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HUMAN HEALTH RISK ASSESSMENT OF SYNTHETIC TURF FIELDS BASED UPON INVESTIGATION OF FIVE FIELDS IN CONNECTICUT

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Questions have been raised regarding possible exposures when playing sports on synthetic turf fields cushioned with crumb rubber. Rubber is a complex mixture with some components possessing toxic and carcinogenic properties. Exposure is possible via inhalation, given that chemicals emitted from rubber might end up in the breathing zone of players and these players have high ventilation rates. Previous studies provide useful data but are limited with respect to the variety of fields and scenarios evaluated. The State of Connecticut investigated emissions associated with four outdoor and one indoor synthetic turf field under summer conditions. On-field and background locations were sampled using a variety of stationary and personal samplers. More than 20 chemicals of potential concern (COPC) were found to be above background and possibly field-related on both indoor and outdoor fields. These COPC were entered into separate risk assessments (1) for outdoor and indoor fields and (2) for children and adults. Exposure concentrations were prorated for time spent away from the fields and inhalation rates were adjusted for play activity and for children's greater ventilation than adults. Cancer and noncancer risk levels were at or below de minimis levels of concern. The scenario with the highest exposure was children playing on the indoor field. The acute hazard index (HI) for this scenario approached unity, suggesting a potential concern, although there was great uncertainty with this estimate. The main contributor was benzothiazole, a rubber-related semivolatile organic chemical (SVOC) that was 14-fold higher indoors than outdoors. Based upon these findings, outdoor and indoor synthetic turf fields are not associated with elevated adverse health risks. However, it would be prudent for building operators to provide adequate ventilation to prevent a buildup of rubber-related volatile organic chemicals (VOC) and SVOC at indoor fields. The current results are generally consistent with the findings from studies conducted by New York City, New York State, the U.S. Environmental Protection Agency (EPA), and Norway, which tested different kinds of fields and under a variety of weather conditions.

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This article and underlying reports were reviewed by staff from the Connecticut Department of Public Health, the Connecticut Department of Environmental Protection, the University of Connecticut Health Center, Section of Environmental and Occupational Medicine, and the Connecticut Agricultural Experiment Station. The Connecticut Academy of Science and Engineering reviewed the underlying reports and made formal recommendations (CASE, June 15, 2010 report), which were incorporated into the final reports and are reflected in this article. The field, laboratory work and CASE review were funded by the Connecticut Department of Environmental Protection.

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Questions have been raised regarding potential exposures and health risks associated with playing on artificial turf fields cushioned with crumb rubber (Brown 2007). This is a form of recycling, as the crumb rubber is produced from the shredding of discarded tires. Tires can be made from natural or synthetic rubber, both of which are complex mixtures of chemicals that include polycyclic aromatic hydrocarbons (PAH), volatile organic chemicals (VOC), nitrosamines, benzothiazoles, latex, and heavy metals. Therefore, there is a potential for exposure and health risk from playing on these fields. Exposure is expected to be greatest via the inhalation route under warm summertime conditions, as some components vaporize into the breathing zone of athletes. Further, running may break down the crumb rubber and emit particles into the air just above the field. The higher ventilation rate during active play further enhances the potential for inhalation exposure. Therefore, this risk assessment focused upon the inhalation route of exposure.

The current investigation adds to a growing body of data describing crumb rubber-based athletic fields (U.S. Environmental Protection Agency [EPA] 2009a; NYSDEC 2009a; TRC 2009; NILU 2006; Norwegian Institute of Public Health 2006; California Office of Environmental Health Hazard Assessment [OEHHA] 2007; KEMI 2006; LeDoux 2007; Bristol and McDermott 2008), and it is unique in providing personal monitoring results for users of the field during active play. Further it is the only apparent study in the United States that assessed an indoor soccer field. Each field was investigated on its own day of fieldwork during July 2009, under sunny, warm, and low wind weather conditions to maximize the potential of detecting off-gassed rubber components. Details of the sampling plan, methodology and results of field testing, analytical chemistry, and off-gas headspace experiments are found in companion papers (Li et al. 2010; Simcox et al. 2011). An independent review of the underlying reports was conducted by the Connecticut Academy of Science and Engineering (CASE 2010).

This project is a follow-up of an earlier pilot study by CAES (2007). A separate analysis of ecological risks associated with rainwater runoff from these fields was performed by the Connecticut Department of Environmental Protection (CTDEP 2010).

The overall objective was to develop a screening level risk assessment in which high-end assumptions for exposure were used for uncertain parameters and surrogate data were employed for chemicals with inadequate toxicity information so that chemicals did not fall 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.

METHODS

Field Investigations

The current project involved investigation at five polyethylene grass fields cushioned with crumb rubber infill located in Connecticut. Four are outdoor fields (designated as A, B, C, D) and one indoors (designated as K). Sampling occurred during July 2009 (Figure 1). Field selection was based upon the ability to gain access, the availability of electrical outlets to run the stationary sampling equipment, and obtaining a variety of field ages (range 2 to 5 yr old). The outdoor fields were all high school football or soccer fields, while the indoor field was a collegiate facility. Three to 4 volunteers played soccer for a 2-h sampling event at each field. The activity consisted of drills and scrimmages with brief breaks taken for water and equipment checks on an as-needed basis. Each player was equipped with a variety of personal sampling devices as listed in Table 1 and as described in more detail elsewhere (Simcox et al. 2011). Volunteers were instructed to avoid personal care products on the day of testing. Soccer was played on a small portion of the field to maximize the local particulate emission and stationary samplers were located in the immediate vicinity of the play.

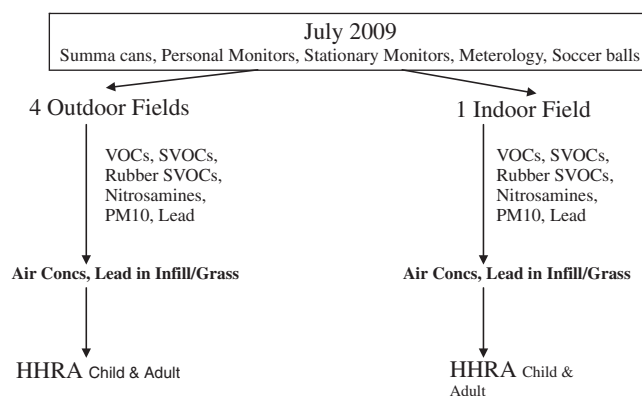


FIGURE 1. Outline of Connecticut synthetic turf air study.

TABLE 1. Numbers of Samples Taken at Each Field

Sample type	VOC	SVOC	Rubber SVOC	Nitrosamines	PM ₁₀
Personal monitor	2	0	2	2	0
Stationary on-field 6 inch	1	0	2	1	0
Stationary on field 3 feet	1	1	2	1	1
Stationary upwind	1	1	2	2	1
Community	— ^a	— ^a	— ^a	— ^a	— ^a

^aThe community field was tested in similar fashion as the synthetic turf fields—three personal monitors plus stationary upwind and on field monitors.

Sampling and analysis were conducted for a wide range of VOC, semivolatile organic compounds (SVOC), rubber-related SVOC, lead (Pb), and particulate matter in the less than 10 μm range (PM₁₀). This included sampling for a suite of 60 VOC, 120 SVOC divided into 22 polycyclic aromatic hydrocarbons (PAH), 5 targeted (potentially rubber-related) SVOC, and 93 miscellaneous SVOC, 7 nitrosamines, and PM₁₀ (Simcox et al. 2011). As shown in Table 1, different samplers were employed in the hopes of identifying analytes originating from the field as opposed to general air pollutants. Personal monitors were attached to the belt of each player to determine what may be in the breathing zone of a young child playing on the field. Stationary monitors located away from the play at 6 inches or 3 ft above the field were intended to study the vertical gradient of contaminants emitted from the field. Upwind samples were taken off the synthetic turf surface (usually on grass) at each field. A separate round of sampling was conducted at a suburban location at a grass field near a busy road to assess general ambient background levels of

the target analytes (community location, designated field L). Soccer was also played at the community grass field to serve as a background data source for the personal monitors; this was needed to evaluate the possibility that some VOC detected in the personal monitors may have originated from the sampling equipment or exhaled breath of the soccer player rather than the field.

