RESEARCH

B08:
ASTHMAGENS IN BUILDING MATERIALS:
THE PROBLEM & SOLUTIONS
ASTHMAGENS IN BUILDING MATERIALS: THE PROBLEM AND SOLUTIONS

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ABSTRACT:
Asthma is a complex, heterogeneous disease, often of multifactorial origin. Asthma rates in the USA have been rising since at least 1980. 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. Nearly 26 million people are affected by chronic asthma, including over eight million children. As asthma affects more people, new strategies need to be considered. Among asthma risk factors, health organizations have identified hundreds of substances that can cause the onset of asthma. Many of these asthmagens are common ingredients of building products like insulation, paints, adhesives, wall panels and floors. This paper identifies asthmagens found in building products, how people can be exposed to these substances, and what is known and yet-to-be known about the impacts of these exposures. Key strategies to minimize exposures to asthmagens in building materials include understanding the composition of building materials; using product ingredient disclosure tools such as those recognized in LEED v4; and, modifying product certification standards, restricted substance lists, indoor environmental testing protocols, and green building incentives.

(Keywords: asthma, respiratory, health, buildings, protocols, IAQ, materials, isocyanates, formaldehyde, phthalates, polyurethane, insulation, PVC, flooring, certifications, FEMA, Katrina)

THE POST-KATRINA ASTHMA DISASTER

The Gulf Coast region understands the relationship between building materials and asthma. After Hurricanes Katrina and Rita struck in 2005, FEMA awarded $2.7 billion in contracts to shelter displaced residents. By August 2006, FEMA purchased 144,000 trailers and mobile homes.[1] A human-made health disaster ensued.

Connections between building materials, formaldehyde air emissions, and asthma were well established when FEMA put the Katrina trailers out to bid.[2] Formaldehyde resins bind composite wood casework, flooring, and wall panels. In 1992, the California Air Resources Board identified formaldehyde resins in these products as major sources of formaldehyde in indoor air. But FEMA's bidding process did not consider the potential health impacts of bringing these materials into a hot and humid climate where formaldehyde is more readily volatilized.

A meta-analysis of seven studies in homes and schools from several different countries concluded that asthma risk in children increased 3-17 percent for every 10 µg/m³ [8.1 parts per billion (ppb)] increase in formaldehyde in indoor air. In these studies, formaldehyde levels varied from very low to > 80 µg/m³ (65 ppb). [3]

A U.S. Centers for Disease Control (CDC) study of Katrina FEMA trailers found formaldehyde well above these levels. The mean for the 519 tested trailers was 77 ppb.[4] In some, air concentrations exceeded 300 ppb. The most common unit - Gulfstream - had a median concentration of 111 ppb, almost seven times the national median of 17 ppb. Using the meta-analysis’ correlation rate, children living in the Gulfstream FEMA trailers had a 35% to 200% elevated risk of having asthma. In a federal health survey of FEMA trailer residents, 31% of the participating children reported having a diagnosis of asthma, nearly three-fold higher than the prevalence of childhood asthma nationally (11% in 2010).[5, 6]

Department of Homeland Security (DHS) Inspector General Richard Skinner reviewed the situation. “All of the units were some form of manufactured housing and therefore tended to have more of the manufactured wood products that can emit formaldehyde gas,” he reported. And the FEMA contracts “did not contain protections against excessive formaldehyde concentrations.”[7]

PATHWAYS FOR EXPOSURE TO ASTHMAGENS IN BUILDING MATERIALS

DHS Inspector General Skinner laid bare this reality: “Although workplace standards and recommendations for allowable exposures to formaldehyde have been implemented to protect workers who are exposed to formaldehyde, there is far less guidance as to what levels should be avoided in residences.”

