Human Health Risk Assessment of Artificial Turf Fields Based Upon Results from Five Fields in Connecticut

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Executive Summary

Questions have been raised about possible exposures when playing sports on artificial turf fields cushioned with crumb rubber infill. Rubber is a complex mixture of various chemicals including volatile organic chemicals (VOCs), semi-volatile organic chemicals (SVOCs) and metals. Some components have toxic and carcinogenic properties. Exposure is possible, primarily via inhalation, given that chemicals emitted from rubber can end up in the breathing zone of players and these players have high ventilation rates. Previous studies from Europe and the United States provide useful data but are limited particularly with respect to the variety of fields and scenarios evaluated. To enhance this database, the State of Connecticut undertook a multi-disciplinary study of artificial turf fields involving field investigation, laboratory offgas studies and human health risk assessment. These reports were reviewed by the Connecticut Academy of Science and Engineering (CASE) and their comments have been incorporated into the final report.

The current investigation involved air sampling at 1 indoor and 4 outdoor artificial turf fields under summer conditions in Connecticut. On-field and background locations were sampled using a variety of stationary and personal samplers. A total of 27 chemicals of potential concern (COPCs) were found to be above background and possibly field-related on both indoor and outdoor fields. These COPCs were entered into separate risk assessments for outdoor and indoor fields and for children and adults. Exposure concentrations were pro-rated for time spent away from the fields and inhalation rates were adjusted for play activity and for children's greater ventilation than adults. Toxicity values (cancer unit risks, RfCs, acute targets) were taken from national databases or derived by CT DPH. In general, conservative public health protective assumptions were made in calculating risks, especially with regard to the inclusion of detects from personal samplers that may not have been field related. As such, this represents a screening level assessment that is likely to overestimate risk.

In spite of the conservative nature of the assessment, cancer risks were only slightly above de minimis levels for all scenarios evaluated including children playing at the indoor facility, the scenario with the highest exposure. The calculated risks are well within typical risk levels in the community from ambient pollution sources and are below target risks associated with many air toxics regulatory programs. Further, the main risk driver, benzene, was only above background in personal monitoring samples and so may be more related to the sampling equipment or host than being field-related. Chronic non-cancer risks were not elevated above a Hazard Index of 1. The Hazard Index for acute risk was also not elevated above 1 but was close to 1 for children playing at the indoor field. The main

contributor to this Hazard Index was benzothiazole, a rubber-related SVOC. This presents an uncertainty regarding the potential for benzothiazole and other volatile irritants to create a slight irritation response in sensitive individuals playing indoors.

Based upon these findings, the use of outdoor and indoor artificial turf fields is not associated with elevated health risks. However, it would be prudent for building operators to provide adequate ventilation to prevent a buildup of rubber-related VOCs and SVOCs at indoor fields. The current study did not evaluate new fields under hot weather conditions and so the potential for acute risks under this circumstance is another uncertainty. The current results are generally consistent with the findings from studies conducted by New York City, New York State, the USEPA and Norway which tested different kinds of fields and under a variety of weather conditions. Thus, it appears that the current results are reasonably representative of conditions that can be encountered at indoor and outdoor crumb rubber fields, although this tentative conclusion could benefit from the testing of additional fields.

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I. Introduction

Questions have been raised about potential exposures and health risks associated with playing on artificial turf fields cushioned with crumb rubber. Rubber is a complex mixture of natural compounds and industrial chemical intermediates, a number of which have the potential to be a health risk if there is sufficient exposure. CTDPH developed a fact sheet in October 2007 regarding the potential exposures and risks associated with offgasing from artificial turf fields highlighting research from Europe and California. The fact sheet identified a number of limitations and uncertainty in the existing database. Since then, a field investigation and risk assessment has been conducted for two fields in NYC (NYSDEC 2009; TRC, 2009) and for 4 fields in various states across the country (USEPA, 2009).

The current field investigation and risk assessment project sampled 5 fields in Connecticut for a wide range of volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), rubber-related SVOCs, lead, and particulate matter in the less than 10 uM range (PM_{10}). The current investigation adds to a growing body of data describing crumb rubber-based athletic fields, and it is unique in providing personal monitoring results for users of the field during active play. Further it is the only study in the US which assesses an indoor soccer field. Each field was investigated on its own day of field work during July 2009 under sunny, warm and low wind weather conditions. The hope was to maximize the detection of offgassed rubber components. Details of the sampling plan, methodology and results of field testing and offgas headspace experiments can be found in the companion report from the University of Connecticut Health Center, Section of Occupational and Environmental Health (UCHC, 2010). An additional report from the Connecticut Agriculture Experiment Station evaluated offgassing from crumb rubber samples from the study fields (CAES 2009). This is a follow-up of an earlier pilot study by CAES (2007).

This HHRA focuses upon the air results for VOCs and SVOCs since other types of analytes were not in an elevated range. The bulk phase lead testing from each field's artificial grass and crumb rubber were uniformly below 400 ppm, the CTDEP Remediation Standard Regulation for lead and the point of departure nationally for concern for housing units and schools. These results were also below the 300 ppm target set by the Consumer Product Safety Improvement Act for lead in products intended to be used by children. The highest lead level found in any sample from the 5 fields was 271 ppm (Field D). Testing for nitrosamines and PM_{10} failed to find detections above background. Lead was a target analyte because of limited test data in New Jersey showing elevated lead in artifical grass samples,

which led to an investigation by CPSC (2009). The lack of elevated lead in our current testing suggests that if lead is elevated in synthetic grass or crumb rubber, that it is not a widespread problem.

The overall objective was to develop a screening level risk assessment in which high end assumptions for exposure were used for uncertain parameters, questionable sampling data were included to avoid exclusion of potentially meaningful chemicals, and surrogate data were used for chemicals with inadequate toxicity information so that chemicals did not drop out of the assessment on the basis of missing data. If the risks projected with this approach are not elevated into a range of concern, then there is little need to refine exposure assumptions or perform a more detailed analysis.

II. Review of Air Sampling Results

The air quality field investigation involved sampling for a suite of 60 VOCs, 120 SVOCs divided into 22 PAHs, 5 targeted (potentially rubber-related) SVOCs, and 93 miscellaneous SVOCs, 7 nitrosamines and PM_{10} (UCHC, 2010). The types of samples taken and analytes measured are summarized in the text table below.

Sample Type	VOCs	SVOCs	Rubber SVOCs	Nitrosamines	PM_{10}
Personal monitor	yes	no	yes	yes	no
Stationary on-field 6 inch	yes	no	yes	yes	no
Stationary on field 3 feet	yes	yes	yes	yes	yes
Stationary upwind	yes	yes	yes	yes	yes
Community	yes	yes	yes	yes	yes

VOCs, targeted SVOCs and nitrosamines were sampled in both stationary and personal samplers while the other analytes were collected in stationary samplers only. All analytes were assessed in the upwind, off-field location and in the community background sample (Site L) using stationary samplers. This lead to 5 types of samples for VOCs and targeted SVOCs: stationary, field height (6”); stationary 3’; personal monitor; off-field upwind, off-field community. For PAH and miscellaneous SVOC there were 3 types of samples: on turf, upwind and community background. Data from the community background sample were combined with the other background samples taken in association with the field investigations to yield a range of background results.

A. Selection of “Contaminants of Potential Concern”

An analyte became a contaminant of potential concern (COPC) if it was detected on the field at higher concentration than in the background samples. Due to the small number of samples and background taken at any one field, there would be low confidence in making decisions about contaminant emissions at a particular field. Therefore, for the 4 outdoor fields, the results were pooled and the highest on-field result (regardless of sample type) was taken to represent what might be coming off the fields. This was then compared to the range of background results. If the highest field result was 25% above the highest background result, the analyte was considered a COPC. This ensures that for an analyte to become a COPC its on-field and background detects do not overlap. The range of background results was inspected to make sure that the highest background was not an outlier, in which case the next highest result would be used. If a contaminant was judged to be a COPC on this basis, its entire concentration was considered to be field-related – there was no background correction, even though in some cases the on-field result was only slightly (albeit >25%) above the background result. All COPCs were carried through the risk assessment process.

Personal sampler results tended to be higher, in some cases much higher, than the field results. This raised the question of whether the finding in personal monitoring samples was field related, especially in cases where the analyte was not detected in any field-related samples (including the indoor field) and in which the CAES and Wisconsin Occupational Health Laboratory (WOHL) headspace studies of crumb rubber from these fields failed to detect the analyte. In these cases, the personal sampler detect was considered to be due to extraneous sources such as the sampling equipment, or host-related factors such as personal care products or exhaled breath. An example of this phenomenon is acrolein (Figure 1) in which substantial detects were found in the personal monitors at three of the fields but no detections were found in stationary on-field or background samples, or in the headspace experiments. While the main source of human exposure to acrolein is considered to be indoor and outdoor air pollution related to cigarette smoke or combustion sources, there are endogenous sources resulting from the processing of sugars, lipids and certain amino acids (Stevens and Maier 2008). Given the volatility of acrolein, a percentage of the endogenous formation would be expected to be found in exhaled breath, and in fact, acrolein has been detected in the exhaled breath of smokers at higher concentration than in non-smokers (Andreoli et al. 2003). In addition to acrolein, there were a number of other analytes which were detected only in the personal monitors and were not included as COPCs. They are listed in Table 1. In other cases, personal monitors yielded considerably higher concentrations of analytes than detected on the field suggesting a contribution from the host in some

manner. Since this percentage is unknown, the personal monitor detects were used to represent what may have been coming off the field for the purpose of the risk assessment.

The indoor field (Field K) is treated as a separate case because the conditions and results are substantially different than outdoors. A quick scan of the data indicated that if the indoor field were lumped in with the other fields, that it would often be the highest detect and the assessment would be driven by results from the indoor field. The greater concentrations indoors provide confidence that measurements from the field were above background in spite of the small sample size. Field K represents something of a worst case for indoor fields because there was no active ventilation at the time of sampling. Table 2 provides a listing of the COPCs for the outdoor fields while Table 3 is the listing for the one indoor field.

Of the 60 VOCs for which analyses were conducted, 14 are considered COPCs at the outdoor field with the same VOCs being COPCs at the indoor field. The indoor detect was greater than any outdoor detect for 9 of the 14 analytes. The personal monitoring result was the greatest detect in all cases for the outdoor VOCs and in most cases for the indoor VOCs. The VOC COPCs were above background at the indoor field and generally at only one of the outdoor fields except for acetone (all 4 outdoor fields), methyl ethyl ketone (MEK, 3 fields), and hexane and toluene (2 fields). In general concentrations in background samples were very low and detects on the field personal monitors were 2 or more times elevated over background. This was not true for benzene and chloromethane (personal monitoring samples) which were only slightly elevated over background at both the indoor and outdoor fields.

Of the targeted, SVOCs, only benzothiazole was detected above background on both the outdoor and indoor fields. The indoor result was 11.7 times greater than the outdoor result which is one of the more dramatic indoor/outdoor differences. Benzothiazole was detected above background at all fields and results on the field were higher than in the personal monitoring sample, an opposite trend compared to the VOCs. One additional targeted SVOC, butylated hydroxytoluene (BHT), was detected in the indoor field. Similar to benzothiazole, BHT was detected above background in all field-related samples at the indoor field, with results higher in the stationary as opposed to personal monitor.

A variety of PAHs were detected above background but at low concentrations (well below 1 ug/m3) at both outdoor and indoor fields. The larger multi-ring PAHs (benzanthracene through chrysene in Table 2) were detected in the outdoor field while the more volatile 2 ring PAHs (naphthalene and its derivatives) were found indoors but generally not outdoors. The one exception was a small increase above background of 1-methylnaphthalene at one outdoor field (Field D). The naphthalene and 1-methylnaphthalene detects at the indoor field were by far the largest PAH detects on any field. Other PAHs (acenapthene, fluoranthene, pyrene) were detected above background both outdoors and indoors. Figures 2-4 graphically depict the across field and outdoor/indoor differences for benzo(a)pyrene, chrysene and naphthalene respectively. An overview of all PAH detects at the 5 fields is present in Figure 5. This figure shows that PAHs were generally in higher content and more numerous at the indoor field.

Miscellaneous SVOCs includes a wide variety of hopanes, pristanes, terpenes, cosanes and other aliphatics derived from fossil fuels or of plant based origin and common in outdoor air (Andreou and Rapsomanikis 2009; Schnelle-Kreis et al. 2007). These air contaminants are particle-bound and while not reported to be present in rubber did show higher concentration on turf than off for 1 analyte on Field A, 1 on Field C and 4 on Field D. The total concentrations of miscellaneous SVOCs that are in excess of background concentrations are shown in Figure 6. These analytes have been totaled for site characterization and risk assessment since there is no toxicological basis for separate analysis. A conservative toxicology value (RfC for pyrene) was used to characterize the entire grouping.

While a variety of carcinogenic and volatile nitrosamines were assessed in field air samples, none were detected which means that nitrosamines were not COPCs in this risk assessment. Nitrosamines were sampled because of their use in rubber manufacture and the potential they could remain in the final product. PM_{10} measurements were made on the fields and at background locations to assess the potential for crumb rubber particulates to be generated by active play and lead to elevated breathing zone concentrations. However, sampling of PM_{10} across 4 of the 5 fields did not find elevated on field concentrations. The on-field result at each field failed to exceed the range of detects found at background locations, 5-10 ug/m3. Field C was an outlier with higher levels of PM_{10} in both the on-field and upwind samples (16-18 ug/m3). A grass field just upwind received pesticide spray during the beginning of the sampling event which could have interfered with PM_{10} and other results. Therefore, PM_{10} was also not considered a COPC. Details of the nitrosamine and PM_{10} testing can be found in the UCHC report.

B. Pattern of VOC and Benzothiazole Detections

The sampling design was especially targeted to detect field-related VOC emissions given the on-field measurement at 6" and 3' heights in stationary samplers compared to background (upwind samplers). The pattern of detects for a number of representative VOCs is presented in Figures 7 to 10. These figures show a consistent pattern of greater detects in personal monitors with the stationary sampler detects being lower and more sporadic. There was generally no indication that the 6" sample had higher results than the 3' sample. While all results were similar for chloromethane (Figure 10), benzene showed a slightly more pronounced detect at one personal monitor (Figure 7), with toluene (Figure 8) and methylene chloride (Figure 9) showing dramatic spikes in a few personal samples.

In contrast to this pattern, benzothiazole detects at the outdoor fields show greater concentration in stationary as opposed to personal monitors with the lower elevation samples showing higher concentration than the 3' samples (Figure 11). This is the expected pattern for chemicals outgassing from the crumb rubber on warm sunny days. The indoor benzothiazole detections are much higher than and overwhelm the outdoor detects and so are added to a separate benzothiazole graph (Figure 12). Once again, a ground level (6") sample was the highest detect although the difference across indoor samples was not as great as in the outdoor setting.

This pattern of VOC and targeted SVOC detections suggests that a substantial portion of the detected VOCs could be coming from the sampling equipment or host as the primary detections were from the personal monitors. In contrast, the benzothiazole detections fit the expected pattern for offgassing from the crumb rubber infill.

III. Exposure Assessment

The primary objective of the field investigation and this risk assessment is to estimate exposures and risks for children playing on the fields. Due to the possibility that adults using these fields could encounter higher exposures due to a longer period of usage, they are also considered as a separate

element of this assessment. As described in the companion field investigation report, study participants played soccer on each field while wearing personal monitors (pumps and summa canisters) at hip height to simulate the breathing zone exposure of young children. The highest result for any sample at that field was used to represent what might be the breathing zone concentration for a child or adult. Given that field sampling occurred in July under sunny, low wind conditions, VOC offgasing from the outdoor fields would be overestimated if the entire 8 month/year exposure period was simulated based upon these results. Instead, we assume that these results (inhalation of measured VOCs) apply to the 4 warmest months with no allowance for days with clouds or high wind which would mitigate exposure. Another conservative assumption is that the highest concentration for each analyte found at any of the outdoor fields is combined across fields to represent a worst case composite. This approach obviates the need for 5 separate risk assessments. Instead we present two sets of risk calculations, one for outdoor fields and one for the indoor field. The results for the indoor field were sufficiently different from the outdoor fields to warrant a separate assessment.

A variety of exposure routes are possible for crumb rubber-related chemicals as follows:

- Inhalation of volatile or semi-volatile chemicals which offgased from the rubber;
- Inhalation of particles and particle-borne chemicals; field methods collected respirable PM_{10} samples and separately a particulate sample was captured on media and assessed in the laboratory for semi-volatile compounds.
- Ingestion of crumb rubber or the dust created from the breakdown of crumb rubber.
- Dermal uptake of chemicals contained in crumb rubber which contact the skin.

The current risk assessment focuses upon the first two pathways, inhalation of offgased and particle-bound chemicals. Ingestion of crumb rubber or dust derived from crumb rubber was not a focus as this pathway has been evaluated elsewhere without being identified as a public health risk (Norwegian Inst Public Health and Radium Hospital, 2006; CalEPA, 2007) and field methods were not designed to measure the amount of dislodgeable dust that could occur on the surface of these fields and end up becoming ingested. The fields do not appear to be especially dusty and the crumb rubber that clings to clothing and body parts is of relatively large size making its ingestion more of an intentional event characteristic of younger age groups and covered by the prior oral crumb rubber risk assessment done in California (CalEPA, 2007). Additionally, the Norway study evaluated chronic inadvertent ingestion of crumb rubber by children playing on the fields (Norwegian Inst Public Health and Radium Hospital,

2006). Dermal exposure was also not a focus as most chemicals in rubber are not a good candidate for dermal absorption: the volatile fraction will tend to revolatilize off skin and thus not remain long enough for substantial dermal penetration; the particle bound fraction will tend to remain bound to the rubber rather than partition into and then penetrate through the skin. There are no data describing the transfer of semi-volatile chemicals from a rubber matrix to skin but this would not appear to be a large uncertainty. Further support for this is that the fields are not highly dusty and players do not become coated with dust particles, although larger rubber particles do cling to clothing and get inside shoes. The Norwegian study evaluated dermal exposure to crumb rubber particles and did not find this to be a significant health risk (Norwegian Inst Public Health and Radium Hospital, 2006).

A. Exposure Scenarios

Two scenarios have been developed for estimating inhalation exposures at these fields, one that simulates exposures in children, and one for adults. Table 4 presents key parameter values used in exposure equations for these two sets of receptors.

