

**Appendix A: Photographs:**



<b>Photo 1</b>	<b>Example of:</b> Suspected Mould Growth (S)	
<b>Description:</b> This photos is an example of suspected mould growth. The base of the walls and below the window show what is presumably water staining, with fungal growth suspected at the base of the far corner and on the backside of the drywall.		



<b>Photo 2</b>	<b>Example of:</b> Minor Mould Growth (1)	
<b>Description:</b> This photo shows an example of minor mould growth (dark staining on top left corner of drywall). Minor mould growth is to be considered <math>< 1\text{m}^3</math>.		

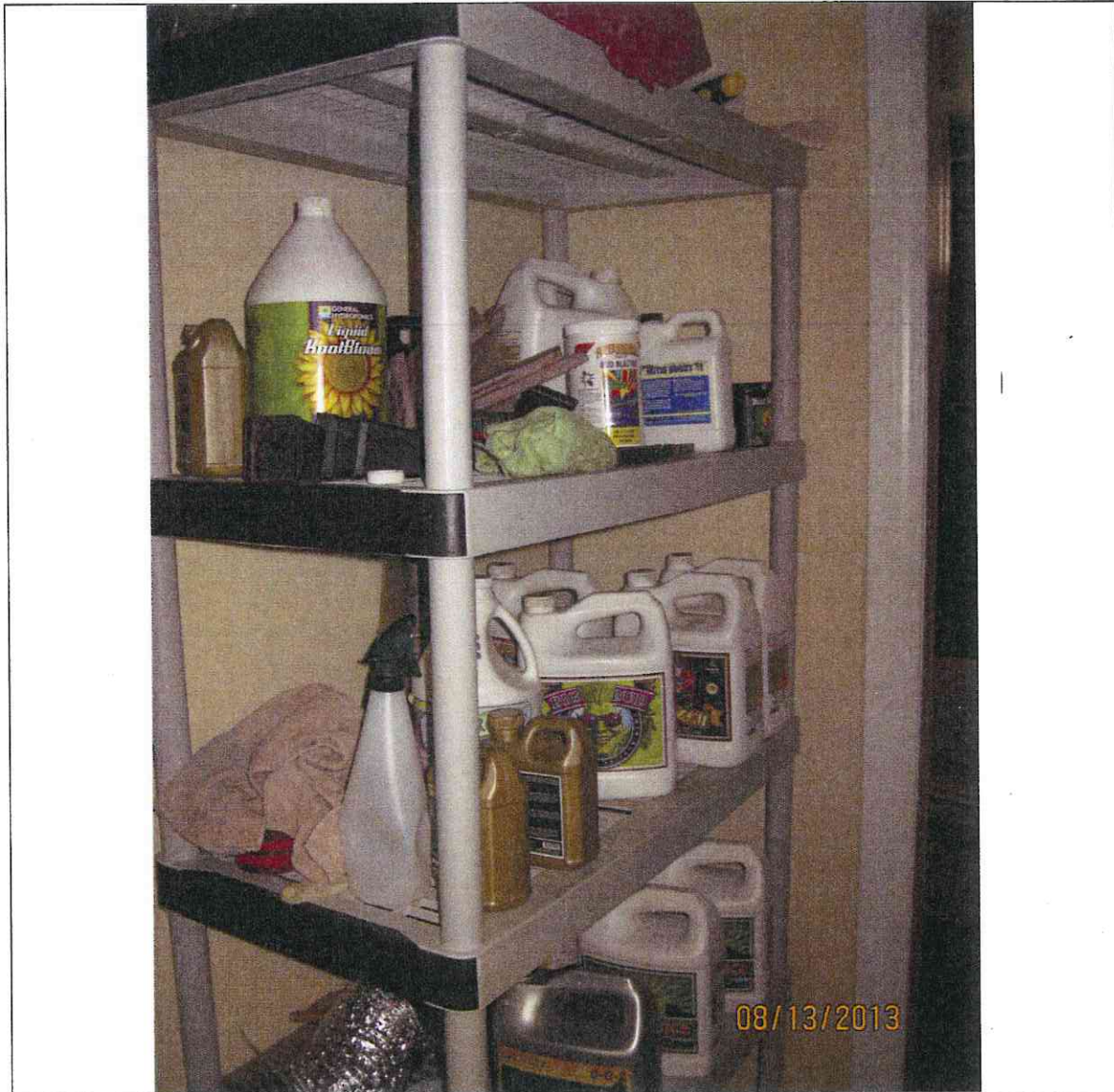


<b>Photo 3</b>	<b>Example of:</b> Major Mould Growth (2)	
<b>Description:</b> This photo shows an example of major mould growth (dark and spotted staining throughout drywall). Major mould growth is to be considered $> 1m^3$ .		



**Photo 4**      **Example of:** Suspected Chemical Container (S)

**Description:** This photo is an example of suspected chemical containers. In our experience, plastic drums (particularly blue-coloured as shown) are frequently used in marijuana grow operations to mix and distribute chemical such as fertilizers. As no labels are present, the contents are unknown.



**Photo 5**

**Example of: Chemical Containers – labels mostly visible**

**Description:** This photos is an example of stored chemical containers presumably related to the marijuana grow operation. Most containers have labels that are at least partially visible.




**Photo 6**      **Example of:** Chemical containers – labels mostly not visible

**Description:** This photo is an example of stored chemical containers presumably related to the marijuana grow operation. Due to the way the containers are being stored, the labels are not visible and therefore the contents cannot be identified by photos along.

## **Appendix B: References**

- American Academy of Allergy, Asthma and Immunology (AAAAI), National Allergy Bureau Scale for Mould Spores and Tree, Weed and Grass Pollen. National Allergy Bureau.
- American Industrial Hygiene Association. Clandestine Indoor Marijuana Grow Operations – Recognition, Assessment, and Remediation Guidance. 2010.
- Bioaerosols: Assessment and Control. American Conference of Governmental Industrial Hygienists (ACGIH). 1999.
- Environmental Protection – Environmental Fate of Pesticides. British Columbia Ministry of Agriculture. [http://agf.gov.bc.ca/pesticides/c\\_2.htm](http://agf.gov.bc.ca/pesticides/c_2.htm) <Accessed on 09/11/2014>
- Fungal Contamination in Public Buildings: Health Effects and Investigation Methods. Health Canada. 2004.
- Fraser Institute. Marijuana growth in BC, 2004:  
<http://www.fraserinstitute.org/newsandevents/commentaries/3481.aspx> <Accessed on 05/18/2010>
- IICRC S520: Standard and Reference Guide for Professional Mold Remediation. Institute of Inspection Cleaning and Restoration Certification. Second Edition: August 2008.
- Mould Remediation in Schools and Commercial Buildings, United States Environmental Protection Agency (EPA). 2001.
- Mould Guidelines for the Canadian Construction Industry. Canadian Construction Association. 2004. Standard Construction Document CCA 82 – 2004.
- Mustard, J., P. Hansen, and G. Wedman: Remediation of Former Marijuana Grow Operations. Presentation at AIHce 2006. Chicago, IL: 2006.
- National Collaborating Center for Environmental Health. Recommendations for Safe Re-occupancy of Marijuana Grow Operations. March 2009.
- New York City Department of Health and Mental Hygiene. Guidelines on Assessment and Remediation of Fungi in Indoor Environments. November 2008.
- Niagara Regional Police Office. Marijuana Home Grow Operations:  
<http://www.nrps.com/community/marijuana.asp> <Accessed on 05/18/2010>
- Occurrence, Fate and Transport Behaviors of Pesticides on Urban Impervious Surfaces, Jiang, W. University of California Riverside, 2012.
- OSU Extension Pesticide Properties Database - National Pesticide Information Centre (NPIC) –  
<http://npic.orst.edu/ingred/ppdmove.htm> <Accessed 09/11/2014>
- Post-remediation Verification and Clearance Testing for Mold and Bacteria: Risk-Based Levels of Cleanliness Assurance. R Brandys and G. Brandys. Occupational and Environmental Health Consulting Services Inc. 2006
- Questions About Pesticide Environmental Fate, Extoxnet FAQs, **University of California at Davis, Environmental Toxicology Department**  
<http://extoxnet.orst.edu/faqs/pesticide/pestfate.htm> <Accessed 09/11/2014>
- Recognition, Evaluation and Control of Indoor Mold. B. Prezant, D. Weeks and J. Miller (eds). American Industrial Hygiene Association (AIHA). 2008.
- Report of Microbial Growth Task Force, American Industrial Hygiene Association (AIHA) Press. 2001.
- Residential Indoor Air Quality Guidelines - Moulds. Health Canada. 2007.



Royal Canada Mounted Police. Marijuana Grow Operations: <http://www.rcmp-grc.gc.ca/fio-ofi/grow-ops-culture-eng.htm> <Accessed on 05/18/2010>

US Environmental Protection Agency (July 24, 2007) What is a pesticide? epa.gov. Retrieved on September 15, 2007.

Texas Department of Public Health. 1991. Clearance Guidelines for Mold Remediation Projects.

Wash-off potential of urban use insecticides on concrete surfaces, Jiang W. *et al.* Environ Toxicol Chem. 2010 Jun;29(6):1203-8 doi:10.1002/etc.184

WorkSafeBC. Guidelines Part 4 – Indoor Air Quality (G4.79 Moulds and indoor air quality). *Occupational Health & Safety Regulation*.

Worldwide Exposure Standards for Mold and Bacteria: Historical and Current Perspectives. 7th Edition. R Brandys and G. Brandys. Occupational and Environmental Health Consulting Services Inc. 2007.





**Appendix C: CV**

Please find included the following CV:

- Shannon McIntosh, BSc, CIH, CRSP – Manager, Industrial Hygiene

## Shannon McIntosh, BSc, DipT OHS, CIH, CRSP

### EXPERIENCE:

May 2013 to  
present

#### **Pacific EHS**

Manager, Industrial Hygiene: Responsibilities include: supervision of hygiene work, focusing on fungal investigations and worker exposure assessments; conducting walkthrough surveys; determining sampling plans; collecting various samples for analyses; interpreting and presenting data and findings in reports; preparation of proposals; communication with clients regarding all aspects of hygiene projects; developing contract specifications; training hygiene technologists, specialists and project managers; and delivering training presentations. Areas of expertise include fungal investigations, sampling for biological contaminants, air sampling for numerous contaminants, indoor air quality investigations, worker exposure and risk assessments, remediation of indoor environments and the assessment and remediation of properties used for the illegal manufacture of controlled substances.