Similarly, authoritative lists of asthmagens are based largely on studies involving worker exposures—hence the commonly used term “occupational asthma.” However, the extent to which chemicals known to cause occupational asthma may have similar effects within the general public, especially children, is often unclear for several reasons:

1. Occupational exposures are often much higher than residential exposures;
2. Dose-response levels are often not well enough established to allow extrapolation to low levels of prolonged exposure;
3. The importance of multiple factors in the origins of asthma, including co-exposure to allergens in residences, may make it more difficult to estimate the contribution of toxic exposures to asthma risk in homes compared to the workplace; and,
4. Occupational asthma generally affects adults whereas most asthma in the general public is among children, who are more vulnerable due to their smaller size and developing immune and respiratory systems.[8]

These challenges clearly pertain to chemicals in building products. Workers exposed to occupational asthmagens during product manufacture or building construction will be at increased asthma risk. But, after construction and building occupancy, exposure levels are generally unknown for most asthmagens.

Building occupants can be exposed to asthmagens in building materials by several pathways. Volatile and semi-volatile asthmagens may volatilize and be emitted into the air to be inhaled. Semi-volatile organic compounds (SVOCs) may migrate from products to dust particles by adsorption, which may in turn be inhaled, ingested, or come into contact with the skin. Non-volatile asthmagens on the surface of a building finish may be
released as dust through degradation or abrasion and be picked up through the skin on contact.

Most concern about asthma risk associated with chemicals in building products in the general population has focused on volatile organic compounds (VOCs). Concern about exposure to less volatile compounds like phthalates and isocyanates has increased for several reasons:

1. Air monitoring and dust studies identify a number of SVOCs that may have originated in building materials;
2. Increasing numbers of epidemiologic studies show correlations between environmental levels of some SVOCs and asthma prevalence; and,
3. In vivo and in vitro laboratory studies have identified mechanisms by which some of these compounds may be related to asthma, including alteration in lung and immune system development after early life exposures.

**EMERGING EVIDENCE OF HARM**

Two SVOCs - isocyanates and phthalates - illustrate different aspects of the mounting evidence.

**ISOCYANATES**

Isocyanates are a family of highly reactive, low molecular weight (MW) chemicals, and are essential ingredients of polyurethane products like adhesives, furniture foam, and spray polyurethane foam (SPF) insulation.

A federal interagency air quality group notes that “agencies have received complaints regarding health effects including severe respiratory irritation, breathing difficulties, dizziness and nausea, resulting from the installation of SPF in homes.”[9]

EPA has an action plan to deal with isocyanates and is leading a multi-agency work group to address SPF.[10] In 2014, the California Department of Toxic Substances’ Safer Consumer Products initiative named SPF as one of its three initial “priority products” due to its “potential for exposure to contribute to or cause significant or widespread adverse impacts.”[11]

Isocyanates are an established cause of occupational asthma through both allergic and irritant mechanisms.[12] They can cause contact dermatitis and lead to respiratory tract sensitization after skin exposure.[13] In a study in mice, isocyanate skin sensitization in females prior to mating resulted in airway inflammation and other features of an asthma phenotype in offspring.[14]

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.”[15]

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. Exposures to isocyanates may also occur in people who enter a building before spray foam insulation is fully cured. In addition, a number of consumer products contain unreacted isocyanates, including adhesives and polyurethane coatings.[16] This means that skin or inhalation exposures may occur fairly regularly within the general population.

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.”[17]

**PHTHALATES**

Phthalates are synthetic diesters of phthalic acid used in many consumer products. These SVOCs 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.

The most common phthalates can be loosely grouped by lower and higher molecular weight (MW). These two groups differ in their industrial uses, environmental fate and transport, and exposure pathways. The dominant plasticizers used in building products like vinyl flooring are higher MW phthalates such as diethyl hexyl phthalate (DEHP) and benzyl butyl phthalate (BBP).

Exposures to phthalates are widespread and come from many sources.[18] This makes it difficult to quantify exposures that can be traced specifically to building materials. Inhalation, ingestion, and transdermal absorption are potential routes of exposure.