Inhalation risk equations are based upon a target airborne concentration such as the reference concentration (RfC in ug/m3) for non-cancer effects and the unit risk factor (risk per ug/m3) for carcinogens. These target concentrations are based upon toxicology studies in animals or human epidemiology studies in which the subjects were at rest or undergoing light exercise (workers). Further these data come from adult animals or humans and so do not necessarily capture the increased exposure and risk possible for younger children (Ginsberg, et al. 2010). Rather than changing the toxicity values for different receptors, the receptor's exposure is adjusted as shown below to account for the difference in ventilation rate during active play and in children. The following equation also presents the time weight-averaging used to calculate inhaled doses that relate to long-term exposure and risk:

$$\text{Inhaled Conc (ug/m3)} = \frac{\text{Measured Conc (ug/m3)} * \text{Hours per Day} * \text{Days per Year} * \text{Years} * \text{Ventilation adj}}{\text{Averaging Time}}$$

The ventilation adjustment for adults is based upon exertion-induced increases in the amount of air inhaled going from the typical assumption of light exercise (0.0148 m3/min or 21.4 m3/day) to the higher ventilation rate associated with sports play as described in USEPA's Exposure Factors Handbook (USEPA 2009b). For this we assume a moderate (0.039 m3/min) level of exercise for the 3 hrs of play which accounts for the fact that periods of time are spent resting or listening to instructions while other periods would elicit an intense level of exercise (0.073 m3/min). This creates an adult ventilation adjustment of (0.039/0.0148 m3/min = 2.64). A further adjustment is made for the ventilation rate in children based upon their greater rate per body weight and respiratory surface area. A recent review and analysis (Ginsberg et al. 2010) points out that a 3 fold factor is appropriate for the first 3 years of life with this decreasing to a 1.5 fold adjustment for ages 4-10. Given that this adjustment applies to a portion of the childhood exposure period simulated in this assessment , the 1.5 fold factor is conservatively applied to the child scenario overall rather than dividing it into two assessements (young vs older child). This factor is applied on top of the adult ventilation adjustment to yield a 3.96 fold adjustment for children (Table 4).

IV. Toxicity Assessment

A broad array of COPCs has been identified, some of which have an extensive toxicology database and others which do not. This toxicity assessment relies upon national databases of toxicity potency values as available from USEPA's Integrated Risk Information System (IRIS) (http://www.epa.gov/iris/), California's Office of Environmental Health Hazard Assessment (OEHHA) (http://www.oehha.ca.gov/risk/chemicalDB/index.asp), and the Agency for Toxic Substances and Disease Registry (ATSDR) (http://www.atsdr.cdc.gov/mrls/) as the primary sources of toxicity information. By convention, IRIS is typically the first choice. However, when values are available from multiple sources they are compared and in cases where there is considerable disagreement (3 fold or greater), CT DPH has evaluated the underlying difference and chosen values that best reflect the most recent and robust treatment of the available science.

Given the screening nature of this risk assessment, toxicity values were assigned in a conservative manner to decrease the potential for the under-reporting of risk. When data were not available for a particular analyte, a related surrogate that has toxicity data was used that reflects a high end of the likely potency. For example, all non-carcinogenic PAHs and other miscellanous SVOCs that lack

RfCs were assigned the RfC for pyrene, which is the lowest RfC available for the general series of PAHs.

Table 5 summarizes the toxicology values used for COPCs in this assessment. Appendix B contains an expanded version of this table with additional details. While we are not providing chemical-specific toxicology monographs, these are commonly available from the cited sources (e.g., IRIS, CalOEHHA, ATSDR). However, we do provide a short monograph on benzothiazole (Appendix A) because it has not been evaluated by the standard governmental sources and is a key component of crumb rubber in terms of field-related exposures.

In addition to chronic cancer and non-cancer toxicity values (unit risks and RfCs respectively), this assessment utilizes acute risk air targets as well. Short term exposure to COPCs could trigger an irritant or neurological response, or some other acute effect. To evaluate this potential requires acute exposure toxicity values that would be the equivalent of a 3 hour RfC. These values typically do not exist. However, for a limited set of chemicals 1 hour acute targets have been derived by California OEHHA (acute RELs) and by Connecticut DPH (CT Acute Exposure Concentrations - AECs). These values have been used along with ATSDR acute MRLs (typically 24 hr continuous exposure basis) to develop 3 hour acute air targets in this assessment. These acute targets have been set based upon evidence of a threshold in short-term studies (often in humans) with the use of variability and uncertainty factors on a case-by-case basis. In consideration of Haber's Law, a 1 hour target developed for other purposes was converted to a 3 hour target by dividing by 3. These 1 hour levels were prioritized over the ATSDR acute MRLs because they relate to a short-term acute exposure while the ATSDR acute values are normalized to a 24 hour continuous exposure basis. The level of conservatism and time factor adjustment in the ATSDR values are not necessarily consistent or always transparent. Thus, the protective value needed for 3 hours is not necessarily 8 fold higher than the ATSDR value in any given case. However, in cases where California or Connecticut acute values are not available, the ATSDR 24 hr acute values are used without adjustment. Another conservative approach was to use the chronic RfC in several cases where an acute value was not available.

As seen in Table 5, all COPCs have been assigned RfCs, 13 have cancer unit risk values and 14 have acute targets. Only those analytes having direct or indirect (e.g., structurally related to carcinogen; mutagenic) evidence of carcinogenicity have been assigned unit risk values. Acute targets have been assigned only for the volatile analytes as the acute effects of particle bound chemicals such as PAHs

have not been well explored but are expected to be minor given that they are not highly reactive and tend to cause chronic effects instead. The following highlight some of the toxicology assessment decisions made in the face of limited or conflicting information:

Benzene – The California OEHHA unit risk is 2.9E-05/ug-m3 which is 3.7 fold above the upper end of the USEPA IRIS unit risk range derived in 2003 (2.2 to 7.8E-06/ug-m3). One difference is that Cal OEHHA developed unit risk values based upon a wide range of animal and human studies and settled upon a value that was near the middle of this range. In contrast, the USEPA risk range came from one human epidemiology study (Rinsky et al., 1987, pliofilm workers) with the range represent different exposure assessment and modeling assumptions. Thus it can be argued that the Cal OEHHA assessment is more robust by taking into account a wider array of datasets. However, there is also a systematic difference in the unit risk calculation as Cal OEHHA's estimate from the Rinsky et al. dataset was 1.4 E-05 which is still above the risk range calculated for that study by USEPA. The details of the IRIS and Cal derivations are not readily available. Because further discrimination is not possible, for the purposes of this risk assessment, the upper bound of the IRIS range and the Cal OEHHA value were averaged to yield 1.84E-05/ug-m3.

Chloromethane – this chemical is generally regarded as a mutagen with limited cancer bioassay data suggesting some activity. However, USEPA and Cal OEHHA have not derived unit risks. Rather than count chloromethane as having no cancer risk, this assessment uses a unit risk developed by the California Proposition 65 committee for the purposes of assessing potential health risks from its presence in consumer products.

Heptane – this solvent lacks an RfC in the standard sources and has not been extensively studied. However, it is known to be less neurotoxic than its congener hexane. As a conservative screening approach, the RfC for hexane was used as a surrogate for heptane.

Styrene – cancer database is limited and conflicting; it has positive mutagenicity data and the main metabolite, styrene oxide is mutagenic. Therefore, an additional uncertainty factor was added to the IRIS RfC to account for the possibility that it has carcinogenic action. Carcinogens typically have much lower de minimis targets than non-persistent noncarcinogens.

Benzothiazole – this agent has very little toxicology data but was positive in one mutagenicity test and has a structural analogue that is carcinogenic (2-MBT). The acute toxicity value was derived based upon analogy with formaldehyde. Both compounds were tested in mouse respiratory depression (RD-50) studies by the same laboratory and found to be irritating. Benzothiazole's potency in this test was 18 fold below that of formaldehyde. Given the uncertainties in extrapolating from an animal screening test to humans and the fact that benzothiazole can cause sensitization (at least on the skin), an additional 10 fold uncertainty factor was applied to derive the acute target. The derivation of cancer, non-cancer and acute toxicity values for benzothiazole is further described in Appendix A.

Butylated Hydroxytoluene – there are no toxicity values but BHT is a common food preservative. The European Union has an acceptable daily intake based upon toxicology concerns of 0.05 mg/kg/d which is 3 fold lower than the USFDA intake limit. The EU value was converted to inhalation by dose route extrapolation for the current purposes.

Dose route extrapolation – this was done in selected cases where an inhalation value was not available and the target site is systemic rather than at the point of contact. Assumptions for dose route conversion are inhalation of 20 m3 per day for a 70 kg adult.

Children's Cancer Potency - According to USEPA's Carcinogen Risk Assessment, Supplemental Guidance for Early Life Stages (USEPA, 2005), children have greater vulnerability to a variety of carcinogens with the evidence particularly strong for those with a mutagenic mode of action. For these carcinogens, the Supplemental Guidances recommends the following enhanced potency factors above the adult potency: 10 fold for 0-2 yr of age and 3 fold for 3-15 yr olds. For the purposes of this assessment, the child exposure scenario (age 12 average; range 6-18) is considered to be at heightened vulnerability and would receive the enhanced 3 fold factor. This applies to all carcinogens that have documented mutagenic or clastogenic activity and included as COPCs: benzene; cloromethane; methylene chloride; benzo(a)pyrene and related carcinogenic PAHs; benzothiazole. The carcinogenicity and mode of action of benzothiazole is uncertain but in very limited testing it was mutagenic and so is included in this list. Naphthalene and its congeners have limited cancer and mechanistic/mutagenic data with its cancer classification not well established. Therefore, we did not apply an additional children's potency factor for naphthalene and its related analytes.

V. Risk Characterization

A. Calculations

This assessment uses standard risk assessment methods to estimate cancer and non-cancer risks. Pro-rated time weight averaged exposures were calculated based upon the highest measured analyte concentration, amount of time playing (3 hrs per day, 138 days per year), exercise-induced breathing rate, and years of exposure (12 or 30). For carcinogens, the lifetime average daily exposure in units of ug/m3, was multiplied by the cancer unit risk or the adjusted unit risk for children, to yield the lifetime cancer risk estimate. Risks for an individual carcinogen were added to other carcinogen risks to yield the total cancer risk associated with playing on the field under the current scenarios and assumptions. Risk estimates above 1E-04 are considered substantially elevated relative to USEPA Superfund guidance (acceptable risk range up to 10^{-4}), background air toxics risk estimates for US census tracts which are typically estimated at a cumulative cancer risk of 1E-05 to 1E-04 (USEPA NATA 2002; Woodruff et al. 1998) and California regulatory air target limits (cancer risk of 1E-05). Cancer risks below 1E-06 are considered de minimis. Cancer risks between 1E-06 and 1E-04 are in an intermediate zone which may require more detailed review of uncertainties and data sources, acquisition of additional data, and under certain circumstances, some type of intervention.

For non-carcinogens, the average daily dose during the exposure window (12 or 30 years) was divided by the RfC to create the Hazard Quotient (HQ). These time frames, 12 and 30 years is considered chronic exposure for the purposes of comparison to the RfC which is usually thought of as the chronic lifetime safe exposure value. HQ values for individual analytes may or may not be additive across analytes depending upon whether target sites and mechanisms of action are similar. However, in lieu of that level of detailed analysis, this assessment assumes that all non-cancer risks are additive across chemicals to yield a cumulative Hazard Index (HI).

For acute risk calculation, the non-pro-rated highest field concentration was adjusted by the enhanced ventilation rate and then divided by the acute air target to create the acute HQ. These acute risks may or may not be additive across chemicals as some are based upon irritation while others on neurological effects, internal organ damage or reproductive effects. As a crude, conservative screen, this

assessment assumed that the individual acute risks were additive across chemicals to yield a cumulative HI_{acute}.

B. Results

Appendix B contains spreadsheets showing calculations for all COPCs across the 4 scenarios – Child Outdoor Field, Child Indoor Field, Adult Outdoor Field, Adult Indoor Field. The appendix also contains a summary table showing the combined risks across all analytes for each scenario. Figure 13 and Table 6 present the key summary information from these tables. A variety of VOCs and SVOCs contribute to both cancer and non-cancer risk estimates. Inhalation exposures were estimated based upon long term chronic averaging to assess cancer and non-cancer risks, and the on-field measurements were used directly (without time pro-rating) to assess acute risks.

Cancer risks are slightly above de minimis in all scenarios, being nearly two fold higher at the indoor field compared to outdoors and being higher for children than adults. Children's greater vulnerability to mutagenic carcinogens combined with their greater exposure rate outweigh the greater amount of time spent on the fields by adults in creating higher cancer estimates for children. The greatest contribution to cancer risk in each scenario is from the VOCs benzene and methylene chloride with much smaller contributions by chloromethane, benzothiazole and PAHs (Table 6, Figure 13). The same array of carcinogenic VOCs contribute to cancer risk at the outdoor and indoor fields in roughly the same proportions, while for SVOCs the benzothiazole contribution to cancer risk is greater at the indoor field. PAHs are minor contributors to cancer risk at both the outdoor and indoor fields. As discussed below, the main contributors to cancer risk, benzene and methylene chloride, were found in personal monitors only and may not be field-related.

The non-cancer risk estimate is below unity for all analytes in all scenarios (Table 6, Figure 14). Even when adding all HQs together, the total is still below unity. The highest HI is 0.48 for children playing at the indoor field. None of the analytes is predominant with the majority of the risk spread between 16 VOCs and targeted SVOCs. PAHs contribute very little to the non-cancer risk. The greatest percent contributor is toluene at just under 20% (Table 6).

The acute risk estimate is also below unity for all analytes and scenarios with the total HI across analytes below unity in all cases (Table 6, Figure 14) . The highest HI is for children at the indoor field, reaching a value just slightly below unity (0.96). This value is driven by benzothiazole (54% of the total) with relatively minor contributions from a variety of other VOCs. Benzothiazole's acute effect of potential concern is respiratory irritation.

C. Discussion

This HHRA detected elevated concentrations of 27 COPCs across the combination of the 4 outdoor fields, and coincidentally, 27 COPCs were also detected at the 1 indoor field. These COPCs were identified on the basis of their being present 25% above background in at least one field-related sample and if the only elevation was in a personal monitoring sample, there also had to be evidence that the analyte was present in head space from a crumb rubber offgas experiment. These criteria are similar to that used in the UCHC report to identify field-related chemicals, although the current criteria are simpler and in some cases more inclusive (e.g., 25% elevation above background instead of two fold). Cancer, chronic non-cancer and acute toxicity values were searched or developed for all COPCs to the extent possible. Cancer potency values (unit risks) were developed for 13 COPCs, non-cancer potency values (RfCs or their equivalent) were developed for all COPCs, and acute targets were developed for 15 volatile COPCs. In most cases these potency values were selected from existing national databases except for acute targets for which a number came from CT DPH acute exposure concentrations (AECs) developed in 2000 and reviewed for the current application.

The list of 27 detected COPCs at the outdoor fields is somewhat different than the list of 27 COPCs at the indoor field. For example certain PAHs were only detected on the outdoor fields while others, tending to be the more volatile PAHs, were only detected at the indoor field. However, the main difference between outdoor and indoor data is that the number of COPCs detected at any one outdoor field was considerably less than indoors (maximum number at a single outdoor field – Field B = 14 COPCs; all 27 detected at Field K), and that concentrations of certain analytes were considerably higher indoors than outdoors: benzothiazole, toluene, acetone, methyl ethyl ketone, methyl isobutyl ketone, and naphthalene and related congeners. UCHC field investigators inquired to the building manager as to possible sources of VOCs and SVOCs in the building from stored materials and products used. This survey failed to uncover any sources that could confound the indoor air results.

While exposure to all detected COPCs is possible, our data are most consistent with a smaller subset actually being field-related. That grouping is led by benzothiazole, a compound known to be used in rubber production. The remaining COPCs are much less specific with many also coming from background combustion sources, and some VOCs may also come from endogenous (within the body) sources and be detected in personal monitoring samples. Based upon the pattern of detection, it appears likely that in addition to benzothiazole, detects of acetone, toluene, methyl ethyl ketone, methyl isobutyl ketone, butylated hydroxytoluene and a variety of PAHs were field-related, with other COPCs less certain to be field related. This is significant because the major contributors to cancer risk are not clearly field-related in either the outdoor or indoor fields.

Table 6 and Figure 13 indicate that benzene is the leading contributor to cancer risk at both indoor and outdoor fields, comprising 51 to 73% of the total risk and being the only analyte to on its own be above de minimus risk. Benzene was only detected in personal monitoring samples and not in stationary field samples suggesting that a substantial portion of the personal monitoring detections comes from the sampling equipment or host and not from the field. In fact, benzene has been detected in the exhaled breath of non-smoking individuals in a number of studies with one study of 20 non-smoking adults finding benzene in the breath of 65% of these people (Buszewski et al. 2008). That study also found methylene chloride in 20% of those individuals (Buszewski et al. 2008). Methylene chloride was also only detected above background in personal monitoring samples. These analytes were included at COPCs because of their detection in WOHL crumb rubber offgas studies (UCHC 2010). Benzene was detected in the head space from two of the five crumb rubber samples while methylene chloride was detected in four of the five. However, those studies were under high temperature (70C for 1 hour) conditions optimized to detect VOCs offgassed from crumb rubber. Further, laboratory blank analyses from those WOHL headspace analyses found 6 VOCs in the lab blanks including benzene, methylene chloride and acetone. This creates additional uncertainty regarding the field-related nature of these VOC detects, but they were still considered as COPCs for the purposes of the current risk assessment.

Two other laboratory crumb rubber offgas studies conducted at high temperature also did not show evidence of chloromethane or methylene chloride in the headspace (CAES 2009; NYSDEC 2009). The NYS study found benzene in the headspace of 0 to 71% of the samples depending upon the source of crumb rubber, while the CAES study failed to detect benzene in any sample. We also note that the Norway indoor field investigation found low amounts of benzene (1-2 ug/m3) in their on-field

measurements that they speculated may have resulted from offgassing over and above background sources (NILU, 2006). That study did not detect chloromethane or methylene chloride in association with artificial turf fields. It thus appears that several contributors to cancer risk in the present study, chloromethane and methylene chloride, are unlikely to be field related while some percentage of the detected benzene and PAHs may have been field related and it is likely that benzothiazole was completely field related.

This conservative screening level risk assessment found cancer risks to be only slightly above de minimis levels at the outdoor and indoor fields, indicating that cancer risks are not elevated into a range of public health concern. The following factors support this conclusion:

a) The level of benzene found in personal monitoring samples at one outdoor field and the indoor field are in the 1-2 ug/m3 range which is often considered the background range for ambient benzene (ATSDR 2007); for example, recent urban and surburban sampling in Tonawanda NY found an average benzene concentration of 1.2 ug/m3 (NYSDEC 2009b).
b) The elevations in VOC cancer risk drivers (benzene, methylene chloride, chloromethane) were from personal monitoring samples which likely had a contribution from the host, and these analytes were not detected in stationary field samples, even at ground level. The fact that benzene has been detected in crumb rubber headspace analyses in several studies suggests that some percentage of the personal monitoring detect may have been field related. However, if the uncertain benzene exposures were not included in the risk assessment, cancer risks would fall below de minimis in 3 of the 4 scenarios with only children/indoor fields still being slightly above 1E-06 risk.
c) The unit risk factor for benzene used in this risk assessment is the average of the upper bound from the IRIS unit risk range and the value from Cal OEHHA. The Cal OEHHA value is well above the IRIS value for reasons not immediately apparent and is not necessarily any more reliable. The value chosen for the current purposes is considered to be conservative and may overestimate benzene potency.
d) The degree of cancer risk presented by these analytes is on the low end of the intermediate cancer risk range (1E-6 to 1E-04) and is below the background level of cancer risk in the general community from air toxics. According to USEPA's National Air Toxics Assessment (NATA) for 2002 the average background level of cancer risk across Connecticut is 2.5 to 5E-05 (USEPA NATA). In fact, the California Air Resources Board (CARB) definition of

significant risk for the total emissions from a facility is 1E-05 or greater (e.g., Santa Barbara APCD, 2009), and the CT DEP multi-contaminant risk target at cleanup sites (Remediation Standard Regulations program) is also 1E-05. The highest cumulative risk from the artificial turf fields, 3.4 E-06, is well within this range of allowable and background risk from environmental toxicants.

e) Benzothiazole and a number of PAHs are the carcinogenic COPCs most likely to be field related. However, they contribute very little cancer risk due to low exposure (PAHs) or low potency (benzothiazole). Benzothiazole has not actually been shown to cause cancer but this is assumed for the purposes of the current risk assessment based upon analogy with 2-MBT.

f) Exposure assumptions for use of these fields are generally conservative (12 or 30 years of exposure at a rate of 138 days per year) and meant to assess the risk at an upper bound of plausible use of this resource. However, the average use rate is likely to be considerably lower and for many athletes would not involve extensive field use in summer months due to town leagues and school teams typically operating in spring and fall only.