August 2012 to  
April 2013

#### **Pacific EHS**

(formerly: Pacific Environmental Consulting)  
125-3001 Wayburne Drive, Burnaby, BC V5G 4W3

Senior Project Manager, Acting Manager, Industrial Hygiene: Responsibilities included: managing and supervising industrial hygiene department, in addition to those responsibilities as a Project Manager and Project Coordinator, outlined below.

June 2007 to  
August 2012

#### **Pacific Environmental Consulting**

Project Manager and Project Coordinator: Responsibilities included: project management of hygiene fieldwork, focusing on fungal investigations; conducting walkthrough surveys; determining sampling plans; collecting various samples for analyses; interpreting and presenting data and findings in reports; preparation of proposals; communication with clients regarding all aspects of hygiene projects; developing contract specifications; delivering training presentations. Areas of expertise include fungal investigations, sampling for biological contaminants, air sampling for numerous contaminants, indoor air quality investigations, worker exposure and risk assessments, remediation of indoor environments and the assessment and remediation of properties used for the illegal manufacture of controlled substances (marijuana and methamphetamine).

March 2006 to  
September 2006

#### **Coca-Cola Bottling Co.**

Viking Way, Richmond, BC

Lockout Specialist: Developed a lockout program for the facility and trained workers in lockout procedures. Also helped with health and safety related tasks such as emergency planning.

### EDUCATION:

2007                      Diploma in Occupational Health and Safety, British Columbia Institute of Technology, Burnaby, British Columbia  
2004                      Bachelor of Science, Biology, University of British Columbia, Vancouver, British Columbia

### ACCREDITATIONS:

2012	Certified Industrial Hygienist (CIH), CP 10126, American Board of Industrial Hygiene
2011	Canadian Registered Safety Professional (CRSP), Registration #11-4236, Board of Canadian Registered Safety Professionals
2008	Certified Ergonomic Specialist, EK Gillin & Associates Inc.

#### CONTINUING EDUCATION:

June 2014	AIHce 2014, San Antonio, Texas
June 2014	Toxicology Strategies for Petroleum Industry Exposure Assessments, Professional Development Course (PDC), San Antonio, Texas
March 2014	AIHA BC Yukon Local Section – Annual General Meeting
May 2013	AIHce 2013, Montreal, Quebec
May 2013	Engineering Controls to Minimize Fugitive Dust – Professional Development Course, Montreal, QB.
March 2013	AIHA BC Yukon Section – Annual General Meeting
October 2012	Ergonomics of Heavy Equipment Design and Whole Body Vibration, the University of British Columbia, Vancouver, BC
September 2012	Wood Dust Explosion Prevention Course, Dalhousie University, Richmond, BC
August 2012	Financial Success Training Part 1 and Part 2, Financial Decision Maker, Inc.
March 2012	Comprehensive Industrial Hygiene Review Course, Ann Arbor, Michigan
May 2011	Toxicology Professional Development Course, Portland, Oregon
May 2011	AIHce 2011, Portland, Oregon
March 2010	AIHA BC Yukon Section – Annual General Meeting
December 2009	BLDC 1500 BC Building Code: Part 9 (SFD)
March 2009	AIHA BC Yukon Section – Annual General Meeting
February 2009	Mold: Exploring Sampling, Analysis & Data Interpretation, AIHA Teleweb
December 2008	Certified Ergonomic Specialist (CES) Designation – E.K. Gillin and Associates Inc.
October 2008	Introduction to Industrial Hygiene and Industrial Hygiene Measurement and Monitoring – E.K. Gillin and Associates Inc.
April 2008	BLDC 3050 Building Envelope Performance – British Columbia Institute of Technology

#### AFFILIATIONS:

Since 2009	American Industrial Hygiene Association – BC Yukon Section (Member)
Since 2010	American Industrial Hygiene Association (Member)
Since 2011	Board of Canadian Registered Safety Professionals (Member)
Since 2012	American Board of Industrial Hygiene (Member)

## Appendix AA: Martyny et al. (2013) Potential Exposures Associated With Indoor Marijuana Growing Operations

*Journal of Occupational and Environmental Hygiene*, 10: 622–639  
ISSN: 1545-9624 print / 1545-9632 online  
Copyright © 2013 JOEH, LLC  
DOI: 10.1080/15459624.2013.831986

### Potential Exposures Associated with Indoor Marijuana Growing Operations

John W. Martyny,<sup>1</sup> Kate A. Serrano,<sup>3</sup> Joshua W. Schaeffer,<sup>2</sup>  
and Mike V. Van Dyke<sup>3</sup>

<sup>1</sup>Occupational Medicine, National Jewish Health, Denver, Colorado

<sup>2</sup>Department of Environmental Health Sciences, Colorado State University, Fort Collins, Colorado

<sup>3</sup>Department of Environmental and Occupational Health Sciences, National Jewish Health, Denver, Colorado

*We entered a total of 30 indoor marijuana grow operations (IMGO) with law enforcement investigators in order to determine potential exposures to first responders. Samples for airborne fungal spores, volatile organic compounds, carbon dioxide, carbon monoxide, and delta-9-tetrahydrocannabinol (THC) were obtained as well as the identification of chemicals utilized in the IMGO. The chemicals utilized within the IMGOs were primarily pesticides and fertilizers with none showing high toxicity. Although several of the IMGOs had CO<sub>2</sub> enrichment processes involving combustion, CO levels were not elevated. THC levels were identified on surfaces within the IMGOs and on the hands of the investigators. Surface levels ranged from <0.1 µg/100 cm<sup>2</sup> to 2000 µg/100 cm<sup>2</sup> with a geometric mean of 0.37 µg/100 cm<sup>2</sup>. THC levels on the hands of officers ranged from <0.10 µg/hwipe to 2900 µg/hwipe with a geometric mean of 15 µg/hwipe. These levels were not considered to be elevated to the point of causing a toxic exposure to responders. A total of 407 fungal spore samples were taken using both slit impactor plates and 400-hole impactors. Both methods identified elevated fungal spore levels, especially during the removal of plants from some of the IMGOs. After plant removal, spore counts increased to levels above 50,000 spores/m<sup>3</sup> with one sample over 500,000 spores/m<sup>3</sup>. In addition, we found that there was a shift in species between indoor and outdoor samples with *Cladosporium* sp. the predominant outdoor species and *Penicillium* sp. the predominant indoor species. We concluded that the potential increase in fungal spore concentrations associated with the investigation and especially removal of the marijuana plants could potentially expose responders to levels of exposure consistent with those associated with mold remediation processes and that respiratory protection is advisable.*

**Keywords:** fungal spores, tetrahydrocannabinol, carbon monoxide, pesticides, microbial growth, pesticides

Address correspondence to John Martyny, Occupational Medicine, National Jewish Health, 1400 Jackson St., Denver, CO 80206, USA; e-mail: johnmartyny@comcast.net

#### INTRODUCTION

In recent years, the United States and Canada have experienced an increase in the number of marijuana growing operations that are being conducted indoors. The National Drug Intelligence Center reported that indoor marijuana growing operation (IMGO) seizures of individual plants had increased from 203,896 in 2004 to 450,986 in 2008.<sup>(1)</sup> Florida was the state with the largest increase during that time with 21,879 plants seized from 246 sites in 2004 and 78,489 plants seized from 1,022 sites in 2008. In Ontario, Canada the number of IMGOs increased by at least 250% between 2000 and 2002 with 650 search warrants issued in 2001.<sup>(2)</sup>

There are several advantages of an IMGO compared to outdoor growing. Indoor growing operations allow for as many as six crop rotations per year, even in colder climates.<sup>(3)</sup> Indoor growth also enables the grower to control light, humidity, and temperature, which enables cloning of plants that have the highest levels of delta-9-tetrahydrocannabinol (THC).<sup>(3,4)</sup> Control of the day-night periodicity and carbon dioxide levels may also aid in producing a higher-quality product than can be grown outdoors.<sup>(3,4)</sup>

The disadvantages of an IMGO include inherent dangers and potential health hazards related to the amount of electrical power required for the lighting systems, carbon monoxide exposure from certain types of carbon dioxide generators, and mold and moisture produced by irrigation and plant growth in less than ideal ventilation conditions. Canadian law enforcement and fire departments report hazards such as electrocution, fire, carbon monoxide exposure, mold exposure, pesticide and chemical exposure, explosion hazards, and booby traps that present a danger to responding enforcement personnel.<sup>(2,5)</sup>

A major concern regarding IMGOs is the potential for significant mold growth within the IMGO. Production

requirements for marijuana include temperatures between 21°C and 32°C, with relative humidity between 50% to 70%.<sup>(4)</sup> In addition, ventilation rates are often suppressed to limit odors emanating from the concealed operation, and irrigation rates are high to maximize production.<sup>(3,4,6,7)</sup> A study of typical ventilation rates in Canadian housing led Miller and Johnson<sup>(8)</sup> to conclude that Canadian houses built after 1980 would be at high risk for moisture- and mold-related damage if used as IMGOs.

A number of health hazards have been associated with indoor mold growth. Johnson and Miller<sup>(6,8)</sup> conclude that increased moisture levels in IMGOs likely cause elevated mold spore levels that could be associated with allergic reactions and allergic bronchopulmonary aspergillosis as well as fungal infections. The Institute of Medicine (IOM) and World Health Organization (WHO) have indicated that the presence of mold in damp indoor environments is associated with upper respiratory symptoms, wheeze, cough, asthma symptoms in sensitized individuals, and hypersensitivity pneumonitis.<sup>(9,10)</sup>

This study was performed at the request of the Colorado Drug Investigators Association (CDIA) to answer the following questions:

- What are the potential exposures associated with the investigation of IMGOs in Colorado?
- What impact do evidence collection and seizure activities have on exposures to investigators in Colorado IMGOs?
- What personal protective equipment (PPE) is necessary to protect law enforcement and other responders involved in IMGO investigations?

