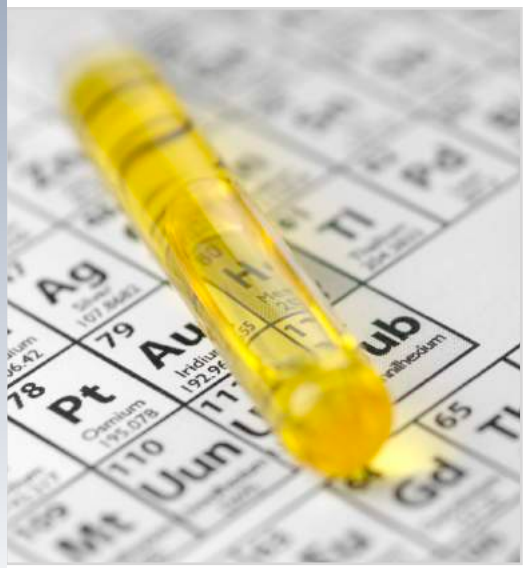
HEALTHY BUILDING NETWORK

Full Disclosure Required:

A Strategy to Prevent Asthma Through Building Product Selection



A Healthy Building Network Report
by Sarah Lott and Jim Vallette
December 2013

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* Institutional affiliation listed for identification purposes only

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1 Executive Summary

Asthma rates in the United States have been rising since at least 1980. Today, nearly 26 million people are affected by chronic asthma, including over eight million children.

These rates are rising despite the proliferation of asthma control strategies, including indoor air quality programs. The Centers for Disease Control (CDC) reported that the number of people diagnosed with asthma grew by 4.3 million during the last decade from 2001 to 2009.[1]

As asthma affects more people, it becomes increasingly clear that new strategies need to be considered, focusing on the prevention of asthma onset. Few strategies are in place that effectively prevents exposure to chemicals that cause asthma. Due to the complexity of this condition conventional efforts have largely focused on asthma management.[2]

Health organizations have identified a number of chemicals that are known to cause the onset of asthma, and are therefore labeled asthmagens. Since these chemicals are common ingredients of many interior finishes, like floors, carpets, and paints, it is possible to improve asthma prevention strategies by reducing or eliminating these chemicals from building materials.

The Healthy Building Network (HBN) took a three-pronged approach that examined how pervasive asthmagen chemicals are in the built environment, what steps have been taken to address them, and what further actions are needed.

This research began by investigating asthmagen chemicals identified through occupational health research. We cross-referenced three authoritative lists of asthmagens maintained by the Association of Occupational and Environmental Clinics (AOEC), Commission de la santé et de la sécurité du travail (CSST), and Collaborative on Health and the Environment (CHE) with the known ingredients of over 1,300 building products. The ingredients of these products are found in the Pharos Building Product Library (Pharos BPL), an online disclosure tool developed by HBN in 2009. This analysis identified 38 asthmagens chemicals used in building materials listed in Pharos.

We also examined emerging evidence that a different set of chemicals may be setting up the conditions for asthma early in life through effects on early childhood development. Children are readily exposed to these suspected asthmagens through building products, such as stain repellants in the fabric of carpets, and plasticizers

that migrate from vinyl floors to household dust that children inhale or touch. There is growing concern that these exposures can disrupt the prenatal and neonatal development of immune systems and lungs, which can in turn lead to the development of asthma. This research identified 12 additional chemicals for further research and prioritization.

We examined scientific and government literature for all 50 of these asthmagen and suspected asthmagen chemicals used in products evaluated by the Pharos BPL. Of these 50 chemicals, 20 chemicals were found to have exposure pathways that may impact building occupants through product installation and/or normal use. These substances, and the products that use them, may be more significant and less controlled factors in the development of asthma than previously understood. However, with this and further research, in many cases it will be possible to reduce and possibly eliminate these chemicals from the built environment as part of a comprehensive asthma prevention strategy.

Key Findings

HBN identified twenty top-priority asthmagens in nine chemical groups that are used in building materials and have high likelihood of occupant exposure.

These asthmagens are found in foam insulation, paints, adhesives, floors and carpets, among many other interior materials (Table 1) to which building occupants routinely come into contact, by touch or inhalation:

- Acid anhydrides (two types)
- Acrylates (four types)
- Ammonium hydroxide
- Bisphenol A Diglycidyl Ether (BADGE)
- Ethanolamines (three types)
- Formaldehyde
- Isocyanates (six types)
- Polyfunctional aziridine
- Styrene

Emerging evidence shows that a dozen chemicals commonly used in building products can impact children at their earliest ages and lead to the development of asthma. We consider eight of these chemicals to be top priorities for consideration in asthma prevention strategies.

Scientists are calling attention to a growing body of research that some chemicals, especially phthalate plasticizers, may disrupt the development of organs such as the lungs. Researchers are finding that exposures can occur before and after birth, and can impair the development of lungs and immune systems. This disruption in turn can lead to the development of asthma.

The association between these chemicals and the onset of asthma is indirect and the chemicals do not meet current clinical criteria to be designated as asthmagens. Therefore, authorities like the AOEC cannot list them as such. Current understanding is that these chemicals may play a role in asthma onset through impacts on lung and immune system development, and do not *directly* cause asthma. However, this should not preclude these chemicals from being considered in precautionary strategies to prevent building materials from contributing to the development of asthma. There is enough evidence of pathways and exposures to suggest that the time for preventative action, particularly for phthalates, is now.

An appendix to this report discusses other asthmagens and suspected asthmagens for which the evidence of exposure pathways for building occupants is less conclusive. These chemicals should be prioritized for further research and possible inclusion in asthma prevention strategies.

Emissions-based building product certification protocols may not adequately address chemicals that cause the onset of asthma.

Building product IAQ certification systems are designed primarily to reduce occupant exposure to VOCs at concentration levels below those known to pose a number of health hazards to building occupants, such as cardiovascular, nervous or reproductive system conditions. Since many VOCs may also exacerbate asthma, these certification systems have been used in strategies to address asthma. However, exposure threshold levels established for VOCs that have been deemed protective against other diseases may not be protective against asthma causation.

Additionally, many asthmagen chemicals are not VOCs, and will not be detected by current emissions testing protocols. As a result, many known and suspected asthmagens can be present even in products that have earned low VOC-emissions certifications.

For example, of the 50 chemicals that we discuss in this report, only three are included in both protocols of the two leading IAQ protocols.

Additional measures must be taken to reduce building occupant exposures to asthmagens, as we outline below.

Recommendations

A precautionary approach that prevents the introduction of asthmagens from building materials into the indoor environment is both possible and an essential part of the strategy to reduce the rate of asthma onset. These are the key elements of this new strategy:

Screen building product contents for asthmagens. Building owners, landlords, interior designers and architects can use the Pharos Building Product Library to identify products that do not contain top priority asthmagens.

Knowing what is in building products is an essential component of any asthma-prevention strategy. Manufacturers should fully disclose all product ingredients so consumers can make informed purchasing decisions. When there is full disclosure, consumers can determine whether asthmagens or chemicals indirectly associated with asthma are present in the product. Consumers should give preference to manufacturers who fully disclose ingredients when purchasing products, and to products that do not contain asthmagens.

Coincident with the release of this report, HBN has expanded its coverage of asthmagens in Pharos. We've added a restricted substance list to the Pharos Chemical & Material Library (Pharos CML) that identifies the twenty top-priority asthmagens and eight high-priority suspected asthmagens identified in this report and a filter to the Pharos BPL that allows Pharos subscribers to identify and select products that do not contain these top-priority chemicals.

Through a combination of manufacturer participation, HBN research, and data quality controls, this tool gives consumers – who live and work in buildings with these materials – the opportunity to discover the substances to which they may be exposed. It is part of our belief at HBN that consumers are entitled to knowledge about such health risks.

The Pharos BPL also evaluates building products by reference to IAQ certifications that measure VOC emissions. Thus it is possible for Pharos subscribers to cross reference IAQ certified products against the new asthmagen filter in order to select for products that perform best at avoiding both type of health hazards.

Every building product purchase presents a series of environmental and human health choices. These choices should be viewed as a whole cloth, rather than piecemeal. The Pharos Project– which cross-references product ingredients against over fifty authoritative hazard lists – provides the clearest pathway for making these choices.

Increase research to understand the potential contributions of chemicals in building materials to the rising epidemic of asthma, and to create safer alternatives to asthmagens.

Environmental, public health, public housing, and other agencies should intensify efforts to identify and prevent avoidable exposures to asthmagens in homes, schools, hospitals and public spaces.

Asthma experts should consider the emerging evidence about phthalates, including how prenatal and early life exposures may play a key role in the onset of asthma. This evidence is of particular importance for environments dedicated to young children such as day care centers, schools, and hospitals, especially in their design of neonatal and pediatric care units.

Authorities should include precautionary actions to avoid exposures to chemical asthmagens as part of a comprehensive asthma reduction strategy.

Manufacturers should work with green chemistry experts to identify and develop suitable alternatives to asthmagens in building products.

Emerging evidence of cellular and epigenetic[a] pathways for chemicals to impact the development of lungs and immune systems speaks to the urgent need for further research into how materials may contribute to early childhood asthma.

Develop protocols and modify indoor air quality certification and building rating systems to address asthma.

The IAQ standards development community should develop testing protocols for the presence and emissions of asthmagens in products and during screening. This will require developing and incorporating consensus standards for laboratory measurement of SVOCs and non-volatiles that can be incorporated into building IAQ standards. Pending the creation of these standards, IAQ certification programs should develop protocols to screen product content declarations to avoid priority asthmagen content.

Product disclosure tools such as the Health Product Declaration should include asthmagens as one of the health hazards in their disclosure protocols, by reference to the AEOC and other authoritative lists.

Building rating systems like the U.S. Green Building Council's Leadership in Energy and Environmental Design (LEED) should develop incentives that reward projects that avoid introducing asthmagens into the indoor environment.

[a] According to the Centers for Disease Control (CDC), "Epigenetics is the study of changes in phenotype caused by something other than changes in the underlying DNA sequence (for example, DNA methylation)." A phenotype is how an organism looks and functions based upon their DNA and the environment. ("Pediatric Genetics: Glossary of Terms." Centers for Disease Control and Prevention. Accessed November 4, 2013. http://www.cdc.gov/ncbddd/pediatricgenetics/glossary.html)

We hope this report provides a frame of reference for further action by the public health and green building communities. Together, we can identify healthy alternatives to building products that expose people to chemicals that cause asthma.

> "When people doubt that we can improve health outcomes, we're going to show them the drawers of unused asthma inhalers in green schools."
>
> *- Rick Fedrizzi, USGBC President and CEO, Plenary Address, Greenbuild 2012*

Table 1. High-priority chemicals

Chemicals in building materials	Lists	Uses in building materials	Exposure pathways for building occupants
ASTHMAGENS (*)			
Acid Anhydrides (Maleic anhydride and Phthalic anhydride)	AOEC (Rs), Quebec CSST, CHE (acid anhydrides; S- strong)	Alkyd and epoxy resins used in coatings, including paints, vanishes, adhesive and high performance coatings (HPCs); and rubber flooring	Inhalation of emissions during installation and inhalation, dermal contact, and ingestion of dust from wear of coatings and floors
Acrylates (MMA, PMMA, Acrylic Acid, TMPTA)	AOEC (Rs), Quebec CSST (MMA and cyanoacrylates), CHE (methacrylates; S- strong)	Paints, lacquers, varnishes, insulation binders, fluid applied floors (FAFs), flooring finishes, and countertops	Inhalation of emissions during installation of wet applied products and dermal contact with residual monomers in solid products
Ammonia hydroxide	AOEC (Rs)	Chalkboard paints and acrylic adhesives	Inhalation of emissions during installation and inhalation, dermal contact, and ingestion of dust from product wear
Bisphenol A Diglycidyl Ether (BADGE)	AOEC (epoxies; G), Quebec CSST, CHE (epoxy resins; S- strong)	Epoxy adhesives, HPCs, and FAFs	Inhalation of emissions during installation and inhalation, dermal contact, and ingestion of dust from wear of coatings and floors
Ethanolamines (Monoethanolamine; 2- dimethylaminoethanol; Triethanolamine)	AOEC (Rs), Quebec CSST, CHE (ethanolamines; S- strong)	Spray polyurethane foam (SPF), adhesives, insulation binder, and HPCs	Inhalation of emissions during installation of wet applied products and insulations
Formaldehyde	AOEC (G), Quebec CSST, CHE (S- good)	Formaldehyde-based resins used in wide range of materials, including laminates, insulations, wallboard, engineered wood, and acrylic/latex adhesives	Inhalation of emissions during installation and product use
Isocyanates (HDI, TDI, pTDI, pMDI, Pure MDI, and 2,4-MDI)	AOEC (G), CHE (isocyanates; S-strong), Quebec CSST (TDI, Pure MDI, and various combinations of TDI, MDI, and HDI)	Polyurethane systems including insulation, binders, FAFs, carpet backing, and foam cushions	Inhalation of emissions during installation of wet applied products and insulation; inhalation and dermal contact with emissions and dusts from polyurethane products
Polyfunctional Aziridine (PFA)	AOEC (Rs) and Quebec CSST	Cross-linking agent (hardener) in wet applied products such as finishes and paints	Inhalation of emissions and dermal contact during installation and dust from wear of coatings
Styrene	AOEC (Rs), Quebec CSST, CHE (S- limited)	Carpets, rubber flooring and other styrene-butadiene rubber products, polystyrene insulation, and HPCs	Inhalation of emissions during installation and product use.
SUSPECTED ASTHMAGENS (**)			
Phthalates (DnHP, DNOP, DBP, DIDP, BBP, DEHP, Dicyclohexyl Phthalate)	CHE (phthalates; S&I -limited) and Quebec CSST (DEHP only)	Various vinyl (PVC) products, lacquers, flooring finishes, adhesives, and FAFs	Inhalation, dermal contact, and ingestion of dust during use of the building product.