Chronic non-cancer hazard indices were uniformly below one even when using screening approaches that are likely to overestimate risk such as adding across analytes with differing target effects and using conservative surrogate toxicity values. This suggests that there is no public health concern for chronic non-cancer effects. The acute hazard indices were also uniformly below one for all four scenarios. However, in the case of children playing at the indoor field, the hazard index was 0.96, with over half of this contributed by benzothiazole and the remainder comprised of small contributions from 14 different VOCs and naphthalenes. Conservative assumptions were used in several of these cases in the face of missing data – the chronic RfC for heptane, hexane and cyclohexane was used as the acute target. However, this did not add substantially to the overall hazard index. Given the limited and inconsistent acute toxicology data available, there is considerable uncertainty regarding the acute effects of benzothiazole, naphthalene and the VOCs found to be field-related. There is also uncertainty regarding how they may interact to cause irritation with at least 5 of these analytes having irritation as their primary acute response. Therefore, the potential for acute toxicity in association with the elevated concentrations seen at the indoor field is a substantial uncertainty for children using this facility.

Benzothiazole is the major rubber-related compound detected at these fields and in laboratory offgas studies. It emerges as the most consistent indicator of crumb rubber contamination of air quality.

However, the toxicology of benzothiazole has not been thoroughly investigated. It has limited mutagenicity data and those data suggest a mutagenic effect; it has a metabolic pathway that could plausibly lead to hydroxylamine formation, which may be a risk for bladder cancer. Further, its structural analogue, 2-mercaptobenzothiazole has shown positive carcinogenicity. Benzothiazole was proposed as a candidate test agent for the National Toxicology Program but this chronic testing has yet to take place. The data gaps are more numerous than actual data for this chemical and toxicity values were based upon analogy with 2-mercaptobenzothiazole for cancer effects and with formaldehyde for acute effects. Based upon this analysis, benzothiazole has conservatively been considered a low dose, mutagenic carcinogen, chronic toxicant and acute irritant. The fact that it has been an approved food additive for many years tends to decrease the level of concern in relation to a source such as artificial turf fields. However, the amount of exposure from food and the possible health effects of that exposure have not been evaluated. Appendix A contains a summary of the toxicology of benzothiazole and 2-mercaptobenzothiazole. Additional studies of benzothiazole's toxic effects would be valuable in improving future risk assessments involving volatile emissisons from crumb rubber fields and possibly other types of products and applications involving rubber.

D. Current Results in Relation to Prior Studies

The current field investigation and risk assessment is similar in approach to several previous assessments, the most relevant being the study of 3 indoor artificial turf fields in Norway (NILU, 2006), 4 outdoor artificial turf fields by USEPA (2009), and two outdoor artificial turf fields in NYC (TRC 2009; NYSDEC 2009). Each of these studies used stationary monitors on or next to the field compared to a representative off-field sample.

The Norwegian study involved an extensive array of VOCs and SVOCs including several specifically targeted because of their presence in rubber (NILU 2006). Their detections of PAHs (nanogram/m3 range), benzothiazole (low ug/m3 range up to 32 ug/m3) and VOCs (up to a high of 85 ug/m3 for toluene) are qualitatively and quantitatively similar to the list of detections in the present investigations, especially with respect to the indoor sampling (Field K – maximum toluene detect 135 ug/m3; maximum benzothiazole detect 14 ug/m3). As described above, the possible detection of benzene at the Norwegian fields in the 1-2 ug/m3 range agrees with the current benzene findings. The risk assessment conducted by the Norwegian government evaluated 3 different age windows for children beginning as early as 7 years of age, and adults (Norwegian Inst Public Health and Radium

Hospital, 2006). Their assessment encompassed inhalation as well as dermal and oral exposure. Cancer risks were driven by benzene with results very similar to the current finding (2E-06 in highest scenario); this risk was described as negligible. Carcinogenic PAHs as exemplified by benzo(a)pyrene were considered due to background sources, and non-cancer risks carried a large margin of safety. Therefore, the Norwegian government considered the exposures to indoor artificial turf fields to be within acceptable limits (Norwegian Inst Public Health and Radium Hospital, 2006). This conclusion encompassed acute risks although a formal assessment of acute exposures against acute inhalation benchmarks was not done.

USEPA performed a scoping-level field monitoring study at 4 fields, one each in Georgia, Ohio, Maryland and North Carolina under summertime conditions in 2008 (USEPA, 2009). VOC, PM_{10} and metals samples were collected on the fields in the vicinity of play activities at 1 meter height at 2 PM. Upwind background samples were also collected. VOC detections on the field were low (less than 1 ppb) with only one VOC (methyl isobutyl ketone) considered to be a field related detection. PM10 results at one field with high play activity were elevated relative to background but the results for PM10 and ambient lead were low relative to National Ambient Air Quality Standards. USEPA's conclusion was that the methodologies were successful for assessing emissions from artificial turf fields and while this study did not find any detections of health concern, the conclusions are limited by its screening nature and small amount of data collection.

Somewhat more extensive testing was conducted at two outdoor New York City artificial turf fields in late August and early Sept 2008 under warm, light-to-moderate wind conditions when ambient temperatures were in the upper 70s to low 80s F and surface temperatures on the fields were as high as 146 F (NYSDEC, 2009). Particulate and VOC samples were collected at 3 foot height above the field and at upwind locations. VOCs were also collected at the field surface. The fields were in active use at the time of sampling. In addition, dust wipe and microvacuum samples were collected from the field. A large array of VOCs, SVOCs and targeted VOCs based upon a laboratory headspace test were analyzed in the samples. The vertical and horizontal gradients of 6 rubber-related analytes were analyzed to determine if these fields show a measureable emission (e.g., higher concentration at the surface than 3 feet, higher concentration on-field than upwind). These tests failed to find clear evidence of a field related gradient and individual VOCs were generally no higher on the field than upwind. An exception was benzothiazole which had a detection of 6.5 ug/m3 on the surface of one of the fields while this analyte was non-detect upwind. Risk assessment of VOC, SVOC and PM_{10} results

failed to find elevated health risks. A separate analysis of these fields contracted by the NYC Dept of Health found similarly few detections of target analytes and in that case, the data did not warrant the conduct of a risk assessment (TRC, 2009).

These prior field investigations included a variety of different weather conditions, field ages, type of crumb rubber, and sampling and analytical procedures. Similar to the current results they did not find many detects that were clearly field-related. When they analyzed field-related and possibly field-related detects, risks were low and within the general background for urban air.

The current analysis adds to this body of research for crumb rubber fields in providing data for 4 additional outdoor fields, one additional indoor field, results for personal monitoring and a formal analysis of acute health risks. While personal monitoring was expected to provide data more relevant to actual users of the fields, the pattern of results indicated that the personal monitors were likely detecting analytes coming from the sampling equipment or host and not necessarily the field (Figure 1). Therefore, this aspect of the design may not make a significant contribution to our understanding of exposures and risks from crumb rubber fields. Our results were otherwise consistent with previous investigations in showing very low detects and risks in outdoor fields with considerably higher detections and somewhat higher risks at the indoor field. The current assessment of acute health risks, while containing a variety of uncertainties, adds to the existing database in showing some potential for acute irritation at the indoor field; this potential appears to be manageable by adequate ventilation at these facilities.

Also consistent with previous results, benzothiazole was the primary marker of rubber-related impacts on air quality. The current assessment provides a more comprehensive review of benzothiazole toxicology than previous analyses with calculations made for cancer, chronic non-cancer, and acute health risks for this analyte. These calculations suggest that benzothiazole is unlikely to be a significant contributor to cancer and chronic non-cancer risk at the concentrations detected, but that there is some uncertainty as to whether benzothiazole could create an acute health risk for children actively playing on poorly ventilated indoor fields.

E. Limitations

This investigation was established as a screen of air quality at 4 outdoor and 1 indoor field in Connecticut. This is a relatively small number of fields and sampling events. Thus, the degree to which the current results are representative of the remaining fields in Connecticut is unclear. This is especially the case for the one indoor field in that there was no active ventilation. We expect this to reflect worst case conditions that may exist in indoor facilities, especially those which do not ventilate except to decrease the buildup of heat in the summertime.

Sampling was conducted in July of 2009 with targeted conditions being sunny, warm and low wind. While this goal was accomplished, we were not able to capture a hot day typical of summer heat waves when the offgassing of VOCs might be maximized. Thus it is possible that we have not captured worst case outdoor conditions. This worst case may involve new crumb rubber as headspace offgas experiments conducted by CAES (2009) indicate that outdoor weathering plays a major role in decreasing the availability of chemicals to offgas from crumb rubber. Thus a hot, sunny, low wind day on a new artificial turf field may present the greatest exposure potential for VOCs. This would only be a potential concern for acute health risks as these conditions would not last very long. Given this potential and the possibility for heat stress to compound the effects on respiration, it is prudent for towns to construct new fields in the cooler months to give them time to weather before warm weather play. However, we emphasize this is more an uncertainty than an actual finding given that these conditions (new crumb rubber, hot weather) were not tested.

The small numbers of samples taken per field presents an additional limitation as statistical comparison between on-field and off-field detections was not possible on a field-by-field basis. However, the combined results across fields and background locations, in combination with results from prior studies, presents a consistent pattern of there being relatively few detections at outdoor fields under the tested conditions.

As stated above, there was no attempt to study the potential for ingestion of rubber-related dust from the fields by players or by young children who may be in attendance with parents watching the play. While the ingestion of crumb rubber contaminants has received some attention in previous risk assessments (California EPA 2007, Norway, 2006) this remains an area of some uncertainty for which a dust monitoring analysis (perhaps using vacuum methods) would indicate the amount of rubber contaminants available on the surface of the fields.

Another limitation is that the current investigation did not attempt to measure latex antigen in the crumb rubber or in the particulate matter collected from on field air samples. The release of latex antigen from the fields via abrasion and release of particulate rubber dust is a theoretical concern given that natural rubber contains this antigen and a substantial fraction of the population may be sensitized. Somewhat mitigating this concern is the fact that current monitoring did not detect elevated PM^{10} on the fields relative to background suggesting that there wasn't a substantial particulate emission from the fields. When this issue was examined by Norway in their artificial turf field investigation, it was described as an uncertainty for which there was insufficient data to assess. A fact sheet by the NY State DEC (2008) discussed latex allergy from the perspective that a California EPA study on guinea pig skin failed to find allergic sensitization from contact with tire rubber and that they were not aware of allergic reactions to the playing fields. A consultant to the rubber industry has published an analysis which suggests that latex allergy will not be a health concern from tire-derived particulate, in part because the way tires are made is much different than latex gloves and other forms of latex that are highly allergenic (Finley et al. 2003). Additional information on this topic is needed.

While the current risk assessment evaluates various types of risk from benzothiazole inhalation exposure, the potential for benzothiazole to induce contact sensitization was not evaluated and is currently unknown in relation to artificial turf fields. There is limited information to suggest that benzothiazole can induce dermal sensitization (see Appendix A). Given that benzothiazole may be available for skin contact from the crumb rubber and from ground crumb rubber dust, this potential for dermal sensitization is theoretically relevant to those using the turf fields. However, the rate of transfer of benzothiazole to the skin from these fields is unknown and may be low as it may tend to partition into the rubber and may require intimate contact with the skin over prolonged periods for yield substantial transfer to the skin. However, this could be facilitated by the presence of sweat.

Given the potential exposure to sensitizing chemicals - latex antigen and benzothiazole – on these fields, it is appropriate for players, coaches, parents and field operators who believe they are witnessing a respiratory or dermal reaction from use of the field to report the incident to their physician and local health department.

F. CASE Review

The Connecticut Academy of Science and Engineering (CASE) evaluated the compiled set of draft reports from the state agencies and provided comments (CASE, 2010). The main areas of CASE comments were 1) the cancer risks calculated by DPH may have been overestimates because of the inclusion of benzene detections that are likely not coming from the playing field but from the players themselves; 2) the uncertainty with respect to the benzothiazole risk assessment since so little toxicology data are available for benzothiazole; and 3) the potential for allergic reactions to occur due to the presence of latex antigen in natural rubber. To address these comments, the risk assessment describes the issues and finds that they do not change the overall conclusions and are unlikely to present added risk. For example, as described above the public is commonly exposed to rubber particles in street dust without obvious reactions to the latex in these particles, so this does not appear to be a major risk at crumb rubber fields. Nevertheless, those who think they are experiencing an allergic reaction to the fields (skin rash, breathing difficulty) should report this to their doctor and to local health officials.

VI. Conclusions

The current investigation was successful in detecting field-related VOCs and SVOCs at both outdoor and indoor fields. In particular, benzothiazole is clearly field-related while certain PAHs and possibly several VOCs may also be field-related. Risk estimates are higher for the indoor as compared to the outdoor fields and higher for children than adults.

Overall, these health risks are low especially given the conservative screening level nature of the assessment, and well within the level of risk from air pollution commonly experienced by the general public. Specifically:

- The major contributors to cancer risk, benzene and methylene chloride, are not clearly field-related and appear to result, at least in part, from factors unique to personal monitoring (offgasing from sampling equipment or exhaled breath);
- Chronic non-cancer hazard indices were uniformly below unity for all scenarios suggesting that there is little concern for chronic non-cancer risk.
- The hazard index for acute effects was also below unity in all cases but was close to unity for children playing at the indoor field. This hazard index is driven by benzothiazole which is

clearly field related. There is considerable uncertainty regarding the acute irritant and other toxic effects of benzothiazole and most VOCs.

The elevated exposures to benzothiazole, naphthalenes and several VOCs found at the indoor field presents the most significant uncertainty stemming from the current investigation and risk assessment. This field was not under active ventilation. It would be prudent for indoor artificial turf fields to receive adequate ventilation to mitigate the additional exposure to rubber-related volatile components possible in the indoor environment. Also, given the potential for weathering to reduce the offgassing of VOCs, it would be prudent for outdoor fields to be established in cooler months, giving them time to weather before the high heat conditions of mid-summer. Finally, dermal or respiratory allergic reactions that occur on the fields and may be field related should be reported to the family physician and local health department.

VII. References

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Tables

Table 1. Analytes found above background in personal monitor samples but not in any other field-related or crumb rubber samples and thus excluded from the COPC List (for all VOC results see UCHC, 2010)

Analyte	Highest Personal Monitor Detection (ug/m3)
1-Ethyl-4-methylbenzene	1.37
1,2,4- & 1,3,5-trimethylbenzenes	2.16
1,2-Dichloropropane	1.14
Acrolein	3.89
Bromoform	13.29
Ethyl acetate	11.87
Propene	0.89
Tetrachloroethylene	3.29
Tetrahydrofuran	3.5
Trichloroethylene	23.4
Vinyl acetate	2.95

Table 2. COPCs at the Four Outdoor Fields (A, B, C, D)

COPC	Max Detect (ug/m3)	Ratio to Highest Background	Location & Type of Sample	# Fields with elevation	Detected Offgas Study
VOCs					
Acetone	52.2	4.2	A: Personal	4	Yes
Benzene	1.56	1.7	B: Personal	1	Yes
Carbon Disulfide[1,2]	0.47	2.9	B: Personal	1	No
Chloromethane[2]	1.7	1.5	A: Personal	1	No
Cyclohexane[2]	17.5	10.3	B: Personal	1	No
Ethyl benzene	4.29	3.6	B: Personal	1	Yes
Heptane	5.72	10.8	B: Personal	1	No
Hexane	31.3	4.2	B: Personal	2	Yes
Methylene Cl	14.1	12.8	B: Personal	1	Yes
MEK	2.94	8.2	B: Personal	3	No
MIBK	3.39	3.3	B: Personal	1	Yes
Styrene	1.96	2.1	B: Personal	1	Yes
Toluene	52.7	34.4	B: Personal	2	Yes
Xylenes	14.7	12.2	B: Personal	1	
Semi-VOCs					
Targeted					
Benzothiazole	1.2	1.7	D: 6 inch	4	Yes
PAHs					
Acenaphthylene	6.6E-03	8.6	D: Stationary	1	NA
Benz(a)anthracene	1.1E-04	3.7	B: Stationary	1	NA
Benzo(a)pyrene	1.9E-04	3.8	B: Stationary	2	NA
Benzo(b)fluoranthene	2.1E-04	3.0	B: Stationary	2	NA
Benzo(e)pyrene	2.6E-04	4.3	B: Stationary	2	NA
Benzo(ghi)perylene	1.4E-04	2.3	A: Stationary	1	NA
Benzo(k)fluoranthene	8E-05	2.0	C: Stationary	1	NA
Chrysene	3.4E-04	4.9	B: Stationary	2	NA
Fluoranthene	6.8E-03	4.6	D: Stationary	2	NA
1-Methylnaphthalene	9.3E-03	1.3	D: Stationary	1	NA
Pyrene	6.9E-03	2.2	D: Stationary	1	NA
Miscellaneous[3]					
Total Sum	1.33	---	D: Stationary	3	NA

[1]Slightly higher Personal Monitor and much higher background detects were found at Field C but that field had pesticide spraying in the background area during sampling.

[2]These volatile analytes were included as COPCs even though they were not detected in the laboratory offgas studies because there was at least some evidence that they were present in field-related samples besides personal monitoring samples.

[3]93 compounds including aliphatics, hopanes, terpenes, pristanes

Table 3. COPCs at the One Indoor Field (K)

COPC	Max On-field Detect (ug/m3)	Ratio to Highest Background	Type of Sample
VOCs			
Acetone	92.5	7.4	Personal
Benzene	1.18	1.3	Personal
Carbon Disulfide[1]	0.9	5.6	Stationary 6", 3'
Chloromethane	1.57	1.4	Personal
Cyclohexane	10.3	6.1	Personal
Ethyl benzene	4.77	3.9	Personal
Heptane	10.22	19.3	Personal
Hexane	11.25	1.5	Personal
Methylene Cl	10.3	9.4	Personal
MEK	44.2	123	Personal
MIBK	36	35	Stationary 6", 3'
Styrene	3.53	3.8	Personal
Toluene	135	88	Personal
Xylenes	15.7	13	Personal
Semi-VOCs			
Targeted			
Benzothiazole	14	19.8	Stationary 6"
Butylated hydroxytoluene	3.9	13.9	Stationary 3'
PAHs			
Acenaphthene	1.74E-02	22.7	Stationary
Acenaphthylene	6.8E-03	8.8	Stationary
Fluoranthene	5.60E-03	3.8	Stationary
Fluorene	5.40E-02	15	Stationary
Naphthalene	1.13E-01	6.6	Stationary
1-Methylnaphthalene	1.14E-01	16.5	Stationary
2-Methylnaphthalene	6.30E-02	19.1	Stationary
2,6-Dimethylnaphthalene	2.90E-02	2.8	Stationary
Phenanthrene	3.20E-02	2.4	Stationary
Pyrene	1.18E-02	3.8	Stationary
Miscellaneous[3]			
Total Sum	4.4	---	Stationary

[1]Much higher background detect at Field C but that field had pesticide spraying in the background area during sampling.