## METHODS

Based on the potential exposures previously described, it was determined that we should monitor each IMGO for the following: volatile organic compounds (VOCs), carbon monoxide, oxides of nitrogen (NO<sub>x</sub>), fungal growth, and THC present in the air and on surfaces within the IMGOs. We were also interested in how levels of airborne fungal spores were affected by the activity of the investigators. To answer that question, fungal sampling was performed twice at IMGOs where plants were removed (pre-removal and post-removal).

IMGOs were identified by a liaison from CDIA. Typically, we met with law enforcement personnel at a location away from the IMGO until the operation was entered and secured. This usually took less than 30 minutes. We entered with the drug investigators and began to sample immediately. No removal operations were conducted until we had collected our baseline samples.

### Chemical Hazards

Potential chemical exposures at the IMGOs were determined utilizing two methodologies: gas chromatograph/mass spectrometer (GC/MS) air samples and a comprehensive inventory of the chemicals present within the IMGO. GC/MS samples were collected utilizing personal sampling pumps cal-

ibrated to a flow rate of 0.1 liters per minute with Carbotrap 300 tubes. Samples were collected at an outdoor location adjacent to the IMGO and inside the grow room. All of the samples were kept cold prior to sending them by overnight shipping to an American Industrial Health Association (AIHA)-accredited laboratory (ALS Laboratory Group, Salt Lake City, Utah) for analysis using EPA method TO-17.

In addition to the GC/MS samples, pictures were taken of the labels of all chemicals observed within the IMGO to allow easy identification when requesting Material Safety Data Sheets (MSDS) from the manufacturers. MSDS information was used to determine the potential health hazards and chemical constituents of the product.

### Carbon Dioxide and Carbon Monoxide Levels

Carbon dioxide and carbon monoxide levels as well as relative humidity and temperatures were measured using an indoor air quality meter (Q-Trak, TSI, Inc., Shoreview, Minn.). Readings were taken outside of the IMGO as well as in all of the growing rooms within the IMGO.

### THC Levels

THC levels were collected from both air and surface locations within the IMGO. Air samples were collected using a high flow personal sampling pump calibrated to a flow rate of 10 L per minute with 37-mm glass fiber filters. The surface samples were collected using a 10 cm × 10 cm cotton wipe on which approximately 2 ml of reagent grade methanol had been added. The wipe samples were collected using a side-to-side and up-and-down wiping method from surfaces using a 100 cm<sup>2</sup> template. Hand wipe samples were collected by asking investigating officers to wipe both of their gloves with a wetted wipe. Each sample was collected while wearing a clean pair of disposable nitrile gloves, and subsequently inserted into a plastic centrifuge tube. All samples were sent to an AIHA-accredited laboratory for analysis (ALS Laboratory Group, Salt Lake City, Utah).

### Fungal Spore Levels

Two methods were utilized to determine airborne fungal spore levels within the IMGOs. A 400-hole impactor (Standard BioStage, SKC, Inc., Eighty-Four, Pa.) was used to collect viable fungal spore samples; Air-O-Cell slit impactors were utilized to collect spores for direct microscopic analysis. The 400-hole impactors were connected to a high-flow sampling pump calibrated to a flow rate of 28.3 liters per minute and run for a period of two minutes. Slit impactors were connected to a high-flow sampling pump calibrated to 15 L per minute and run for a period of 10 minutes. For viable spores, a total of 4 agar plates were taken at each sampling location, two each with malt extract and DG-18 agars. Two slit impactor samples were also collected at each sampling location during the same sampling period as the viable samples.

Sample locations at each IMGO included: outdoors (adjacent to the structure) and in the grow room(s). In IMGOs where the plants were seized, duplicate rounds of samples

were taken at the same locations after removal of the plants and the equipment. The impactors were cleaned using 75% isopropanol wipes between sampling sites. All samples were shipped by overnight delivery to an AIHA-accredited laboratory for analysis (EMSL Laboratories, Cinnaminson, N.J.).

#### Statistical Analysis

Due to the nature of the sampling, descriptive statistics were utilized to analyze the data. Geometric means were utilized since industrial hygiene data are usually log-normally distributed, although these data appeared to be more normally distributed. It was also felt that geometric means would provide

a more conservative result less influenced by single elevated values. Standard deviations, geometric means, means, maximums, and minimums were calculated using Microsoft Excel.

#### RESULTS

A total of 30 IMGOs were sampled during this study (Table I). One instance there were 4 separate IMGOs in a 4-plex residential building and in a second instance there were 4 separate IMGOs in a commercial building. The rest of the IMGOs were in separate structures. Twenty-five of the IMGOs

TABLE I. Characteristics of the 30 IMGOs Investigated During the Study

IMGO #	Structure Type	Number of Plants	CO <sub>2</sub> Generation Method	Grow Room CO <sub>2</sub> Concentration (ppm)	Grow Room CO Concentration (ppm)	Grow Room Relative Humidity (%)
1	Residential 4-Plex <sup>A</sup>	117	None	NM	NM	NM
2	Residential 4-Plex <sup>A</sup>	77	None	NM	NM	NM
3	Residential 4-Plex <sup>A</sup>	58	Combustion	NM	NM	NM
4	Residential 4-Plex <sup>A</sup>	28	None	NM	NM	NM
5	Single-Family Home	160	Combustion	1500	0	60
6	Single-Family Home	65	None	765	0	36
7	Commercial Warehouse	670	CO <sub>2</sub> Tanks	485	0.8	67
8	Single-Family Home	232	None	732	0.1	73
9	Single-Family Home	52	CO <sub>2</sub> Tanks	459	0	41
10	Single-Family Home <sup>B</sup>	37	None	NM	NM	NM
11	Single-Family Home <sup>B</sup>	24	None	NM	NM	NM
12	Single-Family Home <sup>B</sup>	86	None	NM	NM	NM
13	Single-Family Home	28	None	801	0.3	74
14	Single-Family Home	30	Combustion	712	0.1	66
15	Single-Family Home	11	None	690	0	52
16	Single-Family Home	290	CO <sub>2</sub> Tanks	933	0	56
17	Commercial Office Bldg <sup>C</sup>	446	Combustion	591	0.1	36
18	Commercial Office Bldg <sup>C</sup>	323	Combustion	592	0.2	47
19	Commercial Office Bldg <sup>C</sup>	107	CO <sub>2</sub> Tanks	773	0.2	30
20	Commercial Office Bldg <sup>C</sup>	84	None	637	0.3	25
21	Single-Family Home	56	CO <sub>2</sub> Tanks	NM	NM	NM
22	Single-Family Home	NA	None	NM	NM	NM
23	Single-Family Home	188	CO <sub>2</sub> Tanks	713	0	39
24	Single-Family Home	75	None	1328	NM	40
25	Single-Family Home	64	None	750	0.1	23
26	Single-Family Home	100+	Combustion	1370	0	59
27	Single-Family Home	240	CO <sub>2</sub> Tanks & combustion	1196	0	69
28	Single-Family Home	236	CO <sub>2</sub> Tanks	586	0	35
29	Single-Family Home	84	CO <sub>2</sub> Tanks	553	0	47
30	Single-Family Home	168	Combustion	1295	0	94

<sup>A</sup>IMGOs in each unit of a 4-plex

<sup>B</sup>Three adjacent single family homes

<sup>C</sup>Four units in a single commercial office building

NM = not measured.

NA = not available.

were located in a residential structure with 24 of the 25 located in single-family structures.

Five of the IMGOs were located in two commercial buildings. As previously mentioned, one building had 4 separate IMGOs located within the building and the other commercial IMGO was located in a warehouse. The number of plants per IMGO ranged from 11 plants to 670 plants. Thirteen of the IMGOs had over 100 plants. The relative humidity within the IMGOs ranged from 23% to 94% with a mean level of 51%.

Sixteen of the operations tested had an observable method to increase carbon dioxide levels within the grow areas (Table I). Eight of these IMGOs utilized methodologies that involved fossil fuel combustion such as removing the vents from hot water heaters or the use of unvented propane or natural gas burners to increase the carbon dioxide levels. The other eight operations utilized carbon dioxide tanks in order to regulate carbon dioxide levels within the grow area. Carbon dioxide levels within the IMGOs ranged from 485 ppm to 1500 ppm but carbon monoxide levels were found to be at low levels, even when fossil fuel combustion was utilized as the carbon dioxide source.

#### Chemical Hazards

Samples for volatile organic compounds were collected at all of the sites. Since none of the IMGOs that we visited had been using any THC concentration or extraction techniques involving solvents, the presence of high concentrations of solvents was not expected. We detected a number of compounds associated with the smell that we characterize as the marijuana smell.<sup>(11)</sup> These compounds were found to be present in higher quantities (50 to 100 ppb) in the grow rooms and consisted of alpha-pinene, beta-myrcene, beta-pinene, and limonene.

Most of the chemicals observed at the IMGOs fell into one of two categories, pesticides or fertilizers. Most of the compounds observed did not appear to pose a substantial threat to short-duration exposures by law enforcement officers. Pesticides were primarily pyrethroids which have a relatively low toxicity.

#### THC Levels

All of the airborne THC levels were below the detection level (0.10 µg/sample) except for one sample (0.70 µg/sample) that was near the detection limit. A total of 102 wipes for THC were taken in the project. Eighty-one of the wipes were taken on surfaces within the IMGOs and 21 of the wipes were taken on the gloves worn by law enforcement officers conducting the investigations (Table II). The surface wipes ranged from non-detect (<0.10 µg/100 cm<sup>2</sup>) to a high of 2000 µg/100 cm<sup>2</sup> obtained at IMGO #26. The geometric mean of all of the surface wipe samples was 0.37 µg/100 cm<sup>2</sup>, which included the 2000 µg/100 cm<sup>2</sup> sample; however, the geometric mean was reduced to 0.32 µg/100 cm<sup>2</sup> if that result was dropped as an outlier. The THC levels measured on the hands of officers conducting the investigations ranged from non-detect (<0.10 µg/wipe) to 2900 µg/wipe with a geometric mean of 15 µg/wipe.

