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Using a Hierarchy of Evidence to Assess Chemical Health Risks of Artificial Turf

Angela Eykelbosh and Daniel Fong[a]

Key Messages

- The evidence regarding the chemical risks of artificial turf exposure is incomplete or weak due to insufficient research, limitations in current methods, or impracticality of the needed study designs.
- Current literature relies heavily on chemical composition and release studies to estimate risk, whereas evidence from personal exposure monitoring and biomonitoring are less common, but more informative for exposure assessment.
- Studies that examine the chemical risks of artificial turf use a variety of methods and approaches, contributing to the difficulty in assessing the overall state of knowledge and strength of evidence.
- Nevertheless, the strengths and limitations of these approaches can be used to assemble a hierarchy of evidence, useful for ranking evidence in decision-making processes.

Introduction

Over the past 40 years, artificial turf has become common in public and private settings. Compared to natural turf, artificial turf is easier to maintain, requires less water and no fertilizer, and provides year-round access to playing surfaces. This is presumed to have important public health benefits by promoting physical activity and access to recreational space,[1,2] although detailed research into benefits is lacking. However, artificial turf has potential drawbacks that range from environmental risks (e.g., chemical leaching to waterways), physical hazards (e.g., heat exposure and increased rates of injury), and finally toxicological hazards. Public risk perception around artificial turf has been amplified by a recent documentary claiming to have found an increased incidence of cancer among young adults playing soccer on artificial turf.[3]

As a result of this widespread use and growing public concern,[4] public health agencies are frequently asked to weigh the risks and benefits of artificial turf facilities. However, this is challenging given that relatively few studies addressed artificial turf health impacts. Furthermore, data on the presence or release of certain toxic compounds is often discussed without reference to exposure scenarios. The aim of this document is to facilitate public health decision-making by discussing the strengths and limitations of the methods used to study the chemical risks of artificial turf, and how these studies contribute to our developing understanding of artificial turf health risks.

[a] Environmental Health and Knowledge Translation Scientists at the National Collaborating Centre for Environmental Health

"Level of Evidence" in Artificial Turf Research

Not all evidence is created equal. In public health, this is often conceptualized as a pyramid or **hierarchy of evidence**, in which syntheses or meta-analyses of critically appraised epidemiological studies form the pinnacle or highest level of evidence (Figure 1A). In contrast, data that are *not* directly derived from humans—such as animal studies and other laboratory assays conducted without direct reference to humans—fall within the realm of "background information." These background studies are highly useful to explore the potential physicochemical and physiological mechanisms of a toxic exposure, and may allow us to define the hazard and speculate as to what might occur in humans, but they do not provide data on actual human health outcomes.

Unfortunately, in the debate over the safety of artificial turf, epidemiological or human studies are lacking. In fact, the lowest "level" of the pyramid—background information—is currently the highest level of evidence available for discussion and decision-making. Accordingly, it is important to understand the strength of this evidence, what it adds to our scientific understanding, and how this information may be understood by a non-expert audience.