Volatile organic chemicals, targeted SVOC, 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 using stationary samplers. This led to 5 types of samples for VOC and targeted SVOC: stationary, field height (6 inches); stationary, 3 ft; personal monitor; off-field upwind; and off-field community. For PAH and miscellaneous SVOC there were three types of samples: on turf, upwind, and community background. Data from the community background sample were combined with the other background samples taken at each field to yield

a range of background results for comparison to on-field results.

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 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 four 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 ensured that for an analyte to become a COPC its on-field and background detects did not overlap. The range of background results was inspected to ensure 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 COPC were carried through the risk assessment process.

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 did not display active ventilation at the time of sampling; this is typical for indoor fields as they

generally only use the ventilation system to regulate temperature on hot days.

Exposure Assessment

The primary objective of this field investigation and risk assessment was to estimate exposures and risks for children playing on the fields. Due to the possibility that adults using these fields might encounter higher exposures due to a longer period of usage, they were also considered as a separate element. Separate analyses were conducted for data pooled from the outdoor fields vs. the one indoor field yielding four sets of exposure and risk estimates: child-indoors, child-outdoors, adult-indoors, adult-outdoors. Given that field sampling occurred in July under sunny, low wind conditions, off-gassing from the outdoor fields would be overestimated if the entire 8 mo/yr exposure period were simulated based upon these results. Instead, it was assumed that estimates of inhaled VOC apply to the four warmest months with no allowance for days with clouds or high wind which would mitigate exposure. No such adjustment was made for the indoor field, as the results from our 1-day investigation may be representative of each day the facility is operational. Another conservative assumption was that the highest concentration for each analyte found at any of the outdoor fields was combined across fields to represent a worst case composite. This approach obviates the need for five separate risk assessments.

Various exposure routes are possible for crumb rubber-related chemicals as follows:

- Inhalation of volatile or semivolatile chemicals that off-gassed from the rubber.
- Inhalation of particles and particle-borne chemicals.
- Ingestion of crumb rubber or the dust created from the breakdown of crumb rubber.
- Dermal uptake of chemicals contained in crumb rubber that contact the skin.

The current risk assessment focused upon the first two pathways, inhalation of off-gassed and particle-bound chemicals. Ingestion of

crumb rubber or dust derived from crumb rubber was not a focus, as this pathway was evaluated elsewhere without being identified as a public health risk (Norwegian Institute of Public Health and Radium Hospital 2006; California EPA 2007) and field methods were not designed to measure the amount of dislodgeable dust that might occur on the surface of these fields and end up becoming ingested. The fields did 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 conducted in California (California EPA 2007). Dermal exposure was also not a focus as most chemicals in rubber are not a reliable candidate for dermal absorption: The volatile fraction tends to revolatilize off skin and thus not remain long enough for substantial dermal penetration; the particle-bound fraction tends to remain bound to the rubber, rather than partition into and penetrate through the skin. No apparent data were found describing the transfer of SVOC 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 penetrate inside shoes. The Norwegian study evaluated dermal exposure to crumb rubber particles and did not find this to be a significant risk pathway (Norwegian Institute of Public Health and Radium Hospital 2006).

Exposure Scenarios

Table 2 presents key assumptions for the child and adult scenarios used to develop exposure estimates. Exposure was adjusted to account for differences in ventilation rates during active play and in children. This adjusts the exposure concentration rather than the dose received because the toxicity values (reference concentration—RfC; cancer unit risk factor) are in terms of air concentration ($\mu\text{g}/\text{m}^3$) rather than intake dose (e.g., $\text{mg}/\text{kg}/\text{d}$). The

ventilation adjustment is needed because toxicity values are based upon studies in animals or humans in which the subjects were at rest or undergoing light exercise (e.g., workers). Further, these data come from adult animals or humans and thus do not necessarily capture the increased exposure possible for younger children (Ginsberg et al. 2010). The following equation shows the ventilation adjustment (adj) along with the time-weighted averaging used to calculate inhaled doses that relate to chronic exposure and risk. For short-term (acute) exposures, the ventilation-adjusted air concentration (conc) was used directly without time averaging.

Inhaled conc ($\mu\text{g}/\text{m}^3$)

$$= \frac{\text{Measured conc } (\mu\text{g}/\text{m}^3) * \text{hours per day} * \text{days per year} * \text{years} * \text{ventilation adj}}{\text{Averaging time}}$$

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 ($0.0148 \text{ m}^3/\text{min}$ or $21.4 \text{ m}^3/\text{d}$) to the higher ventilation rate associated with sports play as described in the U.S. EPA *Exposure Factors Handbook* (U.S. EPA 2009b). For this one assumes a moderate ($0.039 \text{ m}^3/\text{min}$) level of exercise for the 3 h 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 ($0.073 \text{ m}^3/\text{min}$). This creates an adult ventilation adjustment of 2.64 ($0.039/0.0148 \text{ m}^3/\text{min}$). 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) indicated that a threefold factor is appropriate for the first 3 yr of life, with this decreasing to a 1.5-fold adjustment for ages 4–10 yr. 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 assessments (young vs. older

TABLE 2. Exposure Parameters

Parameter	Child	Adult	Basis
Age (yr)	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 h	3 h	Time for soccer match or practice
Days exposed per year	138	138	4 d/wk for 8 months (spring, fall soccer + 2 mo in summer)
Days exposed per year VOC	69	69	VOC offgas only in the four warm months for outdoor fields; no adjustment for indoor fields
Ventilation adjustment	3.96	2.64	Child—Adult factor ^a child factor Adult—moderate exercise
Averaging time (cancer)	25,550 d	25,550 d	Entire lifespan—70 yr
Averaging time (noncancer)	4380 d	10,950 d	Entire exposure period

child). This factor is applied on top of the adult ventilation adjustment to yield a 3.96-fold adjustment for children (Table 2).

Toxicity Assessment

COPC over a broad array were identified, some of which have an extensive toxicology database and others that do not. This toxicity assessment relies upon national databases of toxicity potency values as available from the U.S. EPA 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 are compared and in cases where there is considerable disagreement (threefold or greater), the Connecticut Department of Public Health (CT DPH) evaluated the underlying difference and selected 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 underreporting 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 noncarcinogenic PAH and other miscellaneous SVOC that lack RfC were assigned the RfC for pyrene, which is the lowest RfC available for the general series of PAH.

Table 3 summarizes the toxicology values used for COPC in this assessment. Chemical-specific toxicology monographs are generally available from the cited sources (e.g., IRIS, California OEHHA, ATSDR). However, this is not the case for benzothiazole, which is of particular interest because it is a known rubber constituent that consistently is found in the air above the outdoor and indoor fields. Benzothiazole received relatively little toxicity assessment in the past; Ginsberg et al. (2011) provide a toxicity assessment for this compound in a companion paper.