[2]These volatile analytes were included as COPCs even though they were not detected in the laboratory offgas studies because there was at least some evidence that they were present in field-related samples besides personal monitoring samples.

[3]93 compounds including aliphatics, hopanes, terpenes, pristanes

Table 4. Exposure Parameters

Parameter	Child	Adult	Basis
Age	12	30	Child – midpoint of 6-18 yr range
Years exposed	12	30	Child – youth to high school soccer; Adult – 90th% residence at one location
Exposure time per event	3 hr	3 hr	Time for soccer match or practice
Days exposed per year	138	138	4 day/wk for 8 months (spring, fall soccer + 2 months in summer)
Days exposed per year VOCs	69	69	VOC offgas only in the 4 warm months for outdoor fields; no adjustment for indoor fields
Ventilation adjustment	3.96	2.64	Child – Adult factor* child factor Adult – moderate exercise
Averaging time (cancer)	25550 days	25550 days	Entire lifespan – 70 yrs
Averaging time (non-cancer)	4380 days	10950 days	Entire exposure period

Table 5. Toxicity Values for COPCs

Analyte	Cancer Unit Risk ($ug/m3^{-1}$)	Source	RfC (ug/m3)	Source	Acute Target (ug/m3)	Source
VOCs						
Acetone	NA	---	**1050**	IRIS RfD for renal tox converted to RfC with 3 fold additional UF due to lower dose effects in gavage study not used by IRIS and lack of RfC.	**8000**	CTAEC for irritation based upon human irritation threshold divided by 3 to convert 1 hr AEC to 3 hr time frame
Benzene	**1.84E-05**	Cal OEHHA value is 3.7 fold higher than IRIS upper bound unit risk – values averaged	**9.6**	ATSDR chronic MRL (2007) for immunotox, which is 3 fold lower than IRIS RfC.	**177**	CTAEC for immunotox/3 to yield 3 hr target; this value is between ATSDR and Cal OEHHA acutes
Carbon disulfide	**NA**	---	**700**	IRIS for peripheral neurotoxicicity	**1000**	CTAEC for neurotoxicity and odor threshold
Chloromethane	**1.7E-06**	Cal Prop 65 only value available - mutagenic	**90**	IRIS for CNS toxicity	**1000**	ATSDR acute MRL for neurotox
Cyclohexane	**NA**	---	**6000**	IRIS for reproductive effects	**6000**	No acute guideline available so RfC used
Heptane	**NA**	---	**700**	No tox values available; Hexane as conservative surrogate	**700**	No acute guideline available so RfC used
Hexane	**NA**	---	**700**	IRIS for neurotoxicity	**700**	No acute guideline available so RfC used
Methylene Cl	**4.7E-07**	IRIS	**400**	Cal OEHHA for cardiovascular and nervous system tox	**4700**	Cal acute REL for neurotox divided by 3 for 1 hr to 3hr tox value conversion

Analyte	Cancer Unit Risk ($ug/m3^{-1}$)	Source	RfC (ug/m3)	Source	Acute Target (ug/m3)	Source
Methyl Ethyl Ketone	**NA**	---	**1000**	Cal OEHHA for reproductive effects; value is 5x < IRIS	**3233**	CTAEC fpr irritation in human chamber study divided by 3 for 1hr to 3 hr conversion
Methyl isobutyl ketone	**NA**	---	**80**	USEPA HEAST, 1997 for liver/kidney tox	**4550**	CTAEC for irritation and headache divided by 3 for 1 hr to 3 hr conversion
Styrene	**NA**	---	**100**	IRIS RfC for neurotox divided by 10 for poss carcinogenicity	**4133**	CTAEC for neurotox divided by 3 for 1 hr to 3 hr conversion
Toluene	**NA**	---	**300**	ATSDR MRL for neurotox which is lower than Cal or IRIS	**3800**	ATSDR acute MRL for neurotox
Xylene	**NA**	---	**100**	IRIS for neurotoxicity	**7333**	Cal acute REL for irritation & neurotox divided by 3 for 1 hr to 3 hr conversion
Targeted SVOCs						
Benzothiazole	**1.8E-07**	Whittaker et al. 2004 unit risk for 2-MBT	**18**	NYS (2009) value based on subchronic oral NOAEL and route extrapolation	**110**	CTDPH value based on 18x higher RD-50 than formaldehyde & 10x UF for data gaps
Butylated hydroxytoluene	**NA**	---	**175**	European ADI of 0.05 mg/kg/d and route extrapolation	**NA**	---
PAHs						
Acenaphthene	**NA**	---	**210**	IRIS RfD for hepatotoxicity with route extrapolation	**NA**	---
Acenaphthylene	**NA**	---	**210**	No data; acenaphthene as surrogate	**NA**	---

Analyte	Cancer Unit Risk ($ug/m3^{-1}$)	Source	RfC (ug/m3)	Source	Acute Target (ug/m3)	Source
Benz(a)anthracene	**1.1E-04**	Unit risk for B(a)P with relative potency of 0.1 from USEPA, 1993	**110**	Pyrene IRIS RfD converted to RfC as surrogate as lowest RfC available	**NA**	---
Benzo(a)pyrene	**1.1E-03**	CalEPA (1999) inhalation unit risk from hamster inhalation bioassay	**110**	Pyrene IRIS RfD converted to RfC as surrogate as lowest RfC available	**NA**	---
Benzo(e)pyrene	**NA**	---	**110**	Pyrene IRIS RfD converted to RfC as surrogate as lowest RfC available	**NA**	---
Benzo(b)fluoranthene	**1.1E-04**	Unit risk for B(a)P with relative potency of 0.1 from USEPA, 1993	**110**	Pyrene IRIS RfD converted to RfC as surrogate as lowest RfC available	**NA**	---
Benzo(k)fluoranthene	**1.1E-05**	Unit risk for B(a)P with relative potency of 0.01 from USEPA, 1993	**110**	Pyrene IRIS RfD converted to RfC as surrogate as lowest RfC available	**NA**	---
Benzo(ghi)perylene	**NA**	---	**110**	Pyrene IRIS RfD converted to RfC as surrogate as lowest RfC available	**NA**	---
Chrysene	**1.1E-05**	Unit risk for B(a)P with relative potency of 0.001 from USEPA, 1993	**110**	Pyrene IRIS RfD converted to RfC as surrogate as lowest RfC available	**NA**	---
Fluoranthene	**NA**	---	**140**	IRIS RfD for kidney, liver, blood effects and route extrapolation	**NA**	---
Fluorene	**NA**	---	**140**	IRIS RfD for blood effects and route	**NA**	---

				extrapolation		

Analyte	Cancer Unit Risk ($ug/m3^{-1}$)	Source	RfC (ug/m3)	Source	Acute Target (ug/m3)	Source
Naphthalene	**3.4E-05**	Unit risk from Cal OEHHA	**3**	IRIS RfC for respiratory hyperplasia	**117**	CTAEC for acute tox to Clara cells in mice divided by 3 to convert 1 hr to 3hr target
1-Methylnaphthalene	**3.4E-05**	No values available, used naphthalene as surrogate	**3**	No values available, used naphthalene as surrogate	**117**	No values available, used naphthalene as surrogate
2-Methylnaphthalene	**3.4E-05**	No values available, used naphthalene as surrogate	**3**	No values available, used naphthalene as surrogate	**117**	No values available, used naphthalene as surrogate
2,6-Dimethylnaphthalene	**3.4E-05**	No values available, used naphthalene as surrogate	**3**	No values available, used naphthalene as surrogate	**117**	No values available, used naphthalene as surrogate
Phenanthrene	**NA**	---	**110**	Pyrene IRIS RfD converted to RfC as surrogate as lowest RfC available	**NA**	---
Pyrene	**NA**	---	**110**	IRIS RfD for renal pathology and route extrapolation	**NA**	---
Miscellaneous SVOCs (aliphatics, hopanes, terpenes, pristanes)	**NA**	---	**110**	No values available, used pyrene as conservative surrogate	**NA**	---

IRIS = USEPA Integrated Risk Information System online database of toxicity values; CalOEHHA = California Office of Environmental Health Hazard Assessment; ATSDR MRL = Agency for Toxic Substances and Disease Registry Minimum Risk Level as provided in the Toxicological Profile; CTAEC = CT DPH acute exposure concentrations for 1 hr exposure developed in 2000 and

updated in 2010 for targeted analytes.

Table 6. Summary of Artificial Turf Field Risks

	Child Outdoor	**Child Indoor**	**Adult Outdoor**	**Adult Indoor**
Cancer Risk	1.9E-06	3.4E-06	1.1-06	2.3E-06
Non-Cancer	0.09	0.48	0.057	0.32
Acute	0.44	0.96	0.29	0.63
Key Analytes				
Cancer	Benzene 73% MethyleneCl 17% Chloro Me 7% BenzothiaZ <1%	Benzene 61% MethyleneCl 14% Chloro Me 8% BenzothiaZ 7%	Benzene 73% MethyleneCl 17% Chloro Me 7% BenzothiaZ <1%	Benzene 51% MethyleneCl 11% Chloro Me 6% BenzothiaZ 6%
Non-cancer	Toluene 19%	Toluene 18%	Toluene 19%	Toluene 18%
Acute	BenzothiaZ 9.8% Benzene 16% Toluene 6.3%	BenzothiaZ 54% Benzene 5.6% Toluene 7.6%	BenzothiaZ 9.8% Benzene 16% Toluene 6.3%	BenzothiaZ 54% Benzene 5.6% Toluene 7.6%

Figures

Figure 1. Acrolein Detects at Artificial Turf Fields (no detects at fields C or D)

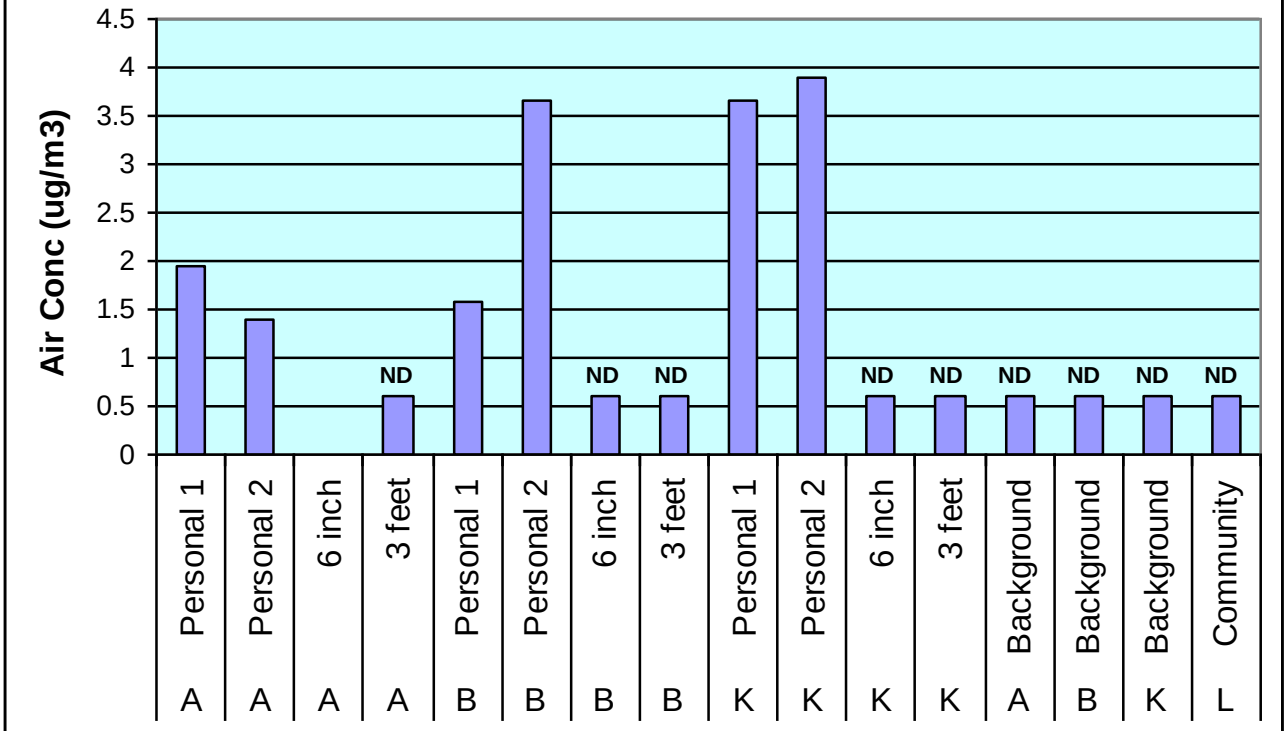
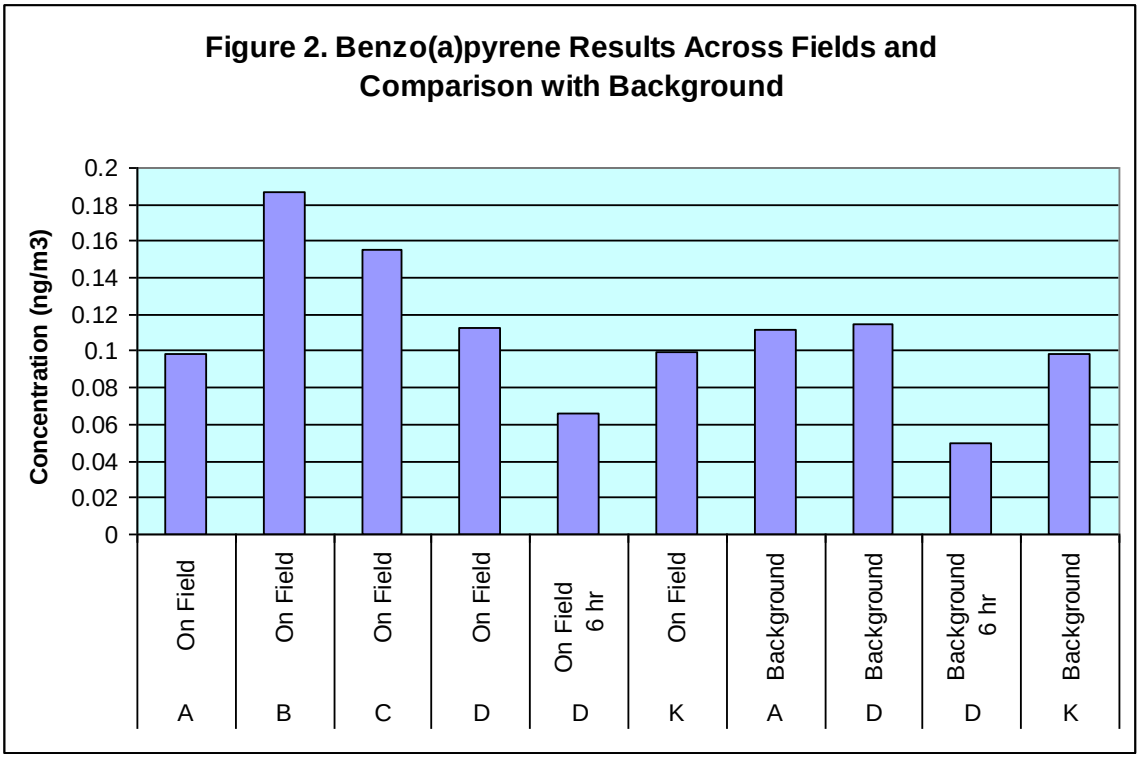


Figure 2. Benzo(a)pyrene Results Across Fields and Comparison with Background



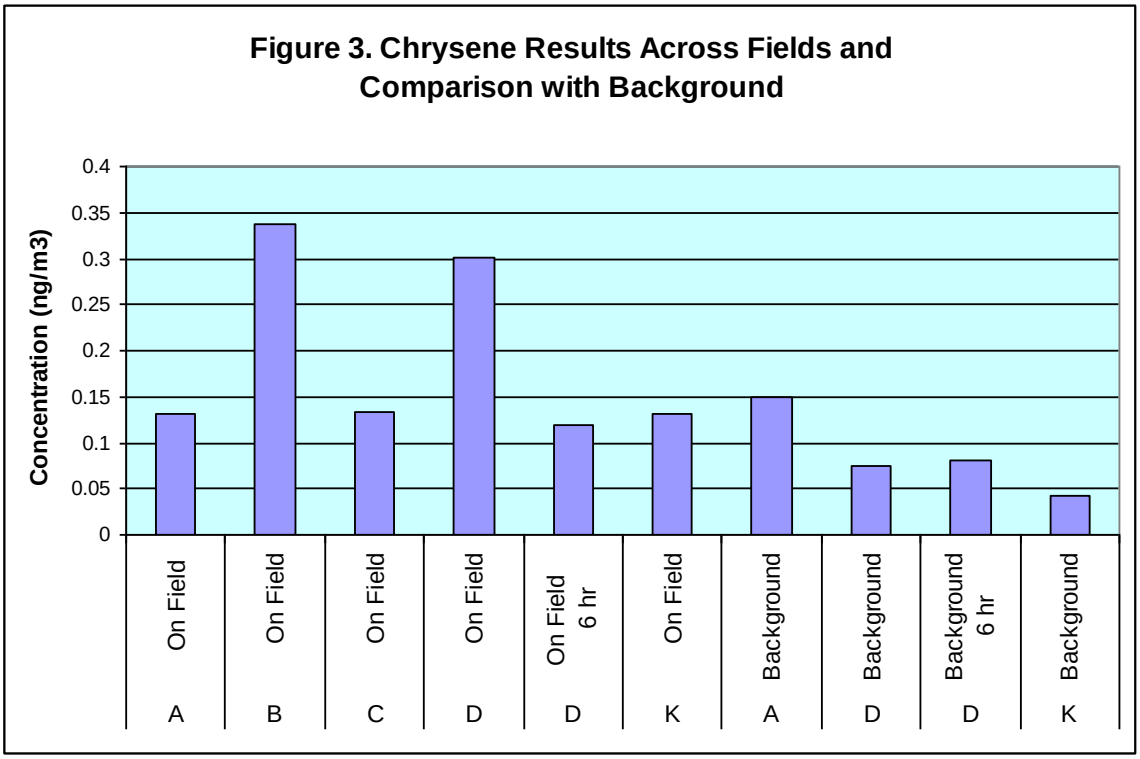
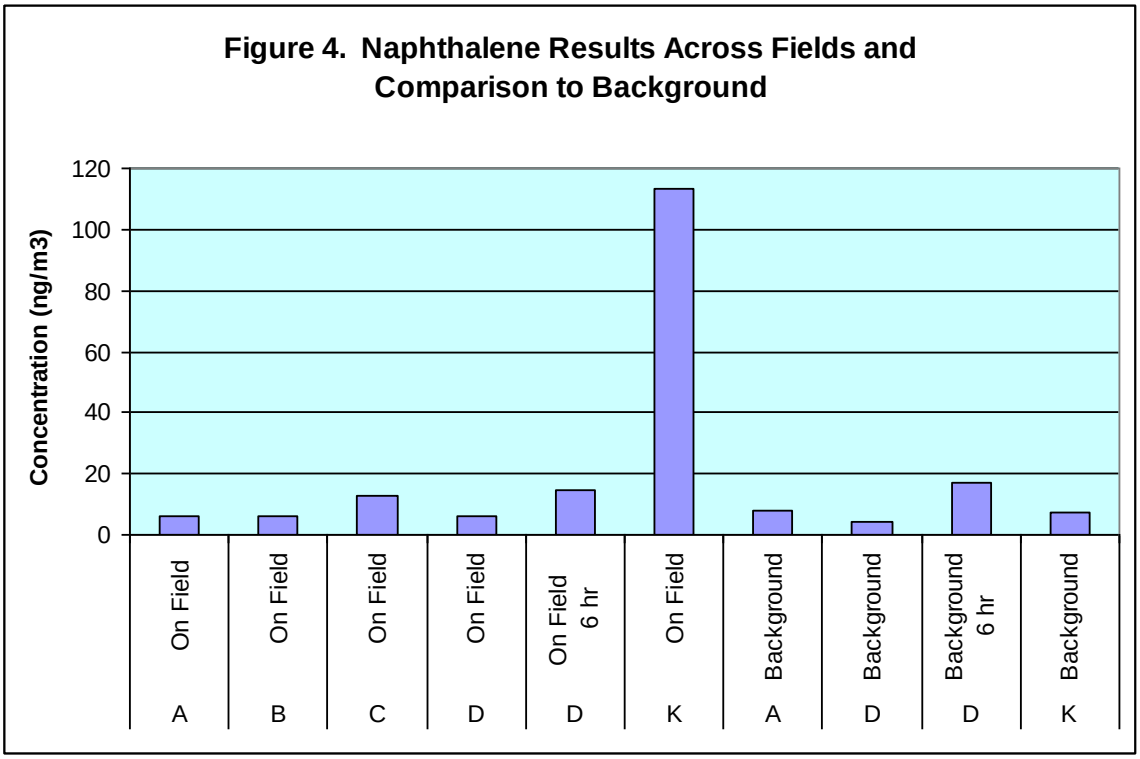


