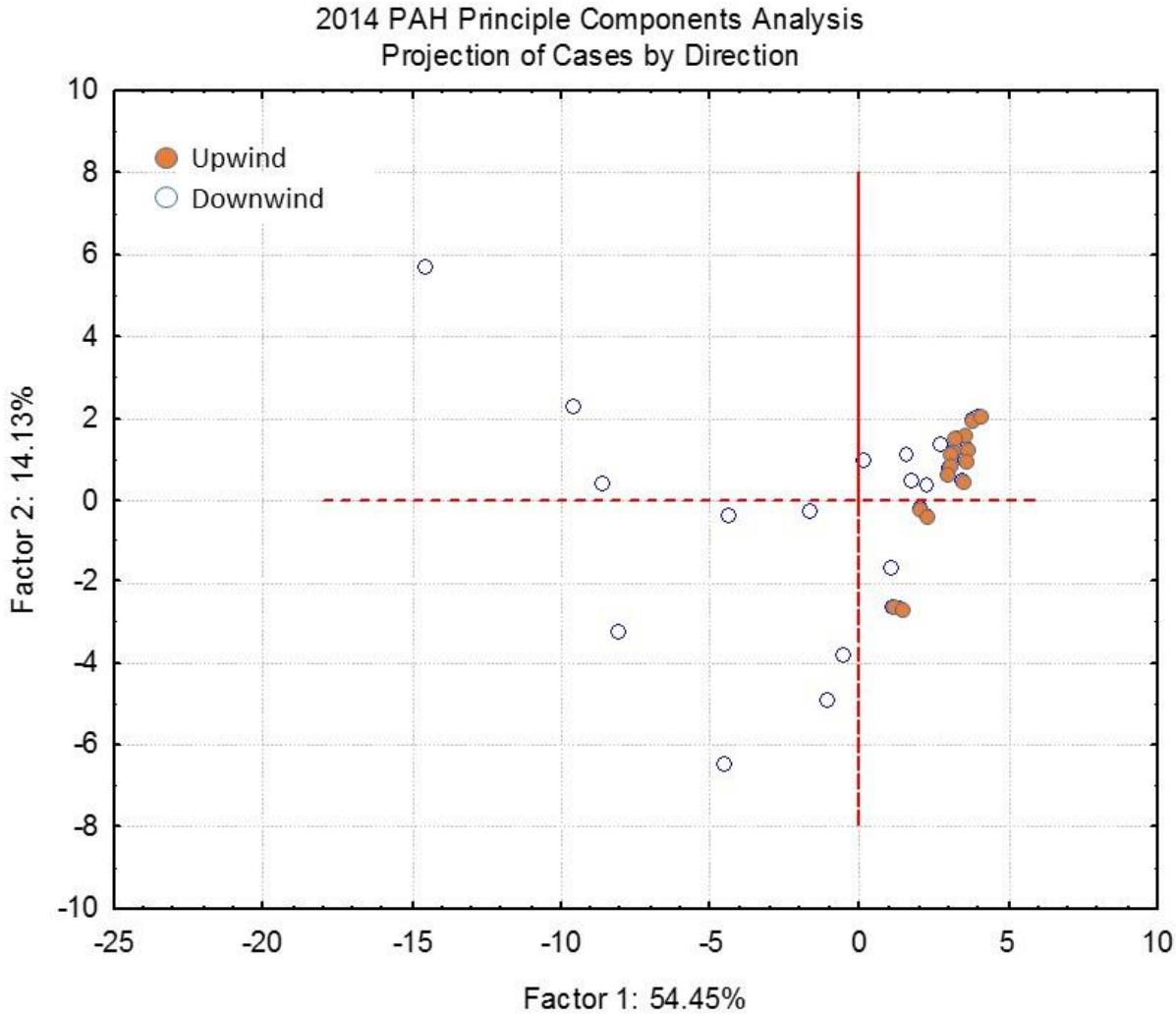


Figure 5.13 Principle components analysis of the 2014 ambient PAH concentrations measured in Curaçao.

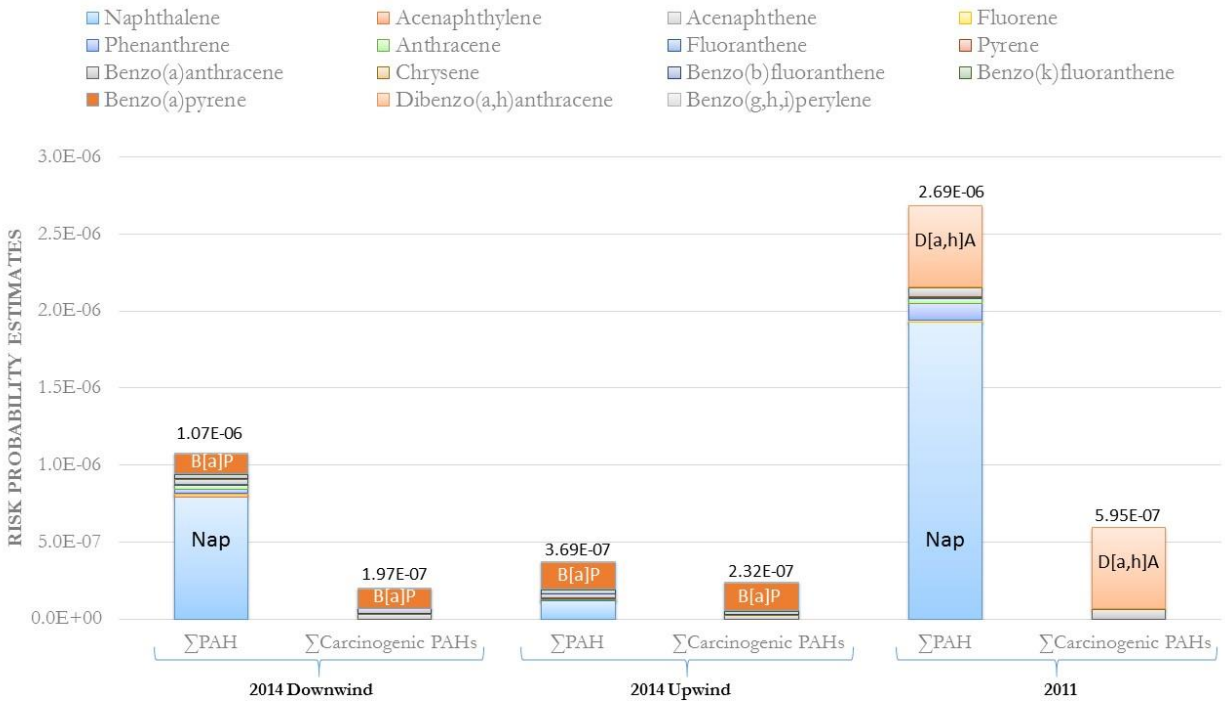


Figure 5.14 Lifetime cumulative cancer risk probability estimates for all PAHs and carcinogenic PAHs measured in ambient air in Curaçao during 2011 and 2014 (upwind and downwind sites). Estimates were calculated using the 95% UCL for the ambient PAH concentrations (μg/m^3). The USEPA's upper bound acceptable risk is 1.0E-4.