In addition to chronic cancer and non-cancer toxicity values (unit risks and RfC respectively), this assessment utilizes acute risk air targets as well. Short-term exposure to COPC might 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-h RfC, since our exposure assessment was for 3 h of play per event. These values

TABLE 3. Toxicity Values for COPC

Analyte	Cancer unit risk ($\mu\text{g}/\text{m}^3$)	Source	RfC ($\mu\text{g}/\text{m}^3$)	Source	Acute target ($\mu\text{g}/\text{m}^3$)	Source
VOC						
Acetone	NA	—	1050	IRIS RfD for renal effect converted to RfC; 3 \times uncertainty factor (UF) added for lower dose effects in a 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 h AEC to 3 h time frame
Carbon disulfide	NA	—	700	IRIS for peripheral neurotoxicity	1000	CTAEC for neurotoxicity and odor threshold
Chloromethane	1.7E-06	California Prop 65	90	IRIS for CNS toxicity	1000	ATSDR acute MRL for neurotoxicity
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 chloride	4.7E-07	IRIS	400	California OEHHA for cardiovascular and nervous system toxicity	4700	California acute REL for neurotox divided by 3 for 1 h to 3 h conversion
Methyl ethyl ketone	NA	—	1000	California OEHHA for reproductive effects; value is 5 \times < IRIS	3233	CTAEC for irritation in humans divided by 3 for time conversion
Methyl isobutyl ketone	NA	—	80	U.S. EPA HEAST, 1997, for liver/kidney toxicity	4550	CTAEC for irritation, headache divided by 3 for time conversion
Styrene	NA	—	100	IRIS RfC for neurotox divided by 10 for possible carcinogenicity	4133	CTAEC for neurotox divided by 3 for time conversion
Toluene	NA	—	300	ATSDR MRL for neurotox—a value lower than California or IRIS	3800	ATSDR acute MRL for neurotoxicity
Xylene	NA	—	100	IRIS for neurotoxicity	7333	California acute REL for neurotox divided by 3 for time conversion
Targeted SVOC						
Benzothiazole	1.8E-07	Whittaker et al., 2004 cancer slope for 2-MBZT and route extrapolation	18	NYS DEC (2009) value based on subchronic oral NOAEL and route extrapolation	110	CTDPH value based on 18 \times higher RD ₅₀ than formaldehyde and 10 \times UF for data gaps

(Continued)

TABLE 3. (Continued)

Analyte	Cancer unit risk ($\mu\text{g}/\text{m}^3$)	Source	RfC ($\mu\text{g}/\text{m}^3$)	Source	Acute target ($\mu\text{g}/\text{m}^3$)	Source
Butylated hydroxytoluene	NA	—	175	European ADI of 0.05 mg/kg/d and route extrapolation	NA	—
PAH						
Acenaphthene	NA	—	210	IRIS RfD for hepatotoxicity with route extrapolation	NA	—
Acenaphthylene	NA	—	210	No data; acenaphthene as surrogate	NA	—
Benz[a]anthracene	1.1E-04	Unit risk for BaP with relative potency of 0.1 from U.S. EPA, 1993	110	Pyrene IRIS RfD converted to RfC as surrogate—lowest RfC available	NA	—
Benzo[a]pyrene (BaP)	1.1E-03	California EPA (1999) unit risk from hamster inhalation bioassay	110	Pyrene IRIS RfD converted to RfC as surrogate	NA	—
Benzo[e]pyrene	NA	—	110	Pyrene IRIS RfD converted to RfC	NA	—
Benzo[b]fluoranthene	1.1E-04	Unit risk for BaP with relative potency of 0.1 from U.S. EPA, 1993	110	Pyrene IRIS RfD converted to RfC as surrogate as lowest RfC available	NA	—
Benzo[k]fluoranthene	1.1E-05	Unit risk for BaP with relative potency of 0.01 from U.S. EPA, 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	NA	—
Chrysene	1.1E-05	Unit risk for BaP with relative potency of 0.001 from U.S. EPA, 1993	110	Pyrene IRIS RfD converted to RfC as surrogate as lowest RfC available	NA	—
Fluoranthene	NA	—	140	IRIS RfD and route extrapolation	NA	—
Fluorene	NA	—	140	IRIS RfD and route extrapolation	NA	—
Naphthalene	3.4E-05	Unit risk from California OEHHA	3	IRIS RfC for respiratory hyperplasia	117	CTAEC for acute tox to Clara cells in mice divided by 3 for time conversion
1-Methylnaphthalene	3.4E-05	Naphthalene as surrogate	3	Naphthalene as surrogate	117	Naphthalene as surrogate
2-Methylnaphthalene	3.4E-05	Naphthalene as surrogate	3	Naphthalene as surrogate	117	Naphthalene as surrogate
2,6-Dimethylnaphthalene	3.4E-05	Naphthalene as surrogate	3	Naphthalene as surrogate	117	Naphthalene as surrogate
Phenanthrene	NA	—	110	IRIS RfD and route extrapolation	NA	—

(Continued)

TABLE 3. (Continued)

Analyte	Cancer unit risk ($\mu\text{g}/\text{m}^3$)	Source	RfC ($\mu\text{g}/\text{m}^3$)	Source	Acute target ($\mu\text{g}/\text{m}^3$)	Source
Pyrene	NA	—	110	IRIS RfD for renal pathology and route extrapolation	NA	—
Miscellaneous SVOC (aliphatics, hopanes, terpenes, pristanes)	NA	—	110	No values available, used pyrene as conservative surrogate	NA	—

Note. Abbreviations: NA, not applicable; IRIS, U.S. EPA Integrated Risk Information System online database of toxicity values; California OEHHA, 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-h exposure developed in 2000 and updated in 2010 for targeted analytes.

typically do not exist. However, for a limited set of chemicals, 1-h acute targets were derived by California OEHHA (acute Reference Exposure Level [REL]) and by Connecticut DPH (CT acute exposure concentrations, AEC). These values were used along with ATSDR acute minimum risk level (MRL) (typically on a basis of 24 h of continuous exposure) to develop 3-h acute air targets in this assessment. These acute targets were 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-h target developed for other purposes was converted to a 3-h target by dividing by 3. The 1-h levels were prioritized over the ATSDR acute MRL because the MRL relate to a longer time frame, 24 h continuous exposure basis. Further, the level of conservatism and time-factor adjustment in the ATSDR values are not necessarily consistent or always transparent. Another conservative screening level approach was to use the chronic RfC in several cases where an acute value was not available.

As seen in Table 3, all COPC were assigned RfC, 12 have cancer unit risk values and 17 have acute targets. Only those analytes having direct or indirect (e.g., structurally related to carcinogen or clearly mutagenic) evidence of carcinogenicity were assigned unit risk values. Acute targets were assigned only for the volatile analytes, as the acute effects of particle bound chemicals such as PAH have not been well explored but are expected to be minor,

given that they are not highly reactive and tend to produce chronic effects instead.

The following highlight some of the toxicology assessment decisions made in the face of limited or conflicting information:

Chloromethane—This chemical is generally regarded as a mutagen with limited cancer bioassay data suggesting some activity. However, the U.S. EPA and California OEHHA did not derive 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 was not 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.

Styrene—Its 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 exerts carcinogenic action. Carcinogens typically have much lower de minimis targets than nonpersistent noncarcinogens that bear an RfC.

Benzothiazole—This agent has little toxicology data but was positive in one mutagenicity

test and has a structural analogue that is carcinogenic (2-mercaptobenzothiazole or 2-MBZT). The acute toxicity value was derived based upon analogy with formaldehyde. Both compounds were tested in mouse respiratory depression (RD₅₀) studies by the same laboratory and found to be irritating. Benzothiazole potency was 18-fold below formaldehyde. Given the uncertainties in extrapolating from an animal screening test to humans and the fact that benzothiazole can produce sensitization (at least on the skin), an additional 10-fold uncertainty factor was applied to derive the acute target. The derivation of cancer, noncancer, and acute toxicity values for benzothiazole is further described in a companion paper (Ginsberg et al. 2011).