(*) Chemicals that directly cause asthma, per the authoritative list of the Association of Occupational and Environmental Clinics (AOEC)

(**) These chemicals are indirectly associated with asthma onset (evidence includes epidemiological studies, epigenetic research, and findings of early life lung and immune system developmental impacts).

2 Building Product Ingredients That Cause Asthma

Asthma is a chronic inflammatory lung condition that makes it difficult to breathe properly. Untreated, inflamed lungs become more responsive to various stimuli. These responses include wheezing, coughing, chest tightness, chest pain, and shortness of breath.

In the United States, twenty-six million people are affected by chronic asthma. After more than doubling between 1980 and 1995, asthma rates continued to increase, at a slower rate, from 1997 to 2010. The percentage of U.S. children "ever diagnosed with asthma" rose from 11.4% of all children in 1997 to 13.6% in 2010. [3b]

Each year in the United States, the condition leads to approximately 13 million visits to physician offices, 1.8 million emergency room visits, and 500,000 hospitalizations. Asthma accounts for over 14 million lost school days for children, and another 14 million lost workdays for adults. Over 3,000 people die from asthma attacks annually.[4] Between medical costs, lost school and work days, and early deaths Asthma costs the US over 50 billion dollars annually.[5]

In this section, we examine the roots of this epidemic, including ways in which building materials may play a significant role in its development.

How Chemicals Cause Asthma

Disease prevention requires understanding how environmental factors contribute to its development. Asthma develops through a variety of complex mechanisms. Many factors influence the development of asthma including dose, duration, physiochemical characteristics of a chemical (such as molecular structure or particle size), and genetic and other factors pertaining to the individual. Once asthma develops, hypersensitivity to many agents may develop.

As a US Department of Health and Human Services report explains, asthma causes an increase in airway responsiveness to a variety of stimuli and if "untreated, the inflammation may lead to irreversible changes in lung structure, known as airway remodeling." [6]

Asthma onset has been broken down into two types: allergic asthma and irritant-induced asthma, which result in the same symptoms but reflect different pathological mechanisms. A person may present with either type or both types depending upon their exposure to asthmagens.

b While increasing percentages of people have been diagnosed with asthma, death and hospitalization rates have declined, by 26% and 24%, respectively, from 1999 to 2009. This success is apparently due to improvements in managing asthma attacks. ("Trends in Asthma Morbidity and Mortality." American Lung Association. Accessed September 2012. http://www.lung.org/finding-cures/our-research/trend-reports/asthma-trend-report.pdf.)

Allergic asthma is the most common form. It is also called immunologic or sensitizer-induced asthma. It involves an immune system response to an allergic agent (sensitizer).[7]

Typically, the initial exposure does not produce immediate symptoms; rather, symptoms develop over time with increased exposure and initiation of the immune response (clinically recognized by creation of specific immunoglobulin E (IgE) antibodies, which elicit airway inflammation). Levels of exposure required to elicit this response and cause asthma are not understood, though some researchers believe the severity of reaction and asthma onset may be resolved if removed from exposure to these asthmagens early on.[8]

Specific mechanisms, involving the immune system, are understood for some, but not all, sensitizing agents.[c]

While inhalation is the primary route, dermal (skin contact) exposures to sensitizing agents have also been found to result in respiratory sensitization.[9] Once a person has asthma, chemicals can exacerbate their symptoms into full-blown asthma attacks or episodes. The more frequent the episodes, the worse the lungs function.

On the other hand, in **irritant-induced asthma** the clinical evidence[d] of asthma is clear, but the immune system is not involved and scientists are still working to understand the mechanism leading to onset. Asthma symptoms due to an irritant do not generally have a latency period, delay between stimulus and response, as is seen with sensitizing agents, and "may be caused from a single exposure to the irritant."[10] However, recently, some have begun to suggest that low level exposures could result in irritant-induced asthma with latency.[11] The levels and frequencies of exposures needed to induce this form of asthma are not well studied.

Emerging evidence of connections between early life exposures and childhood asthma suggests other mechanisms for, and contributing factors to, the development of asthma. This evidence is discussed in Section 3 of this report.

[c] A common mechanism of allergic asthma is the binding of sensitizing agents to antibodies or proteins. Whether a sensitizer binds to an antibody or protein can depend on its molecular weight. High molecular weight substances are large enough to bind with immunoglobulin E (IgE) antibodies. The combining of antigens and IgE antibodies, explains the Massachusetts Toxics Use Reduction Institute, can "produce a cascade of events causing activation of inflammatory cells and the synthesis and release of several mediators that control the inflammatory reaction in the airways." Lower molecular weight agents may bind with other proteins to form an allergenic compound that initiates a similar immune mechanism. ("TUR and Disease Prevention Fact Sheet: Asthma." Massachusetts Toxics Use Reduction Institute. Published 2012. http://www.turi.org/content/download/7403/134641/file/Asthmagens%20fact%20sheet.pdf)

[d] Examples of clinical evidence for irritant-induced asthma can be found in the Association of Occupational and Environmental Clinics (AOEC) Exposure List protocol. ("Revised Protocol: Criteria for Designating Substances as Occupational Asthmagens on the AOEC List of Exposure Codes." Association of Occupational and Environmental Clinics (AOEC). Updated October 2008. http://www.aoec.org/content/Asthmagen_Protocol_10-25-08.pdf)

ASTHMA GLOSSARY

some commonly used and confused asthma terms

ASTHMAGEN A specific agent which causes the onset of asthma in someone who did not previously have the condition. Asthma onset is commonly broken down into two major causes: sensitization or irritation. Asthmagens are not limited to common environmental allergens such as dust mites, but also include many substances that can be found at work or in the home.

SENSITIZATION Sensitizer-induced asthma, more commonly known as allergic asthma, is caused by an immune system response, which is clinically observed through the creation of a specific antibody, immunoglobulin E (IgE).[e] IgE antibodies trigger inflammatory reactions in the airways, which results in asthma symptoms over time. With continued exposure, symptoms worsen, more IgEs are produced and the immune system becomes conditioned to the inflammatory response.

IRRITATION Irritant induced asthma does not involve the immune system and specific mechanisms for onset are largely unknown. Commonly, irritant-induced asthma is induced by single or intermittent high level exposures, and occurs without a latency period. This form of irritant-induced asthma is called Reactive Airway Dysfunction Syndrome (RADS).[12] However, recent research has revealed evidence for irritant-induced asthma via acute exposures with a latency period, though the mechanism of causation is still unclear. [13]

TRIGGER Substances that provoke symptoms in those with asthma, such as coughing, wheezing, bronchoconstriction. These triggers are not necessarily asthmagens.

EXACERBATION Once asthma develops, airway responsiveness can increase to a variety of stimuli. This continued inflammation can lead to worsening of asthma symptoms as well as lead to "irreversible changes in lung structure, known as airway remodeling." In some cases, the development of hypersensitivity has led some to confuse these additional stimuli, which may only exacerbate an existing asthma condition, with asthmagens (i.e. chemicals that contribute to asthma onset).[14]

[e] See other examples of clinical evidence for sensitizer-induced asthma on the AOEC Exposure list protocol. (Revised Protocol: Criteria for Designating Substances as Occupational Asthmagens on the AOEC List of Exposure Codes. *Association of Occupational and Environmental Clinics (AOEC)*. Updated October 2008. http://www.aoec.org/content/Asthmagen_Protocol_10-25-08.pdf)

Identifying asthmagens in building products

Indoor environmental conditions can lead to the development of asthma through exposures from before birth to early childhood to adulthood, and can trigger asthma attacks for those who already have the disease. Building materials used in indoor spaces can contribute to these conditions.

To identify asthmagens involved in building materials we compiled a list of chemicals using three commonly referenced asthma lists: the AOEC Exposure Code List, the CSST List of Agents Causing Occupational Asthma, and the CHE Toxicant and Disease Database (For more information on the Listing organizations and list protocols, see text box). We cross-referenced chemicals on this compiled list with those present in building products in the Pharos BPL.

The Pharos BPL evaluates over 1,300 building products and components. The Pharos BPL characterizes a cross-section of interior finishes that can greatly impact indoor air quality, such as paints, adhesives, composite woods, floors, walls, ceilings, and insulation. Pharos BPL evaluations provide an extensive record of product ingredient information.

Our analysis identified the presence of 50 asthma related chemicals in building products. We reviewed the available scientific and regulatory literature on each, including many of the case studies that asthma experts rely upon for developing the three asthma lists and others like it. Our literature review also revealed emerging evidence for chemicals currently not on asthma lists, for which epidemiological evidence and investigations into new understandings of asthma onset have linked these chemicals to asthma.

We not only considered the case studies and epidemiological evidence available on asthma, but also considered pathways for building occupants to become exposed to these asthmagens during the service life of materials in which they are present.

Building occupants can be exposed to asthmagens in building materials by several pathways:

- Non volatile asthmagens on the surface of a building finish (carpet, color, furniture, wall, etc) may be released from the finish as dust through degradation or abrasion and be picked up through the skin on contact
- Semi-volatile asthmagens may migrate from products on to dust particles by adsorption which may in turn be inhaled, ingested, or come into contact with the skin.
- Volatile and semi volatile asthmagens may volatilize and be emitted into the air to be inhaled.

These pathways will vary in importance for different asthmagens. For some chemicals the evidence is stronger that they can cause asthma through inhalation, for others through dermal contact.

Asthma lists examined for our report

Association of Occupational and Environmental Clinics (AOEC) Exposure Code List

The AOEC is a non-profit organization dedicated to "[facilitating] the prevention and treatment of occupational and environmental illnesses and injuries through collaborative reporting and investigation of health problems." The AOEC Exposure Code List includes asthmagens reported by asthma experts and subsequently reviewed under AOEC criteria. Criteria for designation as an asthmagen include specificity and relevant exposure pathways. Additionally, to determine the type of asthmagen, the AOEC looks for these main clinical indicators in case studies published in scientific journals: specific inhalation challenges with immediate or delayed fall in expiratory flow and/or volume – sometimes called the "gold standard" for diagnosis – evidence of changing airway reactivity in response to exposure and control periods, airway hyperresponsiveness to non-specific stimuli, exposure related asthma symptoms, and presence of specific IgE antibodies (indicates allergic/sensitizer-induced asthma). The AOEC list uses "Rs" for sensitizing agents, "Rr" for Reactive Airways Dysfunction Syndrome (RADS) agents, "Rrs" if both a sensitizing and RADS agent, "R" if the substance has been reviewed and the literature doesn't meet the criteria for either type, or "G" for agents generally accepted as asthmagens.[15]

Commission de la santé et de la sécurité du travail (CSST) List of Agents Causing Occupational Asthma

The CSST is an organization, based in Quebec, that compiles and analyzes scientific information to provide health and safety information for use by Quebec employers. This information includes a list[f] of occupational asthmagens, based on the extensive occupational asthma research of Moria Chan-Yeung and Jean-Luc Malo, who initially published the *Tables of major inducers of occupational asthma* in the 1999 edition of *Asthma in the Workplace*.[16]

The Collaborative on Health and the Environment (CHE) Toxicant and Disease Database

CHE is a project of Commonweal, a health and environmental research institute in Bolinas, CA. Their Toxicant and Disease Database summarizes links between chemicals and more than 200 human diseases or conditions, of which sensitizer- and irritant-induced asthma are included. The database uses human epidemiological studies, health organization data, such as the California Office of Environmental Health Hazard Assessment (OEHHA), and, in some cases, animal data to inform these chemical lists. The chemicals are listed in connection with a disease/ disorder and are then grouped by the strength of evidence available: strong (S), good (G), or (L) limited/ conflicting evidence.[g]

[f] The updated list maintained by CSST is available as a pdf (http://www.asthme.csst.qc.ca/document/Info_Med/IdCauses/Bernstein/AgentsAnglais.pdf) and as an online database (http://www.asthme.csst.qc.ca/document/Info_Gen/AgenProf/Bernstein/BernsteinAng.htm#N1_1).