Figure 3. Chrysene Results Across Fields and Comparison with Background
Concentration (ng/m3)
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
On Field A
On Field B
On Field C
On Field D
On Field 6 hr D
On Field K
Background A
Background D
Background 6 hr D
Background K

Figure 4. Naphthalene Results Across Fields and Comparison to Background



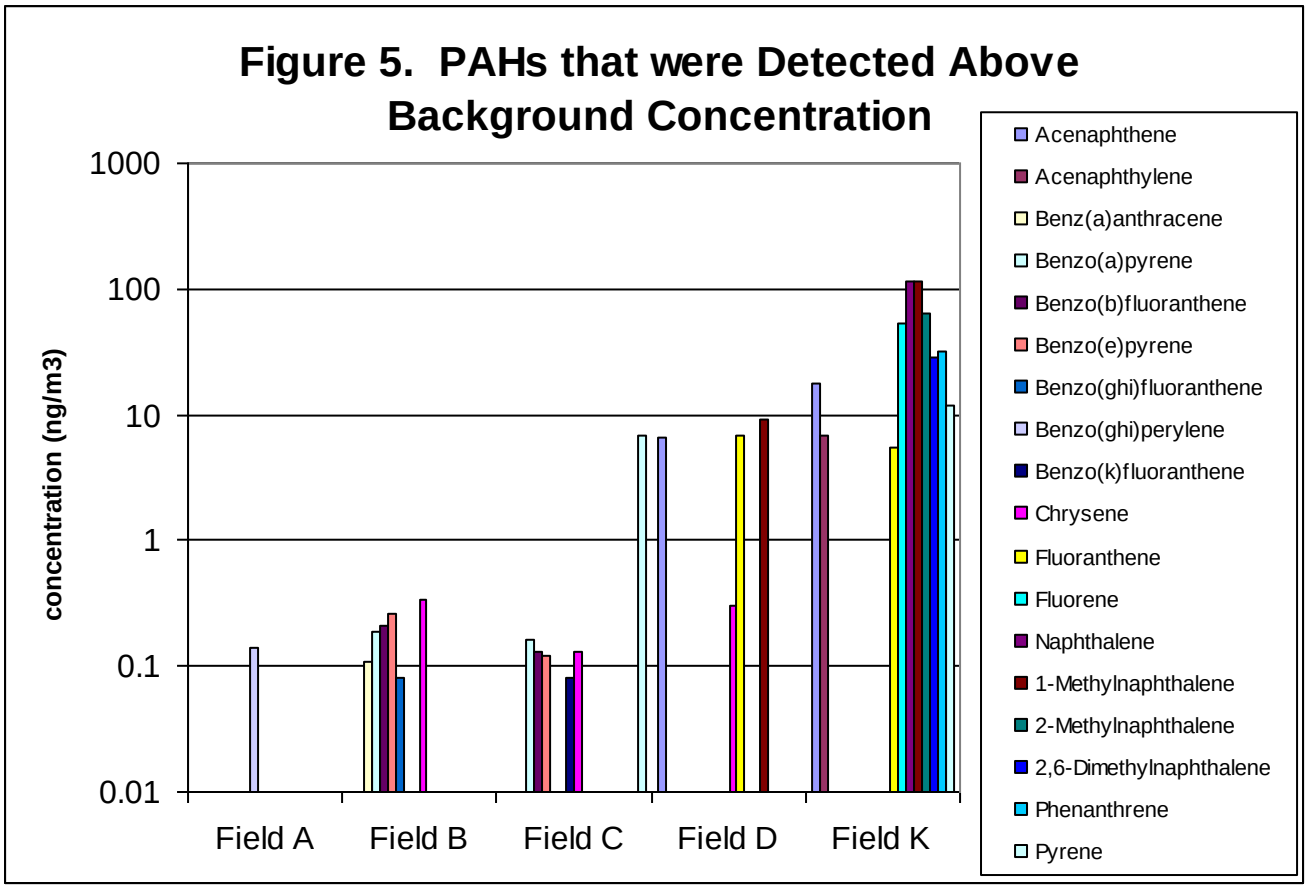


Figure 5. PAHs that were Detected Above Background Concentration
concentration (ng/m3)
1000
100
10
1
0.1
0.01
Field A
Field B
Field C
Field D
Field K
Acenaphthene
Acenaphthylene
Benz(a)anthracene
Benzo(a)pyrene
Benzo(b)fluoranthene
Benzo(e)pyrene
Benzo(ghi)fluoranthene
Benzo(ghi)perylene
Benzo(k)fluoranthene
Chrysene
Fluoranthene
Fluorene
Naphthalene
1-Methylnaphthalene
2-Methylnaphthalene
2,6-Dimethylnaphthalene
Phenanthrene
Pyrene

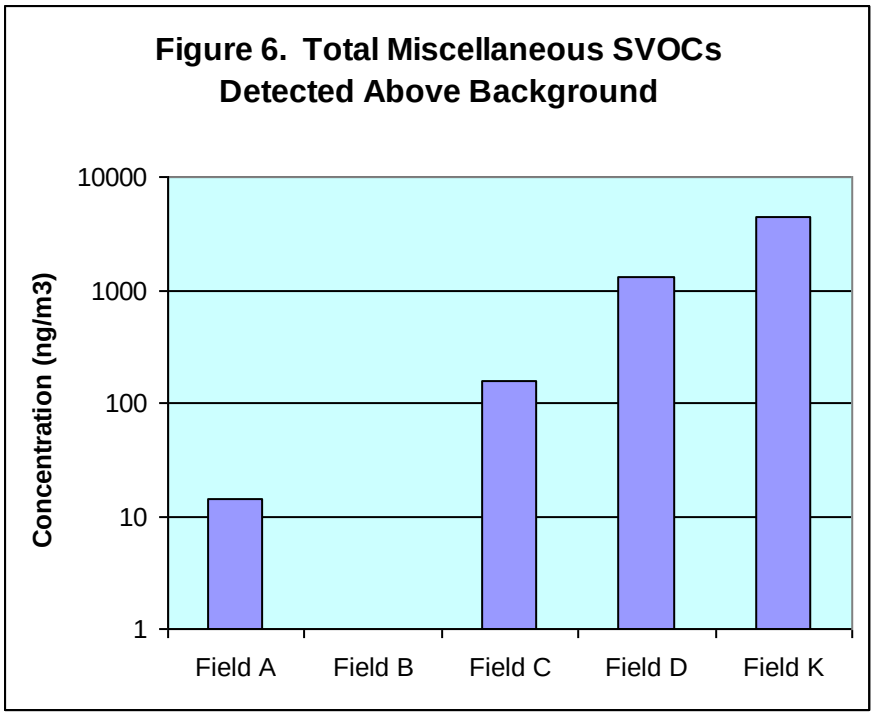
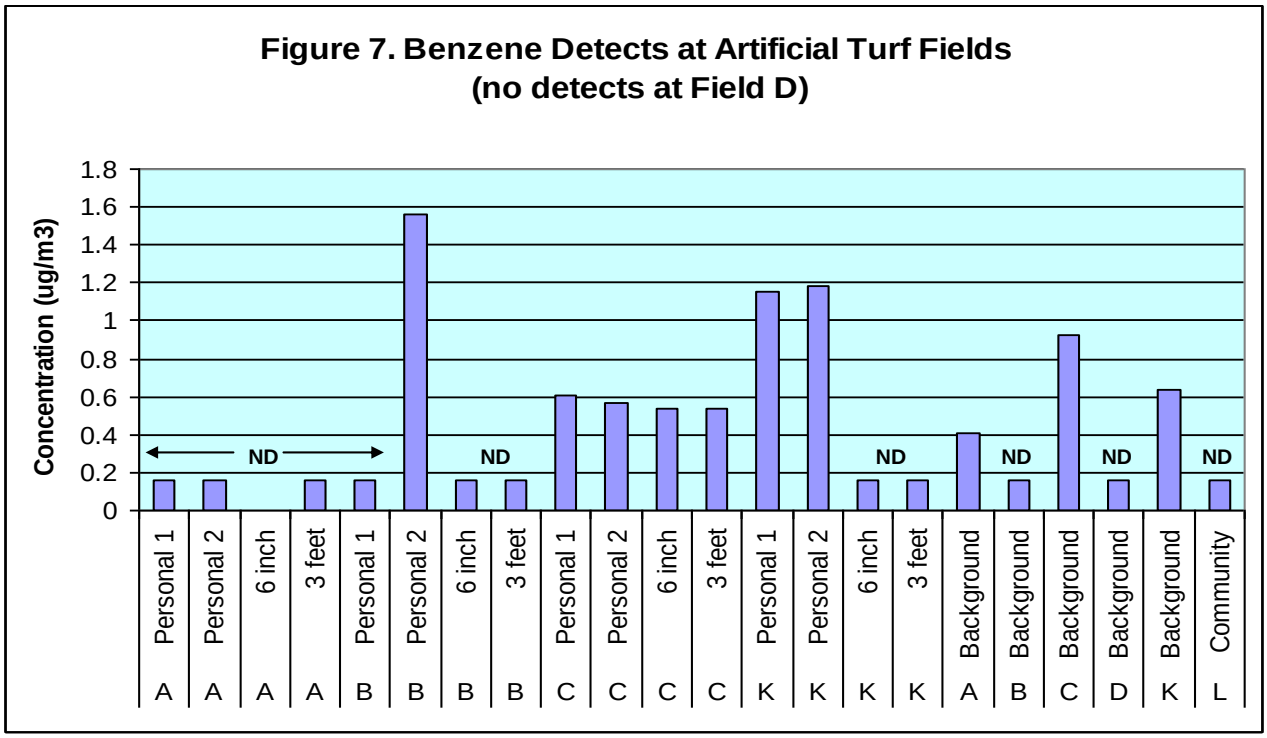


Figure 6. Total Miscellaneous SVOCs Detected Above Background
Concentration (ng/m3)
10000
1000
100
10
1
Field A
Field B
Field C
Field D
Field K

Figure 7. Benzene Detects at Artificial Turf Fields (no detects at Field D)



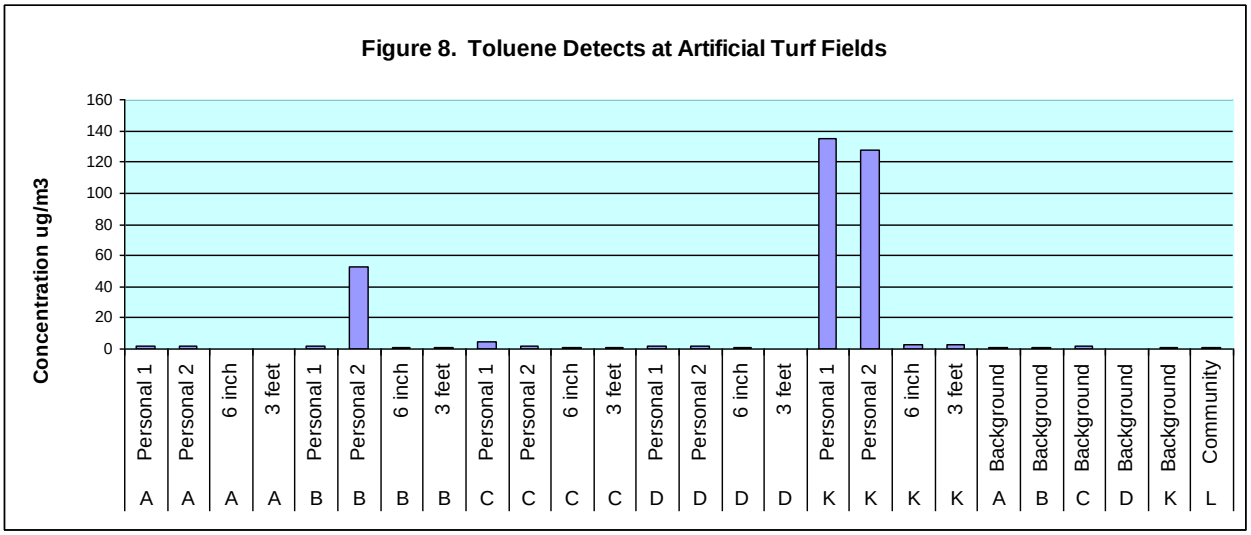
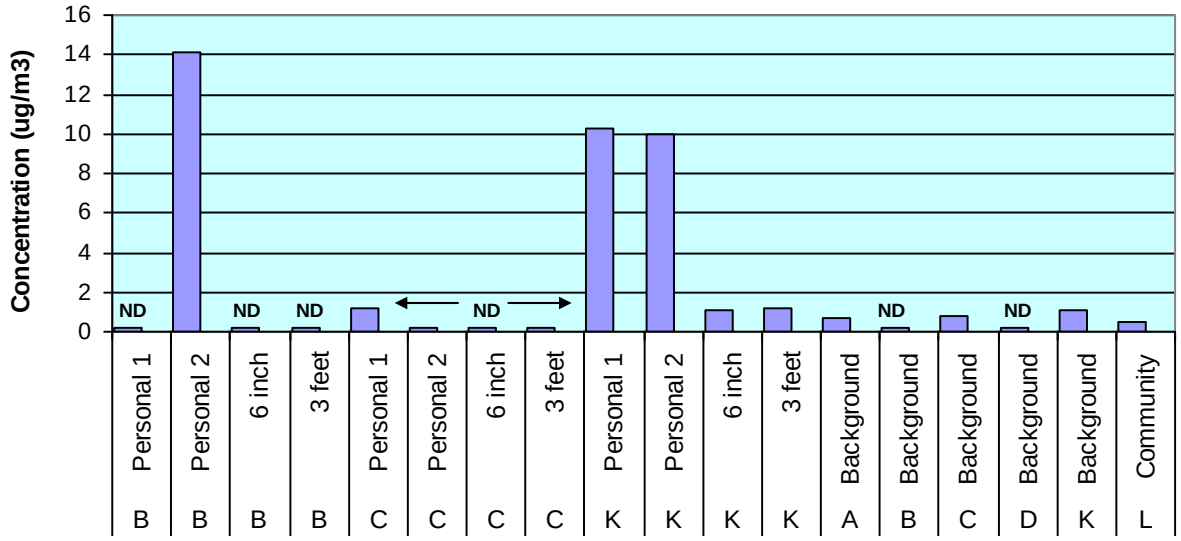


Figure 8. Toluene Detects at Artificial Turf Fields
Concentration ug/m3
160
140
120
100
80
60
40
20
0
Personal 1 A
Personal 2 A
6 inch A
3 feet A
Personal 1 B
Personal 2 B
6 inch B
3 feet B
Personal 1 C
Personal 2 C
6 inch C
3 feet C
Personal 1 D
Personal 2 D
6 inch D
3 feet D
Personal 1 K
Personal 2 K
6 inch K
3 feet K
Background A
Background B
Background C
Background D
Background K
Community L

Figure 9. Methylene Chloride Detects at Artificial Turf Fields (no detects at Fields A or D)



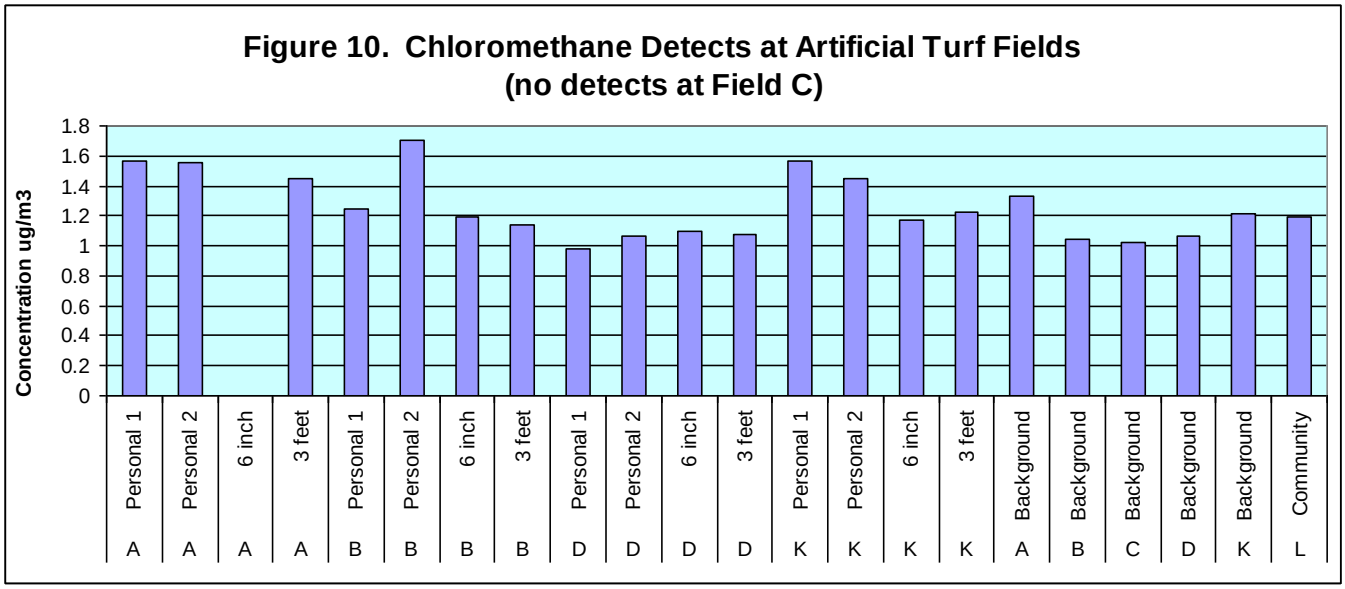


Figure 10. Chloromethane Detects at Artificial Turf Fields
(no detects at Field C)
Concentration ug/m3
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Personal 1 A
Personal 2 A
6 inch A
3 feet A
Personal 1 B
Personal 2 B
6 inch B
3 feet B
Personal 1 D
Personal 2 D
6 inch D
3 feet D
Personal 1 K
Personal 2 K
6 inch K
3 feet K
Background A
Background B
Background C
Background D
Background K
Community L