Air concentrations of phthalates are approximately 10 times higher indoors than outdoors.[19] Sources of phthalates that have been reported to affect indoor air phthalate levels are PVC building materials and furniture.[20] “Exposure to pollutants in the indoor environment has increased with improved insulation and reduced ventilation making many indoor environments act as concentrators of emissions from plastics, paints, and other building materials, reported Barro et al. in 2009. Pollutants like phthalates, they wrote, become concentrated in fine particulate matter with a weight of <2.5 micrograms; that is, dust.[21]

House dust can be inhaled or ingested. Guo et al. recently calculated that house dust could contribute 10% to 58% of total DEHP exposure to residents in a sample from Albany, New York. [22] Another study using biomonitoring data and modeling estimated that 39% of DEHP levels were attributable to indoor dust ingestion and 14% to inhalation.[23]

A public health study after Katrina took dust samples from children’s bedroom floors in FEMA trailers and found measurable levels of BBP and DEHP in all of them, with mean values of 175 ppm for DEHP and 59 ppm for BBP.[24] The levels of phthalates found in FEMA trailers are lower than those identified in older structures. Fromme et al in 2003 studied dust in German apartments and classrooms, and found mean concentrations of DEHP of 775.5 ppm DEHP and 86 ppm BBP.[25] In a recent study of phthalates in dust in 63 daycare centers, Fromme et al. reported median DEHP levels of 888 ppm.[26] They also measured phthalate metabolites in children attending those daycare centers. Using a cumulative risk assessment approach, they concluded that 20 percent of children had exposure levels to phthalates exceeding a safe reference dose. Rudel et al. in 2003 found a median value of 340 ppm DEHP and 45 ppm BBP in over one hundred dust samples from homes in Cape Cod.[27]
PHTHALATES AND ASTHMA

Toxicology studies conducted both in vitro and in vivo animal models have proposed various mechanisms by which phthalates may exert their effect on allergies or airway inflammation.[28] Some phthalates can act as an adjuvant, magnifying the immune and respiratory system response to an allergen.[29] Phthalate exposure may also alter lung and immune system development.[30],[31] In rodent studies, DEHP exposure in utero is associated with delayed lung maturation.[32] The timing and route of exposure also appear to be important. (Similar exposure pathways and early life impacts are found in Bisphenol A Diglycidyl Ether (BADGE), another SVOC commonly used in building products. See Appendix B.)

Numerous studies show an association between exposure to phthalates and respiratory symptoms including asthma or wheeze.[33],[34],[35],[36],[37] The presence of PVC flooring and wall coverings is also associated with increased risk of childhood asthma.[38],[39],[40]

In summary, evidence of a causal relationship between exposure to phthalates and asthma continues to accumulate, particularly for the higher MW compounds commonly used in building products. Many observational studies are limited by cross-sectional design and the challenges of long-term exposure assessment for chemicals with relatively short half-lives. However, in vitro and in vivo laboratory studies have identified plausible mechanisms by which phthalates may influence the risk of allergies and asthma and add to the weight of evidence.

MENDING THE GAP IN BUILDING PRODUCT EVALUATIONS

Over the past two decades, green building advocates have developed powerful tools to test and certify the healthfulness of building materials. Asthma is not yet a major consideration of most programs, despite growing evidence that asthmagens are being released from building materials into indoor environments.

Many green building protocols and regulations reflect the times in which they evolved, when VOCs were considered the primary hazard from products. Regulatory requirements have led manufacturers of wet-applied products to publicly disclose and reduce VOCs since the 1970s. And green building IAQ certifications have measured individual VOC emissions from other non-wet applied products for over a decade.

These programs proved useful in significantly improving many building materials, which today generally contain and emit far fewer VOCs than they did in the 1990s.