[g] The database and its limitations, are further described here: http://www.healthandenvironment.org/tddb_about

We also considered how commonly these chemicals are used in building materials, and in which settings, in order to help prioritize preventative strategies. Depending upon the amount of evidence and potential for building occupant exposure, we categorized the asthmagens into two groups for prioritization in asthmagen prevention strategies. Those chemicals with clear links to asthma causation and pathways of exposure to the building occupant were placed on a list of high-priority asthmagens that should be incorporated into asthma prevention strategies. Chemicals with less conclusive evidence of asthma causation or exposure pathways to the building occupant were placed on a list of low-priority asthmagens, which should be researched further for possible inclusion in asthma prevention strategies.

We determined at least 28 chemicals deserve urgent attention, which in many cases can be avoided through informed selection of interior building products. In the following two sections, we examine the scientific literature that connects these top-priority chemicals to asthma, and the pathways by which building occupants may be exposed to them.

The first set of substance profiles looks at known asthmagens, chemicals with clinical evidence of causing asthma onset. The subsequent section looks at emerging evidence on childhood exposures and new understandings of asthma onset, forms a starting-point for any strategy to avoid asthmagens in building materials. Table 1 summarizes the key data points about each of these chemicals.

An appendix to this report describes 22 other substances of concern that we identified in building products. Some are established asthmagens, but the pathways by which they may cause asthma from building materials in building occupants is not as clear.

Other chemicals described in the appendix have emerging concerns about their potential to damage the development of children's' lungs.

We recommend further investigation on whether building products present pathways for building occupants to be exposed to all of the chemicals addressed in this report, such as through emissions, dusts, and dermal contact.

High-priority asthmagens: Substance Profiles

A. Acid Anhydrides

Chemicals Used in Building Materials: Maleic anhydride, Phthalic anhydride

Authoritative Lists: AOEC (Rs), Quebec CSST, CHE (acid anhydrides; S- strong)

Exposure Pathways for Building Occupants: Inhalation of emissions and dermal contact during installation and dust from wear of coatings and floors.

According to the United Nations Environment Programme, exposure of the general public to anhydrides may occur through releases from indoor products containing the chemical such as varnishes, paints, and coatings.[17]

Cyclic acid anhydrides, such as phthalic and maleic anhydrides, are commonly used in polyester and alkyd resins, and epoxy resin hardeners, which in turn are ingredients of a variety of wet applied products such as paints, varnishes, adhesives, and high performance coatings. These coatings may be factory-applied to other products or site-applied in buildings.[18]

The World Health Organization says anhydrides are irritants and sensitizers. Symptoms of irritation can begin immediately following inhalation or direct skin contact with vapors or dusts.[19]

Sensitization, as opposed to irritation, via anhydrides is mediated by a different mechanism. Symptoms of sensitization (e.g. rhinoconjunctivitis, bronchial hyper-responsiveness, and increased airway resistance) are caused by an immune-mediated mechanism (creation of IgE antibodies which elicits an inflammatory response) and occur after a symptom-free latency period and, most commonly, multiple exposures.[20]

Immune-mediated sensitization (allergic asthma) due to specific anhydrides has been reported and diagnosed in several human and animal studies through specific IgE antibodies, specific inhalation challenges and patch testing.[21]

The World Health Organization notes, "Because of the sensitizing nature of the cyclic acid anhydrides, tolerable concentrations... cannot be established." [h]

B. Acrylates

Chemicals Used in Building Materials: Methyl Methacrylate (MMA), Polymethyl Methacrylate (PMMA), Acrylic Acid, Trimethylolpropane Triacrylate (TMPTA)

Authoritative Lists: AOEC (Rs), Quebec CSST (methyl methacrylate and cyanoacrylates), CHE (methacrylates; S- strong)

Exposure Pathways for Building Occupants: Application of wet applied acrylate materials, such as paints, lacquers, varnishes and adhesives and fluid applied floors. Longer-term exposures are also possible through dermal contact with residual monomers in solid polymeric products.

[h] Human in vitro experiments have found that anhydrides "readily reacts with amino acids, forming protein conjugates such as serum albumin conjugates", further supporting an immune-mediated mechanism (Kim, James H., Herman J. Gibb, Annette Iannucci, "Cyclic acid anhydrides: human health aspects," World Health Organization, 2009, http://www.who.int/ipcs/publications/cicad/cicad75.pdf. (Kim et al 2009)

In animal studies, "significant dose – response relationships have been observed between immune responses and exposure to cyclic acid anhydrides. Antibodies that are usually increased after sensitization and challenge are immuno-globulin E (IgE) and IgG, which are reactive towards the anhydride–albumin conjugate that is being studied" (Organization for Economic Cooperation and Development (OECD), "Phthalic Anhydride: CAS No.: 85-44-9," United Nations Environment Programme, April 5, 2006, http://www.inchem.org/documents/sids/sids/85449.pdf. (OECD 2006)). While not as common, skin sensitization due to maleic and phthalic anhydrides has been reported (UNEP 2006 and WHO 2009).

Occupational asthma among workers who handle wet applications of acrylates, especially dental assistants and fingernail sculptors, is well documented. [22]

Our analysis of Pharos BPL listed products found significant amounts of acrylates in products: 70% or more MMA in acrylic solid surfaces and fluid applied floorings, up to 50% acrylic acid in acrylic paint, up to 40% TMPTA in UV-cured finishes, and 30% or more PMMA in acrylic solid surfaces.

According to the European Chemicals Bureau (ECB), MMA residuals are routinely present in aqueous dispersions like acrylic paints (at between 0.0005% and 0.05% of the product as a whole) and in cast sheets (such as those used in solid surface applications like countertops), at between 0.5% and 1.1% of polymeric product. The ECB notes that two-part reactive acrylic adhesives can contain up to 30% MMA.[23]

The ECB states that workers installing industrial floors are exposed to high levels of MMA (up to 774 mg/m^3) in the air. Dermal exposures to MMA are additional hazards for construction workers applying fluid applied floors and other wet applied products.[24]

Exposures are not restricted to professionals. Consumers may also apply wet acrylics in their homes. Two-component acrylic adhesives, commonly sold by hardware stores, pose an "acute exposure by inhalation to MMA [that] can be neglected," according to the ECB.[25] Additionally, a 1990s study found that a consumer who paints two rooms with water-based acrylic paints would be exposed to "the same order of magnitude" of MMA as that found in industrial settings.[i]

In a 2007 EPA-supported review of indoor residential chemical emissions, Mark Mendell notes that several studies associate painting and renovation with wheezing. It is unclear which paint ingredients are triggering the wheezing. In addition to acrylates, acrylic paints can contain other ingredients that also are asthmagens.[26]

> "Several studies associate painting and renovation with wheezing."

[i] Currently, the Pharos BLP data has not associated MMA with widespread use in paints. However, a MMA safety assessment shows that MMA use in paints may still be common ("Product Safety Assessment: DOW™ Methyl Methacrylate Monomer," Dow Chemical Company, November 20, 2010, http://msdssearch.dow.com/PublishedLiteratureDOWCOM/dh_07f2/0901b803807f2a8f.pdf?filepath=productsafety/pdfs/noreg/233-00665.pdf&fromPage=GetDoc). Thus far, the disclosure of materials in paints has been limited. HBN will be looking into this issue further to see if MMA is still an issue for paints.

C. Ammonium Hydroxide

Authoritative Lists: AOEC (Rs)

Exposure Pathways for Building Occupants: Inhalation of emissions and dermal contact during installation and dust from paint and adhesive product wear.

Ammonium hydroxide is found in a variety of products including household cleaners, textiles, rubber, and paints.[27] Occupational asthma from ammonia hydroxide is common among cleaning workers.[28]

This alkaline agent is very irritating to the eyes, skin, and mucous membranes. Ammonium hydroxide causes necrosis (cell death) in tissues via "saponification of cell membrane lipids resulting in cell disruption and death." Additionally, ammonium hydroxide "extracts water from the cells, and initiates an inflammatory response, which further damages the surrounding tissues," according to the US CDC.[29]

Continuous heavy exposure can cause bronchitis and bronchial hyper-responsiveness.[30] Cases of high-level respiratory irritant exposures from ammonium hydroxide have been reported to "induce new onset of asthma with no latency period, namely, reactive airways dysfunction syndrome" (RADS).[31]

D. Bisphenol A Diglycidyl Ether (BADGE)

Authoritative Lists: AOEC (epoxies; G), Quebec CSST, CHE (epoxy resins; S- strong)[j]

Exposure Pathways for Building Occupants Inhalation of emissions and dermal contact during installation and dust from wear of coatings and floors.

Compounds containing BADGE – made from bisphenol A and epichlorohydrin – are widely used in epoxy adhesives, which AOEC has determined are asthmagens. Skin and respiratory sensitization has been reported from occupational exposures to these compounds in epoxy resins.

A team of Finnish occupational health experts observed the onset of occupational asthma by dermal contact.[32]

Two cases of skin sensitization followed by respiratory sensitization have been reported and confirmed with positive skin prick and patch tests and decreased peak expiratory flow.[33] In one study, specific IgE antibodies were found for BADGE and epoxy resins containing BADGE.[34]

[j] The AOEC and CHE lists do not list BADGE specifically, but list the general term epoxy resins. Epoxy resins, like polyurethane systems, are broad categories that are not based on a specific recipe. Not all polyurethane systems necessarily use isocyanates, and nor are BPA-derived compounds like BADGE pre-requisite for all epoxy resins. See, for example, http://gradworks.umi.com/15/13/1513898.html. In our literature review of epoxies, we found that the term epoxies encompasses many chemicals already identified as specific asthmagens, such as various acid anhydrides. This was also reflected in the CSST list, which lists three anhydrides and BADGE in particular as associated with epoxy resin manufacture. Additional scientific literature supported this listing of BADGE. Skin prick testing for epoxies, for example, provoked the presence of IgE specific to BADGE.

E. Ethanolamines

Chemicals Used in Building Materials: Monoethanolamine (MEA); 2- dimethylaminoethanol; Triethanolamine (TEA)

Authoritative Lists: AOEC (Rs), Quebec CSST, CHE (ethanolamines; S- strong)

Exposure Pathways for Building Occupants: Inhalation of emissions during installation of wet applied products and insulations.

Ethanolamines are widely used in consumer product mixtures like cosmetics and medications, and as building blocks for chemicals like resins and pesticides.[35] In buildings, we find them used in sprayed polyurethane foam, epoxy adhesives, insulation binder and lacquers (Table 1).

"The vapor pressure of the ethanolamines is low in ambient conditions; however, heating and aerosolization can increase potential exposure," notes *Environmental and Occupational Medicine*. "[C]ompounds containing ethanolamine and its derivatives have been reported to induce asthma in multiple industrial settings, including spray painting, detergents, [and] machining with cutting fluids… Occupational Asthma has been reported following exposure to amino-ethyl ethanolamine, dimethyl ethanolamine, ethylenediamine, 2-diethylaminoethanol, monoethanolamine, diethanolamine, and triethanolamine."[36]

Many studies have linked ethanolamines to asthma. For example:

- Belgian researchers conducted specific inhalation challenges in workers with cleaning-related asthma symptoms. They found "a pattern of bronchial reaction consistent with sensitizer-induced occupational asthma." [37]
- A much-cited 1994 article by Savonius et al reviewed three cases of occupational exposure (two metal workers, one custodian) and determined that occupational asthma is caused by ethanolamines.[38]
- In 1998, Piipari et al examined that case of a worker using a cutting fluid with diethanolamine as an ingredient and reporting asthmatic symptoms. Testing exposed the worker to two aerosolized concentrations of DEA (both below ACGIH thresholds). Both concentrations led to constricted airways. Researchers concluded that DEA can "induce occupational asthma by a sensitization mechanism." [39]

F. Formaldehyde

Authoritative Lists: AOEC (G), Quebec CSST, CHE (S- good)

Exposure Pathways for Building Occupants: Emissions from laminates, insulations, wallboard, engineered wood, acrylic/latex adhesives.