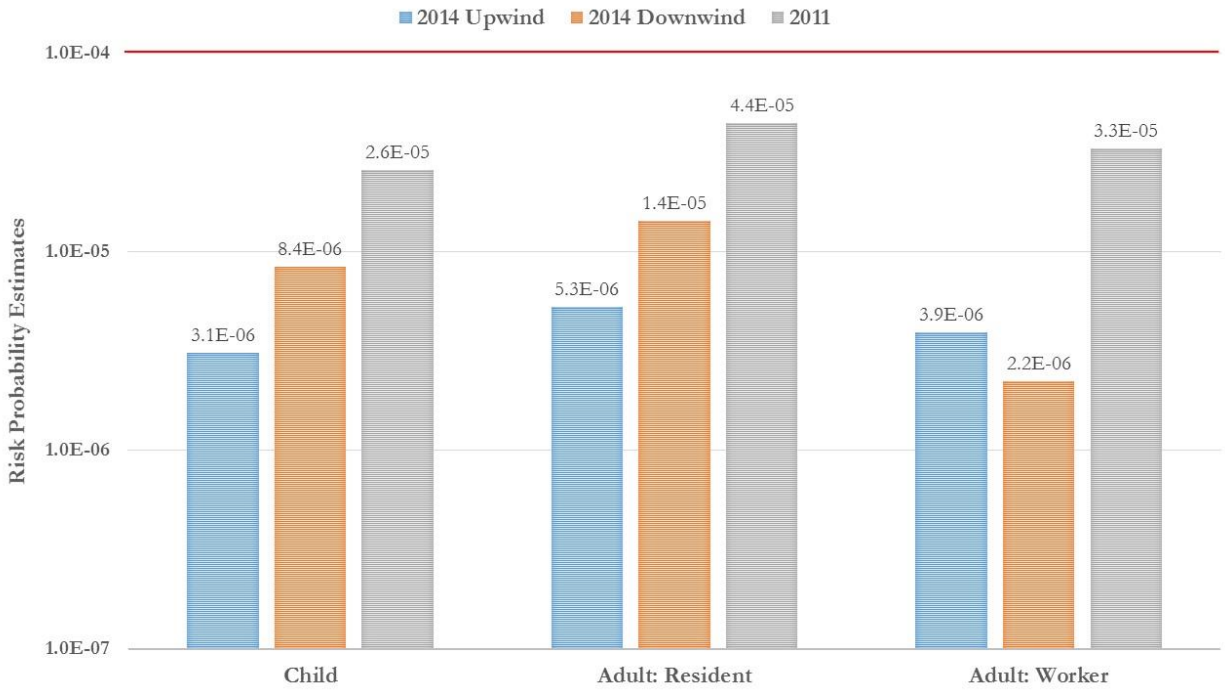


Figure 5.15 Cumulative lifetime risk probability estimates for children, adult residents and adult workers using the 95% upper confidence limits for the 2011 and 2014 ambient PAH concentrations ($\mu g/m^3$). The USEPA's upper bound acceptable risk is marked with the red line at 1.0E-4.

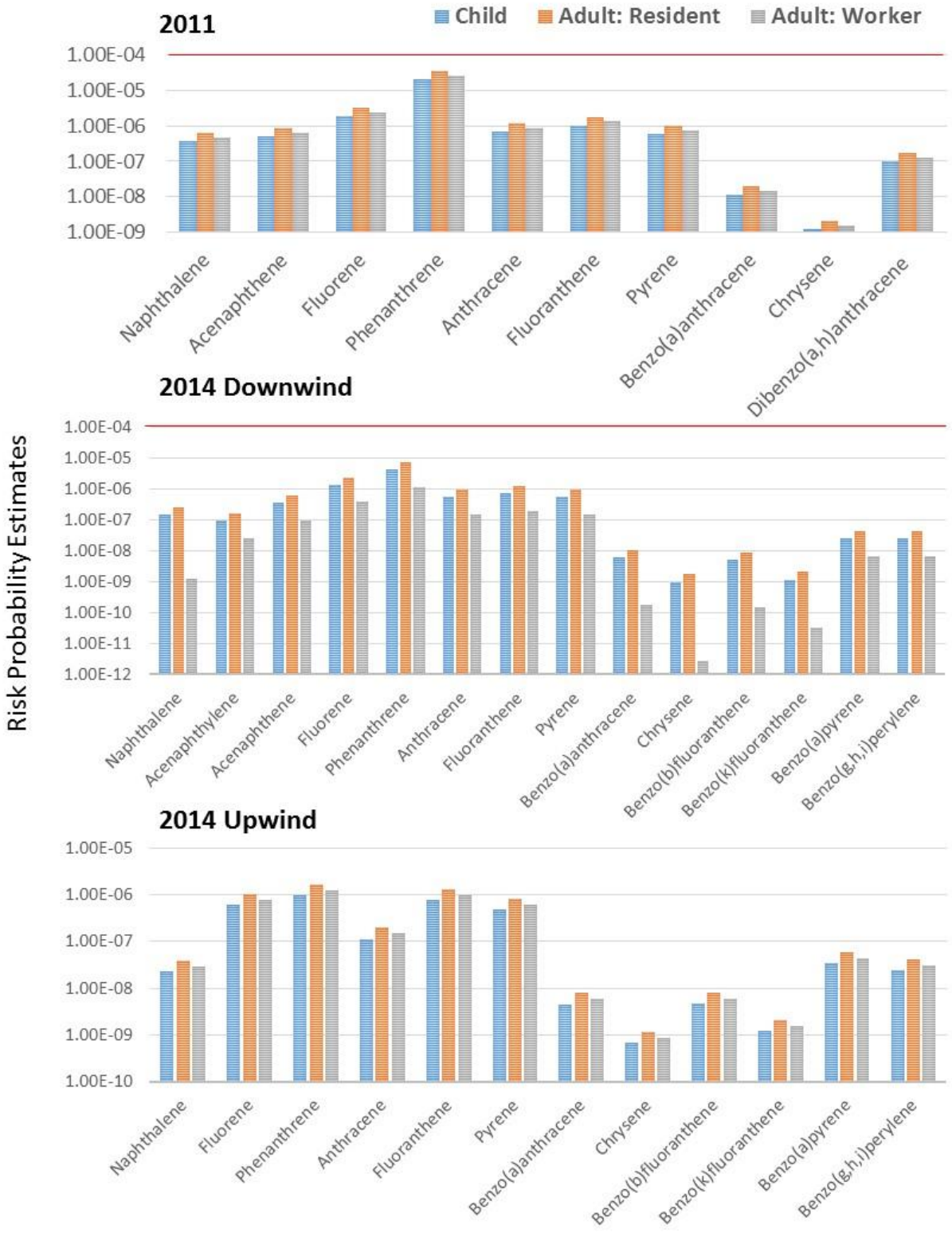


Figure 5.16 Compound specific cumulative lifetime risk probability estimates for children, adult residents and adult workers using the 95% upper confidence limits for 2011 and 2014 downwind and upwind ambient PAH concentrations ($\mu g/m^3$). The USEPA's upper bound acceptable risk is marked with the red line at 1.0E-4.