Butylated hydroxytoluene (BHT)—There are no toxicity values but BHT is a common food preservative. The European Union (EU) has an acceptable daily intake based upon toxicology concerns of 0.05 mg/kg/d, which is threefold lower than the U.S. Food and Drug Administration (FDA) intake limit. The EU value was converted to inhalation by dose route extrapolation for the current purposes.

Naphthalenes—Naphthalene does not have a cancer unit risk on IRIS and is negative in genetic toxicity testing. However, naphthalene produced lung tumors in mice in a National Toxicology Program (NTP) (1992) cancer bioassay and olfactory neuroblastomas in a rat bioassay (NTP 2000). This chemical also has an IARC category 2B cancer classification and is listed by the National Toxicology Program as a carcinogen. California OEHHA derived a unit risk factor based upon the NTP testing, and this is the potency value used in the current assessment. The U.S. EPA risk-based concentration tables also list this unit risk value. The other naphthalenes considered COPC are closely related to naphthalene and lack adequate cancer bioassay data to make separate determinations.

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 the U.S. EPA Carcinogen Risk Assessment, Supplemental Guidance for Early Life Stages (U.S. EPA 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 Guidance recommends the following enhanced potency factors above adult potency: 10-fold for 0–2 yr of age and 3-fold for 3–15 yr of age. For the purposes of this assessment, the child exposure scenario (age 12 yr average; range 6–18) is considered to be at heightened vulnerability and receives the enhanced threefold factor. This applies to all carcinogens that have documented mutagenic or clastogenic activity and included as COPC: chloromethane; methylene chloride; benzo[a]pyrene and related carcinogenic PAH; and benzothiazole. The carcinogenicity and mode of action of benzothiazole are uncertain but in limited testing it was mutagenic and thus is included in this list. Naphthalene and its congeners have limited cancer and mechanistic/mutagenic data with their cancer classification not well established. Therefore, an additional children's potency factor was not applied for naphthalene and its related analytes.

Risk Characterization

This assessment used standard risk assessment methods to estimate cancer and non-cancer risks. Prorated time-weighted average exposures were calculated based upon the highest measured analyte concentration, amount of time playing (3 h/d, 138 d per year), exercise-induced breathing rate, and years of exposure (12 or 30). For carcinogens, the lifetime average daily exposure, in units of micrograms per cubic meter, 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 $1\text{E-}04$ are considered substantially elevated relative to U.S. EPA Superfund guidance (acceptable risk range up to 10^{-4}), background air toxics risk estimates for U.S. census tracts, which are typically estimated at a cumulative cancer risk of $1\text{E-}05$ to $1\text{E-}04$ (U.S. EPA NATA 2005; Woodruff et al. 1998), and California regulatory air target limits (cancer risk of $1\text{E-}05$). Cancer risks below $1\text{E-}06$ are considered de minimis. Cancer risks between $1\text{E-}06$ and $1\text{E-}04$ 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 noncarcinogens, the average daily dose during the exposure window (12 or 30 yr) was divided by the RfC to create the hazard quotient (HQ). These time frames, 12 and 30 yr, are considered chronic for the purposes of comparison to the RfC, which is usually thought of as the lifetime exposure target that is without significant risk for adverse effect. 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, to conservatively screen for noncancer risk issues, this assessment assumes that all noncancer risks are additive across chemicals to yield a cumulative hazard index (HI).

For acute risk calculation, the nonprorated 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 and others are based 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} .

RESULTS

Contaminants of Potential Concern

Of the 60 VOC for which analyses were conducted, 10 are considered COPC at the outdoor fields while 13 are COPC at the indoor field (Tables 4 and 5). Personal monitoring results tended to give the highest VOC detections relative to stationary samplers at both indoor and outdoor fields. The VOC COPC were above the range of background detections at only one of the outdoor fields (not the same field in each case) except for hexane and toluene, in which this was true for two fields. In general, detections in synthetic turf personal monitoring samples were 1.5- to 3-fold greater than background samples at outdoor fields, with the one exception being methylene chloride, in which there was a 12.8-fold elevation (Table 4). Indoor VOC detections tended to have greater elevations relative to background (Table 5).

Personal sampler results tended to be higher, in some cases much higher, than the stationary 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 headspace studies of crumb rubber from these fields failed to detect the analyte (Li et al. 2010; Simcox et al. 2011). The personal monitoring at the grass field community location (site L) also served as a background comparison for these data. Detections in personal monitoring samples that were excluded from the risk assessment are shown in Table 6. In most cases, not only were the analytes absent from headspace studies of crumb rubber, they were also present in background personal monitoring samples from play on grass fields. Acrolein and benzene are examples in which detections may arise spuriously in personal monitors and thus confound a risk assessment (Figures 2 and 3). The pattern showed detections in personal monitors but generally not in other field-related samples, with the results for the background (grass field) personal monitors comparable to the synthetic

TABLE 4. COPC at the Four Outdoor Fields (A, B, C, D)

COPC	Maximum detect ($\mu\text{g}/\text{m}^3$)	Ratio to highest background	Location and type of sample	Number of fields with elevation	Detected off-gas study
VOC					
Carbon disulfide ^{a, b}	0.47	2.9	B: Personal	1	No
Chloromethane ^b	1.7	1.4	B: Personal	1	No
Cyclohexane ^b	17.5	3.6	B: Personal	1	No
Ethyl benzene	4.29	2.0	B: Personal	1	Yes
Heptane	5.72	2.7	B: Personal	1	No
Hexane	31.3	4.2	B: Personal	2	Yes
Methylene chloride	14.1	12.8	B: Personal	1	Yes
Methylisobutyl ketone	3.39	3.3	B: Personal	1	Yes
Toluene	52.7	1.35	B: Personal	2	Yes
Xylenes	14.7	2.1	B: Personal	1	
Semi-VOC					
Targeted					
Benzothiazole	1.2	1.7	D: 6 inch	4	Yes
PAH					
Acenaphthylene	6.6E-03	8.6	D: Stationary	1	NA
Benzo[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 ^c					
Total sum	1.33	—	D: Stationary	3	NA

^aSlightly 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.

^bThese volatile analytes were included as COPCs even though they were not detected in the laboratory off-gas studies because there was at least some evidence that they were present in field-related samples besides personal monitoring samples.

^cFor 93 compounds including aliphatics, hopanes, terpenes, and pristanes.

turf personal samplers. Possible reasons for such detections in personal monitoring samples are discussed in a subsequent section. In other cases, analytes detected in personal monitors were also found in laboratory headspace studies and were higher in synthetic turf as compared to grass personal monitoring samples. These VOC are included in the COPC lists in Tables 4 and 5. It is possible that some percentage of the personal monitoring result in these cases came from the player rather than the field. Since this percent is unknown, the personal monitor detects were used at face value to represent what may have been coming off the field for the purpose of the risk assessment.

Two of the specially targeted SVOC, benzothiazole and butylated hydroxytoluene (BHT), were selected as COPC. Benzothiazole was detected above background on both the outdoor and indoor fields. The maximum indoor result was 11.7-fold greater than the maximum outdoor result, which is one of the more dramatic indoor/outdoor differences (Figure 4). 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 VOC. BHT is a COPC at 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

TABLE 5. COPC at the One Indoor Field (K)

COPC	Maximum on-field detect (μg/m ³)	Ratio to highest background	Type of sample
VOC			
Acetone	92.5	1.7	Personal
Carbon disulfide ^a	0.9	5.6	Stationary 6 inches, 3 ft
Chloromethane	1.57	1.3	Personal
Cyclohexane	10.3	2.1	Personal
Ethyl benzene	4.77	2.2	Personal
Heptane	10.22	4.8	Personal
Hexane	11.25	1.5	Personal
Methylene chloride	10.3	9.4	Personal
MEK	44.2	5.6	Personal
MIBK	36	35	Stationary 6 inches, 3 ft
Styrene	3.53	1.4	Personal
Toluene	135	3.5	Personal
Xylenes	15.7	2.2	Personal
Semi-VOC			
Targeted			
Benzothiazole	14	19.8	Stationary 6 inches
Butylated hydroxytoluene	3.9	13.9	Stationary 3 ft
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 ^b			
Total Sum	4.4	—	Stationary

^aMuch higher background detect at field C but that field had pesticide spraying in the background area during sampling.