More attention needs to focus on other health hazards. This came into sharp relief while the Healthy Building Network (HBN) researched its 2013 report, Full Disclosure Required: A Strategy to Prevent Asthma.[41] This report built from prior works such as Perkins + Will’s 2012 report, Environments: A Compilation of Substances Linked to Asthma, which identifies 374 known or suspected asthmagens in the built environment.[42] Full Disclosure Required narrows the focus to 50 substances, 38 of which are known asthmagens. Twelve chemicals are of emerging concern.

The authors cross-referenced 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 – with materials catalogued in HBN’s Pharos Building Product Library.[43],[44],[45]

Full Disclosure Required names twenty of these asthmagens to be of the highest priority, due to clear pathways for building occupants to be exposed to them after product installation and during normal use. Priority asthmagens include: acid aldehydes (two types); acrylates (four types); ammonium hydroxide; bisphenol A diglycidyl ether (BADGE); ethanolamines (three types); formaldehyde; isocyanates (six types); polyfunctional aziridine; and, styrene. Full Disclosure Required also recommends minimizing the use of phthalates.

Green building certifications standards do not consider most of these chemicals. Many asthmagens are not VOCs, and are not detected by current emissions testing protocols. As a result, high concentrations of asthmagens can be present in products that have earned low VOC-emissions certifications.

Chemical identification is essential for protecting human health. Most green building programs do not identify and therefore do not restrict most asthmagens. Appendix C (Asthmagens in Building Products: Identification by Relevant Standards) reveals:

- Twenty-three (46%) of the substances are not identified by four leading programs (Cradle-to-Cradle, Living Building Challenge, California 01350, and Greenguard Gold).
- California 01350 indoor air emissions testing looks for only three (3) of the substances (formaldehyde, styrene and toluene).
- Greenguard Gold looks for fourteen of these chemicals in its air emission tests. Cradle-to-Cradle and LBC product content requirements identify 11 and 17 of these substances, respectively.
- None of these programs screen for isocyanates or biocides like triclosan.

The global automobile industry has shown that more comprehensive screening is possible. Manufacturers launched a voluntary program, the Global Automotive Declarable Substance List (GADSL), in 2005. Under the GADSL program, carmakers require suppliers to report the presence of 39 of the 50 substances, often at levels far lower than commonly declared in building materials. Most isocyanates must be identified if they are present at over 0.01% of the product by weight. Triclosan must be declared when present at 0.001% of the product.[46]

Gaps in coverage represent opportunities to extend the healthfulness of building materials to other substances of concern, including asthmagens.

Transparency and research deepen the awareness of material contents. Science is associating certain chemicals with emerging health impacts. Chemists and engineers are innovators who can reduce the impacts of products through green chemistry. And leading corporations are recognizing that the future of green building belongs to the triad of creativity, transparency, and healthfulness.
These efforts, bound by independently verified certifications, provide a strong platform for reducing exposures to asthmagens in building materials.

- **Standard-based certifications can help to prevent exposures to asthmagens by defining the product, setting conditions for its evaluation, and standardizing testing protocols.** The end product is a simple label or declaration indicating a specific product meets the standard. Qualified material selection choices can be identified via the standard’s product database or registry.

- **Standards can consider any substance in a product that can harm building occupants.** In the case of asthmagens, standards can be updated or created to recognize the low dose exposures that can cause the onset of asthma, recognizing that for many of these substances researchers have not been able to determine safe thresholds. Emissions based programs can complement their emissions testing protocols with content testing to support avoidance. They can draw from transparency efforts (like the Pharos Project, Declare, and the Health Product Declaration) to ensure that the tests search for any asthmagen known to be present in these products, even at trace levels.

- **Restricted substance lists that drive these certifications can be updated to include priority asthmagens.** New protocols may be developed specifically to address asthmagens or other substances of concern, and require testing for these substances. For example, the European Union this year launched a revised EU Ecolabel for Paints and Varnishes that quantifies and limits SVOC content. It caps SVOC content at 30 grams per liter (g/l) for white paint applied to interior walls, and 60 g/l for some other applications.[47] US-based IAQ programs do not yet quantify SVOCs as a whole.