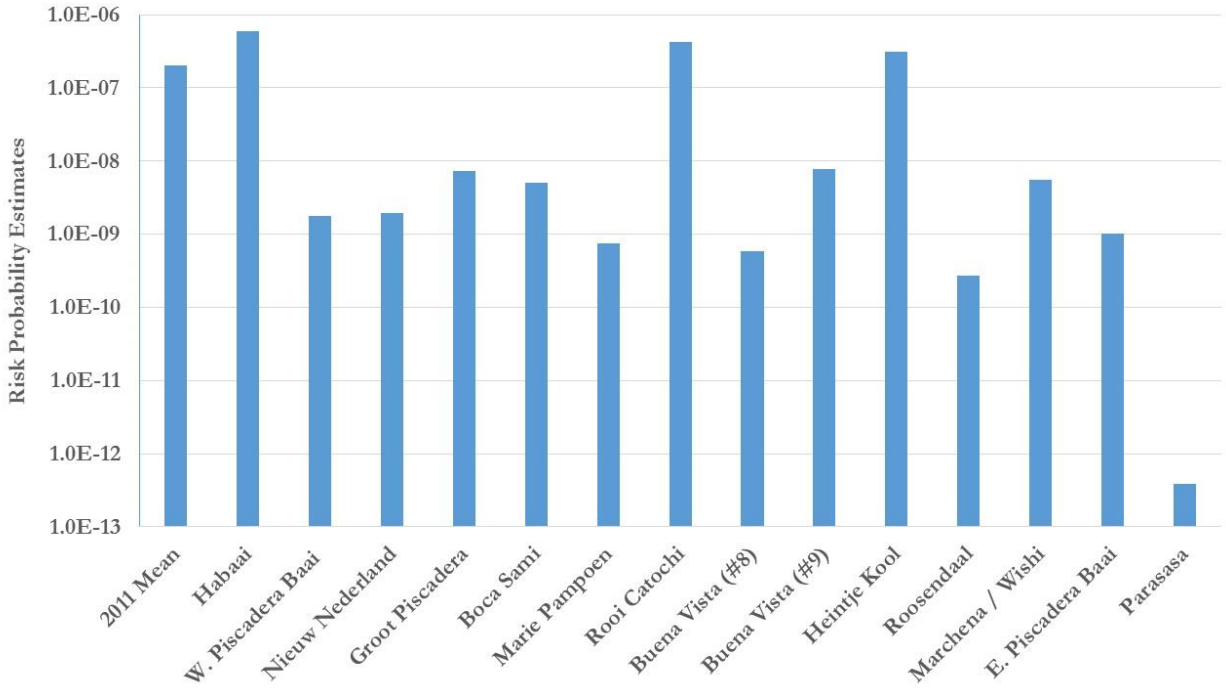


Figure 5.17 Site specific cancer risk probability estimates using the 2011 potency equivalent PAH concentrations (mg-PEQ/m^3).

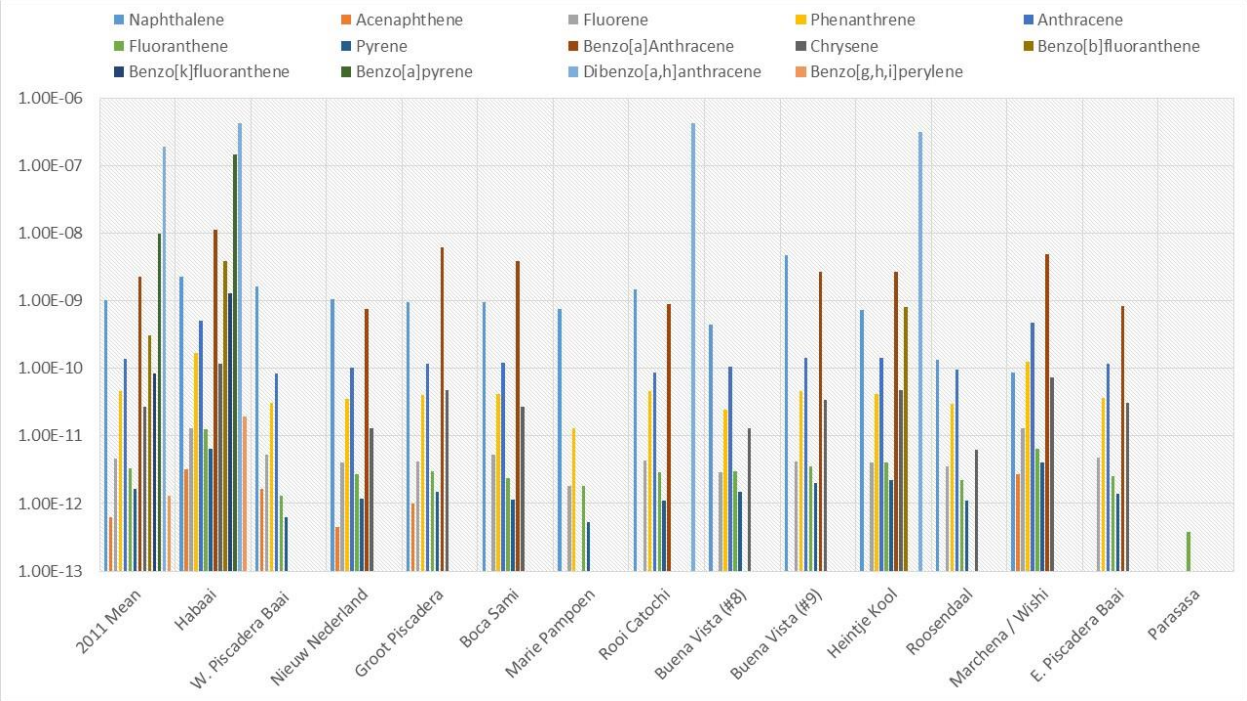


Figure 5.18 Compound specific cancer risk probability estimates for each site using the 2011 potency equivalent PAH concentrations (mg-PEQ/m^3).