OSHA describes formaldehyde as "a sensitizing agent that can cause an immune system response upon initial exposure." It says that formaldehyde "can make anyone exposed cough and wheeze" and that "long-term exposure to low levels in the air or on the skin can cause asthma-like respiratory problems." [40]

A 2011 EPA assessment looked at the off-gassing of formaldehyde from composite woods over the service life of these products. The agency found higher levels of asthma in workers manufacturing particleboard, and noted that the symptoms improved when they were away from the job. [41]

Several studies have found elevated levels of formaldehyde and the presence of particleboard to be associated with asthma diagnoses, chronic bronchitis, wheezing, and other respiratory symptoms.[42]

G. Isocyanates

Chemicals Identified in Building Materials: 1,6-Hexamethylene Diisocyanate; Toluene Diisocyanate (TDI); Polymeric TDI; Polymeric Methylene Bisphenyl Diisocyanate (pMDI); Pure MDI; 2,4'-MDI.

Authoritative Lists: AOEC (G), CHE (isocyanates; S-strong), Quebec CSST (TDI, Pure MDI, and various combinations of TDI, MDI, and HDI)

Exposure Pathways for Building Occupants: Installation of wet applied products and spray polyurethane foam insulation; long-term exposures possible from dermal contact and airborne emissions.

Large proportions of polyurethane building materials are comprised of isocyanates, making them one of the most prevalent asthmagens in building materials (Table 1).

These chemicals are used in adhesives, furniture foam, insulation, flooring finishes and high performance coatings. Many of these polyurethane systems are mixed and applied on site in homes, schools, and offices. These are relatively unregulated and uncontrolled processes in widely varying environments, in which workers and occupants are likely exposed to unreacted components during and after installation.

Isocyanates are the subject of intensive federal government scrutiny:

- The Environmental Protection Agency (EPA) is researching "source issues of spray polyurethane foams (SPF) manufactured on site" and will be issuing an initial report in 2014.[43]
- In June 2013, the Occupational Safety and Health Association (OSHA) initiated a National Emphasis Program to identify and reduce or eliminate the incidence of adverse health effects associated with occupational exposure to isocyanates.[44]

OSHA identifies isocyanates as respiratory, eye, and gastrointestinal irritants. "Hypersensitivity pneumonitis (inflammation in the lungs caused by exposure to an allergen)" has been reported in workers exposed to isocyanates, with symptoms experienced months or even years after exposure ends, according to the agency. "Deaths have occurred due to both asthma and hypersensitivity pneumonitis from isocyanate exposure," according to the agency.[45]

Isocyanate exposures may occur through inhalation or touch. According to OSHA, "Studies indicate that dermal exposure is a significant cause of respiratory sensitization. Thus, workers with skin contact to isocyanates may develop sensitivity, resulting in asthma attacks with subsequent exposures." Isocyanates are also allergic sensitizers, sometimes leading to "cross-sensitization" where exposure to one isocyanate leads to the development of an allergy to another isocyanate.[46]

No industry or government source can be found to clarify how much time is needed until occupants can return to a building in which polyurethane insulation or adhesives have been installed. The National Institute for Occupational Safety and Health (NIOSH) requested field data to help answer this essential question in 2012.

The amount of time it takes to complete the chemical reactions in polyurethane systems, called the "cure rate," appears to be determined by the amine catalysts used in the "B-side" of the two part system. Varying types and amounts of amine catalysts used in a given SPF formulation make predicting cure times, and thus safe re-entry time, difficult.[47]

David A. Marlow, a NIOSH industrial hygiene engineer, says his agency is trying to determine the "actual amount of time before the area is void of harmful levels of vapors. The idea that the area needs to be clear for 24 hours is anecdotal and has no scientific underpinning." [48]

A recent case report may represent the tip of the iceberg of residential exposures to isocyanates. A couple had SPF installed in the attic of their home. An array of asthma-related symptoms started immediately upon their return home after evacuating for the installation.

The couple's doctors reported, "The SPF used in our patients' home was a two-component SPF system (Sealection® 500; Dimilec USA, LLC, Arlington, TX) that contained polymeric diphenylmethane diisocyanate (MDI) (50% to 60%), 4,4'-MDI (35% to 45%), and 2,4'-MDI (1% to 5%) in side A. Both patients were diagnosed with asthma or reactive airway dysfunction syndrome induced by exposure to isocyanates and were treated with

bronchodilators and inhaled steroids.... *Our patients were told to return 4 hours after the application was completed, and thus were likely exposed to high concentrations of MDI.*" [49] (Emphasis added)

Numerous claims against SPF manufacturers and installers have been consolidated into a national class action lawsuit. In July, US District Judge Jan E. DuBois determined at that SPF can off-gas dangerous chemicals into the air, and that "plaintiffs have adequately pled a cause of action for negligence against defendants." [50]

A recent EPA presentation notes that "SPF Insulation component chemicals can migrate to other areas of the building" and that isocyanates "can trigger severe or fatal asthma attacks in sensitized persons upon further exposure, even at very low levels." [51]

In comments submitted to EPA earlier this year, the American Chemistry Council (ACC, the industry's trade association) failed to answer questions about curing rates and safe re-occupancy times.

"There are various ways to define when SPF insulation is fully cured," the ACC wrote to EPA. "Some look at certain physical properties of the installed SPF and believe when these have been achieved the insulation is cured (the SPF is tack-free within several minutes of application, and may achieve its desired physical properties within 24 hours of application). Others may look at the amount of unreacted isocyanate (which appears to be below the limit of detection on the surface of the foam within 15 minutes and below the limit of detection in the air within 2 hours after application). Additional discussion may be needed in this area to agree on an exact definition of cured SPF.... Also, while curing time and re-occupancy time may be related, they are not necessarily one in the same." [52]

While field cured application of isocyanate products appears to be particularly problematic, consumers may also be exposed to isocyanate emissions from polyurethane products that were cured prior to installation, such as polyurethane-backed carpets. A 2010 Berkeley National Laboratory review of chemical emissions from residential materials reported that "emissions of toluene 2,4 diisocyanate – a highly reactive compound – are detected in carpet with polyurethane foam backing." These emissions suggest that polyurethane products continue to contain and release isocyanates long after they have been manufactured.[53]

Into this uncertainty enters another troubling hypothesis: that neonatal dermal exposures to isocyanates in polyurethane products may cause asthma.

In 2003, a research team looked for isocyanates in polyurethane medical materials used in a New Zealand neonatal unit, items like adhesive films, feeding tubes, and disposable diapers. Their tests detected isocyanates in all of these products. They found that the "opportunities for dermal exposures to polyurethane products and isocyanates are numerous." They noted that isocyanates "are notable because of their capacity to elicit respiratory response at extremely low levels of exposure," and that the skin of young children is "thin, delicate, and susceptible to alterations in integrity... Thus, we theorize that neonatal exposure to polyurethane products containing isocyanate residue may contribute to an immune system imbalance and predispose children to asthma development." [54]

Exposure pathways may begin even sooner than early childhood. The new field of epigenetics (as discussed in the section below on Prenatal and Early Life Exposures) is exploring the roles environmental exposures to certain chemicals may have in gene transcription. In a study of mice, the offspring of mothers exposed to TDI before becoming pregnant had an increased susceptibility to asthma.[55]

Isocyanates are low vapor pressure substances and are not considered in IAQ testing protocols. As a result, there are isocyanate-laden products with IAQ certifications. For example:

- The GreenGuard program certifies a flooring finishing system that contains 1,6-HDI.
- Spray foam insulation systems routinely pass California 01350 tests.
- FloorScore certifies recycled rubber floors that contain isocyanate binders.
- The Carpet and Rug Institute's Green Label Plus program has certified hundreds of polyurethane-backed carpets.

Because these air quality programs do not test for isocyanates, they cannot be used to indicate whether a polyurethane building product might cause the onset of asthma.

H. Polyfunctional Aziridine (PFA)

Authoritative Lists: AOEC (Rs) and Quebec CSST

Exposure Pathways for Building Occupants: Installation and wear of wet applied products, including flooring finishes, adhesives, paints, paint primers, topcoats.[k]

PFA has become a common cross-linking (hardening) agent in two-component wet applied products, including interior paints and flooring finishes available in hardware stores. There is considerable evidence that skin contact with products containing PFA can cause asthma.

These "potent skin and respiratory sensitizers"[56] have been found to cause asthma, or precursor allergic contact dermatitis, in workers in an array of occupations. Documented cases include: artificial dry ski slope manufacturers[57]; dye mixers and leather workers[58]; fiberboard painters, parquet layers, and PFA salesmen[59]; wallpaper printers[60]; painters using a primer to protect wood siding[61]; and, spray painters.[62]

In the spray painter's case, scientists from the Harvard School of Public Health documented how an initial dermal exposure in 1992 led to breathing difficulties the following year. This progression exemplifies how skin sensitization and subsequent allergic responses can cause asthma.

[k] Currently, polyfunctional aziridine is only associated with flooring finishes in Pharos BPL listed products, but occupational asthma case studies report use as a hardening agent in many other wet-applied products including, paints, dyes, adhesives, and other finishes.

I. Styrene

Authoritative Lists: AOEC (Rs), Quebec CSST, CHE (S- limited)

Exposure Pathways for Building Occupants: Volatile emissions from styrene-butadiene rubber products, including carpet backings, polystyrene insulation, and high performance coatings.

In 2006, Fernandez-Nieto et al. reviewed cases of styrene and asthma. They concluded, "Although the underlying mechanism causing these events is still unknown, it appears to be similar to other low-molecular-weight agents causing immunologic occupational asthma. The causative role of styrene in occupational rhinitis and asthma should be investigated among workers exposed to this compound." [63]

In 1991, doctors in London documented the case of a Royal Air Force technician who, while working with fiberglass over a twenty-month period, experienced chest tightness, wheezing, and other asthma-related symptoms. "Styrene seems able to initiate asthma by inducing a specific hypersensitivity response," they found. [64]

According to the Agency for Toxic Substances and Disease Registry (ATSDR) toxicological profile for styrene,, "the principal route of styrene exposure for the general population is probably by inhalation of contaminated indoor air.... Emissions of styrene from building materials (carpets, floor tiles, and insulation), office copiers and consumer products... may contribute significantly to indoor air pollution." The profile notes one study calculated a styrene emission rate from glued carpet of 98 ng/minute/square meter. [65]

The ATSDR profile notes that children are exposed to higher amounts of styrene per body weight than adults. "Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume," it notes. A study of elementary school children in Minneapolis found that these exposures led to blood level concentrations of styrene that were generally twice as high as the general population.

Styrene, a VOC, is one of the building material asthmagens measured in standard IAQ tests, including certification programs used as the carpet industry standard. However, these protocols rely upon thresholds established for occupational settings or for health impacts that are not related to the initial onset of asthma. (See later discussion on IAQ programs.)

3 Early Life Chemical Exposures and Asthma

Early-life exposures to some chemicals may contribute to the development of asthma, according to a growing body of evidence. Researchers are finding that exposures that occur before (prenatal) and after (postnatal) birth can impair the development of lungs and immune systems. (See, for example, the evidence described for isocyanates, above.)

Children are particularly vulnerable to asthma. The absence of public policies to prevent chemical exposures early in life is responsible, at least in part, for the rising rates of asthma among children, according to public health scientists Philip Landrigan and Lynn Goldman.[66]

This vulnerability comes from several factors: children's developmental processes are easily disrupted and they have more time to develop chronic diseases. Additionally, children are more vulnerable because their bodies are still developing the capacity to produce the enzymes needed to break down and remove toxic chemicals from the body and they have greater exposures to toxic chemicals for their body weight than adults." [67]

One way that childhood exposures may inflict damage is through disruption of hormonal cell signaling that is responsible for lung development and maturation. Chemical agents, such as phthalates and perflurocarbons (discussed below and in appendix A), have the potential to bind to receptors in cells that control development. Animal studies have revealed that phthalates can bind to hormone receptors involved in initiating expression of genes involved in development and maturation of a number of tissues, including the lungs. Animal pre- and post-natal exposures to phthalates resulted in changes in airway remodeling and allergen response, which can disrupt lung development and function.[68]

> "Asthma can be thought of a failure of development where the normal development of the respiratory and immune systems is altered by the impacts of environmental exposures acting on underlying genetic predispositions."
>
> - P.D. Sly, M. Kusel, P. Franklin, and P.G. Holt, *"Environmental Factors in Children's Asthma and Respiratory Effects: Reference Module in Earth Systems and Environmental Sciences,"* in Encyclopedia of Environmental Health, 2011, 376-379.) (Sly et al 2011)

How chemicals can disrupt lung and immune system development

Epidemiological studies on children with asthma are finding links between adverse influences on lung and immune system development as infants and development of asthma later in life.[69]

At birth, only 30-50% of our alveoli, the air sacks in our lungs responsible for oxygen exchange, are present. After birth, rapid growth of these alveoli occurs. Lung volume doubles by 18 months and again by 5 years of age. Sly et al reports, "Normal lungs grow along trajectories; however, exposure to inflammatory or irritant stimuli can retard lung growth."