TABLE II. THC Levels on Surfaces and the Hands of Investigating Officers

IMGO #	Surface Samples (µg/100 cm <sup>2</sup> )	Officer Hand Samples (µg/sample)
1	0.31, 0.79	NM
2	16.0, 1.2	NM
3	0.61	NM
4	0.28, 0.34	NM
5	0.27, <0.10, 1.4	50
6	0.15, 0.29, 0.14, 0.14	<0.10
7	<0.10, 39.0, 0.83, 6.5, <0.10	11, 1.6
8	2.1, 2.0, 37.0, <0.10	NM
9	1.5, 4.5, 1.5, 0.54	<0.10
10	<0.10, <0.10, 0.45, <0.10	1.4, 1.4
11	<0.10, <0.10, <0.10, 0.46	NM
12	<0.10, <0.10, <0.10	NM
13	<0.10, <0.10, 1.9, <0.10	<0.10
14	NM	NM
15	<0.10, <0.10, <0.10	NM
16	0.76, 0.30, 0.13, 0.77	NM
17	0.80, 59	NM
18	0.49, 0.13	NM
19	NM	NM
20	3.90, 0.94, 0.29	NM
21	NM	NM
22	0.69, <0.10, <0.10	NM
23	<0.10, <0.10, <0.10	NM
24	0.48, 0.73, 0.38	180.0, 40.0
25	0.41, 0.10, 6.1, 0.10, <0.10	11
26	2000, 0.10, <0.10, 0.10	2.40, 5.8
27	43.0, 2.4	1100, 490
28	1.4, 3.2	150, 150
29	<0.10, 0.19	9.2, 120
30	<0.10, 1.1	2900, 1300

Note: NM = not measured.

#### Fungal Spore Levels

A total of 407 fungal spore samples were taken during the IMGO characterization phase (pre-removal phase) of the project. One hundred and eighty-two viable agar plate samples were taken in the indoor grow rooms prior to any removal of plants. Another 92 slit impactor samples were taken simultaneously with the viable samples. Ninety-one viable samples and 42 slit impactor samples were taken as outside controls.

Table III represents the results obtained from the viable fungal spore samples collected at the 30 IMGOs including both the malt extract and DG-18 agars. The range of viable colony counts in the outside air ranged from 72 cfu/m<sup>3</sup> to a high of 4030 cfu/m<sup>3</sup>. Only two IMGOs (#26 and #30) had outdoor viable colony counts exceeding 2,000 cfu/m<sup>3</sup>. For IMGO #26,

TABLE III. Pre-removal Viable Sample Results

IMGO #	Number of Outside/ Grow Room Samples	Outside Range (CFU/m <sup>3</sup> )	Outside Geo. Mean (CFU/m <sup>3</sup> )	Outside Most Prevalent Genera	Grow Room Range (CFU/m <sup>3</sup> )	Grow Room Geo. Mean (CFU/m <sup>3</sup> )	Grow Room Most Prevalent Genera	Ratio of Grow Room/ Outdoor Geometric Means	Ratio of Grow Room/ Outdoor Max.
1	8/4	144-414 <sup>A</sup>	310	Cladosporium sp - 68% Oldiodendrum sp - 7% Penicillium sp - 2%	522-1350	946	Cladosporium - 73% Oldiodendrum sp - 20% Penicillium sp - 2%	3.1	3.3
2	8/4	144-414 <sup>A</sup>	310	Cladosporium sp - 68% Oldiodendrum sp - 7% Penicillium - 2%	1190-2300	1675	Cladosporium sp - 53% Penicillium sp - 20%	5.4	5.5
3	8/4	144-414 <sup>A</sup>	310	Cladosporium sp - 68% Oldiodendrum sp - 7% Penicillium - 2%	486-1080	625	Cladosporium sp - 72% Penicillium sp - 12%	2	2.6
4	8/4	144-414 <sup>A</sup>	310	Cladosporium sp - 68% Oldiodendrum sp - 7% Penicillium - 2%	1640-2270	1954	Cladosporium sp - 89% Yeast - 4%	6.3	5.5
5	4/8	540-1260	890	Cladosporium sp - 81% Yeast - 8%	594-5330	1571	Cladosporium sp - 95% Penicillium sp - 7%	1.8	4.2
6	4/8	360-738	441	Cladosporium sp - 38% Aspergillus sp - 10% Alternaria sp - 10%	396-5870	895	Trichoderma sp - 49% Penicillium sp - 4%	2	8
7	4/10	144-270	183	Cladosporium sp - 31% Penicillium sp - 31% Aspergillus sp - 5%	612-1190	983	Cladosporium sp - 21% Penicillium sp - 93% Aspergillus sp - 2%	5.4	4.4
8	4/16	342-594	459	Cladosporium sp - 36% Yeast - 13%	1750 - > 11300	> 5644	Penicillium sp - 58% Aspergillus sp - 31% Cladosporium sp - 8%	> 12.3	> 19.0
9	4/4	486-1040	711	Cladosporium sp - 38% Penicillium sp - 13% Alternaria - 11%	1640-9790	2858	Penicillium sp - 79% Cladosporium sp - 18% Aspergillus sp - 1%	4	9.4
10	4/4	324-1130 <sup>F</sup>	589	Cladosporium sp - 54% Rhodotorula sp - 16% Penicillium sp - 15%	900 - 1080	947	Cladosporium sp - 59% Penicillium sp - 9% Epitococcus sp - 4%	1.6	0.9





TABLE III. Pre-removal Viable Sample Results (Continued)

IMGO #	Number of Outside/Grow Room Samples	Outside Range (CFU/m <sup>3</sup> )	Outside Geo. Mean (CFU/m <sup>3</sup> )	Outside Most Prevalent Genera	Grow Room Range (CFU/m <sup>3</sup> )	Grow Room Geo. Mean (CFU/m <sup>3</sup> )	Grow Room Most Prevalent Genera	Ratio of Grow Room/Outdoor Geometric Means	Ratio of Grow Room/Outdoor Max.
21	4/8	342-648	303	Cladosporium sp - 61% Penicillium sp - 10% Aspergillus sp - 8%	108-234	141	Cladosporium sp - 61% Penicillium sp - 10%	0.3	0.4
22	4/8	90-162	123	Cladosporium sp - 32% Alternaria sp - 7% Penicillium sp - 7%	198-1730	635	Aspergillus sp - 8% Cladosporium sp - 23% Penicillium sp - 7%	5.2	10.7
23	4/12	252-594	371	Penicillium sp - 46% Aspergillus sp - 4% Cladosporium sp - 38%	144->5920	>1601	Aspergillus sp - 4% Penicillium sp - 94%	>4.3	>9.9
24	4/8	198-684	365	Cladosporium sp - 26% Aspergillus sp - 7% Penicillium sp - 4%	4-1130	240	Cladosporium sp - 49% Penicillium sp - 31%	0.7	1.7
25	4/8	504-1190	759	Aspergillus sp - 4% Cladosporium sp - 68% Aspergillus sp - 7% Yeast - 7%	288 - >6430	>1623	Aspergillus sp - 5% Penicillium sp - 87% Cladosporium sp - 11%	>2.1	>5.4
26	4/8	2180-4030	2999	Penicillium sp - 84% Cladosporium sp - 8% Alternaria sp - 1%	1420 - >10836	>4873	Aspergillus sp - 1% Penicillium sp - 84% Cladosporium sp - 4%	>1.5	>5.0
27	3/4	252-756	388	Cladosporium sp - 66% Penicillium sp - 8% Alternaria sp - 5%	>5980 - >6890	>6405	Myxomycetes sp - 1% Penicillium sp - 84% Cladosporium sp - 10%	>16.5	>9.1
28	4/4	576-1240	836	Cladosporium sp - 68% Yeast - 8% Alternaria sp - 4%	846 - >6220	>1433	Aspergillus sp - 2% Cladosporium sp - 74% Aspergillus sp - 5%	>1.7	>5.1
29	4/4	72-468	240	Cladosporium sp - 72% Penicillium sp - 5% Epiloccum sp - 3%	630-1190	890	Penicillium sp - 9% Penicillium sp - 52% Cladosporium sp - 36%	3.7	2.5
30	4/4	190-3740	961	Penicillium sp - 24% Cladosporium sp - 2% Basidiomycetes - 1%	>3440 - >8410	>6506	Aspergillus sp - 5% Penicillium sp - 80% Cladosporium sp - 4%	>6.9	>2.3
Median <sup>b</sup>			549			>1625		>2.1	>4.3

<sup>a</sup>Outside samples taken outside 4-plex.

<sup>b</sup>Outside samples taken outside 3 adjacent single family homes.

<sup>c</sup>Outside samples taken outside of commercial building.

<sup>d</sup>Median is the rest of all 30 MGOs.

the outdoor sample was more consistent with the inside grow room samples in terms of dominant genera and was likely contaminated during the sampling effort due to the sampling location near the entrance to the IMGO. Grow room levels of viable colony counts were generally higher than outdoor levels with a median ratio of grow room geometric means to outside geometric means of 2.1. The actual ratios of the geometric means ranged from a minimum of 0.3 to a maximum of 16.5. Of the 30 IMGOs, 10 (33%) had indoor/outdoor geometric mean ratios greater than or equal to 5, 12 (40%) had ratios between 1 and 5, and 8 (27%) had ratios less than 1. A comparison of the ratio of the maximum viable colony counts from samples collected in the grow room to the maximum viable colony counts from samples collected outside

the IMGOs ranged from a minimum of 0.2 to a maximum of > 32.3 with a median of 4.3.