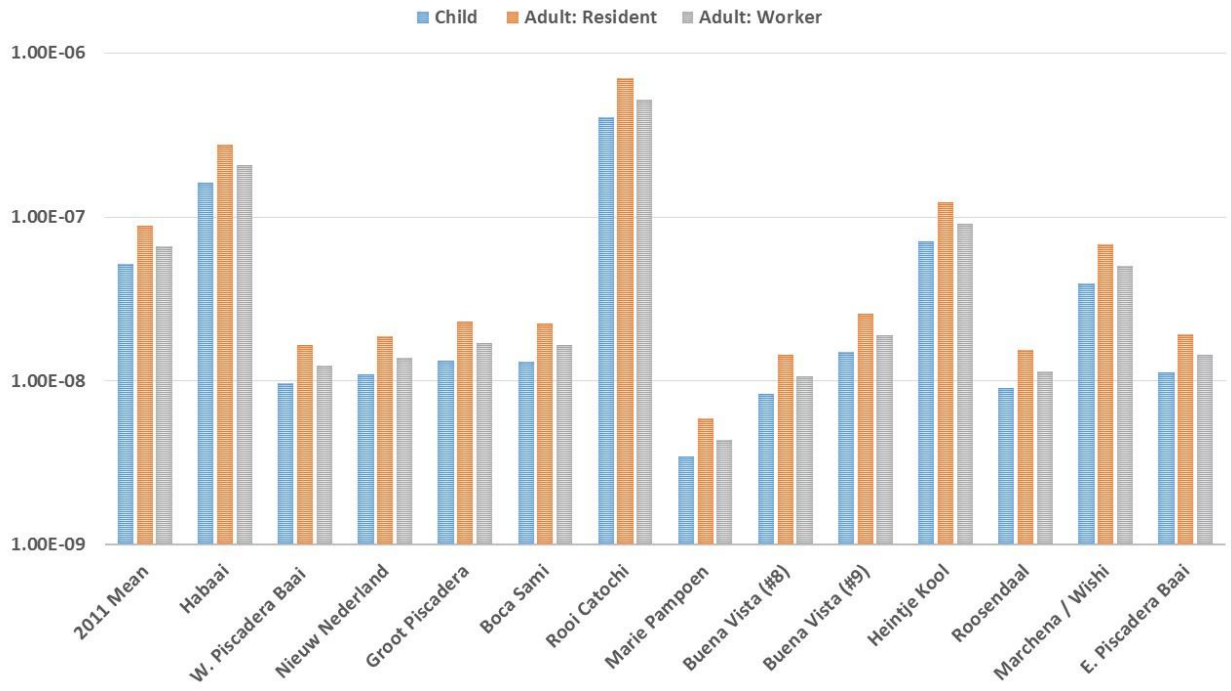


Figure 5.19 Site specific cumulative lifetime risk probability estimates for children, adult residents and adult workers using the 2011 potency equivalent concentrations (mg-PEQ/m^3).

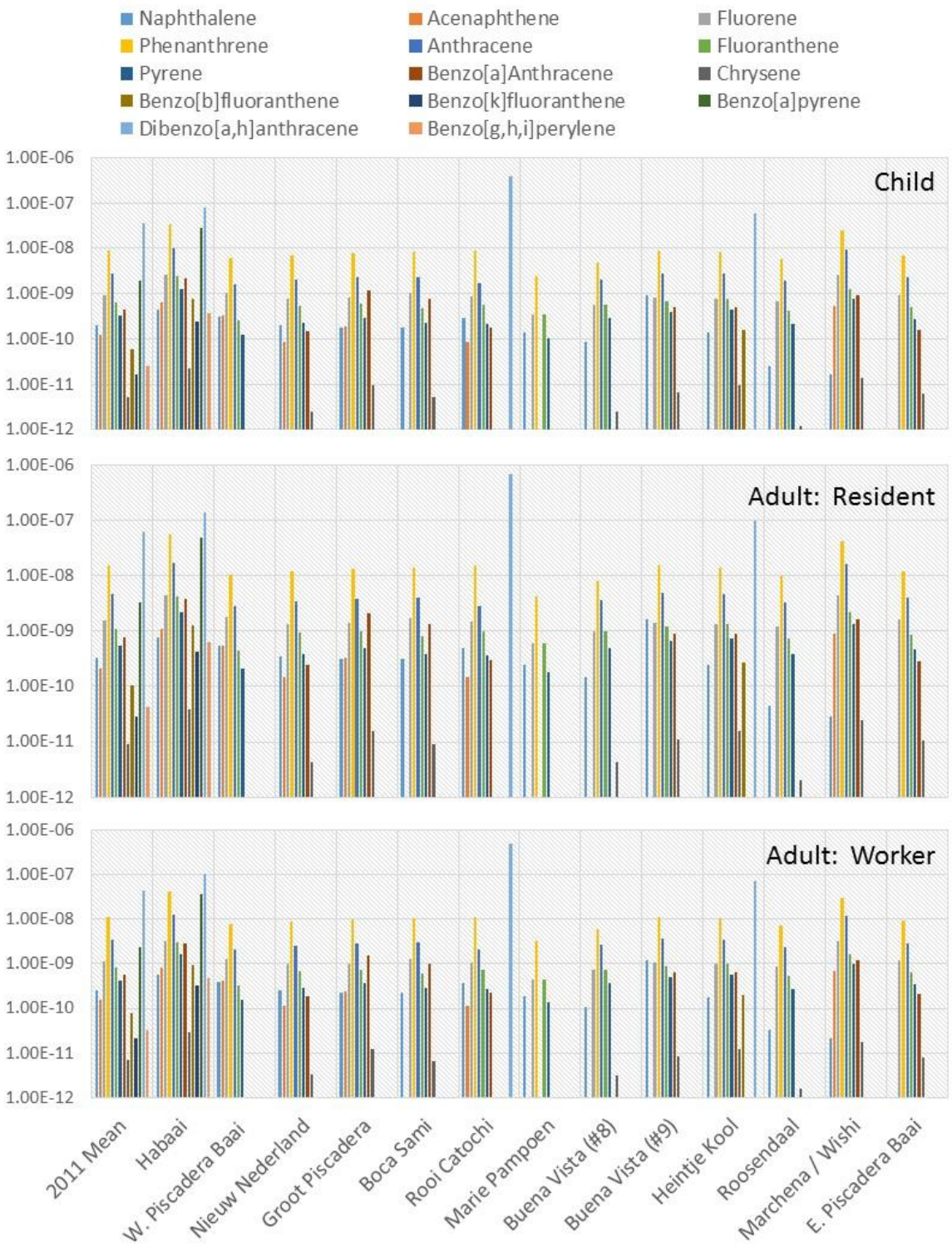


Figure 5.20 Compound specific cumulative lifetime risk probability estimates for children, adult residents and adult workers for each site using the 2011 potency equivalent concentrations (mg-PEQ/m^3).