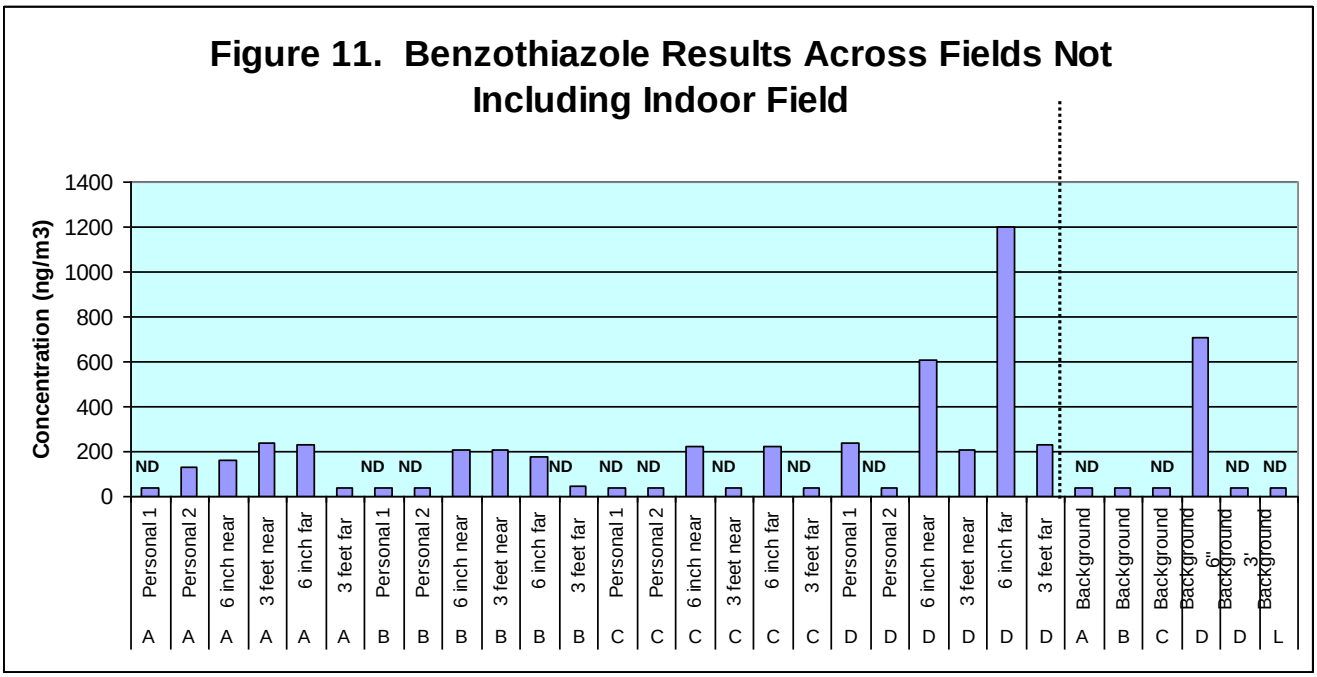


Figure 11. Benzothiazole Results Across Fields Not Including Indoor Field
Concentration (ng/m3)
1400
1200
1000
800
600
400
200
0
ND
Personal 1 A
Personal 2 A
6 inch near A
3 feet near A
6 inch far A
3 feet far A
Personal 1 B
Personal 2 B
6 inch near B
3 feet near B
6 inch far B
3 feet far B
Personal 1 C
Personal 2 C
6 inch near C
3 feet near C
6 inch far C
3 feet far C
Personal 1 D
Personal 2 D
6 inch near D
3 feet near D
6 inch far D
3 feet far D
Background A
Background B
Background C
Background D
6" Background D
3' Background D
Background L

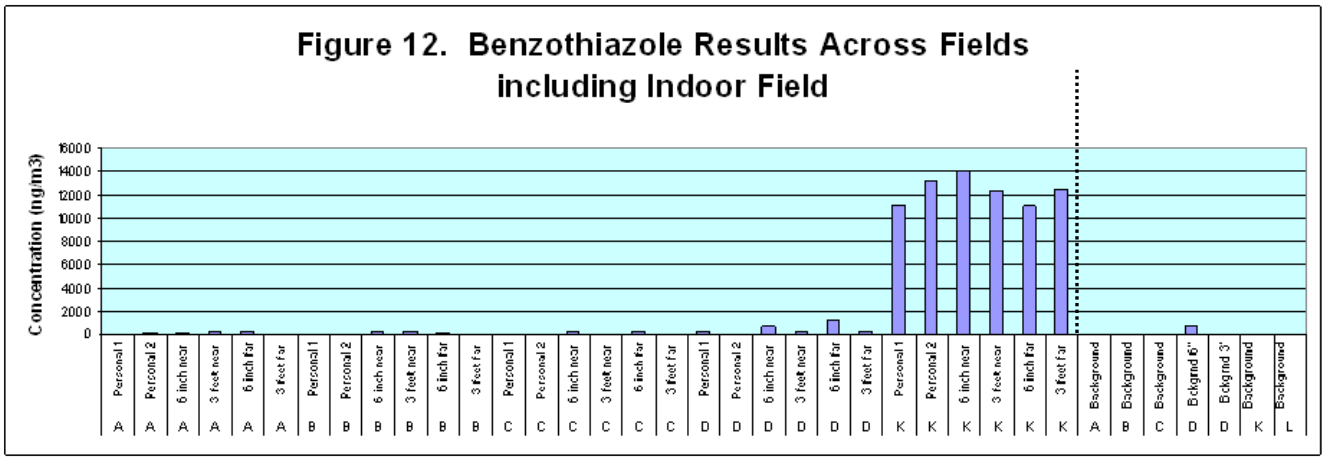


Figure 12. Benzothiazole Results Across Fields including Indoor Field
Concentration (ng/m3)
16000
14000
12000
10000
8000
6000
4000
2000
0
Personal 1
Personal 2
6 inch near
3 feet near
6 inch far
3 feet far
Background
Bckgrnd 6"
Bckgrnd 3'

Figure 13. Cancer Risk Estimates for Indoor and Outdoor Turf Fields

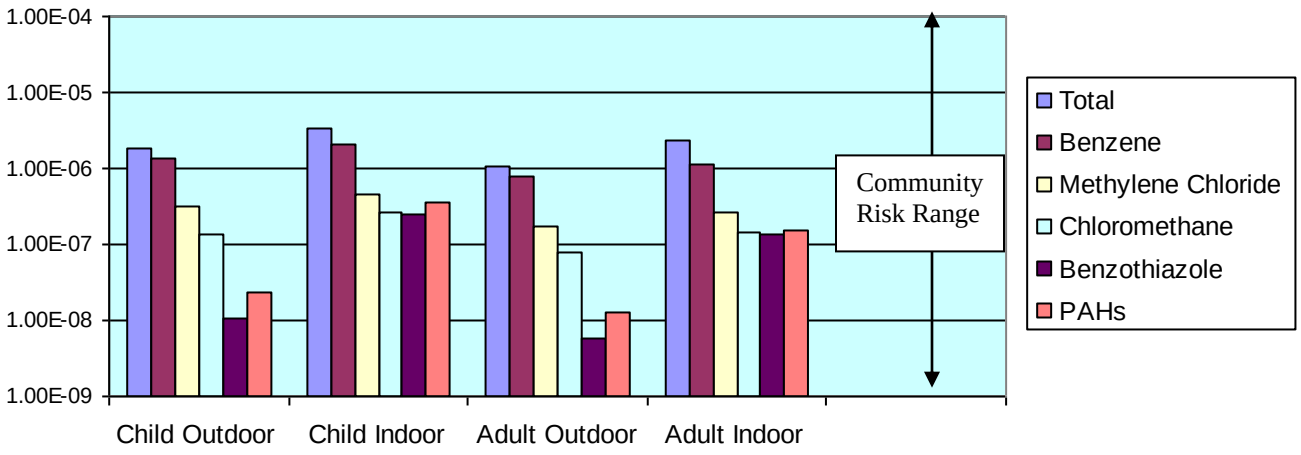
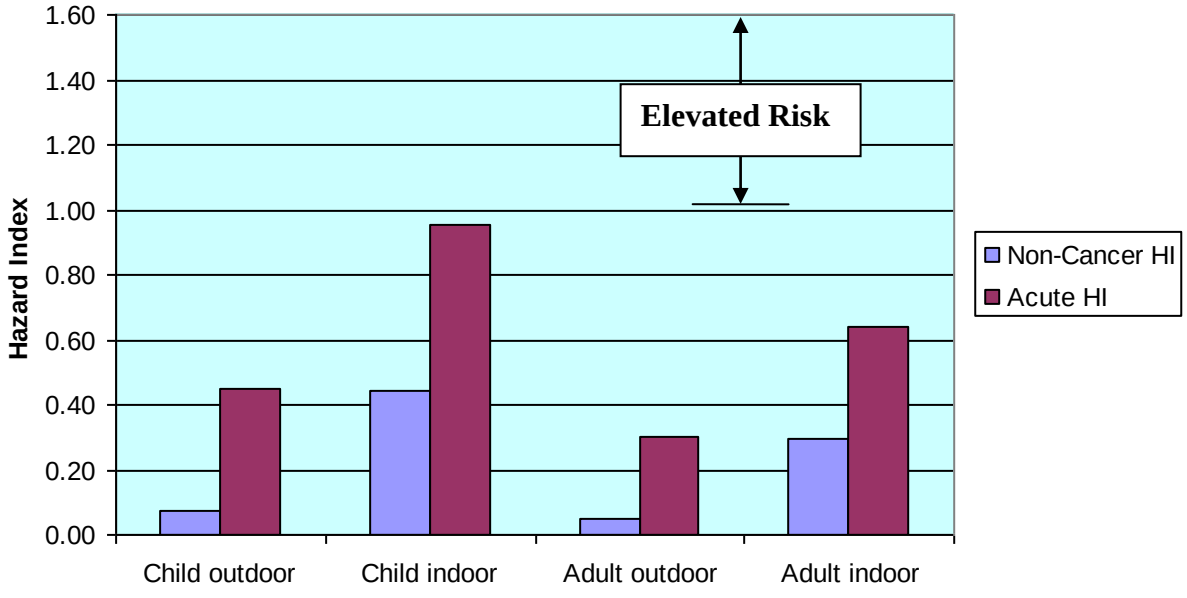


Figure 14. Hazard Indices for Non-Cancer and Acute Risk at Aritifical Turf Fields



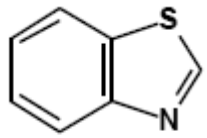
Appendix A

Benzothiazole Toxicity Profile

Properties and Uses:

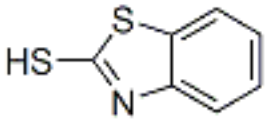
Benzothiazole (BTZ) is a clear yellow liquid with a sulfur or rubbery odor (Lewis, 1993). Its heterocyclic structure provides multiple functionality and opportunites for derivatization making it a good starting material for other industrial chemicals. It is a precursor for rubber accelerators, a component of cyanine dyes ("Summary", 1997), as slimicides in the paper and pulp industry, and is used in the production of certain fungicides, herbicides, antifungal agents and pharmaceuticals (Bellavia et al., 2000; Seo et. al., 2007). It imparts a meaty, nutty or coffee taste and so is used in various foods as a flavoring agent at levels up to 0.5 ppm (Good Scent 2010; "Summary", 1997). It has limited solubility in water (4.3 g/L at 25 C) and has low volatility (vapor pressure 0.014 mm Hg at 25 C).

The toxicology database for BTZ is limited to short-term, sub-acute and mutagenicity studies. A related chemical, 2-mercaptobenzothiazole (2MBT), has been subjected to more extensive testing and so is used as a surrogate for some endpoints. Their structures are presented below:



C_7H_5NS

Benzothiazole: CAS Registry Number 95-16-9



$C_7H_5NS_2$

2-Mercaptobenzothiazole: CAS Registry Number 149-30-4

Exposure to BZT

The most common exposure source is ingestion of foods, beverages, and pharmaceuticals which contain BZT. Inhalation is also common as it is present in tobacco smoke and can be in the atmosphere from the wearing down of tires. Atmospheric forms can include both the particle bound and gaseous states. Workers in rubber processing facilities are particularly likely to receive inhalation and dermal exposure. The amount of background exposure to the general public in the diet or general environment has not been calculated and it doesn't appear to have been the subject of human biomonitoring studies. In addition, there are very few environmental measurements. It has been detected in relation in artificial turf fields in several previous studies. A concentration of 6.5 ug/m3 was found in one air sample taken at the surface of an artificial turf field in New York City under summertime conditions and in full sun. BZT was not detected at this field at the 3 foot height or at the surface of another field tested under similar conditions (NYS, 2009). A Norwegian study (2005) detected 3.4-31.7 ug/m^3 in its air samples at indoor artificial turf fields. In addition, the Connecticut Agricultural Experiment Station (2009) conducted headspace analyses of crumb rubber at elevated temperature (60 C) in which it found BZT at 226 ng/ml, which was much greater than the other detected analytes. Thus, in spite of its limited volatility, BZT has the potential to offgas from crumb rubber used in artificial turf fields and can present as the major rubber-specific component. This makes BZT of particular interest for exposure and risk assessment in the current study.

Toxicokinetics of Benzothiazole and 2MBT:

Both BZT and 2MBT are well absorbed and rapidly excreted, with metabolites appearing primarily in urine and small amounts in feces. Evaluation of BZT metabolism and elimination is limited to one study in guinea pigs which identified metabolites in urine after i.p. administration of 30 mg/kg daily for 4 days ((Wilson et al.1991). Urinary metabolites consisted of the heterocyclic ring scission products 2-methylmercaptoaniline, 2-methylsufinylaniline, 2-methylsulfonylaniline, 2-methylsulphinylphenylhydroxylamine and 2-methylsulphonylphenylhydroxylamine. These BZT metabolites are reactive, particularly with respect to the free amine (NH_2) group and the hydroxylamine (NHOH) group present in several metabolites. Sulphate and glucuronide conjugates of the above metabolites were also recovered in the guinea pig study

.

In contrast to BZT, MBT undergoes metabolism primarily via conjugation of the sulfhydryl moiety at the 2- position. This leads to a variety of 2' glucuronide and sulfate benzothiazole metabolites (El Dareer et al. 1989; Fukuoka and Tanaka 1987; Fukuoka et al. 1995). Thus, instead of ring scission, 2MBT undergoes conjugation of the side chain functional group leaving the ring structure intact.

Toxicity of BZT including relevant data from 2MBT:

A variety of acute studies in animals demonstrate a moderate degree of acute toxicity. The median oral lethal dose (LD_{50}) is between 380 and 900 mg/kg. Intravenous, intraperitoneal and dermal LD_{50s} are lower, ranging from 95 mg/kg to 200 mg/kg. The acute toxicity of BZT is characterized by CNS and respiratory depression as well as liver and kidney toxicity (Bogert, 1931, Zapór , 2005). A repeat dose study in mice recieving 110 mg/kg injections for a week documented liver necrosis and cloudy swelling of the renal tubules (Guess and O'Leary 1969). Higher doses resulted in peripheral vasodilation, extensive salivation and convulsions. 2MBT acute toxicity studies have had variable results with the oral LD_{50} in rats ranging from 100 to 7500 mg/kg. Since most of the reported LD_{50}s are over 1000 mg/kg, 2MBT appears to be somewhat less acutely toxic than BZT.

BZT appears to be a skin allergen as positive dermatitis reactions occurred in 17 of 43 subjects treated topically (Bogert and Husted 1931). The dermatitis was often delayed in appearance and after fading would reoccur on the initial site if BZT were reapplied to a different site. Similarly, 2MBT has demonstrated contact dermatitis and sensitization in humans across a number of studies (Wang and Suskind, 1988 and Fregert and Scog, 1962)**.**

BZT may be a nose and throat irritant based upon anectoal reports of greater irritation of asphalt-rubber workers laying pavement than when non-rubber products are used for this purpose; The greater irritation was attributed to the presence of BZT but other rubber ingredients may have contributed to the effect, especially at the high temperatures used to surface roads (Bustnes et al. 2007).

A study by the U.S. Consumer Product Safety Commission (1996) tested the sensory and pulmonary irritation of various compounds in carpet, one of them being BZT. Mice were exposed to 60 minutes of contaminated air and sensory irritation indicated by a decline in breathing rate. The concentration which caused a fifty percent decrease in respiratory rate (RD-50) for benzothiazole was 235.4 mg/m^3. The positive control in this test was formaldehyde, which had an RD-50 of 12.9 mg/m3. Although this represents a limited dataset, this study suggests that BZT is moderately irritating to the respiratory tract relative to a known irritant such as formaldehyde (18 fold less potent).

Mutagenicity

The only study of BZT mutagenicity was in Salmonella typhimurium in which a mutagenic response was detected in Salmonella strain TA1537 in the presence of a metabolic activation (S9) system (Kinae et al. 1981). 2MBT has been evaluated more extensively with negative results in 4 different Ames tests with and without metabolic activation; none of the Salmonella strains showed evidence of mutagenesis (Whittaker et al. 2004). However, it was mutagenic in two different mouse lymphoma assays with metabolic activation and it was a clastogen in the Chinese Hamster Ovary chromosomal aberration assay. This in vitro finding of clastogenicity did not transfer to in vivo as the

mouse micronucleus test was negative in two different mouse strains (Whittaker et al. 2004). .