^bFor 93 compounds including aliphatics, hopanes, terpenes, and pristanes.

stationary as opposed to personal monitor. BHT was not elevated at the outdoor fields.

A variety of PAH were detected above background and considered COPC at both the outdoor and indoor field. The concentrations were generally low, well below 1 μg/m³. The larger multiring PAH (benzanthracene through chrysene in Table 4) were detected in the outdoor field, while the more volatile two-ring PAH (naphthalene and its derivatives) were found indoors but generally not outdoors (Figure 5). The one exception was 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 (acenaphthene, fluoranthene, pyrene) were detected above background both outdoors and indoors.

Miscellaneous SVOC include a wide variety of hopanes, pristanes, terpenes, cosines, 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 one of the SVOC on field A, one on field C, and four on field D. The total concentrations of miscellaneous SVOC that are in excess of background concentrations are shown in

TABLE 6. Analytes Found in Personal Monitors but Excluded From the Risk Assessment

Analyte	Highest personal monitor detection ($\mu\text{g}/\text{m}^3$)	Reason for exclusion
1-Ethyl-4-methylbenzene	1.37	Absent in headspace, present in grass field personal monitors ^a
1,2,4- and 1,3,5-Trimethylbenzenes	2.16	Absent in headspace, present in grass field personal monitors
1,2-Dichloropropane	1.14	Absent in headspace
Acrolein	3.89	Absent in headspace, present in grass field personal monitors
Benzene	1.56	Grass field personal monitors not different than synthetic turf personal monitors
Bromoform	34.8	Absent in headspace
Ethyl acetate	11.87	Absent in headspace, present in grass field personal monitors
Propene	0.89	Absent in headspace, present in grass field personal monitors
Tetrachloroethylene	3.29	Absent in headspace
Tetrahydrofuran	3.5	Absent in headspace, present in grass field personal monitors
Trichloroethylene	23.4	Absent in headspace, present in grass field personal monitors
Vinyl acetate	2.95	Absent in headspace, present in grass field personal monitors

^aGrass field personal monitor refers to monitored play at the background community site (field L).

Figure 6. These analytes were combined under the COPC category of miscellaneous SVOC in Tables 4 and 5, with the total in excess of background combined across analytes for risk assessment since there is no toxicological basis for chemical-by-chemical 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 and nitrosamines were not COPC in this risk assessment. Nitrosamines were sampled because of their use in rubber manufacture and the potential that 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 produce elevated breathing zone concentrations. However, sampling of PM_{10} across four of the five fields did not find elevated concentrations. The on-field result at each field failed to exceed the range of detects found at background locations, 5–10 $\mu\text{g}/\text{m}^3$. Field C was an outlier with higher levels of PM_{10} in both the on-field and upwind samples (16–18 $\mu\text{g}/\text{m}^3$). A grass field just upwind of field C received pesticide spray during the beginning of the sampling event, which may have interfered with PM_{10} and other results. Therefore, PM_{10} was also not considered a COPC.

Lead (Pb) was a target analyte because of limited data in New Jersey showing elevated Pb in synthetic grass samples, which led to an investigation by the U.S. Consumer Product Safety Commission (CPSC 2009). Our bulk phase lead testing from each field's synthetic grass blades and crumb rubber were uniformly below 400 ppm, the CTDEP Remediation Standard Regulation for Pb, 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 Pb in products intended to be used by children. The highest lead Pb found in any sample from the 5 fields was 271 ppm (field D). The lack of elevated Pb in our current testing suggests that if Pb is elevated in polyethylene synthetic grass or crumb rubber, it is not a widespread problem.

Risk Estimates

Figures 7 and 8 and Table 7 summarize the risk assessment results for the four scenarios

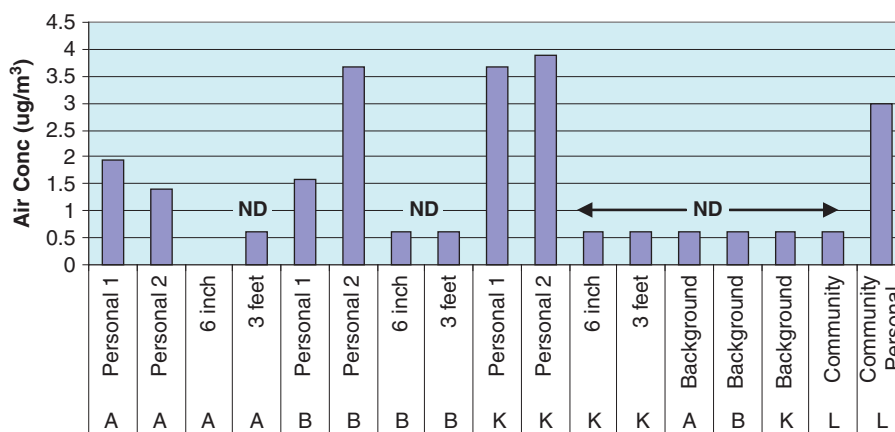


FIGURE 2. Acrolein detects at synthetic turf fields. No detects at fields C and D. ND, not detected (color figure available online).

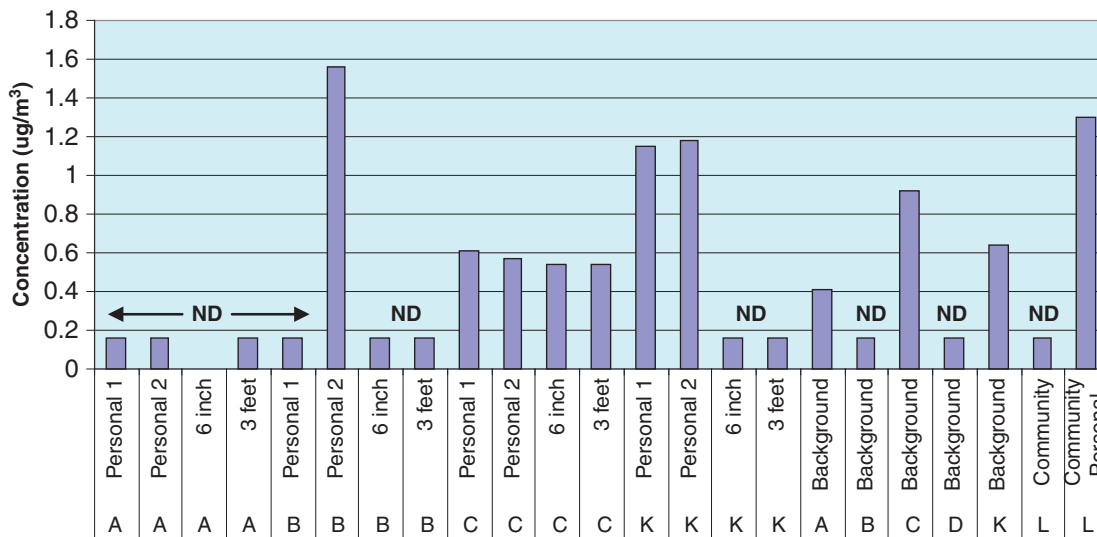


FIGURE 3. Benzene detects at synthetic turf fields. No detects at field D. ND, not detected (color figure available online).