The data in Table III also suggest a shift in the dominant genera between the outside and inside samples for 43% (13) of the IMGOs including 30% (9) that shift from *Cladosporium*-dominant to *Penicillium*-dominant and 10% (3) that shift from *Cladosporium*-dominant to *Trichoderma*-dominant. Table IV provides the viable colony counts of *Penicillium sp.* observed in the outdoor samples compared to viable colony counts observed in the grow room samples. The outdoor levels ranged from non-detect (<9 cfu/m<sup>3</sup>) to a maximum of 3146 cfu/m<sup>3</sup>. Grow room samples ranged from non-detect to >5400cfu/m<sup>3</sup>. Even when IMGO #26 is included, the median outside *Penicillium sp.* viable colony count was only 30 cfu/m<sup>3</sup> as opposed to

TABLE IV. Pre-Removal Viable *Penicillium sp.* Colony Counts

IMGO #	Outside Range (cfu/m <sup>3</sup> )	Outside Gen. Mean (cfu/m <sup>3</sup> )	Grow Room Range (cfu/m <sup>3</sup> )	Grow Room Geo. Mean (cfu/m <sup>3</sup> )	Ratio of Grow Room/Outdoor Geometric Means	Ratio of Grow Room/Outdoor Maximums
1	18-36 <sup>A</sup>	25	9-36	18	0.7	1
2	18-36 <sup>A</sup>	25	324-1116	588	23.5	16.3
3	18-36 <sup>A</sup>	25	9-126	55	2.2	3.5
4	18-36 <sup>A</sup>	25	9-36	21	0.8	1.0
5	9-54	14	28-72	33	2.4	1.3
6	9-54	14	9-198	38	2.7	3.7
7	18-108	42	504-1670	905	21.5	15.5
8	9-342	32	108-9434	650	20.3	27.6
9	9-360	64	2390 - >5400	>4358	>70.9	>15
10	18-198 <sup>B</sup>	68	90-126	76	1.1	0.6
11	18-198 <sup>B</sup>	68	324-882	567	8.3	4.5
12	18-198 <sup>B</sup>	68	54-198	80	1.2	1.0
13	18-90	27	216-1670	456	16.9	18.6
14	72-272	102	255-396	328	3.2	1.5
15	9-18	11	54-126	101	9.2	7.0
16	9-18	11	54-342	104	9.5	19.0
17	9-9 <sup>C</sup>	9	18-108	34	3.8	12.0
18	9-9 <sup>C</sup>	9	9-54	20	2.2	3.0
19	9-9 <sup>C</sup>	9	72-252	124	13.8	28.0
20	9-9 <sup>C</sup>	9	36-126	77	8.6	14.0
21	18-90	42	9-72	21	0.5	1.7
22	9-36	15	9-234	33	2.2	6.5
23	9-72	15	36 - >5400	>881	>38.7	>75
24	36-180	87	9-396	112	1.3	2.2
25	9-36	13	36 - >5400	>554	>42.6	>150
26	2110-3146	2574	1188 - >5400	>3971	>1.5	>2.1
27	36-36	36	>5400	>5400	>150	>34.0
28	9-9	9	9-306	73	8.1	34.0
29	9-54	14	432-522	471	33.6	9.7
30	162 - 972	371	>5400	>5400	>14.6	>14.6
Median <sup>D</sup>		30		189.6	6.3	8.4

<sup>A</sup>Outside samples taken outside 4-plex.

<sup>B</sup>Outside samples taken outside 3 adjacent single family homes.

<sup>C</sup>Outside samples taken outside of commercial building.

<sup>D</sup>Median is the total of all 30 MGOs.

189.6 cfu/m<sup>3</sup> for the grow rooms. The ratios of *Penicillium sp.* viable colony counts in the grow room to the associated counts in the outside air ranged from 0.8 to 150.0 with a median value of 6.3. In fact, for 37% (11) of the IMGOs this ratio exceeded 10. The ratios for the sample maximums are similar and range from 1.0 to 150 with a median of 8.4.

The slit impactor samples also suggest an increase in the microscopic spore counts and types of spores at some of the IMGOs (Table V). The geometric means for the outdoor samples ranged from 93 spores/m<sup>3</sup> to 13,574 spores/m<sup>3</sup> with a median microscopic spore count of 589 spores/m<sup>3</sup>. The microscopic spore counts in the grow rooms ranged from a geometric mean of 98 spores/m<sup>3</sup> to 10,777 spores/m<sup>3</sup> with a median microscopic spore count of 836 spores/m<sup>3</sup>. The ratio of grow room microscopic spore count geometric means to outside microscopic spore count geometric means ranged from 0.1 to 21.5 with a median ratio of 1.2. The ratio of the maximum grow room concentration to the maximum outside concentration ranged from 0.1 to 105.5 with a median ratio of 1.6.

Similar to the viable fungal spore samples, the slit impactor samples appear to show a dominant spore type shift from *Cladosporium sp.* outdoors to *Aspergillus sp./Penicillium sp.* (Asp/Pen) in the grow rooms. Table VI shows the comparison of Asp/Pen spores identified in the grow rooms and the outside samples. The geometric mean of the outside Asp/Pen spore counts (89 spores/m<sup>3</sup>) were similar to the geometric mean of the grow room Asp/Pen spore counts (80 spores/m<sup>3</sup>). For Asp/Pen type spores, the median of the ratio of the grow room geometric mean spore count to the outside geometric mean was 5.1 and ranged from less than 1.0 to 170.5. The maximum grow room Asp/Pen spore type counts exceeded 24,000 spores/m<sup>3</sup> in 13% (4) of the IMGOs compared to a maximum outdoor level of only 2570 spores/m<sup>3</sup>.

#### Mold Levels During Removal

Plant removal by law enforcement occurred in only 10 of the 30 IMGOs. Table VII shows the data for the viable fungal spore samples collected from the sites in which removal occurred. Many of the sites had colony counts exceeding the laboratory's upper counting limit. The median of the geometric means from the outdoor samples was 574 cfu/m<sup>3</sup> while the median for the grow rooms was 1586 cfu/m<sup>3</sup> and the median geometric mean for the samples taken during removal was 5364 cfu/m<sup>3</sup>. The ratios of the removal geometric mean compared to the grow room geometric mean ranged from 0.7 to a maximum of 22. When compared to the geometric means from outside samples, the ratios ranged from a minimum of 1 to a maximum of 27. In 40% of the IMGOs where removal occurred, the geometric means exceeded 10.

The removal viable samples again indicated that the primary fungal genera appeared to be *Penicillium sp.* (Table VIII). Comparison of the geometric means for *Penicillium sp.* from the outside, grow rooms, and the grow rooms after removal were 15 cfu/m<sup>3</sup>, 513 cfu/m<sup>3</sup>, and 4325 cfu/m<sup>3</sup>, respectively. This indicates that removal of the marijuana plants by law enforcement results in a sizable increase in the number of air-

borne *Penicillium sp.* spores. The ratio of the grow geometric means of the *Penicillium sp.* colony counts to the geometric mean before plant removal ranged from 1.0 to 34. The ratio of the geometric mean of the *Penicillium sp.* colony counts after removal to the geometric mean in the outside air ranged from 2 to 415 with a median increase of 72.

Data in Table IX show that there was a sizable increase in the microscopic spore counts due to the removal process. The median of the geometric means for the outside samples was 542 spores/m<sup>3</sup> compared to 3241 spores/m<sup>3</sup> for the initial grow room samples and 28,600 spores/m<sup>3</sup> in the grow rooms after removal. The ratio of the geometric means of the microscopic spore counts for the grow rooms after and before removal ranged from 1 to 34. The ratio for the grow room geometric mean after removal to the initial outside samples ranged from 2 to 76 with a median of 12.3.

The increase in the concentration of the Pen/Asp spores due to plant removal is presented in Table X. The median of geometric means for the outside Pen/Asp spore counts was 94 spores/m<sup>3</sup>, for the grow rooms prior to plant removal it was 752 spores/m<sup>3</sup>, while after the removal, the median of the geometric means was 12,096. The ratios of geometric means for the removal samples to the geometric means of the samples from the grow room prior to plant removal ranged from 1.2 to 273. The ratios of the geometric means for the removal samples to the geometric means for the outside samples ranged from 16 to 1473. These data indicate that the removal of marijuana plants and equipment from an IMGO results in a major increase in both overall spore counts and counts of Pen/Asp type spores. In some instances, the increase is more than 100-fold.

#### DISCUSSION

There have been a number of recent reports indicating that the increase in IMGOs has the potential to expose first responders to a number of hazards.<sup>(2,4-6)</sup> Many law enforcement agencies, especially in Canada, have indicated that specific PPE must be worn to protect responders entering IMGOs.<sup>(2,5,12,13)</sup> This study was conducted to identify the hazards associated with IMGOs in Colorado and to suggest appropriate PPE for entry by law enforcement officers.

We entered 30 Colorado IMGOs that were mostly located in residential structures (83%) (Figure 1). We found that most of the chemicals utilized were low-toxicity fertilizers, pesticides, and growth enhancers (Figure 2). We also determined that airborne THC concentrations were not routinely observed inside IMGOs and that all VOC concentrations were well below levels that might be of concern for short-term exposures.