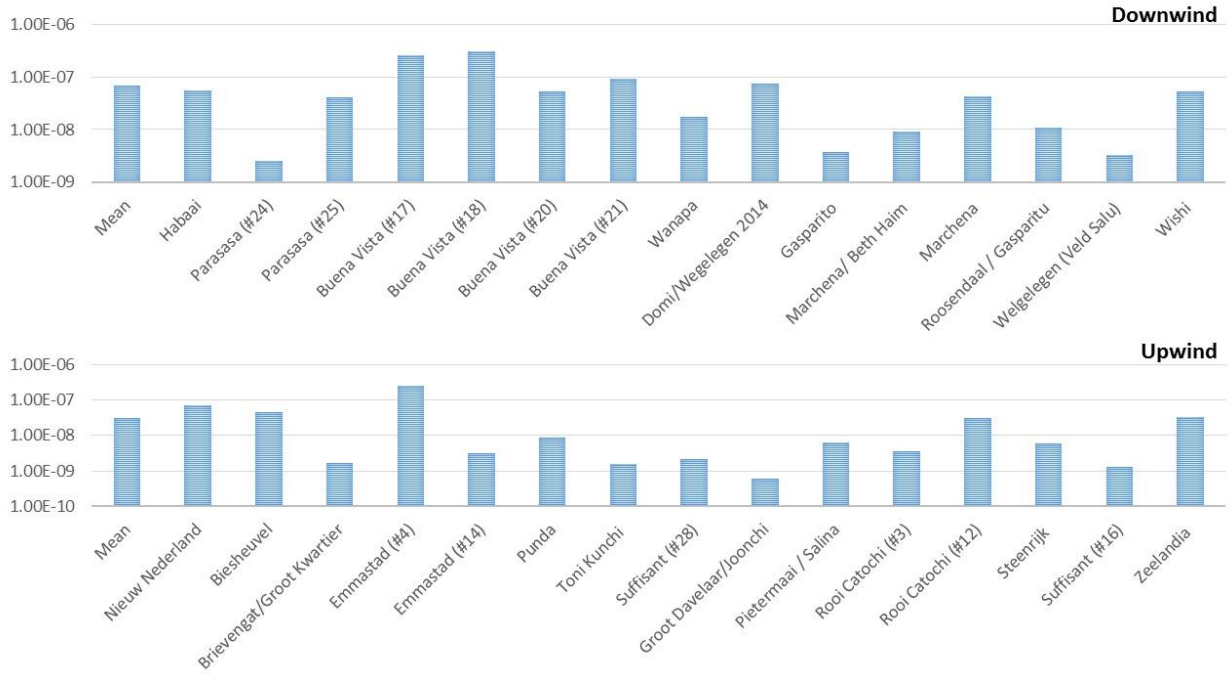


Figure 5.21 Site specific total cancer risk probability estimates for the 2014 downwind and upwind sites using the potency equivalent concentrations (mg-PEQ/m^3).

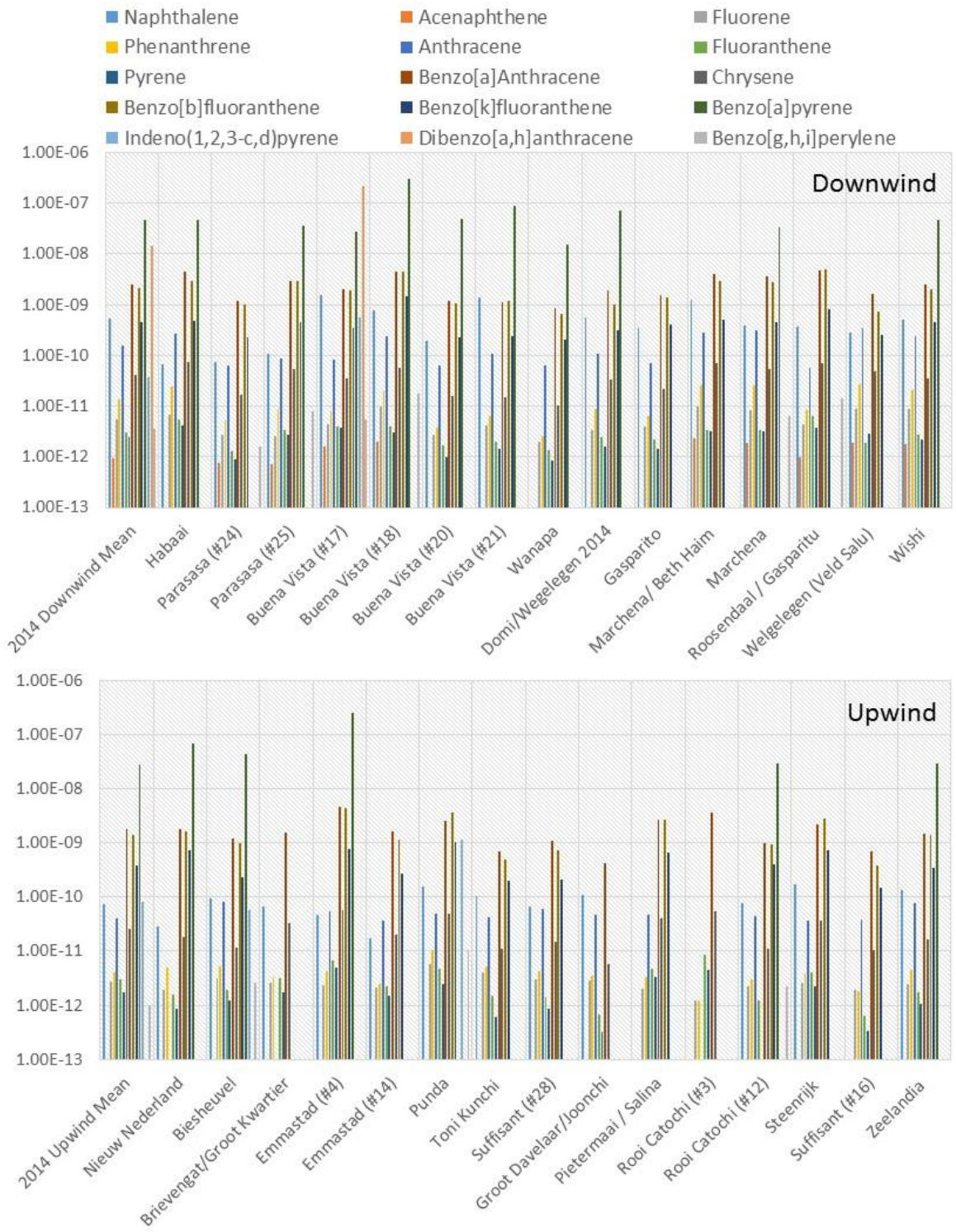


Figure 5.22 Compound and site specific cancer risk probability estimates for the 2014 downwind and upwind sites using potency equivalent concentrations (mg-PEQ/m^3).