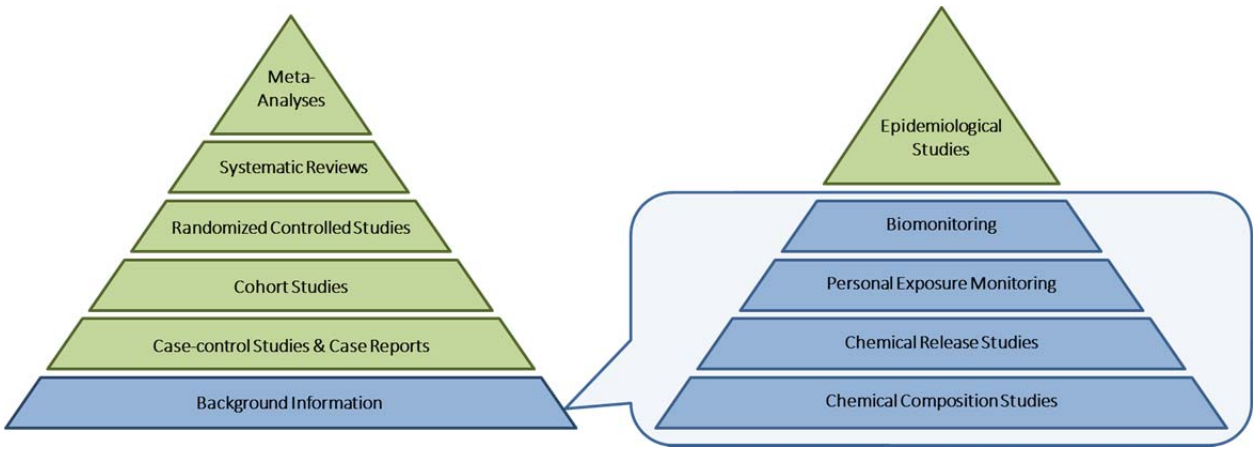

Figure 1. *A) Hierarchy of evidence as typically visualized in evidence-based medicine. B) Modification of the pyramid to show the hierarchy of evidence within the lowest level of the pyramid, as it pertains to artificial turf.*

In this document, we examine the types of evidence that have been used to assess the safety of artificial turf and use them to construct a new hierarchy (Figure 1, right), recognizing the strengths and limitations of each type of evidence. Our objective here is not to comprehensively review the artificial turf literature, as has been done previously,[1,2,5,6] but to show the types of evidence available and provide readers with additional insight into their appraisal.

Chemical composition studies: What is in artificial turf?

Chemical analyses of crumb rubber and artificial turf fibres have found various organic and inorganic substances that are hazardous to human health, including semi-volatile and volatile organic compounds, polycyclic aromatic hydrocarbons (PAHs), and heavy metals.[7-11] Many of these compounds are IARC-classified carcinogens (known, probable, or possible). However, it is important to distinguish between a **hazard** (something that *could* cause harm) and **risk** (the likelihood of coming into contact with that substance and being harmed). Chemical composition analyses inform us regarding hazard, but, for some compounds within artificial turf components, the risk of exposure is very low. For example, heavy metals in crumb rubber are analyzed by first "digesting" the material in pure nitric acid, followed by microwave irradiation (see Menichini et al.[9]), until the metals are fully liberated and can be quantified through other means.

Because such extreme conditions do not reflect what happens in the environment or within the human body, using these values to estimate human exposure may result in a gross overestimation of risk.

Chemical release studies: What gets out of artificial turf?

Chemical release studies examine whether artificial turf-related compounds are likely to be released into the environment under various conditions. Quantifying the type and amount of substances that escape can help to identify hazards, but again provides limited information on risk. Typical chemical release studies include leaching assays (release to water), air quality studies (release to air), and simulated human fluid studies.

Leaching studies examine the release of contaminants into a solvent (typically water). Leaching studies may involve simply mixing the material in the solvent for a specified duration, or may involve complex simulation studies in which the material is layered into a column with soil or other materials. Such column leaching studies are intended to simulate the movement (or retention) of contaminants in soil, and are typically used to explore the potential impacts of artificial turf on receiving waters, such as groundwater or stream water.[12,13] Because these studies are generally of greater relevance to aquatic rather than human toxicology, they are not discussed here.

Leachates from artificial turf components have also been used to perform mutagenicity assays, an analysis that is widely misunderstood. Mutagenicity assays are a screening tool used by toxicologists to determine whether a compound of interest has the *potential* for carcinogenicity. The assay looks at whether exposing bacteria to a compound (or mixture) leads to permanent, transmissible changes or mutations in the cell's genetic material. The assay uses bacteria that cannot survive or grow without nutrient supplementation due to a defective gene, which are incubated in a solution containing the test material. If the solution is mutagenic (causes DNA code changes), then there is a possibility that some bacteria may experience a DNA code change that restores the defective gene to its original functional form. These restored or "revertant" bacteria will then be able to grow without supplementation, and the solution is said to be mutagenic. Further details on the complexities of the mutagenicity assay are available elsewhere.[14]

Several studies have found that artificial turf leachate is indeed mutagenic in bacteria.[15-17] However, mutagenicity in bacteria is not a certain indicator of cancer risk in humans. Although *mutagenicity* (the ability to cause DNA code changes) is often used interchangeably with *carcinogenicity* (DNA code or other changes that lead to cancer), not all mutagens prove to be carcinogenic in animal studies, and not all animal carcinogens are mutagenic in bacteria.[14] Second, mutagenicity assays provide no insight into human exposure or response. That is, how much of the substance will a player receive via inhalation, ingestion, or dermal absorption during normal play? Once inside, how do absorption, metabolism and elimination of the substance modify the risk of ill effects? Clearly, these factors cannot be assessed through a mutagenicity assay. This is mentioned not to downplay or discard mutagenicity data, but rather to place them in an appropriate context as a first-pass screening tool.