Surface THC levels were generally low (geometric mean of 0.37 µg/100-cm<sup>2</sup>) with only one sample at 2000 µg/wipe. The highest levels of THC were observed on the hands of officers involved in the removal of the plants from the IMGOs. The levels on their gloved hands ranged from <0.10 µg/wipe to 2900 µg/wipe. The typical marijuana joint contains approximately 0.5g to 1.0 g of THC with approximately 50% of the THC in a joint absorbed into the bloodstream.<sup>(14)</sup> Oral ingestion

**TABLE V. Pre-Removal Silt Impactor (Air-O-Cell) Results**

IMCO #	Number of Outside/Inside Samples	Outside Range (spores/m <sup>2</sup> )	Outside Geo. Mean (spores/m <sup>2</sup> )	Outside Most Prevalent Spore Types	Grow Room Range (spores/m <sup>2</sup> )	Grow Room Geo. Mean (spores/m <sup>2</sup> )	Grow Room Most Prevalent Spore Types	Ratio of Grow Room/Outdoor Geo. Means	Ratio of Grow Room/Outdoor Max.
1	1/1	241 <sup>A</sup>	NA	Cladosporium sp - 64% Asp/Pen - 17%	711	NA	Cladosporium sp - 91% Asp/Pen - 4%	NA	3
2	1/1	241 <sup>A</sup>	NA	Chaetomium sp - 6% Cladosporium sp - 64% Asp/Pen - 17%	1960	NA	Chaetomium sp - 2% Cladosporium sp - 75% Asp/Pen - 24%	NA	8.1
3	1/1	241 <sup>A</sup>	NA	Chaetomium sp - 6% Cladosporium sp - 64% Asp/Pen - 17%	1410	NA	Myxomycetes - 0.7% Cladosporium sp - 79% Asp/Pen - 17%	NA	5.9
4	1/1	241 <sup>A</sup>	NA	Chaetomium sp - 6% Cladosporium sp - 64% Asp/Pen - 17%	2860	NA	Myxomycetes - 2% Cladosporium sp - 96% Asp/Pen - 3%	NA	11.9
5	0/2	NA	NA	Chaetomium sp - 6% NA	1380-7610	3241	Epicoccum sp - 0.4% Cladosporium sp - 98% Asp/Pen - 1%	NA	NA
6	2/4	274-744	452	Cladosporium sp - 81% Epicoccum sp - 8% Alternaria sp - 4%	505-745	638	Cladosporium sp - 77% Pen/Asp - 4%	1.4	1
7	1/6	161	NA	Cladosporium sp - 39% Asp/Pen - 13% Alternaria sp - 13%	345-2090	183	Alternaria sp - 3% Asp/Pen - 75% Cladosporium sp - 14%	NA	7.4
8	2/8	295-816	491	Cladosporium sp - 49% Pen/Asp - 32% Ascospores - 9%	1960 - 45700	10296	Basidiospores - 3% Asp/Pen - 76% Chaetomium sp - 18%	21.0	56
9	2/4	1370-1570	1467	Cladosporium sp - 52% Ascospores - 24% Basidiospores - 13%	2670-4020	2924	Cladosporium sp - 1% Cladosporium sp - 72% Asp/Pen - 16% Ascospores - 3%	2.0	2.6
10	4/2	928-1050 <sup>B</sup>	589	Cladosporium sp - 54% Rhodotorula sp - 16% Pen/Asp - 15%	780-1020	892	Cladosporium sp - 61% Pen/Asp - 8% Alternaria sp - 11%	1.5	1

(Continued on next page)

TABLE V. Pre-Removal Silt Impactor (Air-O-Cell) Results (Continued)

IMGO #	Number of Outside/Inside Samples	Outside Range (spore/m <sup>3</sup> )	Outside Geo. Mean (spores/m <sup>3</sup> )	Outside Most Prevalent Spore Types	Grow Room Range (spores/m <sup>3</sup> )	Grow Room Geo. Mean (spores/m <sup>3</sup> )	Grow Room Most Prevalent Spore Types	Ratio of (Grow Room/Outdoor) Geo. Means	Ratio of Grow Room/Outdoor Max.
11	4/2	928-1050 <sup>a</sup>	589	Cladosporium sp - 54% Rhodotula sp - 16% Pen/Asp - 15%	471-597	530	Pen/Asp - 53% Cladosporium sp - 28% Myxomycetes - 6% Cladosporium sp - 58%	0.9	0.6
12	4/2	928-1050 <sup>a</sup>	589	Cladosporium sp - 54% Rhodotula sp - 16% Pen/Asp - 15%	465-512	488	Pen/Asp - 9% Epicoecum sp - 17% Asp/Pen - 39% Cladosporium sp - 24% Basidiospores - 26%	0.8	0.5
13	2/4	6690-8170	7393	Basidiospores - 66% Ascospores - 23% Cladosporium sp - 8%	653-2880	1654	Basidiospores - 46% Cladosporium sp - 31% Myxomycetes - 19% Basidiospores - 46% Cladosporium sp - 24%	0.2	0.4
14	2/2	3370-3970	3658	Myxomycetes - 54% Basidiospores - 16% Pen/Asp - 5%	189-369	261	Basidiospores - 46% Cladosporium sp - 31% Myxomycetes - 19% Basidiospores - 46% Cladosporium sp - 24%	0.1	0.1
15	2/2	5960-6190	6074	Basidiospores - 57% Cladosporium sp - 17% Ascospores - 12%	716-850	780	Basidiospores - 46% Cladosporium sp - 24% Alternaria sp - 7%	0.1	0.1
16	2/4	2240-3150	2666	Alternaria sp - 54% Cladosporium sp - 27% Ascospores - 6%	245-654	370	Myxomycetes - 31% Asp/Pen - 16% Alternaria sp - 16% Cladosporium sp - 36% Basidiomycetes - 36%	0.1	0.2
17	2/2	498-507 <sup>c</sup>	502	Basidiospores - 50% Ascospores - 27% Asp/Pen - 2%	0 - 464	NA	Basidiospores - 65% Pen/Asp - 14% Basidiospores - 65% Pen/Asp - 29%	NA	0.9
18	2/2	498-507 <sup>c</sup>	502	Basidiospores - 50% Ascospores - 27% Asp/Pen - 2%	84-274	152	Ascospores - 6% Pen/Asp - 79% Basidiospores - 13% Cladosporium sp - 3%	0.3	0.5
19	2/2	498-507 <sup>c</sup>	502	Basidiospores - 50% Ascospores - 27% Asp/Pen - 2%	323-344	333	Basidiospores - 47% Pen/Asp - 33% Ascospores - 4%	0.7	0.7
20	2/2	498-507 <sup>c</sup>	502	Basidiospores - 50% Ascospores - 27% Asp/Pen - 2%	139-175	156	Basidiospores - 47% Pen/Asp - 33% Ascospores - 4%	0.3	0.4

21	2/4	864-1270	1048	Cladosporium sp - 46% Pen/Asp - 25% Myxomycetes - 16% Myxomycetes - 31% Ascospores - 27%	70-140	98	Pen/Asp - 41% Cladosporium sp - 21% Basidiospores - 16% Cladosporium sp - 32% Alternaria sp - 7% Pen/Asp - 7%	0.1	0.1
22	2/4	273-274	274	Cladosporium sp - 15% Asp/Pen - 63% Cladosporium - 25% Basidiospores - 4%	662-2520	1185	Pen/Asp - 63% Cladosporium sp - 3% Myxomycetes - 2% Cladosporium sp - 54% Myxomycetes - 13% Asp/Pen - 11%	4.3	9.2
23	2/8	681-893	780	Pen/Asp - 51% Cladosporium sp - 41% Basidiospores - 4%	893-25200	6533	Pen/Asp - 63% Cladosporium sp - 3% Myxomycetes - 2% Cladosporium sp - 54% Myxomycetes - 13% Asp/Pen - 11%	8.4	28.2
24	2/4	168-710	343	Pen/Asp - 65% Cladosporium sp - 15% Myxomycetes - 11% Pen/Asp - 68%	365-1490	762	Cladosporium sp - 3% Myxomycetes - 2% Cladosporium sp - 54% Myxomycetes - 13% Asp/Pen - 11%	2.2	2.1
25	2/4	231-1270	542	Cladosporium sp - 17% Alternaria sp - 2%	245-134000	5185	Asp/Pen - 97% Cladosporium sp - 2% Myxomycetes - 0.2% Asp/Pen - 94% Cladosporium sp - 4% Myxomycetes - 1%	9.6	105.5
26	2/4	1350-2330	1774	Cladosporium - 96% Myxomycetes - 1%	5130-9820	6660	NA	3.8	4.2
27	2/4	136-246	176	Cladosporium sp - 2%	NA	NA	NA	NA	NA
28	2/2	1100-16600	13574	Cladosporium sp - 44% Epilobium sp - 15% Ascospores - 11% Pen/Asp - 91% Basidiospores - 8%	2010-2990	2452	Cladosporium sp - 68% Chaetomium sp - 24% Pen/Asp - 22% Asp/Pen - 64% Cladosporium sp - 26% Basidiomycetes - 4% Asp/Pen - 96% Cladosporium sp - 2%	0.2	0.1
29	2/2	77-112	93	Cladosporium sp - 0.4%	766-5210	1998	NA	21.5	46.5
30	2/2	1770-2990	2301	Cladosporium sp - 8%	10100-11500	10777	NA	4.7	3.9
Medhar <sup>d</sup>			589			836		1.2	1.6

<sup>a</sup> Outside samples taken outside 4-apr.

<sup>b</sup> Outside samples taken outside 3 adjacent single-family homes.

<sup>c</sup> Outside samples taken outside of commercial building.

<sup>d</sup> Median is the total of all 30 MGCs.

NA = not available.

TABLE VI. Pre-Removal Slit Impactor (Air-O-Cell) *Aspergillus* sp/*Penicillium* sp. (Asp/Pan) Results

IMGO #	Outside Range (cfu/m <sup>3</sup> )	Outside Geo. Mean (cfu/m <sup>3</sup> )	Grow Room Range (cfu/m <sup>3</sup> )	Grow Room Geo. Mean (cfu/m <sup>3</sup> )	Ratio of Grow Room/Outdoor Geometric Means	Ratio of Grow Room/Outdoor Maximums
1	42 <sup>A</sup>	NA	28	NA	NA	0.7
2	42 <sup>A</sup>	NA	478	NA	NA	11.4
3	42 <sup>A</sup>	NA	246	NA	NA	5.9
4	42 <sup>A</sup>	NA	97	NA	NA	2.3
5	2	NA	28-42	34	NA	21
6	63-105	81	148-1120	360	4.4	10.7
7	21	NA	2-42	16	NA	2
8	21	NA	190-1860	528	NA	88.6
9	359	NA	1080-40100	6967	NA	111.7
10	42-190 <sup>B</sup>	89	63-84	73	0.8	0.4
11	42-190 <sup>B</sup>	89	211-359	275	3.1	1.9
12	42-190 <sup>B</sup>	89	21-42	30	0.3	0.2
13	2-42	9	84-1460	345	38.3	34.8
14	43-356	124	2-105	14	0.1	0.3
15	106-106	106	21-63	36	0.3	0.6
16	2-42	9	42-84	61	6.8	3
17	2-21 <sup>C</sup>	6	2-63	11	1.8	4
18	2-21 <sup>C</sup>	6	21-84	42	7	4
19	2-21 <sup>C</sup>	6	274-2532	263	43.8	13.1
20	2-21 <sup>C</sup>	6	42-63	51	8.5	3
21	127-401	226	21-63	39	0.2	0.2
22	2-21	6	2-211	52	8.7	10
23	422-570	490	317-24900	2652	5.4	43.7
24	21-380	89	63-84	87	1	0.2
25	21-739	125	63-132000	3155	25.2	178.6
26	823-1560	1133	4260-9520	6174	5.4	6.1
27	42-211	94	Overloaded	NA	NA	NA
28	2-63	11	274-844	481	43.7	13.4
29	2-21	6	295-3550	1023	170.5	169.1
30	1770-2570	2132	9960-10800	10371	4.9	4.2
Median <sup>D</sup>		89		80	5.1	4.2

<sup>A</sup> Outside samples taken outside 4-plex.