- **Standards must be transparent.** The vast majority of today’s green building certification programs make their protocols freely available to the public. One notable exception: The Asthma and Allergy Foundation of America recently certified a line of vinyl flooring as “Asthma & Allergy Friendly.”[48] The certification’s standard is not posted on its website, and the foundation did not respond to this paper’s authors requests for information as of August 15, 2014.

While transparency is vital for understanding the potential hazards of a given product, standards can also protect intellectual property. If a standard and certification scheme is trustworthy, it may be generally assumed that the independently verified claims are accurate without full disclosure. The disclosure has been made to the certifier, who assures the conditions of the standard have been met.

- **Asthma prevention strategies can be applied, in turn, to the U.S. Green Building Council’s Leadership in Energy and Environmental Design (LEED) rating system, which should reward projects that avoid introducing asthmagens into the indoor environment.**

USGBC President and CEO Rick Fedrizzi told Greenbuild 2012 attendees, “When people doubt that we can improve health outcomes, we’re going to show them the drawers of unused asthma inhalers in green schools.” [49] The green building movement finds solutions. By focusing more of its collective resources - materials research, product testing, certifications and incentives - on asthmagens in building materials, it can solve this problem, too.

**ACKNOWLEDGEMENTS**

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**REFERENCES**


ASTHMA GENS IN BUILDING MATERIALS: THE PROBLEM AND SOLUTIONS (cont.)


ASTHMAGENS IN BUILDING MATERIALS: THE PROBLEM AND SOLUTIONS (cont.)

APPENDIX A.
BACKGROUND ON ASTHMA
Asthma is a complex and heterogeneous disease. Various combinations of genetic predisposition and exposures to environmental agents, including allergens, chemicals, psychosocial stress, and different dietary patterns, can result in the development of asthma. Airway narrowing and wheeze characterize asthma. Inflammation in the lungs is usually present although there is some variability in the remodeling of airways accompanying the disease.

We use term “asthmagen” to refer to a chemical that can initiate asthma onset in someone who did not previously have the disease. Exposures to asthmagens can combine with other risk factors to make the disease more likely. An asthma trigger is an environmental agent that can cause an asthma attack in someone who has the disorder.

Two kinds of asthma that can be related to chemical exposures:
1. One kind is allergic or sensitizer induced asthma; the other is
2. Irritant-induced asthma.
3. Sometimes they are mixed in a single person.

Allergic or sensitizer induced asthma involves an immune system response to the chemical exposure. If it’s not recognized early, the sensitization can become more widespread to include a number of additional chemical substances.

Chemicals may also contribute to the risk of sensitizer-induced asthma by serving as an adjuvant—an agent that boosts the response to an allergen, making sensitization more likely. For example, laboratory animal studies suggest that the plasticizer DEHP may act as an adjuvant, thereby enhancing the risk of allergic sensitization to an allergen.[1]

Irritant induced asthma can be caused by a single, fairly large exposure to the chemical irritant. Airway irritation is followed by airway narrowing and wheezing. This may then lead to reactive airway disease and more frequent wheezing episodes with an ultimate diagnosis of asthma.