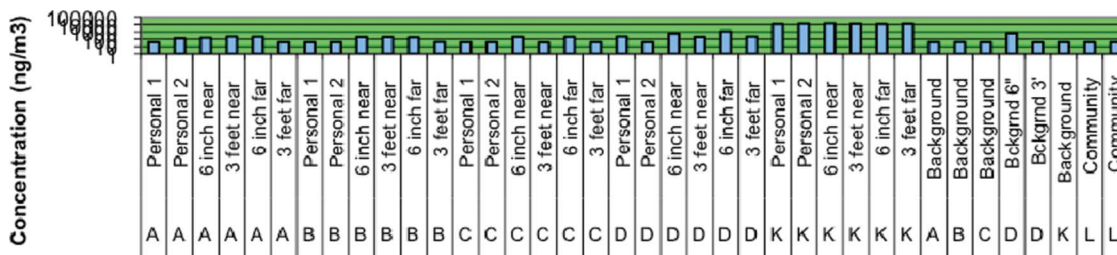


FIGURE 4. Benzothiazole results across indoor and outdoor fields. ND, not detected (color figure available online).

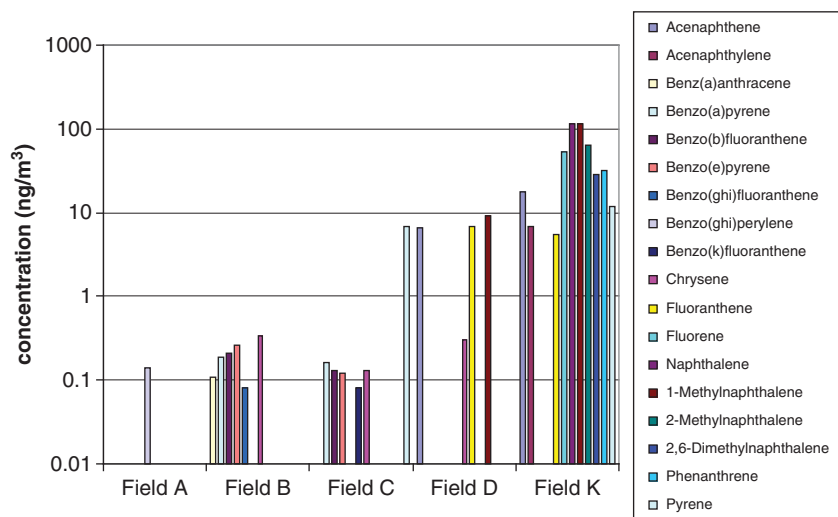


FIGURE 5. Polycyclic aromatic hydrocarbons detected above background concentration (color figure available online).

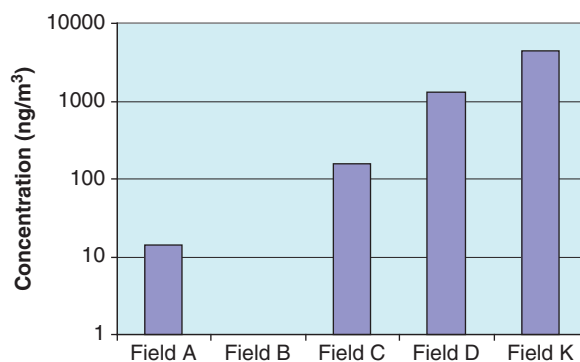


FIGURE 6. Total miscellaneous SVOC detected above background (color figure available online).

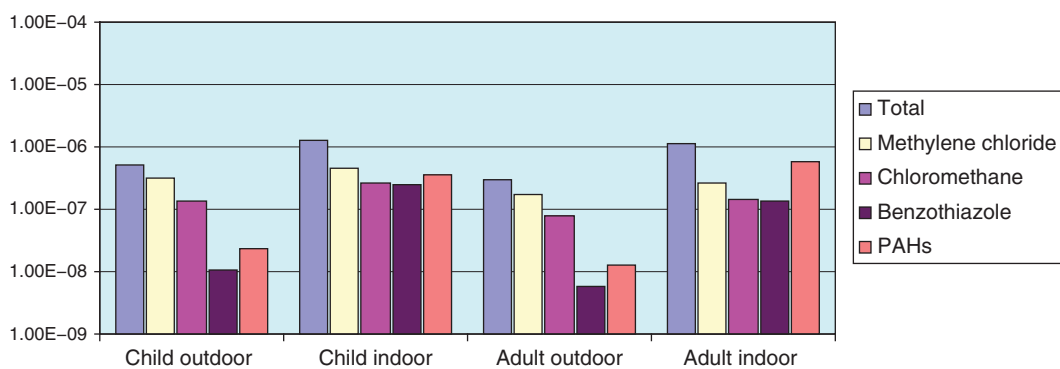


FIGURE 7. Cancer risk estimates from indoor and outdoor synthetic turf fields (color figure available online).

evaluated. Various VOC and SVOC contribute to both cancer and noncancer risk estimates. Cancer risks are at or below de minimis (1 in a million) levels in all scenarios, with the greatest

cumulative cancer risk found indoors for children (1.3E-06). Various analytes were detected at greater concentration indoors relative to outdoors, which translated into a two to 3-fold

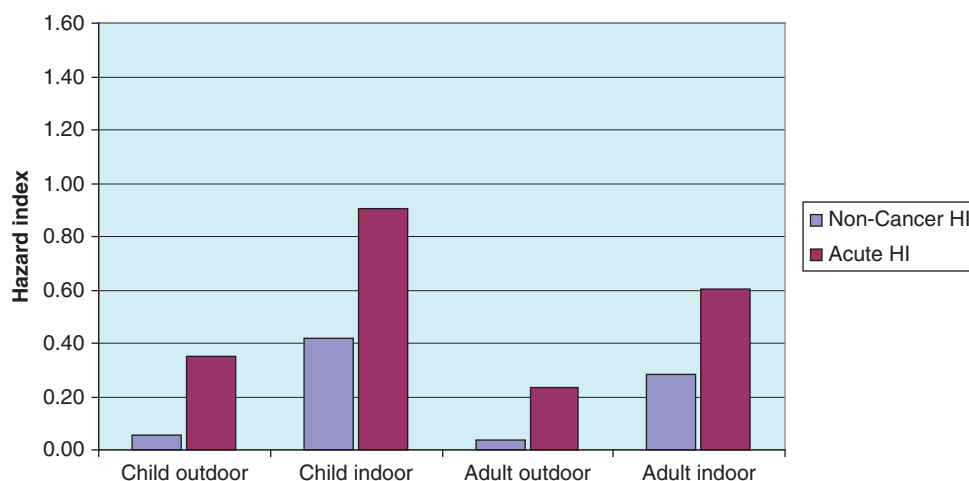


FIGURE 8. Hazard indices for noncancer and acute risk at synthetic turf fields (color figure available online).

TABLE 7. Summary of Artificial Turf Field Risks

	Child outdoor		Child indoor		Adult outdoor		Adult indoor	
Cancer risk	5.00E-07		1.30E-06		2.90E-07		1.10E-06	
Noncancer	0.05		0.42		0.04		0.28	
Acute	0.35		0.9		0.23		0.6	
Key analytes								
Cancer	PAH	6.7%	PAH	26%	PAH	10%	PAH	52%
	MethyleneCl	64%	MethyleneCl	35%	MethyleneCl	61%	MethyleneCl	23%
	Chloro Me	28%	Chloro Me	20%	Chloro Me	27%	Chloro Me	12.8%
	BenzothiaZ	2%	BenzothiaZ	18%	BenzothiaZ	<1%	BenzothiaZ	12.1%
Noncancer	BenzothiaZ	12%	BenzothiaZ	35%	BenzothiaZ	11.5%	BenzothiaZ	35%
	Toluene	30%	Toluene	20%	Toluene	30%	Toluene	20%
Acute	BenzothiaZ	12.4%	BenzothiaZ	56%	BenzothiaZ	12.4%	BenzothiaZ	56%
	Toluene	8.0%	Toluene	7.9%	Toluene	8.0%	Toluene	7.9%

Note. PAH: polycyclic aromatic hydrocarbon; Methylene Cl: methylene chloride; BenzothiaZ: benzothiazole; Chloro Me: chloromethane.

greater cancer risk indoors (Figure 7). Risk estimates for children were somewhat higher than adults due to their greater ventilation rate and vulnerability to mutagenic carcinogens, but this was partially offset by the greater number of years of exposure in the adult scenario. The greatest contribution to cancer risk at the outdoor fields was from the VOC methylene chloride, while indoors there was a more uniform spread of contributing analytes, with PAH making a more prominent contribution (Table 7 and Figure 7). None of the individual contributors approached 1 in a million cancer risk.