<sup>B</sup> Outside samples taken outside 3 adjacent single family homes.

<sup>C</sup> Outside samples taken outside of commercial building.

<sup>D</sup> Median is the total of all 30 IMGOs.

NA = not available.

results in less absorption with blood concentrations reaching only about 25%-30% of the levels obtained by inhaling an equivalent dose in the smoke.<sup>(14,15)</sup> Occasional or naïve users may experience a "high" after absorbing as little as 2-3 mg of THC with frequent users needing higher doses to experience a "high."<sup>(13)</sup>

Based upon the highest level that we measured (2900 µg) on the gloved hands of a law enforcement officer, if the entire amount was ingested, the resultant dose would be about 30% or 870 µg of THC.<sup>(14)</sup> That level would be approximately 44% of the minimum dose necessary to result in a "high" in an adult. In addition, the amount of THC expected to be absorbed from a marijuana joint would be approximately 0.25 g (1/2 of

0.5 g THC/joint) or approximately 250,000 µg, which is nearly 100 times the dose from ingesting the entire amount of THC on the glove. Based on this information, we do not believe that the THC levels observed on the surfaces of the IMGOs or on the gloves of the law enforcement officers pose a significant health risk to those involved under normal conditions.

The principal health concern that we identified in the Colorado IMGOs was mold or moisture issues. While there is still uncertainty regarding the potential health effects of exposure to environments with mold or moisture issues, there have been two major evaluations of the current literature by the Institute of Medicine (IOM) in 2004<sup>(9)</sup> and WHO in 2009.<sup>(10)</sup> The report by the IOM indicated that the presence of mold in damp



**TABLE VII. Post-Removal Viable Sample Results**

IMGO #	Outside Range (CFU/m <sup>3</sup> )	Outside Geometric Mean (CFU/m <sup>3</sup> )	Grow Room Range (CFU/m <sup>3</sup> )	Grow Room Geometric Mean (CFU/m <sup>3</sup> )	Removal Range (CFU/m <sup>3</sup> )	Removal Geometric Mean (CFU/m <sup>3</sup> )	Ratio of Removal/Grow Room Geometric Means	Ratio of Removal/Outside Geometric Means
5	540-1260	890	594-5330	1571	1010-1400	1149	0.7	1
22	90-162	123	198-1730	635	1090->6840	>2255	3.6	18
23	252-594	371	144->5920	>1601	198 - >5690	>1598	1.0	4
24	198-684	365	4-1130	240	270 - >11300	>5290	22.0	14
25	504-1190	759	288->6430	>1623	>5800 - >5890	>5837	3.6	8
26	2180-4030	2999	1420->10836	>4573	>5400 - >5740	>5559	1.2	2
27	252-756	388	>5980->6890	>6405	>5890 - >6620	>6268	1.0	16
28	576-1240	836	846->6260	>1433	1710-3940	2541	1.8	3
29	72-468	240	630-1190	890	>5620-7810	>6552	7.4	27
30	180 - 3740	961	>5400->8410	>6606	>5400 - >5490	>5437	0.8	6
Median <sup>A</sup>		574		1586		5364	1.5	6.7

<sup>A</sup> Median is the total of all 10 MGOs.

indoor environments is associated with upper respiratory tract symptoms, wheeze, cough, asthma symptoms in sensitized individuals, and hypersensitivity pneumonitis.

The WHO study confirmed the associations reported in the IOM and added that indoor dampness-related agents are associated with asthma development, dyspnea, current asthma, and respiratory infections. WHO concludes the following: "Microbial growth may result in greater numbers of spores, cell fragments, allergens, mycotoxins, endotoxins, beta-glucans, and volatile organic compounds in indoor air. The causative agents of the adverse health effects have not been identified conclusively, but an excess level of any of these agents in the indoor environment is a potential health hazard."<sup>(16)</sup> There are

multiple lines of evidence that indicate that there was increased microbial growth in many of the IMGOs evaluated in this study.

During initial law enforcement investigation activities, both viable and microscopic spore sampling identified elevated fungal spore levels in marijuana grow rooms as compared to outdoor levels. Forty-percent (12) of the IMGOs had at least five-fold higher grow room levels of viable airborne fungal colonies or countable airborne spores as demonstrated by the ratios of the geometric means of the grow room and outdoor concentrations. This type of increased fungal spore level as compared to outdoors is not a common finding in U.S. buildings. In fact, it is generally accepted that the concentration of fungal spores in indoor air is typically similar or lower than outdoor air.<sup>(16)</sup>

**TABLE VIII. Post-Removal Viable *Penicillium* sp. Colony Counts**

IMGO #	Outside Range (CFU/m <sup>3</sup> )	Outside Geometric Mean (CFU/m <sup>3</sup> )	Grow Room Range (CFU/m <sup>3</sup> )	Grow Room Geometric Mean (CFU/m <sup>3</sup> )	Removal Range (CFU/m <sup>3</sup> )	Removal Geometric Mean (CFU/m <sup>3</sup> )	Ratio of Removal/Grow Room Geometric Means	Ratio of Removal/Outside Geometric Means
5	9-54	14	28-72	33	18-144	74	2.2	5
22	9-36	15	9-234	33	648->5400	>1123	34.0	75
23	9-72	15	36->5400	>881	90->5400	>1031	1.2	69
24	36-180	87	9-396	112	162->5420	>3457	30.9	40
25	9-36	13	36 - >5400	>554	>5400->5400	>5400	9.7	415
26	2110-3146	2574	1188->5400	>3971	>5400->5400	>5400	1.4	2
27	36-36	36	>5400->5400	>5400	>5400->5400	>5400	1.0	150
28	9-9	9	9-306	73	486-1220	780	10.7	87
29	9-54	14	432-522	471	4900->5400	>5193	11.0	371
30	162 - 972	371	>5400 - >5400	>5400	>5400 - >5400	>5400	1.0	15
Median <sup>A</sup>		15		513		4325	6	72

<sup>A</sup> Median is the total of all 10 MGOs.

**TABLE IX. Post-Removal Slit Impactor (Air-O-Cell) Results**

IMGO #	Outside Range (Spores/m <sup>3</sup> )	Outside Geometric Mean (Spores/m <sup>3</sup> )	Grow Room Range (Spores/m <sup>3</sup> )	Grow Room Geometric Mean (Spores/m <sup>3</sup> )	Removal Range (Spores/m <sup>3</sup> )	Removal Geometric Mean (Spores/m <sup>3</sup> )	Ratio of Removal/ Grow Room Geometric Means	Ratio of Removal/ Outside Geometric Means
5	NA	NA	1380-7610	3241	2080	NA	NA	NA
22	273-274	274	662-2520	1185	1970-7090	3653	3.1	13
23	681-893	780	893-25200	6533	5440-15900	8849	1.4	11
24	168-710	345	365-1490	762	7240->82300	26144	34.3	76
25	231-1270	542	245-134000	5785	3250-4310	3743	0.7	7
26	1350-2330	1774	5130-9820	6660	19700-534000	113255	17.0	64
27	126-246	176	NA	NA	NA	NA	NA	NA
28	11100-16600	13574	2010-2990	2452	28600	28600	11.7	2
29	77-112	93	766-5210	1998	190	190	0.1	2
30	1770-2990	2301	10100-11500	10777	107000-136000	120632	11.2	52
Median <sup>A</sup>		542		3241		2860	7.1	12.3

<sup>A</sup> Median is the total of all 10 MGOs.  
 Note: NA = not available.



**FIGURE 1.** IMGO located in a basement of a residential structure. (color figure available online)

More quantitative comparisons can be made by comparing the data from IMGOs to the work of Shelton et al.<sup>(17)</sup> examining air samples from over 1700 U.S. buildings. Shelton et al.<sup>(17)</sup> determined that the ratios of the median indoor culturable fungal spore concentrations compared to the median outdoor concentrations were 1:1 or lower for 85% of the buildings and 2.8 or lower for 95% of the buildings. Fourteen of the ratios of the geometric means of grow room to outdoor culturable fungal spore concentrations were above the 2.8 ratio. In terms of culturable colony counts, Shelton et al.<sup>(17)</sup> determined that the median indoor fungal concentration was approximately 80 cfu/m<sup>3</sup> and 95% of the buildings tested had a median concentrations less than 1,300 spores/m<sup>3</sup>. The geometric means from viable fungal spore samples in the grow rooms ranged from 355 spores/m<sup>3</sup> to 6606 spores/m<sup>3</sup> and 37% (11) of the IMGOs had geometric mean grow room concentrations exceeding 1300 spores/m<sup>3</sup>.