Air quality studies aim to identify and quantify gases that escape artificial turf materials under a range of conditions (e.g., temperature and UV exposure, age and source of crumb, etc.). These studies may be performed in a laboratory using specialized chambers to capture escaping gases, or through field studies in which air sampling equipment is installed over actual artificial turfs.[13] These data can then be applied in risk assessments to estimate inhalation exposure. Volatilized contaminants appear to be more concentrated in indoor playing fields,[18] appear higher during continuous vs. intermittent play,[9] and appear to diminish over time with weathering.[19]

Finally, **simulated human fluid studies** are used to determine whether turf components leach toxins when in contact with human fluids, such as sweat,

gastric fluid, or lung fluid.[11,20,21] The test material (e.g., crumb rubber, dust, etc.) is incubated in the fluid, after which the fluid is analyzed for contaminants. The amounts of contaminants recovered are used to estimate **bioaccessibility** or release into body compartments. Bioaccessibility data are then used in risk assessments to estimate exposure to specific substances via absorption through these compartments. For example, Pavilonis et al.[20] found that although new turf fibers contained relatively high amounts of both lead and chromium, only lead was released into simulated fluids, which informed subsequent risk analyses. However, it is important to note that bioaccessibility (the amount recoverable in body fluid) is not equivalent to **bioavailability** (the amount absorbed into the circulatory system) or **bioactivity** (the response at the tissue target site). Thus, bioaccessibility remains only a rough approximation of internal dose.

Personal exposure monitoring

Although air quality studies identify what might be present in the environment, there is a great deal of uncertainty as to what degree humans will come into contact with it. Important considerations include how humans interact with the environment, as well as transport and transformation of the contaminant.

Personal exposure monitoring attempts to reduce this uncertainty by estimating exposure in the player's immediate surroundings, typically in their personal breathing zone (for inhaled toxins). The importance of personal exposure monitoring becomes apparent when comparing data from stationary samplers vs. those from personal samplers on artificial turf, natural turf, and the background urban environment.[9,18,22] Simcox et al.[18] found a great deal of variation in the type and quantity of VOCs found above various indoor, outdoor, and artificial turfs. To complicate matters, the authors also reported that personal air samplers attached to players' waistbands showed *higher* concentrations for some VOCs than stationary samplers positioned six inches above the surface, and that some compounds were found at higher concentrations above grass fields than artificial turfs. These findings called into question which of the > 20 chemicals studied were truly derived from artificial turf, rather than the urban background or the players themselves (e.g., from sports equipment or the use of personal care products). It was only through a combination of gas emission measurements in the laboratory, stationary field samplers, and personal samplers, that researchers were able to identify artificial turf VOCs. [23] This work highlights the importance of careful sample collection and data validation when estimating players' inhalation exposure.

Although there has been some work on characterizing dermal exposure (skin abrasion rates while playing),[24] experimental studies to support personal exposure through ingestion are lacking.

Biomonitoring: What gets into human bodies?

Despite data regarding chemical constituents and their release into the environment, there are very few data regarding the presence of these substances within humans as a result of playing on artificial turf. *Biomonitoring* refers to the analysis of known biomarkers of chemical exposure, typically either the contaminants themselves or their metabolites, which are collected from the human body (e.g., from blood, urine, or other biological samples). Biomonitoring is a more powerful means to assess exposure because it integrates all pathways of exposure and accounts for the moderating effects of absorption, metabolism, and elimination. However, biomonitoring studies have limitations, principally the time and expense required to fully understand and validate a biomarker before it can be used, as well as the ethical and logistical issues associated with human sampling.[25] Biomarkers may also be specific to a compound or associated with a group of compounds, which can make it difficult to attribute exposures.

Only one study has evaluated change in a single biomarker of artificial turf exposure. Van Rooij and Jongeneelen[26] analysed 1-hydroxypyrene, a metabolite related to PAH exposure, in the urine of seven adult, non-smoking, soccer players, before, during and after playing on artificial turf. This analysis did not detect evidence of play-related 1-hydroxypyrene above levels due to normal dietary or environmental exposure. This study shows that although players were likely exposed to a mixture of the many PAHs that have been detected at low levels over artificial turf, playing on that turf did not result in additional 1-hydroxypyrene within the urine. Further work is needed to study a wider variety of contaminants and their biomarkers and to apply this methodology in children.

Epidemiological studies: Is exposure connected to health outcomes?