The chronic noncancer risk estimate is below unity for all analytes in all scenarios

(Table 7 and Figure 8). Even when adding all HQ together, the total is still below unity. The highest HI is 0.42 for children playing at the indoor field. None of the analytes predominate with the majority of the risk spread between 16 VOC and targeted SVOC. PAH contribute little to the noncancer risk. The greatest percentage contributors are benzothiazole and toluene (Table 7).

The acute risk estimate is below unity for all analytes and scenarios; this is also true when the individual chemical risks are totaled to a cumulative HI (Table 7 and Figure 8). The highest HI is for children at the indoor field, reaching a value just below unity (0.9). This value is driven by benzothiazole (56% of the

total), with relatively minor contributions from a variety of other VOC. The benzothiazole-induced acute effect of potential concern is respiratory irritation.

DISCUSSION

This risk assessment utilized conservative screening level assumptions to assess the sampling results from four outdoor and one indoor synthetic turf field. Full-scale detections of analytes were used without background correction, surrogate chemicals were used to fill in missing toxicity data, risks were added across chemicals regardless of target effect, and high-end assumptions were used regarding time spent playing on the fields. Acute risk was evaluated along with the more traditional chronic risk estimates. While our calculations are likely to overestimate risk, the risk estimates were still uniformly at or below de minimis levels. The greatest cancer risk calculated was $1.3E-06$ for children playing on the indoor field, while the acute and chronic hazard indices were all below unity.

The major area of uncertainty identified is with respect to the acute irritation potential at the indoor field. The HI was near unity (0.9), with benzothiazole the major risk driver. Of all the detected analytes, benzothiazole is most clearly field-related and its levels at the indoor field were 12-fold higher than outdoors. The toxicity data gaps for benzothiazole are more numerous than actual data, and toxicity values used in this assessment were based upon analogy with 2-mercaptobenzothiazole for cancer effects and with formaldehyde for acute effects. The derivation of benzothiazole potency values for acute, chronic, and carcinogenic endpoints is detailed in our companion article (Ginsberg et al. 2011). 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 for other benzothiazole exposures. However, the amount of exposure from food and the possible adverse health effects of that exposure have not

been evaluated. Further, the inhalation route of exposure may be qualitatively distinct from ingestion in food.

The limited inhalation data for benzothiazole indicate activity in the mouse RD_{50} assay, suggesting irritation potential for humans. This assay has been a useful screen for assessing occupational exposure levels for respiratory irritants and is a way to scale the relative potency of one chemical versus another (Bos et al., 1992). Therefore, extrapolation of the ratio of benzothiazole to formaldehyde RD_{50} results (CPSC, 1996) to an acute target concentration for benzothiazole is justifiable. A 10-fold uncertainty factor was added to account for the nature of the extrapolation (across species, across chemicals) and for the potential that benzothiazole may be a sensitizing agent (Ginsberg et al. 2011). The fact that this approach yielded a borderline acute risk for benzothiazole in combination with other irritants found at the indoor field (naphthalene and several VOC) indicates this to be an area of uncertainty.

What is clear is that air concentrations are considerably higher for some analytes at the indoor field. The study team enquired to the building manager as to possible sources of VOC and SVOC in the building from stored materials and products used. This survey failed to uncover any sources that might confound the indoor air results. Thus, it was concluded that the elevated detections came from the field itself. This field has a ventilation system but it was not turned on on the day of testing, which is typical of indoor synthetic turf facilities. The ventilation system is primarily in place to vent excess temperatures in the summertime and thus not running much of the year. The day this facility was tested it was moderate summer weather (outdoor temperature 75°F), so would be unlikely to have triggered running of the ventilation system. After learning of our results, the management at this field decided to vent the indoor air whenever the field is in use.

Aside from lack of ventilation, another reason for elevated concentrations indoors is lack of weathering. Figure 9 shows the rate of weathering of benzothiazole and other analytes

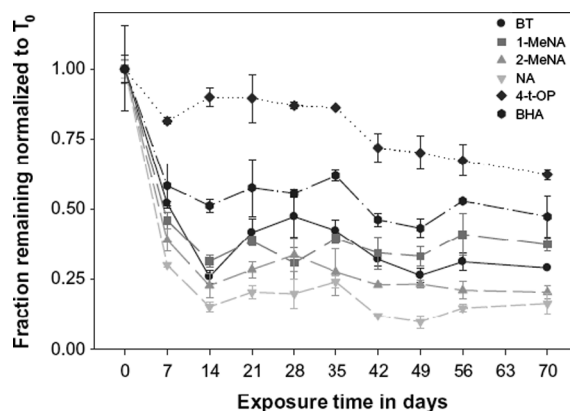


FIGURE 9. Decrease in chemical out-gassing from a crumb rubber sample under natural weathering conditions. Crumb rubber sample was exposed outdoors for the indicated periods of time beginning August 3, 2009. Fraction remaining at each time point was normalized to day 0. Standard deviations of duplicate samples are shown as error bars. Chemical abbreviations: BT, benzothiazole; 1Me-NA, 1-methylnaphthalene; 2Me-NA, 2-methylnaphthalene; NA, naphthalene; 4-t-OP, 4-*tert*-octylphenol; BHA, butylated hydroxyanisole. Reprinted from Li et al. (2010) with permission.

off-gassing from a new crumb rubber sample that had undergone different times of outdoor weathering (Li et al. 2010). Approximately 75% of benzothiazole is lost from the crumb rubber sample within the first 2 wk of weathering, a factor that might make an indoor field retain high concentrations of rubber-related SVOC longer than an outdoor field.

While exposure to all detected COPC is possible, our confidence is greatest for a smaller subset being field-related. That grouping is led by benzothiazole, a compound known to be used in rubber production. The remaining COPC are less specific, with many also coming from background combustion sources, and some VOC may also derive from endogenous (within the body) sources and be detected in personal monitoring samples. Based upon the pattern of detection and presence of laboratory off-gas samples (Li et al. 2010; Simcox et al. 2011), it appears likely that in addition to benzothiazole, detects of toluene, methyl ethyl ketone, methyl isobutyl ketone, BHT, methylene chloride, and a variety of PAH (especially the naphthalenes) were field related. That does not imply that the entire amount detected originated from the field in these cases, but this assumption adds a degree of conservatism to the risk estimates.

An unexpected finding was the number of VOC that were elevated in the personal

monitoring samples relative to the stationary monitors. This raises a concern that some of these detections might not be field-related. In particular, acrolein and benzene were detected in personal monitors at levels that might potentially be risk drivers but were not likely to be derived from the synthetic turf because the grass field personal monitoring showed these analytes at comparable levels, and in the case of acrolein, it is not known to be a component of rubber. While the main source of human exposure to acrolein is considered to be 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 endogenous formation would be expected to be found in exhaled breath, and in fact, acrolein was detected in the exhaled breath of smokers at higher concentration than in nonsmokers (Andreoli et al. 2003). Similarly, benzene was detected in the exhaled breath of nonsmoking individuals in a number of studies with one study of 20 nonsmoking adults finding benzene in the breath of 65% of these individuals (Buszewski et al. 2008). Therefore, use of personal monitoring devices to screen for VOC needs to have appropriate controls that capture the background coming from the equipment or individual wearing the device.