Sub-Chronic and Chronic Toxicity and Cancer

CTDPH could not find subchronic or chronic studies for BZT in the published literature or governmental reports. However, an unpublished 90 dietary study conducted in 1971 was submitted by the Flavor and Extract Manufacturer's Assoc. to the World Health Organization (WHO). WHO 2003 cites this study (Morgareide 1971) as providing evidence that groups of 15 FDRL rats/sex were dosed in their diet with BZT at 5.1 mg/kg/d. Animals were observed for clinical signs of toxicity and at 6 and 12 weeks blood was taken for standard hematology and clinical chemistry parameters. Histopathology was performed at study termination. Further methodological details are not available. WHO reports that the test diet was well tolerated with no alterations in blood parameters, organ weights or histopathology due to this level of BZT. Therefore, the oral dose of 5.1 mg/kg/d was considered a NOAEL. As described below, this NOAEL was used by New York State to develop an inhalation toxicity value for BZT of 18 ug/m3 in their 2009 risk assessment.

2MBT has been more thoroughly tested, with its database covering 90 day and 2 year studies in rats and mice by the oral dose route. The NTP series of studies on 2MBT involved gavage exposure in corn oil vehicle. The most sensitive effect in the 90 day studies was hepatomegaly in the livers of male rats seen at the lowest dose (188 mg/kg/d) and higher (NTP, 1988). A 20 month dietary study in mice found 2MBT associated with microscopic changes in the kidney at a dose of 58 mg/kg/d and higher with the NOAEL reported to be 14 mg/kg/d (Whittaker et al. 2004).

BZT has not been subjected to cancer bioassay testing but has been listed as a high priority for such testing by the National Toxicology Program (NTP, 1997). However, this ranking has yet to be followed up with actual testing. The high ranking appears to stem from the potential for widespread exposure in food and certain occupations, as well as the single positive mutagenicity test (Kinae et al. 1981). 2MBT has been fully tested by NTP for carcinogenic potential in two year rat and mouse studies. Gavage doses of 188 or 375 mg/kg/d 5 days a week in female rats or 375 or 750 mg/kg/d in male rats yielded a variety of compound-related tumors including tumors of the adrenal gland (both sexes), pituitary gland (both sexes), pancreas, preputial gland and leukemia (males only). Further, male rats had a low incidence of renal transitional cell tumors that appear to be compound related due to the fact that these tumors are rare in the controls. In mice dosed by gavage with 375 or 750 mg/kg/d, the only positive response was in female liver and this was only at the low dose.

The carcinogenicity of 2MBT has been evaluated in several epidemiological studies

involving workplace exposure. Bladder cancer excess has been seen in studies of the rubber industry in relation to worker exposure to vulcanization inhibitors, accelerators, antioxidants and other specialty chemicals (Sorahan 2008). A chemical factory in North Wales has been a particular focus because it produces chemicals for the rubber industry. Departments working with aromatic amines (aniline, o-toluidine, phenyl-beta-naphthylamine) and 2MBT were the main focus, with excess bladder cancer risk seen for o-toluidine and 2MBT exposure (Sorahan 2008). A follow-up study of 363 of these workers exposed to 2MBT found higher bladder cancer incidence (SRR= 253, range 131-441) and mortality (SMR = 374, range 162-737) relative to national rates for this gender and age group. 2MBT exposure was also associated with intestinal cancer and multiple myeloma in these workers (Sorahan 2009). A study of 600 West Virginia rubber chemical workers with exposure to 2MBT found a large increase in bladder cancer mortality for workers exposed to both 2MBT and 4-aminobiphenyl (SMR=27.1, 95% CI 11.7-53.4) but not to 2MBT alone (Collins et al. 1999). This suggests an interactive effect with aromatic amines, a factor which may have also been at work in the North Wales cohort.

Teratogenicity & Reproductive Effects

BZT has not been tested in developmental or reproductive studies. 2MBT has been tested in a range of studies with mixed results. This may be because a variety of different test protocols were used. A one generation range finding study in rats administered 2MBT in the diet found effects on body weight at all dose levels with the LOAEL reported to be 357 mg/kg/d. However, the followup developmental study was conducted by gavage and found no body weight effects and a NOAEL of 300 mg/kg/d for non-specific clinical effects (urine staining, salivation) (Monsanto, undated, as summarized in Whittaker et al. 2004). Rabbit developmental studies via gavage did not find fetotoxicity or teratogenicity in spite of evidence of maternal toxicity (decreased body weight at all doses down to 150 mg/kg/d; maternal lethality at 1000 mg/kg/d). A rat developmental study also did not find gross external malformations from gavage doses as high as 2200 mg/kg/d to dams during gestation, although this was only a range finding study without detailed examination of fetuses (Monsanto, undated – as summarized in Whittaker, et al. 2004). When rats were administered 200 mg/kg/day 2-MBT via i.p. injection for days 1-15 of gestation, there was no evidence of maternal toxicity, fetal toxicity or teratogenesis (Hardin et al., 1981).

In a 2- generation dietary study rats were administered 4 different concentrations of 2-MBT ranging from 179 to 1071 mg/kg. Exposure began 10 weeks before mating and continued until 88 days postweaning. The LOAEL for decreased body weight gain was determined to be 179 mg/kg/day across the two generations but there were no effects on fertility or other reproductive parameters (Springborn, undated).

However, other studies suggest fetotoxic and teratogenic effects. Up to 20% of the chicken embryos injected with 0.10-2.0 umol/egg of 2MBT were found to have malformations such as eye, neck and back defects as well as open coelom (Korhonen et

al. 1983). A study in mice evaluated the response of several different strains to doses up to 464 mg/kg/day on day 6-15 of gestation. 2MBT was associated with fetal malformations in two of the strains with confirmatory results for one of the strains in a followup study (Bionetics Research Labs, 1968). In a high dose study in mice, subcutaneous injection of 4176 mg/kg/day of 2-MBT on days 6-14 of gestation yielded fetotoxicity and a number of fetal malformations involving the ears, eyes and gastrointestinal tract (National Technical Information Service, 1990).

Toxicity Values for Cancer and Non-Cancer Effects

There are no regulatory criteria or guidelines for BZT in drinking water or ambient air. The European Food Safety Authority has a limit for BZT in food of 0.5 ppm but this is not associated with a specific toxicity value or acceptable daily intake (ADI). Toxicity values of 3 types are possible for BZT as follows:

1) Acute non-cancer – the main concerns from short-term exposure are the potential for ocular and respiratory irritation and the potential for sensitization. While there is no information on whether BZT is a respiratory sensitizer, limited data on its respiratory irritant effects exist in mice. The RD-50 studies from CPSC (1996) indicate that BZT is 18 times less irritating in this mouse model system than is formaldehyde, a reactive irritant gas that also causes hypersensitivity. While indoor and outdoor air targets vary for formaldehyde, the CT DPH has a general guideline value for homes and schools of 50 ppb (61.5 ug/m3) which is intended to prevent irritant and hypersensitivity reactions. Based upon the ratio of RD-50 results, a target BZT acute air guideline would be in the vicinity of 1100 ug/m3. However, the acute database for BZT is very limited with no data in humans. This and the considerable uncertainty in the extrapolation across chemicals, especially with regard to relative sensitization potential, leads to a 10 fold database uncertainty factor and a short-term air target of **110 ug/m3**.
2) Chronic non-cancer – an RfD type value has been derived by New York State as part of its artificial turf exposure and risk assessment report (NYS 2009). They use the unpublished and EFSA-reviewed 1971 study with BZT in which the only dose level (5.1 mg/kg/d) was without effect to derive an RfD of 5 ug/kg/d based upon a cumulative 1000 fold uncertainty factor. This target is based upon the only BZT repeat dose study available and that study has very limited reporting of data and only one dose level. However, it is consistent with and supported by the NOAEL for kidney effects in the 2 year NTP bioassay of 2-MBT: 14 mg/kg/d. If a cumulative 1000 fold were applied to that NOAEL (10 for cross-species, 10 for intra-species, 10 for datagaps and extrapolation across chemicals), the oral target woulg be 14 ug/kg/d, similar to the 5 ug/kg/d derived from the only BZT study. This value can be converted via dose-route extrapolation to an **RfC of 18 ug/m3**.
3) Cancer Unit Risk – there is uncertainty with respect to the potential carcinogenicity of BZT given its positive mutagenicity and the carcinogenic effects of 2MBT. A 10 fold uncertainty factor could be applied to the RfC

described above, but that would make the cumulative uncertainty factor 10000 fold which is higher than the range commonly used by USEPA in establishing RfDs (up to 3000 fold). Use of a 3 fold carcinogen uncertainty factor leads to an RfC of 6 ug/m3 or 1.7E-03 mg/kg/d. That target is coincidentally the 1 in a million cancer risk level based upon the 2MBT cancer slope factor derived by Whittaker et al (2004). That calculation is:
1.7E-03 mg/kg/d * 6.34E-04/mg-kg-d = 1.08 E-06 cancer risk.

The cancer slope factor for 2MBT of **6.34E-04/mg-kg-d** is based upon the rat renal tumor response in the NTP bioassay described above. Whittaker et al. (2004) used linear multi-stage modeling from a benchmark dose point of departure as is standard practice for low dose modeling for genotoxic carcinogens. Whittaker et al. claim this to be the most sensitive endpoint but they did not show cancer slope comparisons for the other tumor targets in the NTP study. DPH converted the oral slope factor to an inhalation unit risk by assuming 20 m3 air breathed per day for a 70 kg adult.

Discussion

Overall, the studies conducted on BZT and 2-MBT demonstrate that BZT may pose a health risk at sufficiently high exposure. Exposure to BZT may result in CNS depression, liver and kidney damage, dermatitis and pulmonary irritation. BZT has the potential to be mutagenic and carcinogenic. This latter conclusion is predicated to some degree on analogy with 2MBT, an imperfect comparison due to differences in structure and metabolic pathways. The mechanistic concern with BZT is ring opening from oxidative metabolism with the formation of hydroxylamines, which are known risk factors for bladder cancer. 2MBT undergoes side chain conjugation leaving the ring structure intact. In spite of these metabolic differences, the main cancer target of 2MBT in human studies has been the bladder, with renal cancer a key target in rats. Therefore, there may be overlap in the spectrum of toxic and carcinogenic effects caused by these related thiazoles.

The large degree of uncertainty in the toxicology database is somewhat mitigated by the fact that BZT exposure is common in foods and has a relatively high acceptable daily intake as set by FDA. However, studies of BZT exposure or health effects from food consumption have not been reported.

The toxicity values derived presently for BZT for acute and longer term exposure are likely to be health protective. The RfD for BZT derived by NYS (2009) makes reasonable use of the only repeat dose study and the RfD so derived is consistent with a possible RfD derivation for 2MBT. The lack of cancer bioassay data for BZT would normally preclude its entry into cancer risk assessment, creating the implicit assumption that it has zero potency. Our use of the potency factor for 2MBT allows this potential carcinogenicity to be factored into the risk assessment. While there remains a large

degree of uncertainty in BZT toxic effects and potency, the current approaches are a reasonable starting point for including BZT in a crumb rubber risk assessment.

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Appendix B

Toxicology Values for COPCs - Cancer

Chemical	Cancer IRIS	CalEPA	Other	Selected Value
VOCs				
Acetone	---	---	---	---
Benzene	7.80E-06	2.90E-05		1.84E-05
Carbon Disulfide	---	---	---	---
Chloro methane	---	---	1.70E-06	1.70E-06
Cyclohexane	---	---	---	---
Heptane	---	---	---	---
Hexane	---	---	---	---
Methylene Chloride	4.70E-07	1.00E-06		4.70E-07
Methyl ethyl ketone	---	---	---	---
Methyl isobuytyl ketone	---	---	---	---
Styrene	---	---	---	---
Toluene	---	---	---	---
Xylenes	---	---	---	---
SVOCs - Targeted				
Benzothiazole	NA	NA	1.81E-07	1.81E-07
BHT	---	---	---	---
SVOCs - PAHs				
Acenaphthene	---	---	---	---
Acenaphthylene	---	---	---	---

Benz(a)anthracene	---	---	1.10E-04	1.10E-04
Benzo(a)pyrene	---	1.10E-03	---	1.10E-03
Benzo(b)fluoranthene	---	---	1.10E-04	1.10E-04
Benzo(e)pyrene	---	---	---	---
Benzo(ghi)perylene	---	---	---	---
Benzo(k)fluoranthene	---	---	1.10E-05	1.10E-05
Chrysene	---	---	1.10E-06	1.10E-06
Fluoranthene	---	---	---	---
Fluorene	---	---	---	---
Naphthalene	---	3.40E-05	---	3.40E-05
1-Methylnaphthalene	---	---	3.40E-05	3.40E-05
2-Methylnaphthalene	---	---	3.40E-05	3.40E-05
2,6-Dimethylnaphthalene	---	---	3.40E-05	3.40E-05
Phenanthrene	---	---	---	---
Pyrene	---	---	---	---

SVOCs - Miscellaneous (aliphatics, hopanes, pristanes, terpenes)

Total SVOC miscellaneous	---	---	---	---

Toxicology Values for COPCs – Non-Cancer

Chemical	IRIS	ATSDR	CalEPA	Other	Selected Value
Acetone	3150	31000	na		1050
Benzene	30	9.6	60		9.6
Carbon Disulfide	700	930	700		700
Chloro methane	90	103	---		90
Cyclohexane	6000	NA	NA		6000
Heptane	NA	NA	NA		700
Hexane	700	2100	---		700
Methylene Chloride	---	1043	400		400
Methyl ethyl ketone	5000	NA	1000		1000
Methyl isobuytyl ketone	NA	NA	NA	80	80
Styrene	1000	850	900	100	100
Toluene	400	300	400		300
Xylenes	100	217	700		100
Benzothiazole	NA	NA	NA	18	18
BHT	NA	NA	NA	175	175
Acenaphthene	210				
Acenaphthylene	210				

Benz(a)anthracene							110			
Benzo(a)pyrene							110			
Benzo(b)fluoranthene							110			
Benzo(e)pyrene							110			
Benzo(ghi)perylene							110			
Benzo(k)fluoranthene							110			
Chrysene							110			
Fluoranthene		140								
Fluorene		140								
Naphthalene		3		3.7		9			3	
1-Methylnaphthalene	NA		NA		NA		3		3	
2-Methylnaphthalene	NA		NA		NA		3		3	
2,6-Dimethylnaphthalene	NA		NA		NA		3		3	
Phenanthrene							110			
Pyrene		110					110			
Total SVOC miscell		110					110			

Toxicology Values for COPCs – Acute Targets

Chemical	Non-Cancer - ACUTE AEGL-1	ATSDR	CalEPA	CTDPH	Selected Value
Acetone		62000	na	24000	8000
Benzene		28.8	1300	530	176.6666667
Carbon Disulfide		---	6200	1000	1000
Chloro methane		1000	NA	10000	1000
Cyclohexane		NA	NA	NA	6000
Heptane		NA	NA	NA	
Hexane		NA	NA	NA	700
Methylene Chloride		10000	14000	21000	4666.666667
Methyl ethyl ketone			13000	9700	3233.333333
Methyl isobuytyl ketone		na	na	13650	4550
Styrene		8500	21000	12400	4133.333333
Toluene		3800	37000	45000	3800
Xylenes		4300	22000	22000	7333.333333
Benzothiazole		NA	NA	110	110
BHT		NA	NA	NA	---

Acenaphthene	---	---	---	---
Acenaphthylene	---	---	---	---
Benz(a)anthracene	---	---	---	---
Benzo(a)pyrene	---	---	---	---
Benzo(b)fluoranthene	---	---	---	---
Benzo(e)pyrene	---	---	---	---
Benzo(ghi)perylene	---	---	---	---
Benzo(k)fluoranthene	---	---	---	---
Chrysene	---	---	---	---
Fluoranthene	---	---	---	---
Fluorene	---	---	---	---
Naphthalene	---	---	350	117
1-Methylnaphthalene	---	---	---	117
2-Methylnaphthalene	---	---	---	117
2,6-Dimethylnaphthalene	---	---	---	117
Phenanthrene	---	---	---	---
Pyrene	---	---	---	---
Miscell SVOCs	---	---	---	---

Children's Exposure and Risk Calculations

Indoor Field (K)

Concentration Adjustment for Child exposure Scenario

Chemical	Max Conc	Loc of Max	Hrs per day	Exp Freq	Exp Dur	Child Vent Adjment	Hrs per day	Avg Time - Cancer	Avg Time - Non-Cancer	Adj Conc - Cancer	Adjusted Conc - Non-Cancer	Cancer Unit Risk	RfC	Acute RfC	Cancer Risk	Non-Cancer Risk	Acute Risk
VOCs (ug/m3)																	
Acetone	92.5	NA	3	138	12	3.96	24	25550	4380	2.9677	17.3114	---	1050	8000	---	0.0165	0.0458
Benzene	1.18	NA	3	138	12	3.96	24	25550	4380	0.0379	0.22084	5.52E-05	9.6	88	2.09E-06	0.023	0.0531
Carbon Disulfide	0.9	NA	3	138	12	3.96	24	25550	4380	0.0289	0.16844	---	700	1000	---	0.0002	0.0036
Chloro methane	1.57	NA	3	138	12	3.96	24	25550	4380	0.0504	0.29383	5.10E-06	90	1000	2.57E-07	0.0033	0.0062
Cyclohexane	10.3	NA	3	138	12	3.96	24	25550	4380	0.3305	1.92765	---	6000	6000	---	0.0003	0.0068
Ethyl benzene	4.77	NA	3	138	12	3.96	24	25550	4380	0.153	0.89271	---	700	700	---	0.0013	0.027
Heptane	10.22	NA	3	138	12	3.96	24	25550	4380	0.3279	1.91268	---	700	700	---	0.0027	0.0578
Hexane	11.25	NA	3	138	12	3.96	24	25550	4380	0.3609	2.10545	---	700	700	---	0.003	0.0636
Methylene Chloride	10.3	NA	3	138	12	3.96	24	25550	4380	0.3305	1.92765	1.41E-06	400	4666	4.66E-07	0.0048	0.0087
Methyl ethyl ketone	44.15	NA	3	138	12	3.96	24	25550	4380	1.4165	8.2627	---	1000	3233	---	0.0083	0.0541
Methyl isobutyl ketone	36	NA	3	138	12	3.96	24	25550	4380	1.155	6.73742	---	80	4550	---	0.0842	0.0313
Styrene	3.53	NA	3	138	12	3.96	24	25550	4380	0.1133	0.66064	---	100	4133	---	0.0066	0.0034
Toluene	135	NA	3	138	12	3.96	24	25550	4380	4.3312	25.2653	---	300	7500	---	0.0842	0.0713
xylenes	15.66	NA	3	138	12	3.96	24	25550	4380	0.5024	2.93078	---	100	7333	---	0.0293	0.0085