APPENDIX A SOURCES:

APPENDIX B.
Bisphenol A Diglycidyl Ether (BADGE): Early life exposures
Bisphenol A diglycidyl ether (BADGE) is a member of the family of glycidyl ethers that have been widely used as components of epoxy resins for decades. In the built environment, they are found in high concentrations in epoxy fluid applied floors, adhesives and sealants, as well as paints, toys, compact discs, electronic equipment, and printed circuit boards. Volatilization and/or release of bisphenol analogues from these products are considered to be sources of contamination of indoor dust.[1]

BADGE and its hydrolysis products have been reported in indoor dust. Wang et al. in 2012 studied 158 dust samples from four countries. The study found mean concentrations of 2 to 3 parts per million (ppm) of BADGE and its hydrolysis products in indoor dust, and considered BADGE-based epoxy resin as a source.[2] Rudel et al. in 2003 found a median value of 0.821 ppm bisphenol A in 118 dust samples from homes in Cape Cod.[3],[4]

Workers who spray BADGE have elevated urinary levels of bisphenol A (BPA), which suggests that BADGE may generate BPA endogenously.[5] Exposure to BPA in the general population is widespread.[6] Dietary intake from food contamination is a major source, but dermal contact as well as indoor dust ingestion and inhalation contribute significantly. The estimated median daily intake of bisphenol analogues through dust ingestion in the U.S., China, Japan, and Korea was 12.6, 4.61, 15.8, and 18.6 ng/kg body weight/day, respectively, for toddlers and 1.72, 0.78, 2.65, and 3.13 ng/kg bw/day, respectively, for adults.[7]

Some of the highest levels of BPA reported in human specimens (8.3 ng/ml) occur in amniotic fluid between weeks 15-18 of pregnancy.[8] In some animal studies, developmental exposure to bisphenol A is associated with alteration of the immune response and in some cases, increased sensitization to allergens.[9],[10] In a laboratory study of mice, maternal dietary exposure to BPA during gestation and lacta-
tion resulted in increased allergic sensitization to an allergen (ovalbumin) and airway inflammation in newborn offspring.[11] In a similar study, after maternal exposure to BPA in drinking water during gestation and lactation, offspring were studied in adulthood after being on a BPA-free diet following weaning.[12] Allergic sensitization to ovalbumin in infancy remained increased in BPA-exposed animals without evidence of increased lung inflammation.

But not all studies in mice have similar findings.[13],[14] A recent study using a similar, widely used experimental model of asthma in mice concluded that the impacts of BPA on immune system development and allergic asthma response is dependent on timing and duration of exposure.[15] Lifelong exposure from birth, but not pre-natally, until the last antigen challenge increased inflammation in the lung, airway hyper-reactivity and antigen-specific serum IgE levels in OVA-sensitized adult mice compared to mice without BPA exposure.

A study in female non-human primates found that exposure to environmentally-relevant doses of BPA during late gestation accelerates secretory cell maturation in the proximal conducting airways.[16]

In one prospective cohort study of 398 mother-infant pairs, higher prenatal BPA exposure was associated with increased odds of wheeze in early life.[17] Another long-term prospective cohort study of children in New York found that urinary BPA concentrations at ages 3, 5, and 7 years were associated with increased asthma risk assessed at ages 5 to 12 years, whereas maternal gestational exposures had an inverse relationship to asthma risk.[18]

Collectively, these studies show that developmental exposures to BPA can alter both immune system and lung development in ways that may make asthma more likely. Some developmental periods appear to be more responsive than others, making the timing and duration of exposure important considerations.

### APPENDIX C.

**ASTHMAGENS IN BUILDING PRODUCTS: IDENTIFICATION BY RELEVANT STANDARDS**

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<td>Methyl Methacrylate (80-62-6)</td>
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<td>Trimethylolpropane triacrylate (15625-89-5)</td>
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<td>2-AmiNoethaNol (141-43-5)</td>
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<td>TriethaNolamine (102-71-6)</td>
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**GREEN BUILDING STANDARDS IDENTIFICATION**
## APPENDIX C.