During this time the infant immune system is also developing. Prenatal and early postnatal immune systems are biased toward producing IgE antibodies that activate inflammatory cells.[70] Unimpaired, individuals lose this bias in favor of IgG antibodies that protect against sensitization.[71]

Early exposures to sensitizing agents, such as those found in building materials, thus can disrupt the development of lung and immune systems, leaving airways stunted and the immune system biased toward producing IgE.

The field of **epigenetics**[l] is exploring another pathway for pre- and post- natal onset of asthma. As a recent Pesticide Action Network report explains, "Many environmental pollutants can strip or add chemical tags to DNA, locking the expression of genes on or off and changing how they function. These changes are called 'epigenetic tags,' and have been linked to various health effects."[72] Singh and Shoei-Lung Li (2012) add that alterations in gene expressions, induced by exposures to toxicants, "may persist throughout life."[73]

Maternal exposures to asthmagens can cause epigenetic (see textbox) changes that have impacts on the child's immune system development and function.[74] In one study, mice were exposed, dermally, to toluene diisocyanate (a common building material ingredient) before becoming pregnant. Their offspring had an increased susceptibility to asthma.[75]

Epigenetic changes have also been associated with EDCs, specifically Bisphenol A and phthalates, although the implications for health impacts such as asthma development are currently unclear.[76]

These emerging associations of asthma with cellular and epigenetic pathways show an imperative for further research into these mechanisms as a way to prevent and potentially treat childhood asthma.

l For more background on gene expression and genetics, many resources are available at: http://www.biochemweb.org/genes.shtml

Preventing early life childhood exposures

Some chemicals commonly used in building materials disrupt the prenatal and neonatal development of organs such as lungs. This disruption, as discussed above, in turn can lead to the development of asthma.

Because the association between these chemicals and the development of asthma is indirect, this relationship does not define the chemicals as asthmagens – that is, they do not directly cause asthma. However, this indirect association should not preclude these chemicals from being considered in strategies to prevent building materials from contributing to the development of asthma. There is enough evidence to suggest that the time for preventative action is now.

For this report, and for developing an asthma prevention filter in the Pharos BPL, we considered pathways for people to become exposed to these chemicals during the service life of materials in which they are present.

Based on their indirect associations with asthma, we concluded that phthalate plasticizers should be considered as high priorities in asthma prevention strategies.

In addition to phthalates, we have compelling evidence of concern that perfluorocarbons (PFCs, commonly used as stain repellants in carpets) also may be causing asthma though similar pathways. A 2013 study of 456 Taiwanese children found that children with asthma had significantly higher concentrations of PFCs in their blood than children without asthma.[77] Children are readily exposed to PFCs through hand-to-mouth transfer from treated carpets. There is an urgent need for more research on the relationship between asthma and PFCs, especially the C-6 stain repellants that have replaced the better known C-8 compounds, PFOA and PFOS. See Appendix A for further information.

Building owners, landlords, interior designers and architects can use the Pharos Building Product Library to identify products that do not contain top priority asthmagens.

Suspected Asthmagens: Substance Profiles

A. Phthalates

Chemicals Identified in Building Materials: Di-n-hexylphthalate (DnHP), Di-n-octyl phthalate (DNOP), Dibutyl Phthalate (DBP), Diisodecyl Phthalate (DIDP), Butyl Benzyl Phthalate (BBP), Diisononyl Phthalate (DINP), Di(2-ethylhexyl)phthalate (DEHP), Dicyclohexyl phthalate

Authoritative Lists: CHE (phthalates; S&I -limited) and Quebec CSST (DEHP only)

Exposure Pathways for Building Occupants: These semi-volatile organic compounds may be released throughout the service life of building products such as such vinyl flooring, vinyl carpet backing, lacquers, flooring finishes, adhesives, and fluid applied floors. These emitted compounds become attached to household dust to which people are readily exposed.

Phthalates are some of the most widely used and abundant semi-volatile organic chemicals (SVOCs) in indoor environments. The vast majority comes from polyvinyl chloride (PVC) products. Inherently rigid, PVC requires softening plasticizer additives – typically phthalates – to make it flexible enough for use in products like wall covering, flooring and upholstery. Because they are not permanently bound to the vinyl, phthalates are "slowly emitted from the products to air or other media."[78]

Multiple studies have associated PVC products with increased levels of phthalate exposure, with DEHP being the most common.[79]

There is considerable evidence that the endocrine-disruptive characteristics of phthalates may include the abnormal development of lung tissues.

In animal studies, early exposure to DEHP has been shown to bind to the PPAR hormone receptor in the body, and cause lung tissue to form abnormally, with larger air spaces.[80] In some mice subjects, DEHP exposure resulted in severe changes similar to those seen in children and animal models with bronchopulmonary dysplasia.[81] This is a respiratory condition that makes people more likely to develop asthma in the future.[82] Human lungs have the same PPAR gene receptor, which researchers speculate[83] may account for higher rates of asthma in children exposed to DEHP through household dust.[84]

Prenatal exposures to phthalates are also possible. One study suggests that "fetuses may be exposed to phthalates and their monoesters in the amniotic fluid of their mothers."[85] Just et al., 2012 found that prenatal exposure to BBP may have an influence on the risk of developing early childhood eczema, which further supports the potential for this pathway.

4 How to Avoid Asthmagens through Materials Selection

The building industry utilizes product IAQ certification systems to evaluate and select building products that have low emissions of hazardous chemicals[m]. These certification programs function by measuring emissions from products and determining if the resulting concentrations in a building will be below the concentration levels at which specific human health hazards have not been observed.

In general, these programs primarily assess volatile organic compounds (VOCs)[n], that "off-gas" from materials and can be measured in a laboratory chamber test with relative ease. Our research has revealed, however, some VOCs that cause asthma require different, more targeted testing protocols than those used to screen for other hazards such as cardiovascular, nervous or reproductive system conditions, and there are no consensus IAQ standards for assessing these types of chemicals for building material use at this time. Further, building products contain a number of chemicals that are released into the indoor air and can cause asthma, but which are not VOCs, and therefore not covered by VOC-based IAQ testing standards.

Two concentration lists are primarily used for this determination in the United States. Threshold Limit Values (TLVs) are established by the American Conference of Governmental Industrial Hygienists (ACGIH) to provide guidelines for exposures in the workplace. Chronic Reference Exposure Levels (CRELs)[o], established by the California Office of Environmental Health Hazard Assessment are more conservative thresholds that address exposures to a wider range of populations, including infants and children. The TLV and CREL concentration levels are determined based on a large set of human health criteria, but frequently do not include asthma onset[p].

[m] Such as UL GreenGuard, RFCI FloorScore, CRI GreenLabel and SCS Indoor Advantage and other programs based upon the California Standard Practice (01350)

[n] CA 01350 defines VOCs as "carbon-containing compounds (excluding carbon monoxide, carbon dioxide, carbonic acid, metallic carbides and carbonates and ammonium carbonate) with vapor pressures at standard conditions approximately ranging between those for n-pentane through n-heptadecane" (Standard Method For The Testing And Evaluation Of Volatile Organic Chemical Emissions From Indoor Sources Using Environmental Chambers Version 1.1," California Department of Public Health, Updated February 2010, http://www.cal-iaq.org/phocadownload/cdph-iaq_standardmethod_v1_1_2010%20new1110.pdf (CPDH 2010)). This generally includes carbon compounds with 5-17 carbon atoms (CPDH 2010). GreenGuard defines VOCs similarly in their protocol, referring to a range defined by the characteristics of n-hexane and n-hexadecane, including boiling points between 60-290°C ("GREENGUARD Certification Program Method for Measuring and Evaluating Chemical Emissions From Building Materials, Finishes and Furnishings." Underwriter Labs, March 29, 2013. https://www.comm-2000.com/productdetails.aspx?sendingPageType=BigBrowser&CatalogID=Standards&ProductID=ULE2821_1_G_20130402 (ULEnvironment)).

[o] Up-to-date list of CRELs are available at: http://www.oehha.ca.gov/air/allrels.html.

[p] As seen in the CREL summaries on individual chemicals, data rarely exists to determine thresholds for asthma onset de novo. However, research on asthmatics has informed thresholds to help prevent asthma exacerbation. Therefore, many CRELs have addressed respiratory issues, including asthma exacerbation, but have not been able to address thresholds for asthma onset. ("Appendix D. Individual Acute, 8-Hour, and Chronic Reference Exposure Level Summaries," California Department of Public Health (CPDH), Updated August 2013, http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD1_final.pdf#page=5 (D1), http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD2_final.pdf#page=18 (D2), and http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=453 (D3).

Our analysis indicates that over 70% of the asthmagens that we have identified in building materials are not presently covered by the leading IAQ testing standards. Of the 50 chemicals we identified in building materials listed below in Tables 3-5, 36 (72%) are not covered by either CA 01350 or GreenGuard testing protocols. CA 01350 covers 3 (6%) asthmagens from our list, while GreenGuard covers an additional 11 (28%), including 4 of the 8 phthalates associated with building products in the Pharos BPL. (Appendix B tables 3 – 5)

For those asthmagens that are covered by leading IAQ testing protocols, the testing thresholds are not designed to be protective for asthma onset even though they are protective against many important health problems, including cancer, reproductive and developmental health, and respiratory problems such as asthma exacerbation.

Additional evaluation of products based upon not only emissions, but also chemical content is required to avoid chemicals in building products that can cause the onset of asthma. This can be accomplished with building materials by obtaining information on the contents of building materials under consideration for a project and selecting those that do not include asthmagens. The Pharos Project's Building Product Library (BPL) is uniquely suited to support this kind of material selection process.

Coincident with the release of this report, HBN has expanded its coverage of asthmagens in Pharos. We've added a restricted substance list – the HBN Priority Building Material Asthmagen List – to the Pharos Chemical & Material Library that identifies the 28 top-priority asthmagens identified in this report. We've also added a filter to the Pharos Building Product Library that allows Pharos subscribers to identify and select products that do not contain these top-priority chemicals: the 20 asthmagens and the eight phthalates (See sections 2 and 3). The filter screens products for both the intentional ingredients and for chemicals that are likely to appear as contaminants in the product as residuals from the manufacturing process, including monomers, nonreactive additives and other contaminants from feed stocks. Products must be fully disclosed in order to ensure a complete screening and clear the filter. This filter will support selection of products with increased confidence of avoiding the top priority chemicals associated with asthma in building materials.

Conclusion

Asthma is a complex disease, for which researchers are still working to determine onset mechanisms. However, the presence of asthmagens in building materials and exposures to building occupants are known and steps can be taken now to prevent exposures even without this information.

1. Building owners, architects, and designers should screen building product contents for asthmagens. Our report identified 28 top-priority asthmagens. Using the HBN Priority Building Material Asthmagen List users can filter products in the Pharos BPL to identify those products without these chemicals.
2. More research is needed to fully understand asthma onset mechanisms and potential contributions of asthmagens in building materials to the increasing incidence of asthma.
3. IAQ testing protocols and rating systems need to develop new protocols that take asthma onset into account. Our research and anaysis showed that current IAQ protocols are not designed to prevent asthma onset.

Appendix A:
Other Common Asthmagens in Building Materials

The AOEC considers all substances on their asthmagens list to be disease-causative. Its protocol notes that "the AOEC asthmagen criteria do not reflect a specific exposure scenario, which will alter the risk of asthma from a particular substance (e.g. encapsulated or airborne form, enclosed or open process, low or high concentration)."

The presence of asthmagens in materials inherently represents potential exposures to building occupants. We examined the underlying cases and studies behind the asthmagens in this appendix that are used in building materials. More research is needed to determine whether, in normal conditions of use in building materials, exposure pathways for these substances are as significant as those we identify above as top priorities for asthma disease prevention.