SVOCs (ug/m3)																
Benzothiazole	14	NA	3	138	12	3.96	24	25550	4380	0.4492	2.62011	5.40E-07	18	110	2.43E-07	0.1456	0.504
Butylated hydroxytoluene	3.9	NA	3	138	12	3.96	24	25550	4380	0.1251	0.72989		210	---	---	0.0035	---
PAHs (ug/m3)																
Acenaphthene	1.70E-02	NA	3	138	12	3.96	24	25550	4380	0.0005	0.00318	---	210	---	---	2E-05	---
Acenaphthylene	0.0068	NA										---	210	---			---
Benz(a)anthracene	ND	NA	3	138	12	3.96	24	25550	4380	---	---	3.30E-04	110	---	---	---	---
Benzo(a)pyrene	ND	NA	3	138	12	3.96	24	25550	4380	---	---	3.30E-03	110	---	---	---	---
Benzo(b)fluoranthene	ND	NA	3	138	12	3.96	24	25550	4380	---	---	3.30E-04	110	---	---	---	---
Benzo(e)pyrene	ND	NA	3	138	12	3.96	24	25550	4380	---	---	---	110	---	---	---	---
Benzo(ghi)perylene	ND	NA	3	138	12	3.96	24	25550	4380	---	---	---	110	---	---	---	---
Benzo(k)fluoranthene	ND	NA	3	138	12	3.96	24	25550	4380	---	---	3.30E-05	110	---	---	---	---
Chrysene	ND	NA	3	138	12	3.96	24	25550	4380	---	---	3.30E-06	110	---	---	---	---
Fluoranthene	5.60E-03	NA	3	138	12	3.96	24	25550	4380	0.0002	0.00105	---	140	---	---	7E-06	---
Fluorene	5.40E-02	NA	3	138	12	3.96	24	25550	4380	0.0017	0.01011	---	140	---	---	7E-05	---
Naphthalene	1.13E-01	NA	3	138	12	3.96	24	25550	4380	0.0036	0.02115	3.40E-05	3	117	1.23E-07	0.007	0.0038
1-Methylnaphthalene	1.14E-01	NA	3	138	12	3.96	24	25550	4380	0.0037	0.02134	3.40E-05	3	117	1.24E-07	0.0071	0.0039

2-Methylnaphthalene	6.30E-02	NA	3	138	12	3.96	24	25550	4380	0.002	0.01179	3.40E-05	3	117	6.87E-08	0.0039	0.0021
2,6-Dimethylnaphthalene	2.90E-02	NA	3	138	12	3.96	24	25550	4380	0.0009	0.00543	3.40E-05	3	117	3.16E-08	0.0018	0.0010
Phenanthrene	3.20E-02	NA	3	138	12	3.96	24	25550	4380	0.001	0.00599	---	110	---	---	5E-05	---
Pyrene	1.18E-02	NA	3	138	12	3.96	24	25550	4380	0.0004	0.00222	---	110	---	---	2E-05	---
SVOCs - Miscellaneous (aliphatics, hopanes, pristanes, terpenes)																	
Total miscellaneous SVOCs	4.4	NA	3	138	12	3.96	24	25550	4380	0.1412	0.82346	---	110	---	---	0.0075	---

Children's Exposure and Risk Calculations

Outdoor Field - Child

Concentration Adjustment for Child exposure Scenario

Chemical	Max Conc	Location of Max	Hrs per day	Exp Freq	Exp Dur	Child Vent Ad	Hrs per day	Avg Time - Cancer	Avg Time - Non-Cancer	Adjusted Conc - Cancer	Adjusted Conc - Non-Cancer	Cancer Unit Risk	RfC	Acute RfC	Cancer Risk	Non-Cancer Risk	Acute Risk
VOCs (ug/m3)																	
Acetone	52.2	A - Personal	3	69	12	3.96	24	25550	4380	0.83737	4.88463	---	1050	8000	---	0.00465	0.0258
Benzene	1.56	B - Personal	3	69	12	3.96	24	25550	4380	0.02502	0.14598	5.52E-05	9.6	88	1.38E-06	0.01521	0.07
Carbon Disulfide	0.5	C - Personal	3	69	12	3.96	24	25550	4380	0.00802	0.04679	---	700	1000	---	6.7E-05	0.001
Chloro methane	1.7	B - Personal	3	69	12	3.96	24	25550	4380	0.02727	0.15908	5.10E-06	90	1000	1.39E-07	0.00177	0.0067
Cyclohexane	17.5	B - Personal	3	69	12	3.96	24	25550	4380	0.28073	1.63757	---	6000	6000	---	0.00027	0.011
Ethyl benzene	4.29	B - Personal	3	69	12	3.96	24	25550	4380	0.06882	0.40144	---	700	700	---	0.00057	0.0242
Heptane	5.72	B - Personal	3	69	12	3.96	24	25550	4380	0.09176	0.53525	---	700	700	---	0.00076	0.0323
Hexane	31.3	B - Personal	3	69	12	3.96	24	25550	4380	0.5021	2.92891	---	700	700	---	0.00418	0.1770
Methylene Chloride	14.1	B - Personal	3	69	12	3.96	24	25550	4380	0.22618	1.31941	1.41E-06	400	4666	3.19E-07	0.0033	0.0119
Methyl ethyl ketone	2.94	A - Personal	3	69	12	3.96	24	25550	4380	0.04716	0.27511	---	1000	3233	---	0.00028	0.0036
Methyl isobutyl ketone	3.39	B - Personal	3	69	12	3.96	24	25550	4380	0.05438	0.31722	---	80	4550	---	0.00397	0.002
Styrene	1.96	B - Personal	3	69	12	3.96	24	25550	4380	0.03144	0.18341	---	100	4133	---	0.00183	0.0018
Toluene	52.7	B - Personal	3	69	12	3.96	24	25550	4380	0.84539	4.93142	---	300	7500	---	0.01644	0.0278
xylenes	14.7	B - Personal	3	69	12	3.96	24	25550	4380	0.23581	1.37556	---	100	7333	---	0.01376	0.0079

SVOCs (ug/m3)																
Benzothiazole	1.2	D - 6" - far	3	69	12	3.96	24	25550	4380	0.01925	0.11229	5.40E-07	18	110	1.04E-08	0.00624	0.04
Butylated hydroxytoluene	ND					3.96											
PAHs (ug/m3)																	
Acenaphthene	6.60E-03	D Turf	3	138	12	3.96	24	25550	4380	0.00021	0.00124	---	210	---	---	5.9E-06	---
Acenaphthylene	ND											---	210	---			---
Benz(a)anthracene	1.10E-04	B Turf	3	138	12	3.96	24	25550	4380	3.5E-06	2.1E-05	3.30E-04	110	---	1.16E-09	1.9E-07	---
Benzo(a)pyrene	1.90E-04	B Turf	3	138	12	3.96	24	25550	4380	6.1E-06	3.6E-05	3.30E-03	110	---	2.01E-08	3.2E-07	---
Benzo(b)fluoranthene	2.10E-04	B Turf	3	138	12	3.96	24	25550	4380	6.7E-06	3.9E-05	3.30E-04	110	---	2.22E-09	3.6E-07	---
Benzo(e)pyrene	2.60E-04	B Turf	3	138	12	3.96	24	25550	4380	8.3E-06	4.9E-05	---	110	---	---	4.4E-07	---
Benzo(ghi)perylene	1.40E-04	A Turf	3	138	12	3.96	24	25550	4380	4.5E-06	2.6E-05	---	110	---	---	2.4E-07	---
Benzo(k)fluoranthene	8.00E-05	C Turf	3	138	12	3.96	24	25550	4380	2.6E-06	1.5E-05	3.30E-05	110	---	8.47E-11	1.4E-07	---
Chrysene	3.40E-04	B Turf	3	138	12	3.96	24	25550	4380	1.1E-05	6.4E-05	3.30E-06	110	---	3.60E-11	5.8E-07	---
Fluoranthene	6.80E-03	D Turf	3	138	12	3.96	24	25550	4380	0.00022	0.00127	---	140	---	---	9.1E-06	---
Fluorene	ND					3.96						---	140	---			---
Naphthalene	ND					3.96						3.40E-05	3	117			---
1-Methylnaphthalene	9.30E-03	D Turf	3	138	12	3.96	24	25550	4380	0.0003	0.00174	3.40E-05	3	117	1.01E-08	0.00058	0.00
2-Methylnaphthalene	ND					3.96						3.40E-05	3	117			---
2,6-Dimethylnaphthalene	ND					3.96						3.40E-05	3	117			---
Phenanthrene	ND					3.96						---	110	---			---

Pyrene	6.90E-03	C Turf	3	138	12	3.96	24	25550	4380	0.00022	0.00129	---	110	---	---	1.2E-05	---
SVOCs - Miscellaneous (aliphatics, hopanes, pristanes, terpenes)																	
Total miscellaneous SVOCs	1.33	D Turf	3	138	12	3.96	24	25550	4380	0.04267	0.24891	---	110	---	---	0.00226	---

Adult Exposure and Risk Calculations

Outdoor Field

Concentration Adjustment for Child Adult Scenario

Chemical	Max Conc	Location of Max	Hrs per day	Exp Freq	Exp Dur	Adult Vent Adjment	Avg Time - Cancer	Avg Time - Non-Cancer	Adjusted Conc - Cancer	Adjusted Conc - Non-Cancer	Cancer Unit Risk	RfC	Acute RfC	Cancer Risk	Non-Cancer Risk	Acute Risk
VOCs (ug/m3)																
Acetone	52.2	A - Personal	3	69	30	2.64	25550	10950	1.395609	3.256422	---	1050	8000	---	0.0031	0.017
Benzene	1.56	B - Personal	3	69	30	2.64	25550	10950	0.041708	0.097318	1.84E-05	9.6	88	7.67E-07	0.01014	0.047
Carbon Disulfide	0.5	C - Personal	3	69	30	2.64	25550	10950	0.013368	0.031192	---	700	1000	---	4.5E-05	0.001
Chloro methane	1.7	B - Personal	3	69	30	2.64	25550	10950	0.045451	0.106052	1.70E-06	90	1000	7.73E-08	0.00118	0.004
Cyclohexane	17.5	B - Personal	3	69	30	2.64	25550	10950	0.467877	1.091712	---	6000	6000	---	0.00018	0.008
Ethyl benzene	4.29	B - Personal	3	69	30	2.64	25550	10950	0.114697	0.267625	---	700	700	---	0.00038	0.016
Heptane	5.72	B - Personal	3	69	30	2.64	25550	10950	0.152929	0.356834	---	700	700	---	0.00051	0.022
Hexane	31.3	B - Personal	3	69	30	2.64	25550	10950	0.836831	1.952605	---	700	700	---	0.00279	0.118
Methylene Chloride	14.1	B - Personal	3	69	30	2.64	25550	10950	0.376975	0.879608	4.70E-07	400	4666	1.77E-07	0.0022	0.008
Methyl ethyl ketone	2.94	A - Personal	3	69	30	2.64	25550	10950	0.078603	0.183408	---	1000	3233	---	0.00018	0.002
Methyl isobutyl ketone	3.39	B - Personal	3	69	30	2.64	25550	10950	0.090634	0.21148	---	80	4550	---	0.00264	0.002
Styrene	1.96	B - Personal	3	69	30	2.64	25550	10950	0.052402	0.122272	---	100	4133	---	0.00122	0.001
Toluene	52.7	B - Personal	3	69	30	2.64	25550	10950	1.408977	3.287614	---	300	7500	---	0.01096	0.019
xylenes	14.7	B - Personal	3	69	30	2.64	25550	10950	0.393016	0.917038	---	100	7333	---	0.00917	0.005

SVOCs (ug/m3)																
Benzothiazole	1.2	D - 6" - far	3	69	30	2.64	25550	10950	0.032083	0.07486	1.80E-07	18	110	5.77E-09	0.00416	0.029
Butylated hydroxytoluene	ND					2.64										
PAHs (ug/m3)																
Acenaphthene	6.60E-03	D Turf	3	138	30	2.64	25550	10950	0.000353	0.000823	---	210	---	---	3.9E-06	---
Acenaphthylene	ND										---	210	---			---
Benz(a)anthracene	1.10E-04	B Turf	3	138	30	2.64	25550	10950	5.88E-06	1.37E-05	1.10E-04	110	---	6.47E-10	1.2E-07	---
Benzo(a)pyrene	1.90E-04	B Turf	3	138	30	2.64	25550	10950	1.02E-05	2.37E-05	1.10E-03	110	---	1.12E-08	2.2E-07	---
Benzo(b)fluoranthene	2.10E-04	B Turf	3	138	30	2.64	25550	10950	1.12E-05	2.62E-05	1.10E-04	110	---	1.24E-09	2.4E-07	---
Benzo(e)pyrene	2.60E-04	B Turf	3	138	30	2.64	25550	10950	1.39E-05	3.24E-05	---	110	---	---	2.9E-07	---
Benzo(ghi)perylene	1.40E-04	A Turf	3	138	30	2.64	25550	10950	7.49E-06	1.75E-05	---	110	---	---	1.6E-07	---
Benzo(k)fluoranthene	8.00E-05	C Turf	3	138	30	2.64	25550	10950	4.28E-06	9.98E-06	1.10E-05	110	---	4.71E-11	9.1E-08	---
Chrysene	3.40E-04	B Turf	3	138	30	2.64	25550	10950	1.82E-05	4.24E-05	1.10E-06	110	---	2.00E-11	3.9E-07	---
Fluoranthene	6.80E-03	D Turf	3	138	30	2.64	25550	10950	0.000364	0.000848	---	140	---	---	6.1E-06	---
Fluorene	ND					2.64					---	140	---			---
Naphthalene	ND					2.64					3.40E-05	3	117			---
1-Methylnaphthalene	9.30E-03	D Turf	3	138	30	2.64	25550	10950	0.000497	0.00116	3.40E-05	3	117	1.69E-08	0.00039	2E-04
2-Methylnaphthalene	ND					2.64					3.40E-05	3	117			---
2,6-Dimethylnaphthalene	ND					2.64					3.40E-05	3	117			---
Phenanthrene	ND					2.64					---	110	---			---
Pyrene	6.90E-03	C Turf	3	138	30	2.64	25550	10950	0.000369	0.000861	---	110	---	---	7.8E-06	---
SVOCs - Miscellaneous (aliphatics, hopanes, pristanes, terpenes)																
Total miscellaneous SVOCs	1.33	D Turf	3	138	30	2.64	25550	10950	0.071117	0.16594	---	110	---	---	0.00151	---

Adult Exposure and Risk Calculations - Field K

Concentration Adjustment for Adult exposure Scenario

Chemical	Max Conc	Hrs per day	Exp Freq	Exp Dur	Adult Vent Adjment	Hrs per day	Avg Time - Cancer	Avg Time - Non-Cancer	Adjusted Conc - Cancer	Adjusted Conc - Non-Cancer	Cancer Unit Risk	RfC	Acute RfC	Cancer Risk	Non-Cancer Risk	Acute Risk
VOCs (ug/m3)																
Acetone	92.5	3	138	30	2.64	24	25550	10950	4.9461	11.541	---	1050	8000	---	0.011	0.03053
Benzene	1.18	3	138	30	2.64	24	25550	10950	0.0631	0.1472	1.84E-05	9.6	88	1.16E-06	0.0153	0.0354
Carbon Disulfide	0.9	3	138	30	2.64	24	25550	10950	0.0481	0.1123	---	700	1000	---	0.0002	0.00238
Chloro methane	1.57	3	138	30	2.64	24	25550	10950	0.084	0.1959	1.70E-06	90	1000	1.43E-07	0.0022	0.00414
Cyclohexane	10.3	3	138	30	2.64	24	25550	10950	0.5508	1.2851	---	6000	6000	---	0.0002	0.00453
Ethyl benzene	4.77	3	138	30	2.64	24	25550	10950	0.2551	0.5951	---	700	700	---	0.0009	0.01799
Heptane	10.22	3	138	30	2.64	24	25550	10950	0.5465	1.2751	---	700	700	---	0.0018	0.03854
Hexane	11.25	3	138	30	2.64	24	25550	10950	0.6016	1.4036	---	700	700	---	0.002	0.04243
Methylene Chloride	10.3	3	138	30	2.64	24	25550	10950	0.5508	1.2851	4.70E-07	400	4666	2.59E-07	0.0032	0.00583
Methyl ethyl ketone	44.15	3	138	30	2.64	24	25550	10950	2.3608	5.5085	---	1000	3233	---	0.0055	0.03605
Methyl isobutyl ketone	36	3	138	30	2.64	24	25550	10950	1.925	4.4916	---	80	4550	---	0.0561	0.02089
Styrene	3.53	3	138	30	2.64	24	25550	10950	0.1888	0.4404	---	100	4133	---	0.0044	0.00225
Toluene	135	3	138	30	2.64	24	25550	10950	7.2187	16.844	---	300	7500	---	0.0561	0.04752
xylenes	15.66	3	138	30	2.64	24	25550	10950	0.8374	1.9539	---	100	7333	---	0.0195	0.00564
SVOCs (ug/m3)																
Benzothiazole	14	3	138	30	2.64	24	25550	10950	0.7486	1.7467	1.80E-	18	110	1.35E-	0.097	0.336

											07			07		
Butylated hydroxytoluene	3.9	3	138	30	2.64	24	25550	10950	0.2085	0.4866	---	210	---	---	0.0023	---
PAHs (ug/m3)																
Acenaphthene	1.74E-02	3	138	30	2.64	24	25550	10950	0.0009	0.0022	---	210	---	---	1E-05	---
Acenaphthylene	0.0068										---	210	---			---
Benz(a)anthracene	ND	3	138	30	2.64	24	25550	10950	---	---	1.10E-04	110	---	---	---	---
Benzo(a)pyrene	ND	3	138	30	2.64	24	25550	10950	---	---	1.10E-03	110	---	---	---	---
Benzo(b)fluoranthene	ND	3	138	30	2.64	24	25550	10950	---	---	1.10E-04	110	---	---	---	---
Benzo(e)pyrene	ND	3	138	30	2.64	24	25550	10950	---	---	---	110	---	---	---	---
Benzo(ghi)perylene	ND	3	138	30	2.64	24	25550	10950	---	---	---	110	---	---	---	---
Benzo(k)fluoranthene	ND	3	138	30	2.64	24	25550	10950	---	---	1.10E-05	110	---	---	---	---
Chrysene	ND	3	138	30	2.64	24	25550	10950	---	---	1.10E-06	110	---	---	---	---
Fluoranthene	5.60E-03	3	138	30	2.64	24	25550	10950	0.0003	0.0007	---	140	---	---	5E-06	---
Fluorene	5.40E-02	3	138	30	2.64	24	25550	10950	0.0029	0.0067	---	140	---	---	5E-05	---
Naphthalene	1.13E-01	3	138	30	2.64	24	25550	10950	0.006	0.0141	3.40E-05	3	117	2.05E-07	0.0047	2.55E-03
1-Methylnaphthalene	1.14E-01	3	138	30	2.64	24	25550	10950	0.0061	0.0142	3.40E-05	3	117	2.07E-07	0.0047	2.57E-03
2-Methylnaphthalene	6.30E-02	3	138	30	2.64	24	25550	10950	0.0034	0.0079	3.40E-05	3	117	1.15E-07	0.0026	1.42E-03
2,6-Dimethylnaphthalene	2.90E-02	3	138	30	2.64	24	25550	10950	0.0016	0.0036	3.40E-05	3	117	5.27E-08	0.0012	6.54E-04
Phenanthrene	3.20E-02	3	138	30	2.64	24	25550	10950	0.0017	0.004	---	110	---	---	4E-05	---
Pyrene	1.18E-02	3	138	30	2.64	24	25550	10950	0.0006	0.0015	---	110	---	---	1E-05	---
SVOCs - Miscellaneous (aliphatics, hopanes, pristanes, terpenes)																
Total miscellaneous SVOCs	4.4	3	138	30	2.64	24	25550	10950	0.2353	0.549	---	110	---	---	0.005	---