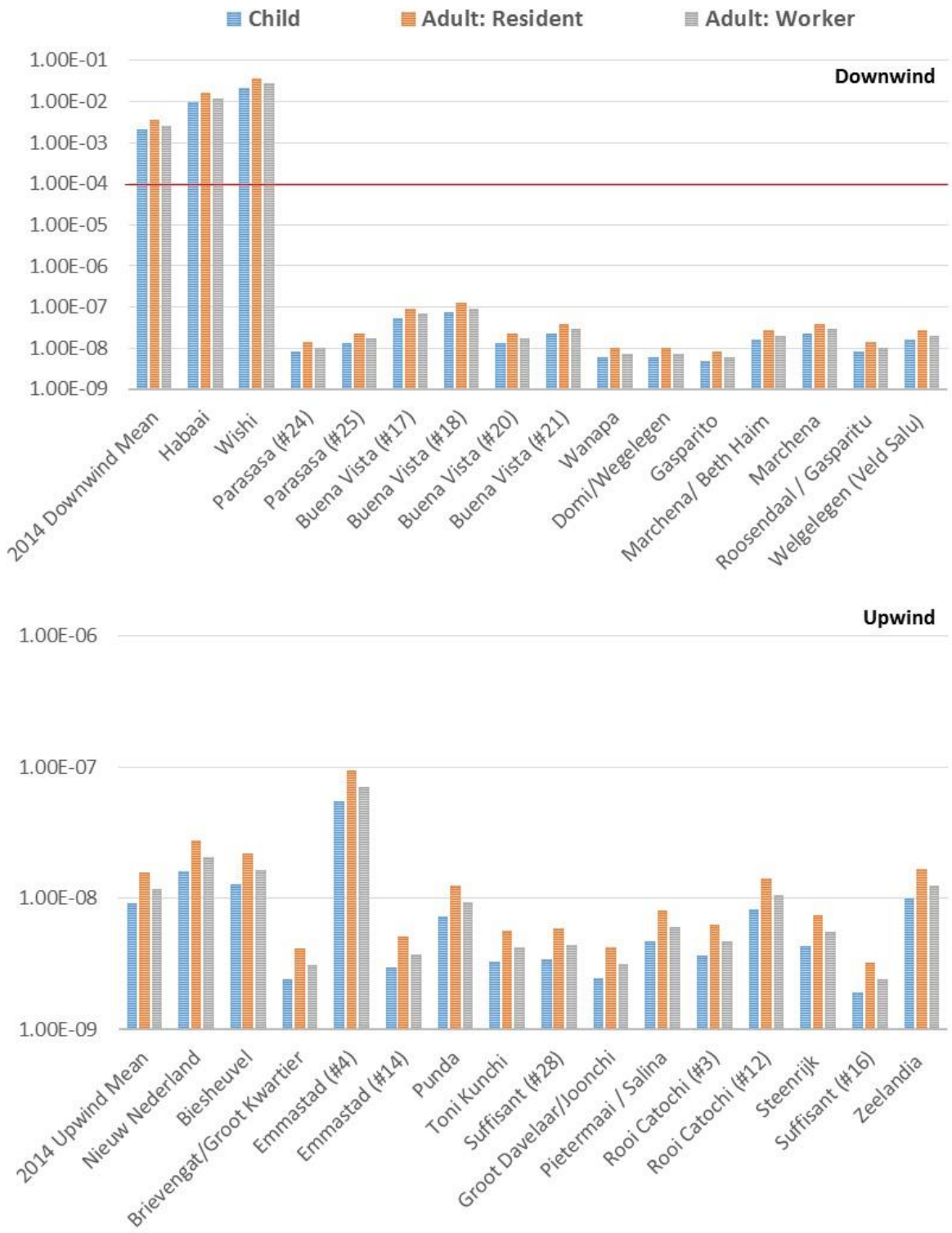


Figure 5.23 Site specific cumulative lifetime risk probability estimates for children, adult residents and adult workers for the 2014 specific potency equivalent concentrations (mg-PEQ/m^3). The USEPA's upper bound acceptable risk is marked with the red line at 1.0E-4.

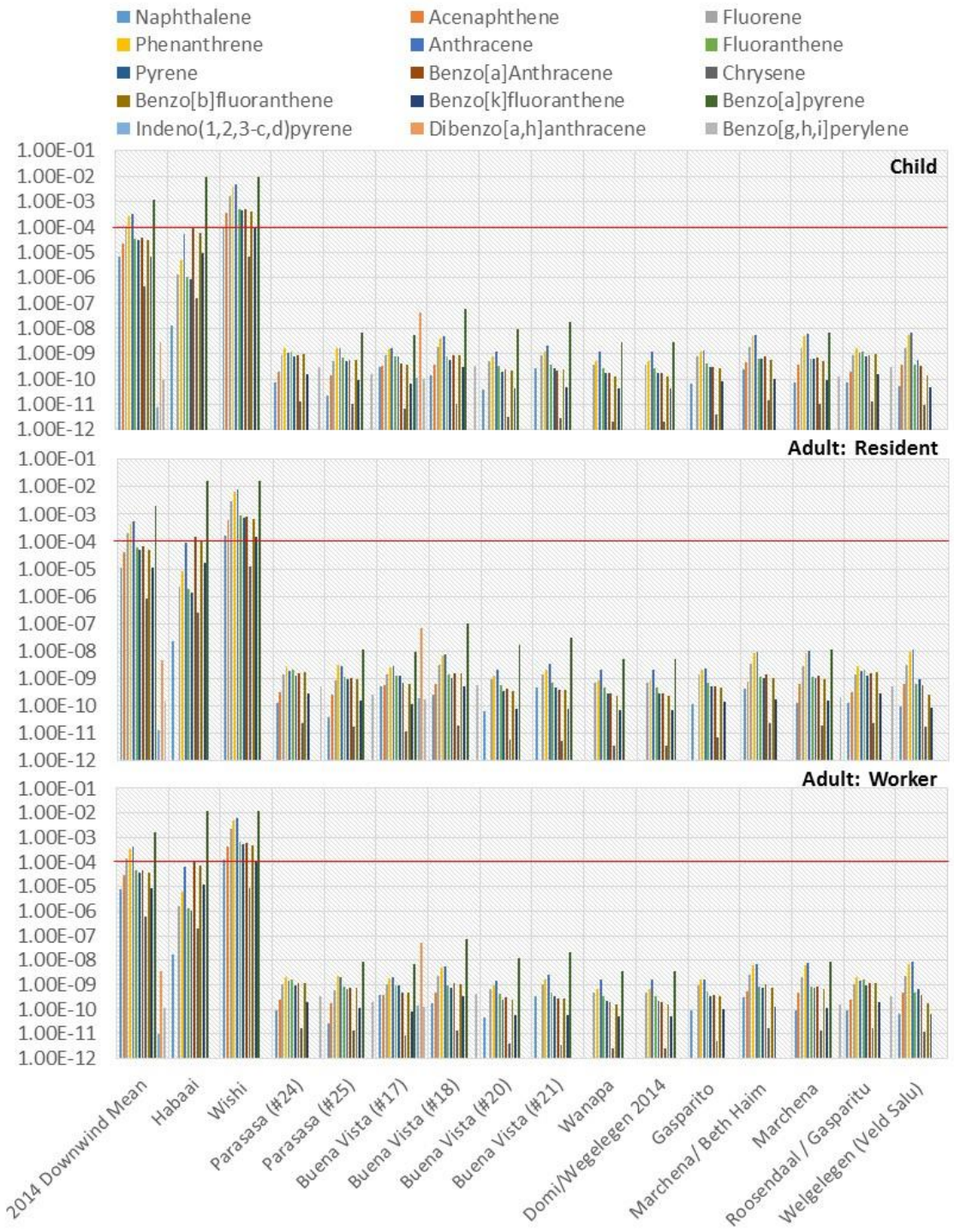


Figure 5.24 Compound specific cumulative lifetime risk probability estimates for children, adult residents and adult workers for the 2014 downwind sites using potency equivalent concentrations (mg-PEQ/m^3). The USEPA's upper bound acceptable risk is marked with the red line at 1.0E-4.