Table 2. Other substances of concern

Chemical group	Lists	Uses in building materials*
Acetic acid, glacial	AOEC (Rrs) and Quebec CSST	Silicone caulking and HPCs
Ammonia	AOEC (Rr), CHE (I- strong)	Carpet backing, fiberglass insulation, and latex
Biocides (in particular, Didecyl dimethyl ammonium chloride (DDAC); tetrachloroisophthalonitrile; 1,2-Benzisothiazolin-3-one (BIT); Hexamethylenetetramine; Triclosan**)	AOEC (Rs; DDAC only), Quebec CSST (Tetrachloroisophthalonitrile, BIT, and hexamethylenetetramine only), CHE (I- limited; tetrachloroisophthalonitrile only)	Treated wood floors and carpets, wet applied products, such as paints and natural oil flooring stains
Bisphenol A (BPA)	None**	Epoxies (HPCs, FAFs, and adhesives)
Gum Rosin (Colophony)	AOEC (colophony, G), Quebec CSST, CHE (S- strong)	Linoleum and adhesives
Metal Dusts (Al, AlOx, Ch, Co, Ni, V, ZOx)	AOEC (Rs), Quebec CSST (Al, Ch, Co & Ni), CHE (S- strong; except AlOx & ZOx)	Steel and aluminum alloys, carpet backing fillers, and flooring finishes
Perfluorocarbons (PFCs, in particular PFBS and PFHxA)	None**	Stain repellants in carpets and textiles
Polyvinyl Chloride	AOEC (Rs), Quebec CSST, CHE (S- strong)	Resilient flooring, wall protection, ceilings, carpet backing and roofing membranes
Tall Oil Rosin	AOEC (Rs), Quebec CSST	Linoleum and adhesives
Toluene	CHE (I- limited)	Adhesives and lacquers
Western Red Cedar, other wood dust and bark	AOEC, Quebec CSST, CHE (Wood dust; S- Strong)	Composite wood products and laminates

*As identified by cross-referencing asthmagen lists with over 1,300 products in the Pharos BPL.

** These chemicals are present in building products in Pharos, but are not present on asthmagen lists. However, these chemicals were added to this analysis due to emerging evidence of links to asthma found in scientific literature.

A. Acetic Acid, Glacial

Authoritative Lists: AOEC (Rrs) and Quebec CSST

Exposure Pathways for Building Occupants: Inhalation of emissions from silicone caulking.

Glacial acetic acid is commonly used as a raw material in the manufacture of solvents and plastics such as cellulose acetate, rayon, polyethylene terephthalate (PET), and latex; for dying, printing and cleaning solutions[86]; and, this chemical is used in building materials such as high performance coatings and caulking.

OSHA says acetic acid is a "major indoor air pollutant," and identifies silicone caulking compounds as a primary source.[87]

This chemical is irritating and corrosive to the skin, eyes, and upper respiratory tract. Exposures to high concentrations of vapors may result in chest pain, difficulty breathing, coughing, bronchopneumonia, and, following single high concentration exposures, RADS.[88]

B. Ammonia

Authoritative Lists: AOEC (Rr), CHE (I- strong)

Uses in Building Materials: Carpet backing and fiberglass insulation

Ammonia is well known as a respiratory irritant in humans. Ammonia is a volatile chemical, with a boiling point of -33°C, Chronic low-level exposures have little effect on pulmonary function, while high (500ppm+) single and chronic exposures are known to cause more serious respiratory effects, including chemical burns. Reported respiratory effects include: nasal irritation; epiglottal, laryngeal, pharyngeal, tracheal, and pulmonary edema; dyspnea; wheezing; coughing; rhonchi; pneumonia; and cardio-respiratory arrest. However, these exposures are unlikely to occur in normal residential or workplace situations.[89]

The potential for ammonia to cause respiratory irritation potential of ammonia may be due to its volatile nature and its breakdown in water, such as on skin and mucous membranes, to ammonium hydroxide (see description in Top Priorities section of this report).

C. Biocides

Chemicals Identified in Building Materials: Didecyl dimethyl ammonium chloride (DDAC), tetrachloroisophthalonitrile, 1,2-Benzisothiazolin-3-one (BIT), Hexamethylenetetramine, and Triclosan

Authoritative Lists: AOEC (Rs; DDAC only), Quebec CSST (Tetrachloroisophthalonitrile, BIT, and hexamethylenetetramine only), CHE (I- limited; tetrachloroisophthalonitrile only)

Uses in Building Materials: Treated wood floors and carpets, wet applied products, such as paints and natural oil flooring stains

The functional class of chemicals called biocides is designed to kill living organisms, like microorganisms and pests. It should not be surprising, then, that biocides used in interior building materials can be hazardous to humans.

Certain biocides that treat interior wood products, preserve paints, and kill microbes in carpets cause asthma.

Lumber yards often treat their wood products with anti-sapstain mixtures. While preventing visible blemishes, anti-sapstain treatments drastically change the toxicological profile of otherwise benign products like sustainably harvested lumber. The AOEC lists DDAC as an asthmagen. Tetrachloroisophthalonitrile is not yet on that list, but in one documented case, a farmer had episodes of dyspnea, shortness of breath and wheezing after exposure to mist tetrachloroisophthalonitrile. A bronchial provocation test was positive, and animal tests showed a "dose-response" statistically significant correlation.[90]

Very limited literature is available on indoor exposures to preservatives used on interior wood products. However, a recent German Federal Environment Agency study warns that these surface treatments "may result in significant emissions" and that interior air is the "primary receiving environmental compartment for treated floors."[91]

1,2-Benzisothiazolin-3-one (BIT) is commonly used to preserve wet applied products, especially paint. These are also used in some flooring stains.

Occupational lung doctors from Newcastle General Hospital (U.K.) described the occurrence of asthma in an isothiazolinone manufacturing plant and concluded that it was "likely that this was the result of sensitization to isothiazolinone."[92] This finding corresponded with a contemporaneous but independent report of occupational asthma and rhinitis caused by inhalation of BIT.[93]

Also of potential concern: an impurity in paint preservatives called hexamethylene tetramine. A 1963 study reported respiratory sensitization ("wheezing, chest tightness, asthma") to hexamethylene tetramine in seven paint industry workers."[94]

The connections identified between these biocides and asthma may represent only the leading edge for this class of substances. There is also emerging concern about the widely-used biocide, triclosan.

Triclosan, a halogenated phenolic compound known as triclosan, has come into focus as a known endocrine disruptor, for example, in the groundbreaking World Health Organization/United Nations report on EDCs released earlier this year.[95]

Doctors from the Johns Hopkins Medicine Division of Allergy and Clinical Immunology evaluated data from the 2005-2006 National Health and Nutrition Examination Survey and concluded that levels of triclosan "were

significantly associated with allergic sensitization. The potential role of antimicrobial EDCs in allergic disease warrants further study as they are commonly used in Western society."[96]

Further study and action are urgent. There is a growing burden of triclosan in human bodies. According to the CDC, in 2009-10, the mean level of triclosan in the U.S. population's urine was 14.5 µg/L, up from 13 µg/L in 2003-04.[97]

D. Bisphenol A (BPA)

Authoritative Lists: None

Uses in Building Materials: High performance epoxy coatings, FAFs, and adhesives

BPA is a potent developmental toxicant. Emerging evidence has begun to associate BPA with asthma in children. Thus far, two studies have found significant associations between asthma symptoms such as wheezing in children and levels of BPA urine metabolites in pregnant mothers (prenatal exposures) and children (postnatal exposure).[98]

Spanier et al 201297 found that prenatal exposure to BPA was associated with increased odds of wheezing at 6 months of age. However, these effects did not persist over time and the window of vulnerability appeared to be only during the first 16 weeks of pregnancy, which does not coincide with fetal lung development.[99]

Donohue et al 2012 also found associations in BPA exposure and wheezing and physician diagnosed asthma. While prenatal exposures did not appear to contribute to asthma symptoms in this study, postnatal exposures did.[100]

Some animal studies have had opposite results. For example, mice exposed to BPA in utero (prenatally) displayed symptoms of allergic asthma. However, those exposed postnatally via breast milk did not present with asthma.

Epoxy adhesives containing BPA-derived compounds, however, are known asthmagens, and are discussed in the Top Priority section of this report.

While the mechanism for the onset of asthma due to BPA exposure is still unknown, associations between BPA and asthma symptoms in children are emerging and occupational exposures to BPA containing compounds present support for an immune system mechanism. Further research is needed to clarify the mechanism of asthma onset and why symptoms from early-life exposures do not appear to continue into adulthood. Additionally, further research is needed to determine the role of building materials in BPA exposure, especially for pregnant women and children.

E. Gum Rosin (Colophony)

Authoritative Lists: AOEC (colophony; G), Quebec CSST, CHE (S- strong)

Uses in Building Materials: Linoleum flooring; Adhesives

Gum rosin, or colophony, is a natural product obtained from coniferous trees. It may be used in a range of products including linoleum flooring, adhesives, ink, paints and soldering fluxes.

The potential of colophony to cause asthma has been reported since the 1970s, especially among electronics workers who are exposed to colophony via heated soldering fluxes.[101] The majority of cases of occupational asthma from colophony have been from heated colophony at high temperatures (300-450°C). Therefore, it is unclear if colophony or its degradation products are the cause of asthma.[102]

One occupational case study exists for exposure to unheated colophony particles. Monitoring of the patient's lung function (peak expiratory flow) at work and in testing showed marked drops in lung function and elicited an asthmatic reaction that lasted for three days and required treatment. Additionally, some isolated reports exist for occupational asthma due to lower temperature exposures to colophony at 180 and 90°C.[103]

Mechanisms for the onset of asthma from colophony are uncertain. However, emerging evidence support the idea that colophony is a respiratory sensitizer that causes allergic asthma. Elms et al 2005[104] found that "serum from exposed symptomatic individuals showed increased binding of specific IgE antibodies to a range of colophony-cell protein conjugates." Additionally, several cases of skin sensitization have been reported in occupationally exposed workers. Skin tests (patch, prick) of these workers with rosin elicited positive allergic skin reactions. These studies suggest an immune system reaction that is characteristic of allergic asthma.

F. Metal Dusts

Metals: Aluminum; Aluminum Oxide; Chromium; Cobalt; Nickel; Vanadium; Zinc Oxide.

Authoritative Lists: AOEC (Rs), Quebec CSST (Aluminum, Chromium, Cobalt & Nickel), CHE (S- strong; except Aluminum Oxide & Zinc Oxide)

Uses in Building Materials: Steel; Aluminum Alloys; Filler for carpet backing; Flooring finishes (Aluminum Oxide, Epoxies).

Occupational exposures to metal dusts, especially via welding, have been linked to respiratory symptoms in a multitude of cases. Inhalation of metal dusts and fumes can induce a wide range of lung pathology, including airways disorders, cancer, and parenchymal diseases.

Metals may be found in a variety of indoor finishes including ceilings, wall protection, and as hardening agents in flooring finishes. However, while significant occupational evidence exists for exposure to metal dusts, exposures levels and pathways to building occupants via metal building materials are largely unknown. Taber Abrasion Tests, which measure the service life of a product by weight loss over time, could help indicate the potential for exposures from metal surfaces as well as flooring finishes containing aluminum oxide. Further research is needed to determine the potential levels of exposure to metals from building materials and if those levels can cause the onset of asthma.

G. Perfluorocarbons (PFCs)

Chemicals Used in Building Materials: Perfluorobutane sulfonate (PFBS) and perfluorohexanoic acid (PFHxA)

Authoritative lists: None.

Exposure Pathways for Building Occupants: Contact with carpets and textiles with these stain treatments.