Environmental epidemiology is the study of the relationship between exposures and the occurrence of disease in human populations. This evidence can be used to inform decision-making on artificial turf by feeding into decision tools like human health risk assessment.[27] One limitation of epidemiological studies is that they cannot answer precisely *why* disease occurs. Although epidemiological studies may find a statistical association between certain risk factors (e.g., exposure to artificial turf) and a health outcome, they cannot rule out or control for all other potential causes of or contributions to that outcome. Demonstrating cause and effect requires examining the exposure under carefully controlled experimental conditions, and comparing exposed and non-exposed individuals (work done using animals, for ethical reasons). In addition to lack of causation, well-designed epidemiological studies can be time-consuming and expensive, and as such may be impractical within the time available for a specific decision.

To date, no epidemiological evidence is available regarding artificial turf. In response to the 2015 documentary,[3] the Washington Department of Health is following up with some of those identified to better understand their exposure to artificial turf and, ultimately, to understand whether certain cancers are more prevalent among young adult soccer players compared to non-soccer players.[28] Although results are not expected until the end of 2016,[29] this work represents the first epidemiological study of human health outcomes associated with artificial turf exposure.

Making Decisions: Synthesizing Evidence and Projecting Risk

Once the available evidence has been collected and reviewed, additional methods or decision tools are required to aggregate this information and predict future risk to the public. **Human health risk assessment (HHRA)** is a process through which real-world data are used to construct or calculate this theoretical risk, based on a number of assumptions. Methods vary, but generally include four key steps: 1) the health hazards of a specific contaminant set are identified; 2) the effects of exposure at different levels are assessed (typically based on animal studies, but potentially also epidemiological studies); 3) a realistic estimate of human exposure is generated; and finally 4) human health risk is characterized. The final risk characterization step produces an estimate of the likelihood of an adverse human health outcome for the populations of interest. For non-carcinogenic toxins, a risk exists if the estimated exposure dose is greater than an established reference dose or threshold above which adverse effects are possible (i.e., a hazard quotient > 1).

Cancer risk is not based on a reference value, as it is assumed that there is no threshold for cancer effects. That is, even a low exposure generates a small increase in the excess lifetime cancer risk. In the US, where most artificial turf studies have been performed, the risk is deemed "*de minimis*" or negligible to the population at large if it is below 1 in 1,000,000 (1×10^{-6}, or 1 additional case of cancer per 1,000,000 exposed persons). Risks estimates greater than 1×10^{-6} generally require further research, and risk reduction measures are typically recommended when the risk estimate exceeds 1 in 10,000 (1×10^{-4}).

The strength of most HHRAs is that they allow investigators to explore the potential risks of multiple exposures (singly or together) in various exposure scenarios, something that would be prohibitively expensive, impractical, or unethical to investigate in real life. However, HHRAs must rely on a combination of real-life data and expert judgements or assumptions to come up with a final risk estimate. As more and better data are collected, and fewer assumptions are required, the result is strengthened.

HHRAs for artificial turf can be complex. Turf components emit a mixture of potentially toxic compounds. In the absence of toxicological data for a specific chemical, researchers may use data for a similar compound presumed to act in the same way. Even when good data are available, these data (generated in animals for ethical reasons) may not be fully representative of humans. A much greater issue, however, is that exposure characterization requires data-based estimates that reflect differences in sex, age, amount of playing time, type of contact (running, diving, sliding), field type (newer, off-gassing fields vs. weathered fields), field material (conventional crumb rubber vs. the many new alternatives), meteorological conditions (hot fields vs. cool fields), and so on. The HHRA must also consider how humans might be exposed to the contaminant in other normal activities. These data are currently lacking.

Although some assumptions are required in any HHRA, a well-designed study will make these assumptions explicit and conservative. That is, investigators use values that are likely to overestimate the risk, such that the "real risk" falls somewhere safely below the risk estimate produced by the analysis. Conservatism can be built into an HHRA in a number of ways: 1) by basing analyses on high-end data (e.g., 95th percentile values, rather than mean or median values; 2) overestimating the exposure (e.g., assuming an athlete will be on the field for 12 hours a day for 30 years); 3) by applying an "uncertainty factor" to any parameter for which the data are less reliable; and 4) summing risk estimates for different compounds, even when they are mechanistically unlikely to have an additive effect.