### ASTHMAGENS IN BUILDING PRODUCTS: IDENTIFICATION BY RELEVANT STANDARDS (cont.)

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<th>Substance of Concern (CAS No.)</th>
<th>Related Building Materials</th>
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<td>Methylene Bisphenyl Diisocyanate (101-68-8)</td>
<td>Recycled tire flooring, adhesives, composite wood binder, fluid flooring, spray foam insulation, carpet backing</td>
<td>0.01% (BGO)</td>
<td>No</td>
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<td>Methylenebisphenyl diisocyanate &amp; related compounds (26447-40-5)</td>
<td>SPF, thermal insulation, mineral board, composite woods, solid surfaces, high performance coatings, adhesives</td>
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<td>Toluene diisocyanate (26471-62-5)</td>
<td>Carpet backing</td>
<td>0.01% (BGO)</td>
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<td>Polymeric TDI (9017-01-0)</td>
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<td>1,6-Hexamethylene Diisocyanate (822-06-0)</td>
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<td>4,4’-MDI homopolymer (25686-28-6)</td>
<td>Spray foam insulation</td>
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<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Diphenylmethane-2,4’-diisocyanate (2,4’-MDI) (5873-54-1)</td>
<td>Spray foam insulation, polyurethane carpet backing</td>
<td>0.01% (BGO)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Polymeric MDI (9016-87-9)</td>
<td>Cork flooring, fluid flooring, engineered wood binder, adhesives, spray foam insulation, polyurethane carpet backing, underlayment</td>
<td>0.01% (BGO)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td><strong>PHTHALATES</strong></td>
<td></td>
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</tr>
<tr>
<td>Benzyl butyl phthalate (85-68-7)</td>
<td>PVC flooring (sheet and VCT), floor- ing adhesives, PVC roofing membrane, carpet backing</td>
<td>0.01% (BGO); prohibited over 0.25%</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Di-2-ethylhexyl phthalate (117-81-7)</td>
<td>Roofing membranes, vinyl carpet backing, PVC flooring (VCT and sheet)</td>
<td>0.01% (BGO); prohibited over 0.3%</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Di-n-hexyl phthalate (84-75-3)</td>
<td>Vinyl composition tile</td>
<td>0.1% (Renault)</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Dibutyl phthalate (84-74-2)</td>
<td>Flooring finishes, casework adhesive, lacquers</td>
<td>0.01% (BGO); prohibited over 0.25%</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>DiisoNonyl phthalate (28553-12-0)</td>
<td>Vinyl composition tile, vinyl carpet backing</td>
<td>0.1% (BGO)</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>DiisoNonyl phthalate (DINP-1, mixture of isomers as manufactured) (68515-48-0)</td>
<td>Carpet (backing), membrane roofing, VCT flooring</td>
<td>0.1% (BGO)</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Dicyclohexyl phthalate (84-61-7)</td>
<td>Methyl methacrylate flooring</td>
<td>0.1% (BGO)</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Diisodecyl phthalate (117-81-7)</td>
<td>Flooring adhesive, vinyl composition tile</td>
<td>0.1% (BGO)</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Di-n-octyl phthalate (117-84-0)</td>
<td>PVC membrane roofing</td>
<td>0.1% (BGO)</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>OTHERS</strong></td>
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</tr>
<tr>
<td>Ammonium hydroxide (1336-21-6)</td>
<td>Paints, adhesives</td>
<td>0.01% (BGO)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Bisphenol A Diglycidyl Ether (BADGE, 1675-54-3 and 25085-99-8)</td>
<td>Adhesives, High Performance Coatings, Grouts and Mortars</td>
<td>0.01% (BGO)</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Formaldehyde (50-00-0, and various compounds)</td>
<td>Laminate, thermal insulation, mineral board, SPF, wallboard, engineered wood, acrylic/latex adhesives</td>
<td>Any level must be reported (GADSL 2014) prohibited over 0.1%</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Polyfunctional aziridine (64265-57-2)</td>
<td>Floor finish, high performance coating</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Styrene (100-42-5)</td>
<td>High performance coatings, polystyrene insulation, foam board insulation</td>
<td>0.1% (BGO)</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>
## APPENDIX C.