PFCs are produced for use as surfactants in many applications, including carpets and textiles, to enhance water, grease, and soil repellency. These fluorosurfactants are also used in paints, adhesives, inks and other coatings.[105]

Measurable levels of these synthetic compounds have also been found in people's liver and blood.[106]

Most industry, regulatory and consumer attention on PFCs has focused on longer-chain compounds – especially the eight-carbon compounds, perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS) – because bioaccumulation potential increases with chain length. The US carpet industry has replaced these eight-carbon PFCs with shorter chain compounds, such as perfluorobutane sulfonate (PFBS) and perfluorohexanoic acid (PFHxA). Biomonitoring data finds these and other perfluoroalkyl compounds in human blood serum.[107]

Major pathways that enable PFCs to get into human blood are not fully understood, but, according to the US CDC, "one study has proposed that the major exposure pathways for PFOS for the general population in Europe and North America were food and water ingestion, dust ingestion, and hand-to-mouth transfer from mill-treated carpets." Exposures to children "from hand-to-mouth transfer from treated carpets is expected to be much larger," according to the CDC.[108]

A recent epidemiological study correlated PFC serum concentrations with asthma. Dong et al (2013) analyzed serum samples for ten PFCs in 456 Taiwanese children (ages 10-15), with and without asthma. The National Science Council in Taiwan-supported study found that children with asthma had significantly higher concentrations of PFBS and PFHxA (among other PFCs) in their blood. These and most other PFCs were also "positively associated with serum IgE concentrations, absolute eosinophil counts (AEC), eosinophilic cationic protein (ECP) concentrations, and asthma severity scores among asthmatics."[109]

Dong et al concluded that the conclusion "suggests an association between PFC exposure and juvenile asthma. Because of the widespread exposure to these chemicals, these findings may be of potential public health concern."

Further, as Dong et al notes, prenatal pathways are possible with PFCs. Animal studies have linked prenatal exposures to PFOS to changes in lung function and development that appear to be regulated via a PPAR-mediated mechanism.[110]

H. Polyvinyl Chloride (PVC)

Authoritative Lists: AOEC (Rs), Quebec CSST, CHE (S- strong)

Uses in Building Materials: PVC is used in a wide variety of building specifications including resilient flooring, wall protection, ceilings, and carpet backings.

Occupational exposures to PVC have been associated with cases of occupational asthma; however, the onset of asthma in these cases may have been caused by certain additives, not the resins as a whole.[111]

PVC has long been associated with asthma in occupational settings. "Meat-packers asthma," as it was called, resulted from exposure to PVC films heated to 180-300°C. However, in these cases it was unclear whether additives (especially plasticizers and stabilizers) or breakdown products (especially hydrogen chloride) were responsible.[112]

Experimental research by Tuomainen et al. 2006, found that subjects experienced significant respiratory symptoms when they were exposed to degrading PVC flooring material. However, by using finished PVC, the researchers were unable to conclude if the PVC resin itself was the cause of these symptoms. Their experiment found 2-ethylhexanol in the test chamber, and in the breaths of all subjects. According to Wieslander et al. 1999, 2-ethylhexanol is a degradation product of the common plasticizer di-2-ethylhexyl phthalate (DEHP). This suggests plasticizer exposure occurred through exposure to this flooring material.

Studies into residential exposures of PVC products and incidence of asthma have found associations between risk and diagnosis of asthma in young children.[113] However, levels of PVC products in homes have also been associated with increased levels of phthalates, the most common being DEHP.[114] Therefore, it is unclear whether the PVC resin or its additives are responsible for these associations.

While building occupant exposures to PVC are possible through natural wear and maintenance of PVC products such as resilient flooring, there is little research available on the levels of plastic dusts found in indoor spaces.

Taber abrasion tests can help us understand the volume lost through commercial wear, however these are rarely reported for plastic products. One study done by the USGBC PVC task group collected Taber abrasion testing

data on various flooring materials[115] including multiple vinyl products. They found that vinyl composition tile (VCT) loses most volume per year at 7.69 mm^3, in comparison to heterogeneous sheet vinyl (3.80 mm^3 loss per year) and homogeneous sheet vinyl (2.66 mm^3 loss per year). Aside from normal wear, particles may be generated through flooring maintenance. For example, VCT is typically stripped and re-finished annually in commercial spaces.[116]

I. Tall Oil Rosin

Authoritative Lists: AOEC (Rs), Quebec CSST

Uses in Building Materials: Linoleum flooring; Adhesives

Tall oil rosin, like colophony, is a natural substance used in linoleum and adhesives. Cases of occupational exposure to tall oil are found in the cleaning and tall oil production industries.

Respiratory symptoms occurred in response to cleaning products containing tall oil, as evidenced by decreases in forced expiratory flow (FEV) of up to 60% in response to cleaning products and 17% for tall oil alone. Therefore, it is unclear if other chemicals may be causing the onset of asthma in these workers.

The mechanism for the onset of asthma due to tall oil is unknown. Pathways of exposure to tall oil for building occupants are unknown. However, due to its use in a variety of consumer products such as, paper products, soaps, polishes, and lubricants, there is a high potential for exposures to consumers and children.[117]

J. Toluene

Authoritative Lists: CHE (I- limited)

Uses in Building Materials: Adhesives and lacquers

Toluene is a volatile organic compound (VOC) and a known respiratory irritant. Case studies of varied toluene exposures in animals and humans have resulted in limited and conflicting results. Some studies found limited effects on respiratory passages at small (40-100 parts per million [ppm]) and high (>800+ ppm) exposures. Others observed irritation of the nose and throat along with some non-respiratory symptoms at concentrations of 1-200ppm.

"Occupational asthma has occurred in some workers exposed to toluene levels considered safe in the workplace" and may have occurred to genetic factors that may make some people more sensitive to the effects of inhaled solvents.[118]

Animal studies have shown morphological changes in nasal and tracheal epithelial cells at low exposures to toluene. Lung irritation, inflammation, pulmonary distress and lesions were only observed at high exposures (1000+ ppm).[119]

In children, toluene has been associated with increased risk factors for the diagnosis of asthma. In research on individual VOCs, Rumchev et al 2004 found the highest odds ratios for asthma due to benzene, ethylbenzene and toluene. "For every 10 unit increase in the concentration of toluene and benzene (mg/m^3) the risk of having asthma increased by almost two and three times, respectively."[120] Additionally, toluene is associated with increased IgE sensitization to total IgE or foods, Mendell notes. This could indicate an immune system response as the mechanism responsible for asthma onset.[121]

Further research is needed to confirm respiratory symptoms due to specific acute and chronic exposures to toluene.

K. Western Red Cedar dust or bark, other wood dust

Authoritative Lists: AOEC, Quebec CSST, CHE (Wood dust; S- Strong)

Uses in Building Materials: Plywood, MDF, engineered wood flooring, laminate.

It has long been understood that Western Red Cedar wood dusts cause asthma. A 1981 epidemiologic study found a "high prevalence of occupational asthma was observed among workers exposed to WRC wood dust. A dose-response relationship between total WRC dust level and prevalence of asthma was noted with employees in jobs with the greatest dust exposure, i.e. sawyers, packers, chippers, and splitters showing the highest prevalence of disease."[122]

A 1986 study of western red cedar workers confirmed a "causal relationship" between asthma and plicatic acid (a low molecular weight compound present in the heartwood).[123]

In British Columbia, plicatic acid has been identified as the province's leading cause of occupational asthma. It reportedly affects five percent of exposed workers.[124]

For other wood species, causal relationships are less certain. The California Department of Public Health notes that, "Most types of wood dust can irritate your lungs and cause other breathing problems. Sometimes wood dust can cause asthma or make it worse. Some woods contain chemicals that make allergic reactions like asthma more likely. A few common examples are California redwood, teak, Western red cedar, oak, and ash...The hazard depends on the amount of wood dust that gets in the air, the size of the dust particles, the type of wood, the levels of asthma-causing substances in the wood, the additives in the wood, how long you are exposed to the dust, and your own body's resistance."[125]

Appendix B:
Asthmagens and IAQ Testing Protocols

Following our examination of listed asthmagen chemicals in the Pharos BPL, we examined the IAQ testing protocols of the two leading certification systems – GreenGuard Gold and California 01350 – and determined that most asthmagens in building materials are not covered by either.

Out of the 50 chemicals listed below in Tables 3-5, which includes 12 chemicals identified via scientific literature review with emerging links to asthma, 36 (72%) chemicals are not covered by either CA 01350 or GreenGuard testing protocols. Only 3 (6%) are covered by both CA 01350 and GreenGuard Gold testing protocols and 11 additional chemicals (28%), including 4 phthalates associated with building products in the Pharos BPL, by GreenGuard Gold.

Most asthmagens fall outside these tests because agencies have not established TLVs or Chronic REL thresholds for these chemicals, or because these substances have low vapor pressures, medium/high boiling points, are inorganic, or have carbon content that falls outside the ranges specified in the IAQ VOC testing protocols.

The following tables describe the chemical formulations, volatilities, boiling points, and threshold values for the chemicals discussed in this report. These values relate to the protocols used by the two leading protocols. A chemical's formula is relevant for GreenGuard's Total Volatile Organic Compound (TVOC) measurement, which includes only those chemicals whose carbon content falls within the C6 – C16 range. Vapor pressure is a measure of volatility. Boiling point is another indicator, but only in part, that a chemical is volatile.

Of the 50 chemicals that we discuss in this report, only three are included in both protocols of the two leading IAQ protocols.

Table 3. Asthmagens Covered in Both GreenGuard Gold and 01350 Protocols[126]

Substance	CAS No.	Formula	Vapor pressure (mm Hg @ 25°C)[a]	Boiling point (°C)[b]	1/100 TLV (µg/m³)[c]	½ Chronic REL (µg/m³)[d]	Authoritative lists that include these chemicals[e]	Content in building materials[f]
Formaldehyde	50-00-0	CH_2O	3,890	-19	3.7	Full REL: 9	AOEC (G), Quebec CSST, CHE (S- good)	Traces found in laminate, insulation wallboard, composite wood, and adhesives
Styrene	100-42-5	C_8H_8	6.40	145	850	450	AOEC (Rs), Quebec CSST, CHE (S- limited)	Up to 50% in binder; also in insulation and coatings
Toluene	108-88-3	C_7H_8	28.4	111	1880	150	CHE (I- limited)	Up to 15% in adhesives and lacquers

a. Data from the Hazardous Substances Data Bank (HSBD), available at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
b. Data from Pharos Chemical and Materials Library (CML)
c. TLVs established by the American Conference of Governmental Industrial Hygienists (ACGIH). TLV data from GreenGuard Certification protocol, available for download at: https://www.comm-2000.com/productdetails.aspx?sendingPageType=BigBrowser&CatalogID=Standards&ProductID=ULE2821_1_G_20130402(ULEnvironment)
d. CRELs established by California Department of Public Health (CPDH). List of full CRELs available here: http://www.oehha.ca.gov/air/allrels.html. List of CRELs used in CA 01350 emissions testing protocol available here: http://www.cal-iaq.org/phocadownload/cdph-iaq_standardmethod_v1_1_2010%20new1110.pdf
e. Up-to-date asthmagen lists can be found here: http://www.aoecdata.org/ExpCodeLookup.aspx (AOEC), http://www.asthme.csst.qc.ca/document/Info_Gen/AgenProf/Bernstein/BernsteinAng.htm#N2_12 (CSST), and http://www.healthandenvironment.org/tddb (CHE)
f. As identified by cross-referencing asthmagen lists with over 1,250 products in the Pharos BPL.