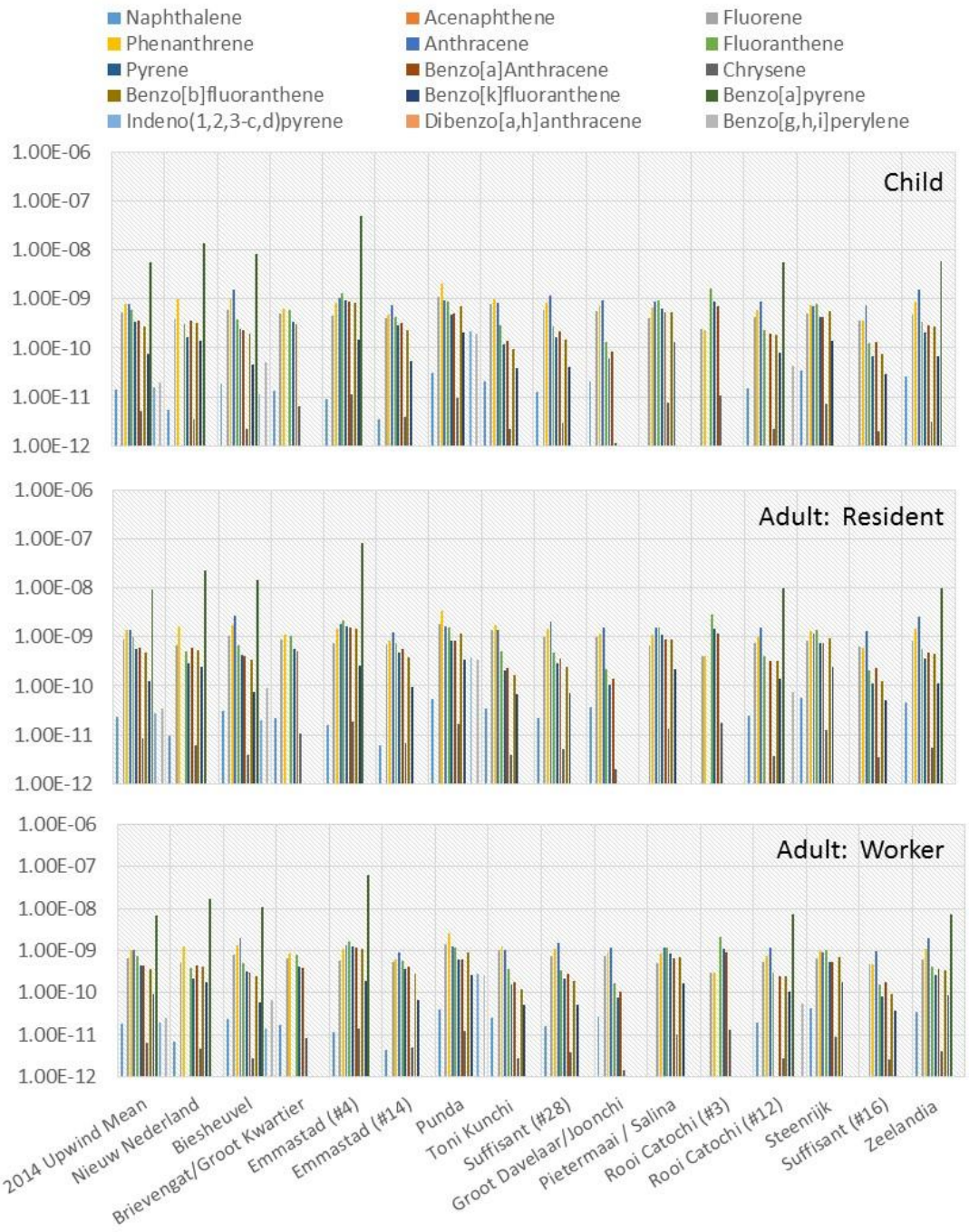


Figure 5.25 Compound specific cumulative lifetime risk probability estimates for children, adult residents and adult workers for the 2014 upwind sites using potency equivalent concentrations (mg-PEQ/m^3).

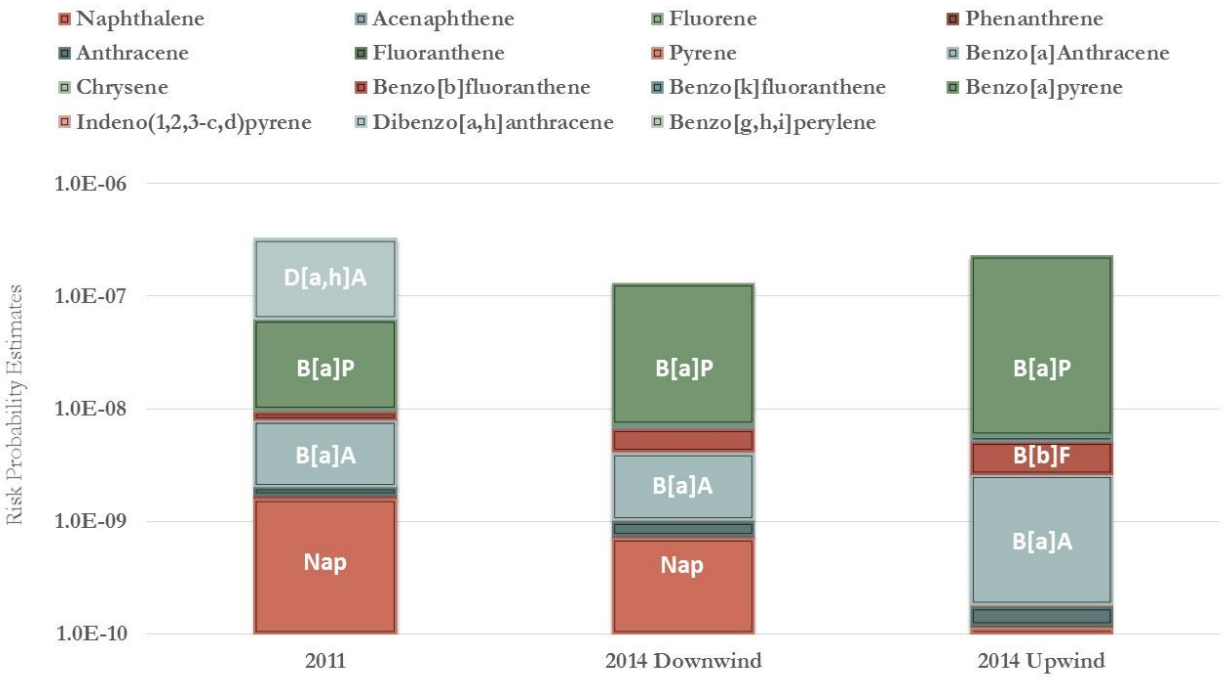


Figure 5.26 Cancer risk probability estimates for all PAHs measured in ambient air in Curaçao during 2011 and 2014 using the 95% UCLs for the potency adjusted concentrations (μg-PEQ/m^3).