A number of studies (Appendix A) have estimated the cancer and non-cancer-related risks from the most toxic artificial turf contaminants, with varying degrees of complexity in the exposure assessment. To date, most studies have reported at or below *de minimis* risk levels for cancer and non-cancer effects for most users, including children, teens, and adults.[9,10,13,20,21,23,24,30,31] These results are striking in that very conservative assumptions were made that would greatly overestimate risk. However, as mentioned, the assumptions required within such RAs create some degree of uncertainty. Thus, even when risk estimates are low, the uncertainties identified are important for further data collection. For example, Pavilonis et al.[20] found that although artificial turf-derived metals and SVOCs posed negligible cancer or non-cancer risks across a wide age range, lead levels in turf products were so variable that further data collection was deemed necessary to verify whether the values used in the assessment were indeed representative.

Health impact assessment (HIA) is another tool that has recently been applied to artificial turf. HIAs differ from HHRAs in several ways: they use both quantitative and qualitative evidence to assess risks; 2) they consider both the positive and negative potential impacts (including toxicity) of an activity or policy, and 3) they take a community perspective. Methods vary, but generally consist of a six-step process (screening, scoping, appraising, reporting, and monitoring and evaluation).[32] The aims of an HIA are both to maximize benefits (and reduce risks) to the general population, as well as to reduce health inequities in cases where risks and benefits are unevenly or unfairly distributed across a community.[33]

Because HIAs attempt to capture the complexity of interacting factors that affect community health, they are highly attractive as a decision-making tool for public health policy. However, they are also more challenging to undertake, and have been criticized for lack of methodological rigour,

insufficient evidence base, and (subsequently) difficulty in generating a reliable assessment of risks and benefits.[32,34,35] Nevertheless, HIAs are becoming increasingly common in public policy. Toronto Public Health[1] used HIA to discuss the potential harms and benefits of artificial turf due to environmental factors (creation of urban heat islands, heat-related injuries, toxic exposures, and storm-water management), built environment and lifestyle factors (access to recreational space, access to green space, and neighbourhood impacts), and equitable access for disadvantaged communities and people with disabilities, compared to natural turf. The report concluded that although there does not seem to be evidence of toxic exposures, artificial turfs should only be installed in locations where natural turf is not practical. Natural turfs were valorized for their ability to sequester carbon, capture storm-water, and cool the urban environment, all of which were considered to improve resilience in the face of climate change, while at the same time providing access to natural, more biodiverse greenspace. As is often the case in HIA, the lack of data for either artificial or natural turf means that these assertions are open to challenge. Despite this, HIA remains a valuable exercise as it highlights the myriad ways in which the use of artificial vs. natural turf could impact determinants of health beyond simple toxic exposures.

Conclusions

The aim of this document is not to comprehensively review the evidence of the health effects associated with artificial turf, which has been covered elsewhere,[1,2,5,6] but rather to critically examine the types of evidence available. Given that no epidemiological studies regarding artificial turf exist at this time, decision- and policy-makers must instead attempt to understand artificial turf risk based on a combination of laboratory analyses, bioassays, and limited human exposure data, which have been used to produce human health risk assessments of varying designs and quality. Here, we attempt to order these types of evidence into a hierarchy of evidence, and to highlight some of the strengths, limitations, and points of misinterpretation for each.

It is hoped that future initiatives will further improve the evidence available. The upcoming *Federal Research Action Plan*,[36] as well as the California OEHHA study,[37] will greatly increase data available for exposure assessment, although the biomonitoring component is limited. As these new data are released, reference to this document may help decision-makers categorize new information into a hierarchy of evidence, and facilitate decision-making concerning artificial turf. However, it should be noted that although the upcoming data will help to reduce uncertainties in risk assessment, uncertainty with human health risks will remain. Epidemiological studies may serve to further reduce this uncertainty, but cannot completely eliminate uncertainty given the complex exposures that humans receive on a daily basis.

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Appendix A – Human Health Risk Assessments