### ASTHMAGENS IN BUILDING PRODUCTS:
IDENTIFICATION BY RELEVANT STANDARDS (cont.)

<table>
<thead>
<tr>
<th>Substance of Concern (CAS No.)</th>
<th>Related Building Materials</th>
<th>Auto Industry</th>
<th>C2C</th>
<th>LBC</th>
<th>OIT350</th>
<th>GG GOLD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BIOCIDES</strong></td>
<td></td>
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</tr>
<tr>
<td>Benzisothiazolin-3-one (BIT) (2634-33-5)</td>
<td>Paints, adhesives</td>
<td>0.01% (BGO)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Didecyl dimethyl ammonium chloride (DDAC) (7173-51-5)</td>
<td>Anti-sapstain treatments</td>
<td>0.01% (BGO)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Hexamethylenetetramine (100-97-0)</td>
<td>Preservatives</td>
<td>0.01% (BGO)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Triclosan (3380-34-5)</td>
<td>Paints, carpets, engineered wood, ceramic tile</td>
<td>0.001% (BGO)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td><strong>METAL DUSTS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aluminum (dust) (7429-90-5)</td>
<td>Window frames, siding, board insulation</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Aluminum oxide (dust) (1344-28-1)</td>
<td>Resins, finishes, grouts, laminate surfaces</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Chromium &amp; chromium compounds (dust) (7440-47-3; 18540-29-9 (Chromium VI))</td>
<td>Stainless steel; aluminum alloys; carpet backings (via fly ash filler)</td>
<td>0.001% (Renault) prohibited above 0.1%</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Cobalt &amp; cobalt compounds (dust) (7440-48-4)</td>
<td>Paints, flooring stains, fly ash, porcelain tile</td>
<td>0.001% (BGO)</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Nickel &amp; nickel compounds (dust) (7440-02-0)</td>
<td>Wall guards; carpet backings (via fly ash)</td>
<td>0.001% (BGO)</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Vanadium (dust) (7440-62-2)</td>
<td>Porcelain tile</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Zinc Oxide (dust) (1314-13-2)</td>
<td>Paints, carpet backing, glazes, adhesives</td>
<td>0.01% (BGO)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td><strong>PERFLUOROCARBONS</strong></td>
<td></td>
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</tr>
<tr>
<td>Perfluorooctanesulfonic acid &amp; salts (PFOS [C8], 1763-23-1)</td>
<td>Stain-blocking carpet treatments</td>
<td>0.001% (BGO); prohibited over 0.1% (Renault)</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Perfluorooctanoic acid &amp; its salts (PFOA [C8], 335-67-1)</td>
<td>Stain-blocking carpet treatments</td>
<td>0.01% (BGO)</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Perfluorohexanoic acid (PFHxA [C6], 307-24-4)</td>
<td>Stain-blocking carpet treatments, grout</td>
<td>0.1% (Renault)</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td><strong>OTHERS</strong></td>
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</tr>
<tr>
<td>Acetic acid, glacial (64-19-7)</td>
<td>Silicone caulking, High performance coatings</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Bispheneol A (80-05-7)</td>
<td>High-performance concrete hardeners, fluid-applied flooring hardeners, epoxies, dry-erase paint</td>
<td>0.01% (BGO)</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Colophony (gum rosin) (8050-09-7)</td>
<td>Linoleum and adhesives</td>
<td>0.01% (Renault)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Polyvinyl Chloride (9002-86-2)</td>
<td>VCT flooring, corner guards, ceiling panels, carpet backing, roofing membrane</td>
<td>0.1% (BGO)</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Tall Oil Rosin Ester (8002-26-4)</td>
<td>Linoleum and adhesives</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Toluene (108-88-3)</td>
<td>Adhesives, lacquers, engineered wood (binder and finish), paint</td>
<td>0.1% (BGO)</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Wood dust, esp. Western Red Cedar (No CAS No.)</td>
<td>Composite wood products and laminates</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>