CHAPTER SIX:

CONCLUSION

The objectives of this investigation were to 1) establish baseline levels of select petrochemical emissions (i.e., PM_{10}, SO_2, and PAHs) in ambient air surrounding Willemstad, Curaçao; 2) determine if temporal and spatial trends exist in the measured concentrations; 3) identify emission sources; 4) determine if the levels exceed public health guidelines for petrochemical emissions and finally 5) to identify potential health risks. In conclusion, concentrations of PM_{10} and SO_2 in Curaçao are among the highest reported globally and demonstrate an increasing trend over time. Levels of both PM_{10} and SO_2 exceeded the annual and 24-hour guidelines recommended by Curaçao, the European Commission, World Health Organization and the USEPA. Furthermore, both the 24-hour and annual mean concentrations of PM_{10} and SO_2 measured in Curaçao were within the ranges often associated with cardiovascular and respiratory effects and mortality as a result of short-term exposures. Therefore, it is plausible that residents of Curaçao may experience health effects, however, the epidemiological evidence is inadequate to infer causality between health effects and chronic, long-term exposures to PM_{10} and SO_2.

In general, ambient PAH concentrations in Curaçao were consistent with other urban and industrialized regions of the world, however, the levels measured downwind of Isla Refineriá were among some of the highest reported ambient PAHs globally. The mean ambient PAH levels downwind of the refinery were significantly higher than those located upwind with no statistical differences between 2011 and 2014 levels.

The ambient air concentration profiles were dominated by the low molecular weight 2-3 ring PAH compounds, yet the carcinogenic 4-6 ring PAH compounds accounted for the majority of the carcinogenicity of the quantified PAHs. Source characterization through the use of concentration and distribution profiles, binary diagnostic ratios and factor analysis revealed both petrogenic (i.e., refinery) and pyrogenic (i.e., vehicular) emission sources. The sources of ambient PAHs in 2011 and in the 2014 downwind locations were dominated by petrogenic emission sources and to a lesser degree pyrogenic emissions. Whereas, the 2014 upwind locations appear to be equally influenced by both petrogenic and pyrogenic emissions sources.

Benzo[a]pyrene levels exceeded the recommended guideline (0.1 ng/m^3) in Habaai during the 2011 sampling event and in Buena Vista and Emmastad during 2014. In addition, the majority of the locations exceeded the recommended fluoranthene guideline (2 ng/m^3) in both 2011 and 2014. Exceeding these recommended WHO guidelines would theoretically lead to one extra cancer case in 100,000 (1.0E-05) exposed individuals. Using the risk calculation methodology recommended by the USEPA resulted in cancer risk estimates that were below yet approaching the USEPAs upper bound acceptable risk level (1.0E-04) for ambient PAH concentrations. The cumulative lifetime exposure risk estimates for children and adults were within the acceptable range (1.0E-06 to 1.0E-04) for the levels measured in 2014, yet were approaching levels warranting action (1.0E-04) in 2011. In addition, the potency adjusted concentrations for the 2011 and 2014 site specific risk estimates were all considered negligible (<1.0E-06). In contrast, if considering potential worse case scenarios, extrapolating cumulative lifetime cancer risks using the potency adjusted PAH concentrations without using 95% UCLs, resulted in the 2014 downwind overall mean for Habaai and Wishi exceeding the action level (1.0E-04) by up to two orders of magnitude. Currently, the USEPA considers excess cancer risks below 1 in a million (1.0E-06) to be so small as to be negligible, excess cancer risks that

range between 1.0E-06 and 1.0E-04 are generally considered acceptable, and risks at or above 1E-04 are sufficiently large to warrant remediation (USEPA).

This investigation is not without its limitations. First, ambient PAHs were only measured over a three month period during the spring and early summer months (Feb-June). Although the temperature in Curaçao is in the mid-80s year round, there is distinguishable dry and rainy seasons (Oct – Feb), which can affect petrochemical emission concentrations in the ambient air. Secondly, only the vapor phase of PAHs were measured in this study using passive samplers, albeit, previous studies have determined that both the vapor and particulate phases are captured using passive sampling [83, 86]. Site specific validation and year-round sampling is warranted to determine if the ambient PAHs in Curaçao are underestimated, thereby potentially underestimating potential health risks. Thirdly, the risk estimates presented are only based on inhalation and do not characterize additional exposure routes such as dermal and ingestion. Fourth, quantitative cancer risk estimates of PAHs are uncertain due to the lack of useful, good-quality data and the difficulty in assessing the toxicity of complex mixtures [70].

Finally, and more importantly, the accepted consensus is that the true value of cancer risk or threshold posed by chemical exposures is unknown and therefore could essentially be as low as zero [197]. This is acknowledged by the USEPA which cautions that linear models lead to plausible upper limits associated with mechanisms of carcinogenesis however they are not necessarily realistic predictions. Risk assessment can be a powerful tool for protecting public health but it cannot be used to determine an individual's risk, incidence of disease or types of effects exposures can have on humans. Nevertheless, this is the first known assessment quantifying ambient PAH concentrations, elucidating potential sources and estimating potential public health risks as a result of petrochemical constituents in Curaçao. This research underscores the importance for further research as well as management assessment of current local air quality guidelines.

Future research needs in Curaçao include expanding the air monitoring efforts to include areas upwind of the refinery as well as additional petrochemical emissions, including but not limited to sulfur dioxide, particulate matter (PM_{10} and $PM_{2.5}$), benzene, as well as both the vapor and particulate phases of ambient PAHs. Additional environmental studies are encouraged to evaluate the extent of contamination in a variety of biota and matrices (i.e., water, sediment, and fish). As previously mentioned, 60% of 3230 children (ages 0-14) in Curaçao had asthma which is more than four times the global average for children [193]. As such, a more complete human health risk assessment is recommended to include dermal, inhalation and dietary exposure pathways. In addition, a more rigorous epidemiological study involving clinical assessments are needed to evaluate health effects and disease associations with air quality parameters.

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