Table 4. Asthmagens Covered by GreenGuard Gold Protocol Only

Substance	CAS No.	Formula	Vapor pressure (mm Hg @ 25°C)[a]	Boiling point (°C)[b]	1/100 TLV (µg/m³)[c]	½ Chronic REL (µg/m³)[d]	Authoritative lists that include these chemicals[e]	Content in building materials[f]
Acetic Acid, Glacial	64-19-7	$C_2H_4O_2$	15.7	118	250	none	AOEC (Rrs), Quebec CSST	Up to 5% in HPCs; also found in silicone caulking
Acrylic Acid	79-10-7	$C_3H_4O_2$	3.97	141	59	3	AOEC (Rs)	Up to 50% in paint; also found in composite wood binders
2- Aminoethanol	141-43-5	C_2H_7NO	0.404	171	75	none	AOEC (Rs), Quebec CSST, CHE (ethanol-amines; S- strong)	Up to 5% in adhesives
Butyl Benzyl Phthalate (BBP)	85-68-7	$C_{19}H_{20}O_4$	0.0000082	250	none	none	CHE (phthalates; S&I-limited)	Up to 4% in VCT, carpet backing, and adhesive
Dibutyl Phthal-ate (DBP)	84-74-2	$C_{16}H_{22}O_4$	0.0000201	340	none	none	CHE (I- limited)	up to 5% in flooring finishes, and lacquers
Di(2-ethyl-hexyl) Phthalate (DEHP)	117-81-7	$C_{24}H_{38}O_4$	0.000000142	384	none	none	CHE (phthalates; S&I -limited)	Up to 50% in roofing membrane; 16% in vinyl carpet backing; 4% in vinyl tile
Di-n-Octyl Phthalate (DNOP)	117-84-0	$C_{24}H_{38}O_4$	0.0000001	242	none	none	CHE (phthalates; S&I -limited)	Up to 10% in roofing membrane
Maleic Anhydride	108-31-6	$C_2H_2(CO)_2O$	0.25	202	4	0.035	AOEC (Rs), Quebec CSST, CHE (acid an-hydrides; S- strong)	Up to 50% in HPCs, paints, and adhesives; up to 1.5% in carpet backing
Methyl Methacrylate	80-62-6	$C_5H_8O_2$	38.5	100	2050	none	AOEC (Rs), Que-bec CSST (methyl methacrylate and cyanoacrylates), CHE (methacrylates; S- strong)	Up to 100% in solid surfaces, 70% in fluid-applied floors, 20% in insulation binder
Phthalic Anhydride	85-44-9	$C_6H_4(CO)_2O$	0.000517	295	61	10	AOEC (Rs), Quebec CSST, CHE (acid an-hydrides; S- strong)	Up to 50% in HPCs, paints, and adhesives; up to 1% in rubber flooring
Triethanolamine	102-71-6	$C_6H_{15}NO_3$	0.00000359	335	50	none	AOEC (Rs), Quebec CSST, CHE (ethanol-amines; S- strong)	Up to 17% in binder; also in HPCs

a. Data from the Hazardous Substances Data Bank (HSBD), available at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
b. Data from Pharos Chemical and Materials Library (CML)
c. TLVs established by the American Conference of Governmental Industrial Hygienists (ACGIH). TLV data from GreenGuard Certification protocol, available for download at: https://www.comm-2000.com/productdetails.aspx?sendingPageType=BigBrowser&CatalogID=Standards&ProductID=ULE2821_1_G_20130402(ULEnvironment)
d. CRELs established by California Department of Public Health (CPDH). List of full CRELs available here: http://www.oehha.ca.gov/air/allrels.html. List of CRELs used in CA 01350 emissions testing protocol available here: http://www.cal-iaq.org/phocadownload/cdph-iaq_standardmethod_v1_1_2010%20new1110.pdf
e. Up-to-date asthmagen lists can be found here: http://www.aoecdata.org/ExpCodeLookup.aspx (AOEC), http://www.asthme.csst.qc.ca/document/Info_Gen/AgenProf/Bernstein/BernsteinAng.htm#N2_12 (CSST), and http://www.healthandenvironment.org/tddb (CHE)
f. As identified by cross-referencing asthmagen lists with over 1,250 products in the Pharos BPL.

Table 5. Substances Not Covered by Either IAQ Protocol (*)

Substance	CAS No.	Formula	Vapor pressure (mm Hg @ 25°C)[a]	Boiling point (°C)[b]	½ Chronic REL (µg/m³)[c]	Authoritative lists that include these chemicals[d]	Content in building materials[e]
Aluminum	7429-90-5	Al	1 mm Hg @ 1284°C	2337	none	AOEC (Rs), Quebec CSST, CHE (S-strong)	Up to 100% in ceiling tiles, corner guards, and metal laminates, 15% in insulation
Aluminum Oxide	1344-28-1	Al_2O_3	1 mm Hg @ 2158°C	2997	none	AOEC (Rs)	Up to 20% in UV-cured finishes; 5% or less in paint and laminates
Ammonia	7664-41-7	NH_3	7510	-33	100	AOEC (Rr), CHE (I- strong)	Traces found in vinyl and latex carpet backing; insulation
Ammonium hydroxide	1336-21-6	H_5NO	2160	100	none	AOEC (Rs)	0.2% in chalkboard paint and primer
Benzisothiazolin-3-one (BIT)	2634-33-5	C_7H_5NOS	Not determined	154 (Melting point)	none	Quebec CSST	Up to 1% as a preservative in paints, stains, and sealants
Bisphenol A	80-05-7	$C_{15}H_{16}O_2$	4.0×10^{-8}	361	none	None**	Up to 30% in epoxy adhesives and sealants
Bisphenol A Diglycidyl Ether (BADGE)	1675-54-3/ 25085-99-8	$C_{21}H_{24}O_2$	1.1×10^{-7}	8 (Melting point)	none	AOEC (epoxies; G), Quebec CSST, CHE (epoxy resins; S- strong)	Up to 50% in epoxy fluid applied floors, up to 40% in epoxy adhesives and sealants
Chromium, elemental	7440-47-3	Cr	1 mm Hg @ 1616°C	2642	none	AOEC (Rs), Quebec CSST, CHE (S- strong)	Up to 1.2% in steel alloys; traces in fly ash and aluminum products
Cobalt	7440-48-4	Co	1 Pa @ 1517°C	2927	none	AOEC (Rs), Quebec CSST, CHE (S- strong)	Traces in fly ash
Didecyl dimethyl ammonium chloride (DDAC)	7173-51-5	$C_{22}H_{48}ClN$	2.3x10-11	NA	none	AOEC (Rs)	Up to 75% in anti-sapstain wood treatment
Diisodecyl Phthalate (DIDP)	26761-40-0	$C_{28}H_{46}O_4$	0.000000528	250	none	CHE (phthalates; S&I-limited)	Up to 4% in VCT; also in adhesives
Diisononyl Phthalate (DINP)	28553-12-0/ 68515-48-0	$C_{26}H_{42}O_4$	0.000054	252	none	CHE (phthalates; S&I-limited)	Up to 13% in carpet backing; also in VCT
2- dimethylamino-ethanol	108-01-0	$C_4H_{11}NO$	21 mm Hg @ 20°C	134	none	AOEC (Rs), Quebec CSST, CHE (ethanolamines; S- strong)	5% in Part B of SPF insulation; Also found in coatings
Di-n- hexylphthalate (DnHP)	84-75-3	$C_{20}H_{30}O_4$	0.000014	185	none	CHE (phthalates; S&I-limited)	4% in VCT
Diphenylmethane-2,4'- diisocyanate (2,4'-MDI)	5873-54-1	$C_{15}H_{10}N_2O_2$	typically for pMDI <0.0001[f]	376	none	CHE (isocyanates; S- strong)	Up to 16% in carpet backing; also in SPF insulation

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Substance	CAS No.	Formula	Vapor pressure (mm Hg @ 25°C)[a]	Boiling point (°C)[b]	½ Chronic REL (µg/m³)[c]	Authoritative lists that include these chemicals[d]	Content in building materials[e]
Gum Rosin	8050-09-7	$C_{20}H_{30}O_2$	negligible	280	none	AOEC (colophony, G), Quebec CSST, CHE (S- strong)	Up to 12% linoleum flooring; also in adhesives
1,6- Hexamethylene Diisocyanate (HDI)	822-06-0	$C_8H_{12}N_2O_2$	0.05	213	none	AOEC (G), Quebec CSST (in combination with other isocyanates), CHE (isocyanates; S- strong)	Found in polyurethane paint, coatings, carpet backing and flooring finishes
Hexamethylenetetramine	100-97-0	$C_6H_{12}N_4$	4.0×10^{-3}	536	none	Quebec CSST	Used in small amounts as an in-can preservative in paints
4,4'-MDI Homopolymer; Diphenylmethane Diisocyanate; Methylene Biphenyl Diisocyanate (pure MDI)	101-68-8/ 25686-28-6/ 26447-40-5	$C_{15}H_{10}N_2O_2$	5.0×10^{-6}	314	0.035	AOEC (101-68-8 only, G), Quebec CSST (in combination with other isocyanates), CHE (isocyanates; S- strong)	Up to 60% of SPF (Part A); Up to 30% in acrylic resins; up to 16% in carpet backing; also in recycled rubber flooring, fluid-applied flooring and engineered wood binders
Nickel	7440-02-0	Ni	1 mm Hg @ 1810 °C	2730	0.007	AOEC (Rs), CHE (S- strong)	Up to 5% in steel; traces in fly ash
Perfluorobutane sulfonate (PFBS)	375-73-5	$C_4HF_9O_3S$	Not determined	235	none	None**	Up to 0.1% in waterborne finishes
Perfluorohexanoic acid (PFHxA)	307-24-4	$C_6HF_{11}O_2$	Not determined	157	none	None**	Up to 70% in stain and soil repellant treatments, Up to 0.1% in waterborne finishes
Polyfunctional Aziridine	64265-57-2	$C_{24}H_{41}N_3O_6$	Not determined	200	none	AOEC (Rs), Quebec CSST	Up to 97% in flooring finishes and HPCs
Polymethyl Methacrylate (PMMA)	9011-14-7	$C_5H_8O_2$	Not determined	200	none	AOEC (Rs), CHE (methacrylates; S- strong)	Up to 100% in solid surfaces; Also used in chalkboard paint and as binder in engineered wood
Polymeric MDI (pMDI)	9016-87-9	$[C_6H_3(NCO)CH_2]_n$	<0.0001	300	none	AOEC, CHE (isocyanates; S- strong)	Up to 50% in engineered wood binder; 30% in SPF insulation
Polymeric TDI (pTDI)	9017-01-0	$C_9H_6N_2O_2$	0.025 [g]	251	0.035	CHE (isocyanates; S- strong)	Up to 7% in cork flooring
Polyvinyl Chloride (PVC)	9002-86-2	$(C_2H_3Cl)_n$	NA	NA	none	AOEC (Rs), Quebec CSST, CHE (S- strong)	Up to 70% in sheet flooring; 60% in membrane roofing; 40% in corner guards; 20% in Ceilings; 14% in VCT; 10% in Vinyl carpet backing
Tall Oil Rosin Ester	8002-26-4	$C_{18}H_{36}O_2$	<0.001 mm Hg @ 20°C	100	none	AOEC (Rs), Quebec CSST	Up to 23.5% in linoleum floors

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Substance	CAS No.	Formula	Vapor pressure (mm Hg @ 25°C)[a]	Boiling point (°C)[b]	½ Chronic REL (µg/m³)[c]	Authoritative lists that include these chemicals[d]	Content in building materials[e]
Toluene Diisocyanate (TDI)	26471-62-5	$C_9H_6N_2O_2$	0.023	251	0.07	Quebec CSST, CHE (isocyanates; S- strong)	Up to 16% in polyurethane carpet backing
Triclosan	3380-34-5	$C_{12}H_7Cl_3O_2$	0.0000052	120	none	None**	Up to 1% in treated floors and carpets, and wet applied products, such as paints
Trimethylolpropane Triacrylate/ 2- Hydroxypropyl Acrylate	15625-89-5	$C_{15}H_{20}O_6$	0.00059	200	none	AOEC (Rs), CHE (acrylates; S- strong)	Up to 40% in UV-cured finishes
Vanadium	7440-62-2	V	2.34x10-2 mm Hg at 1,916 °C (extrapolated)[i]	3407	none	CHE (S- Strong)	Less than 1% in cold rolled steel and fly ash
Zinc Oxide	1314-13-2	ZnO	0 (approx)	NA	none	AOEC (Rs)	Up to 10% in flooring, 5% in paints, 2% in adhesives and roofing membranes, and 1% in nylon

a. Data from the Hazardous Substances Data Bank (HSBD), available at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
b. Data from Pharos Chemical and Materials Library (CML)
c. CRELs established by California Department of Public Health (CPDH). List of full CRELs available here: http://www.oehha.ca.gov/air/allrels.html. List of CRELs used in CA 01350 emissions testing protocol available here: http://www.cal-iaq.org/phocadownload/cdph-iaq_standardmethod_v1_1_2010%20new1110.pdf
d. Up-to-date asthmagen lists can be found here: http://www.aoecdata.org/ExpCodeLookup.aspx (AOEC), http://www.asthme.csst.qc.ca/document/Info_Gen/AgenProf/Bernstein/BernsteinAng.htm#N2_12 (CSST), and http://www.healthandenvironment.org/tddb (CHE)
e. As identified by cross-referencing asthmagen lists with over 1,250 products in the Pharos BPL.
f. Data from: "Product Safety Summary for MDI, Polymeric MDI, and MDI-based Products," *Bayer Material Science*, Updated December 31, 2012, http://www.productsafetyfirst.bayer.com/~/media/Product%20Safety%20First/Documents/Product%20Safety%20Summary%20MDI.ashx?la=en
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* Includes asthmagens as well as EDCs that may be indirectly associated with asthma onset.

** These chemicals are present in building products in Pharos, but are not present on asthmagen lists. However, these chemicals were added to this analysis due to emerging evidence of links to asthma found during scientific literature review.

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A Strategy to Prevent Asthma Through Building Product Selection

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Healthy Building Network
2001 S Street NW, Suite 570
Washington, DC 20009
tel: (202) 741-5717 or (877) 974-2767
fax: (202) 898-1612
info@healthybuilding.net
www.healthybuilding.net