Current Results in Relation to Prior Studies

The current field investigation and risk assessment are similar in approach to several previous assessments, the most relevant being the study of three indoor synthetic turf fields in Norway (NILU 2006), four outdoor synthetic turf fields studied by the U.S. EPA (2009a), and two outdoor synthetic turf fields in New York City (TRC 2009; NYSDEC 2009a). 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 VOC and SVOC, including several specifically targeted because of their presence in rubber (NILU 2006). Chemical detections of PAH (ng/m³ range), benzothiazole (low µg/m³ range up to 32 µg/m³) and VOC (up to a high of 85 µg/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 µg/m³; maximum benzothiazole detect 14 µg/m³). The risk assessment conducted by the Norwegian government evaluated 3 different age windows for children beginning as early as 7 yr of age, and adults (Norwegian Institute of Public Health and Radium Hospital 2006). Their assessment encompassed inhalation as well as dermal and oral exposure. Cancer risks were driven by benzene, which was detected in both on field and background samples with the background-corrected worst case estimate 1.4 µg/m³ corresponding to 2E-06 cancer risk in their highest exposure scenario; this risk was described as negligible (Norwegian Institute of Public Health and Radium Hospital 2006). Our benzene detections were of a similar magnitude, but since similar results were obtained between background and synthetic turf samples, benzene was not considered a COPC. The Norway study found carcinogenic PAH, as exemplified by benzo[a]pyrene, to be from background sources, and non-cancer risks carried a large margin of safety. Therefore, the Norwegian government considered the exposures to indoor synthetic turf

fields to be within acceptable limits (Norwegian Institute of Public Health and Radium Hospital 2006). This conclusion encompassed acute risks, although a formal assessment of acute exposures against acute inhalation benchmarks was not undertaken.

The U.S. EPA performed a scoping-level field monitoring study at four fields, one each in Georgia, Ohio, Maryland, and North Carolina, under summertime conditions in 2008 (U.S. EPA 2009a). VOC, PM₁₀, and metals samples were collected on the fields in the vicinity of play activities at 1 m height. 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 Pb were low relative to National Ambient Air Quality Standards. The U.S. EPA concluded that the methodologies were successful for assessing emissions from artificial turf fields. 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 synthetic turf fields in late August and early September 2008 under warm, light to moderate wind conditions when ambient temperatures (°F) were in the upper 70s to low 80s and surface temperatures on the fields were as high as 146°F (NYSDEC 2009a). Particulate and VOC samples were collected at 3-ft height above the field and at upwind locations. VOC 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 VOC, SVOC, and targeted VOC based upon a laboratory headspace test were analyzed in the samples. The vertical and horizontal gradients of six rubber-related analytes were analyzed to determine if these fields show a measureable emission (e.g., higher concentration at the surface than 3 ft, higher concentration on-field

than upwind). These tests failed to find clear evidence of a field-related gradient and individual VOC were generally no higher on the field than upwind. An exception was benzothiazole, which had a detection of $6.5 \mu\text{g}/\text{m}^3$ on the surface of 1 of the fields while this analyte was nondetect 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 New York City Department of Health found similarly few detections of target analytes, and in that case, 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. Studies did not find many detects that were clearly field-related and when they analyzed health risks, but detections 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 four 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 (e.g., Figure 2). Therefore, this aspect of the design needs to be carefully controlled, and our results point out the value of stationary monitors since they will introduce less confounding. Our results are generally consistent with previous investigations in showing low concentrations 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 and demonstrates some potential for acute irritation at the indoor field; this potential is likely to be manageable by adequate ventilation at these facilities.

In agreement 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 noncancer, and acute health risks for this analyte. These calculations suggest that benzothiazole is unlikely to be a significant contributor to cancer and chronic noncancer risk at the concentrations detected, but that there is some uncertainty as to whether benzothiazole might contribute to an acute health risk for children actively playing on poorly ventilated indoor fields.

Limitations

This investigation was established as a screen of air quality at four outdoor fields and one 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 or elsewhere is unclear. This is especially the case for the one indoor field in that there was no active ventilation or open doors or windows. These fields often get used for youth soccer in colder times of the year, so the volatile emissions may be lower than what was measured. Regarding the outdoor fields, sampling was conducted in July 2009 with targeted conditions being sunny, warm, and low wind. While this goal was accomplished, it was not possible to capture a hot day typical of summer heat waves when the off-gassing of VOC might be maximized. Thus, it is possible that worst-case outdoor conditions were not captured. This worst case may involve new crumb rubber, as headspace off-gas experiments (Figure 9) indicating that outdoor weathering plays a major role in decreasing the availability of chemicals to off-gas from crumb rubber. Thus, a hot, sunny, low-wind day on a new artificial turf field may present the greatest exposure potential for VOC. This would only be a potential concern for acute health risks, as these conditions would not last 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, it needs to be emphasized that 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, present a consistent pattern of there being relatively few detections at outdoor fields under the tested conditions.

As stated earlier, 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; Norwegian Institute of Public Health and Radium Hospital 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. However, it is important to note that our analysis of Pb in synthetic blades and crumb rubber failed to find elevations at any of the fields.

Another limitation is that the current investigation did not attempt to measure latex antigen in the crumb rubber or in the PM₁₀ 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 PM₁₀ on the fields relative to background, suggesting that there was not 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. A fact sheet by the New York State Department of Environmental Conservation (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 rubber industry analysis suggests that latex allergy might not be a health concern from tire-derived particulate, in part because the way tires are made is different than latex gloves and other forms of latex that are highly allergenic (Finley et al. 2003). However, the extensive dermal contact with crumb rubber and potential for low level PM₁₀ exposure during active play leaves open the possibility of exposure to latex allergen on these fields.

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 these fields. There is limited information to suggest that benzothiazole induces dermal sensitization (Ginsberg et al. 2011). Given that benzothiazole may be available for skin contact from the crumb rubber and from ground crumb rubber dust, there is a potential basis for dermal reactions. The rate of transfer of benzothiazole to the skin from crumb rubber is unknown but expected to be low, as benzothiazole is likely to remain in the rubber rather than partition into skin. However, this could be facilitated by intimate contact with the skin over prolonged periods in the presence of sweat. Given the potential for exposure to sensitizing chemicals, latex antigen, and benzothiazole, the possibility that these fields may be associated with respiratory or dermal allergic reactions remains an uncertainty.

CONCLUSIONS

The current investigation was successful in detecting field-related VOC and SVOC at

both outdoor and indoor fields. In particular, benzothiazole is clearly field related and certain PAH and possibly several VOC may also be field related. Risk estimates were at or below de minimis levels for all endpoints for all four scenarios evaluated. The elevated exposures to benzothiazole, naphthalenes, and several VOC found at the indoor field present the most significant uncertainty stemming from the current investigation and risk assessment. This indoor field was not under active ventilation.

While elevated risks were not found at the sampled fields, the uncertainties identified earlier suggest certain measures to decrease the potential for exposure. Indoor artificial turf fields would benefit from adequate ventilation while in use. Given the potential for weathering to reduce the off-gassing of VOC, it would be prudent for outdoor fields to be established in cooler months, giving them time to weather before the high heat conditions of midsummer. Finally, any dermal or respiratory reactions that appear to be field related need to be reported to medical personnel and local health authorities.

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