Authors	Material Studied	Contaminants	Users	Exposure Pathways	Supporting Data	Findings
Ginsberg et al. 2011[23]	Air sampled from four indoor and one outdoor artificial turf fields.	VOCs, SVOCs, nitrosamines, PM_{10}, Pb.	Children (12 years old) + adult (30 years old).	Inhalation	Stationary on-field and off-field monitoring, personal monitoring, and laboratory off-gassing studies.	Although a number of substances were found to be higher on artificial turf fields (especially indoors) compared to off-field locations, the cancer and non-cancer risk derived from those concentrations were at or below *de minimis* levels for both children and adults. Comprehensive air quality analysis was necessary to differentiate emissions from artificial turf vs. urban contaminants or emissions from the players themselves.
Kim et al. 2012[30]	Rubber chips from play-grounds.	Metals (Pb, Cr, Cd, Hg, and Zn), toluene, ethylbenzene, and phthalates.	Elementary, middle school, and high school children, as well as adults and children with pica.	Inhalation, ingestion, dermal	Measured chips in 50 schools.	Cancer and non-cancer risk at or below *de minimis* for all users, except children with pica who showed low-level cancer (1×10^{-4}) and non-cancer risk.
Lim and Walker, 2009[13]	Rubber crumb from two outdoor fields, on hot summer days.	SVOCs, VOCs, and PM.	Children	Inhalation	Upwind, on-field, downwind, and laboratory off-gassing studies to identify artificial turf-derived components. Also used simulated play during PM measurements.	Under hot weather conditions, SVOC, VOC, and PM emissions unlikely to pose a public health risk compared to contaminant exposures off-field. However, temperatures on the field were markedly higher compared to grass and sand surfaces.

Authors	Material Studied	Contaminants	Users	Exposure Pathways	Supporting Data	Findings
Menichini et al. 2011[9]	Rubber granules from 13 outdoor fields.	PAHs	Children	Inhalation	Stationary on-field and off-field samplers, as well as personal samplers worn at waist height to simulate exposure to children.	Estimated cancer risk for benzo[a]pyrene exposure at *de minimis* level (1×10^{-6}).
Nilsson et al. 2008 [10]	Desk study	Benzothiazole; dicyclohexylamine, cyclohexanamine, dibutyl phthalate.	Young adults (16-19 years).	Dermal and oral only.	Chemical and leaching studies to select substances of concern.	No effects for the four chemicals studied, although maybe risk of allergic sensitization.
NIPH 2006[31]	Two indoor artificial turf facilities.	VOCs, dust, phthalates, and alkyl phenols.	Children (7-11 years), older children (12-15 years), juniors (16-19 years), adults (20-40 years).	Oral, dermal, and inhalation for children only; dermal and inhalation for all other groups.	Composition analyses, leaching, laboratory off-gassing studies, and dust wipe sampling.	Neither inhalation, nor dermal, nor oral exposure to the contaminants listed were considered to pose a public health risk, although authors note the unknown risk of developing asthma or allergy to compounds present in dust in indoor facilities.
Pavilonis et al. 2014[20]	New fibers, new rubber crumb, and rubber crumb from 7 existing fields.	PAHs, VOCs, SVOCs, metals (Ag, As, Be, Cd, Cr, Cu, Mg, Pb, Se, Ti, V).	Young children (6-10 years), older children (11-15 years), teenagers (16-18 years), adults (≥ 19 years).	Dermal, ingestion, and inhalation.	Bioassays, laboratory off-gassing studies.	Results did not identify cancer or non-cancer risk from metals or SVOCs, although it was noted that lead levels varied widely and lead was found to be bioaccessible in simulated human fluid studies.

Authors	Material Studied	Contaminants	Users	Exposure Pathways	Supporting Data	Findings
Vidair 2010[24]	Crumb rubber from outdoor fields.	$PM_{2.5}$, bound metals, and VOCs on in-use and (separately) hot outdoor fields, compared to natural turf.	Children (5-15 years), young adults (16-18 years) and adults (19-55 years).	Inhalation and skin infection.	Air emissions during hot weather; bacteria present and abrasion rates on artificial turf compared to grass and sand fields.	No public health risk due to $PM_{2.5}$, lead in dust, or VOCs, although higher skin abrasion rates could increase the risk of skin infection. However, fewer bacteria were found on artificial turf compared to natural turf.
Vidair et al. 2007[21]	Playground surfaces.	Al, As, Ba, Cd, Co, Cu, Cr, Fe, Pb, Mn, Hg, Ni, Se, Zn; VOCs, SVOCs.	Children (1-12 years).	Oral and dermal exposure.	Wipe sample of playground surfaces and gastric digestion assays.	Found *de minimis* levels of risk for one-time ingestion of 10 g of tire shreds by a three-year-old, based on values from the literature and a gastric digestion bioassay; found slightly above *de minimis* cancer risk for chronic ingestion of chrysene; no skin sensitization in guinea pigs.

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200 – 601 West Broadway
Vancouver, BC V5Z 4C2

Tel.: 604-829-2551
contact@ncceh.ca

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