There was a clear shift in the dominant fungal genera from *Cladosporium sp.* outdoors to *Penicillium sp.* in the grow rooms that is consistent with indoor fungal colonization. Fifty-seven percent (17) of the IMGOs had at least five-fold higher grow room levels of viable *Penicillium sp.* or *Aspergillus/Penicillium* type spores demonstrated by the ratios of the geometric means of the grow room and outdoor concentrations. In homes and buildings not impacted by water damage or visible mold, two studies have demonstrated the similarity between indoor and outdoor fungal spore samples both in terms of numbers of spores or colonies and in terms of the genera of fungi present, suggesting this genera shift is not common in the absence of indoor mold growth.<sup>(18,19)</sup>

Other studies involving moldy buildings support that this shift to a higher proportion of *Aspergillus/Penicillium*-type spores in indoor samples is indicative of indoor mold growth.<sup>(19,20)</sup>

The removal of marijuana plants and equipment from IMGOs increased the levels of airborne fungal spores. After removal operations, airborne spore concentrations increased up to 34-fold with a median ninefold increase compared to baseline conditions. The difference in airborne spore levels was more dramatic compared to outdoor air with a median 53-fold increase. Geometric mean spore concentrations ranged from 190 spores/m<sup>3</sup> to 113,255 spores/m<sup>3</sup> with a median level of 28,600 spores/m<sup>3</sup>. The median level of viable fungal spores was >5363 cfu/m<sup>3</sup> after removal operations which underestimates the true median due to the overloading of many of the culture plates. Concentrations in grow rooms after removal are within the range of those observed by Rautiala et al.<sup>(21)</sup> during repair work on moldy buildings in which viable airborne fungal spore levels ranged from 1,000 cfu/m<sup>3</sup> to >190,000 cfu/m<sup>3</sup> with an average of 23,000 cfu/m<sup>3</sup>. However, the grow room removal concentrations are lower than the 2,200,000 spores/m<sup>3</sup> and 32,000,000 spores/m<sup>3</sup> those measured by Morey and Hunt<sup>(22)</sup> during demolition of buildings with severe mold contamination.

Based on the information obtained during our study, we believe that the indoor growing of marijuana has a significant potential to increase the fungal spore levels to levels that may result in adverse health conditions to unprotected individuals. While there is still uncertainty regarding the health effects of mold exposure, there is unanimous consensus by all

TABLE X. Post-Removal Slit Impactor (Air-O-Cell) *Aspergillus sp./Penicillium sp.* (Asp/Pen) Results

IMGO #	Outside Range (Spores/m <sup>3</sup> )	Outside Geometric Mean (Spores/m <sup>3</sup> )	Grow Room Range (Spores/m <sup>3</sup> )	Grow Room Geometric Mean (Spores/m <sup>3</sup> )	Removal Range (Spores/m <sup>3</sup> )	Removal Geometric Mean (Spores/m <sup>3</sup> )	Ratio of	Ratio of
							Re- moval/ Grow Room Geometric Means	
5	2	NA	28-42	34	0	NA	NA	NA
22	2-21	6	2-211	52	1010-5070	2234	43.0	372
23	422-570	490	317-24900	2652	4120-15400	7991	3.0	16
24	21-380	89	63-84	87	6040->82300	23780	273.3	267
25	21-739	125	63-132000	3155	3250-4310	3743	1.2	30
26	823-1560	1133	4260-9520	6174	18300-534000	110945	18.0	98
27	42-211	94	2-2	2	Ovl	NA	NA	NA
28	2-63	11	274-844	481	16200	16200	33.7	1473
29	2-21	6	295-3550	1023	21	NA	NA	NA
30	1770-2570	2132	9960-10800	10371	107000-135000	120187	11.6	56
Median <sup>A</sup>		94		752		12096	18.0	97.9

<sup>A</sup> Median is the total of all 10 MGOs.

Note: OVL = overloaded;

NA = not available.

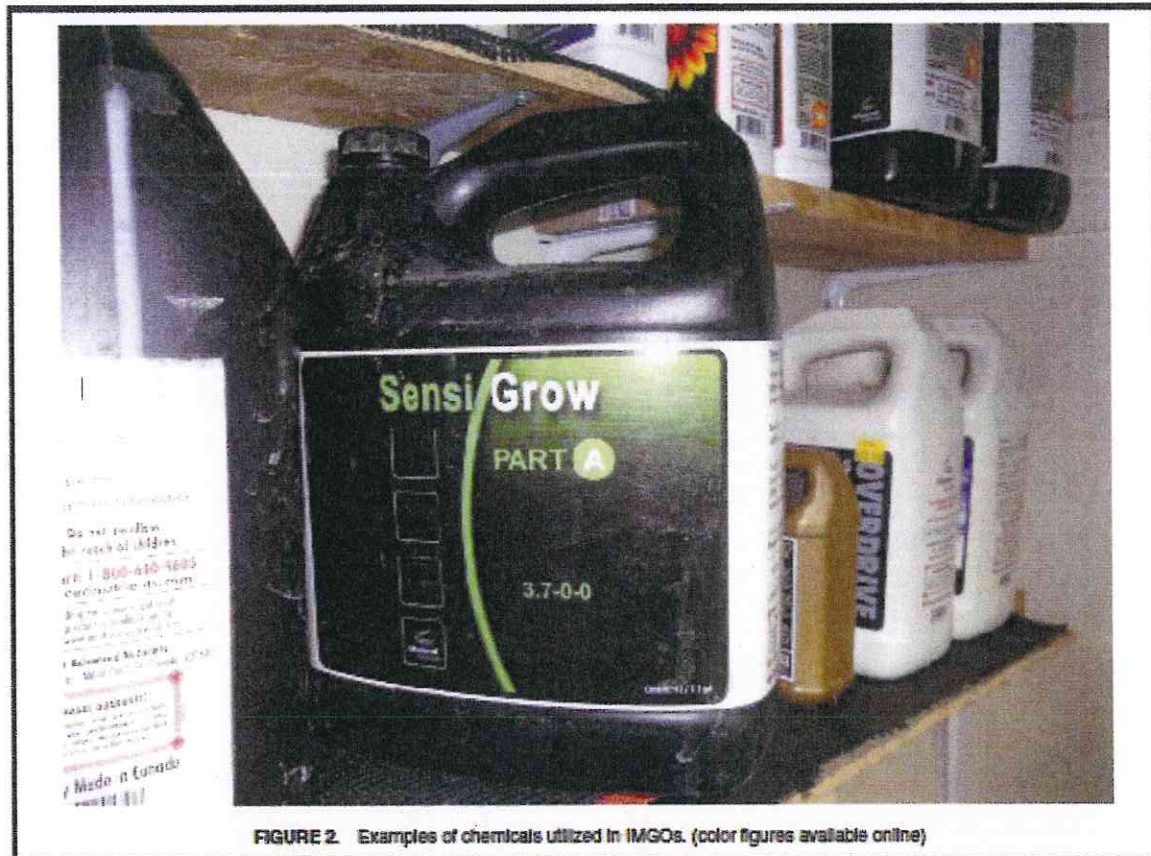


FIGURE 2. Examples of chemicals utilized in IMGOs. (color figures available online)

frequently cited mold remediation guidelines that respiratory protection is recommended for even small mold remediation jobs.<sup>(4,8,16,23)</sup> Therefore, it is reasonable to recommend similar (PPE) for law enforcement officers. It is also feasible that exposure levels from IMGO plant removal could account for the symptoms currently being reported by these officers.<sup>(22)</sup>

### CONCLUSION

There are a number of physical and safety hazards that have the potential to cause acute injury to law enforcement personnel entering IMGOs. In terms of health hazards, our results indicate that the primary hazard of concern to these officers is the potential for exposure to airborne fungal spores produced by indoor mold growth. During marijuana plant removal operations, these exposures are significantly increased and are consistent with those that would be experienced in mold remediation activities. Although there is a possibility of exposure to pesticides and carbon monoxide as reported by some Canadian authorities,<sup>(2)</sup> we did not observe similar exposures in the 30 IMGOs we investigated. However, we did frequently observe combustion-powered equipment, used to

increase carbon dioxide concentrations, that has the potential to increase carbon monoxide exposures.

The levels of fungal spores present within the IMGOs are frequently consistent with those considered to be indoor air quality issues and associated with health effects. It is our recommendation that law enforcement investigators take steps to protect themselves against these exposures including the use of filtering facepiece N-95 respirators if extended time periods are spent in an IMGO. Fungal spore exposures during marijuana plant removal appear to be consistent with exposures during medium-size mold remediation jobs. For removal operations, we recommend that proper respiratory, skin, and eye protection be worn as outlined in current mold remediation guidelines published by the U.S. Environmental Protection Agency<sup>(8)</sup> Office of the Fire Commissioner, Canada,<sup>(25)</sup> and AIHA.<sup>(4)</sup>

### ACKNOWLEDGMENTS

This project was supported by Grant No. 2010-DJ-BX-0316 awarded by the Bureau of Justice Assistance, a component of the U.S. Department of Justice's Office of Juvenile Justice and Delinquency Prevention and the Office for

Victims of Crime. The points of view or opinions expressed in the document are those of the author and do not represent the official position or policies of the Department of Justice. This project was also supported by grants from the Colorado Chiefs of Police and the Association of Colorado County Sheriffs. The Colorado Drug Investigators Association and the North Metro Task Force in Denver, Colorado, were instrumental in providing IMGs in which to conduct the sampling as well as personnel to maintain security and assistance. Commander Jerry Peters and Sergeant Jim Gerhardt of the North Metro Task Force and the City of Thornton Police Department were instrumental in coordinating the project and providing law enforcement knowledge to the project.

## REFERENCES

1. National Drug Intelligence Center (NDIC): Domestic Cannabis Cultivation Assessment. U.S. Department of Justice. Product #2009-1.0848-001A. July 2009.
2. La Barge, A.P., and K. Neakes: Indoor marijuana growing operations. *The Police Chief* 72(3):28-31 (2005).
3. Melaren, J., W. Swift, P. Dillon, and S. Ailsop: Cannabis potency and contamination: A review of the literature. *Addiction* 103:1100-1109 (2008).
4. Koch, T.D., C. Chambers, S. Buchert, J. Martyny, J. Cotner, and S. Thomas: *Clandestine Indoor Marijuana Grow Operations—Recognition, Assessment, and Remediation*. Fairfax, VA: American Industrial Hygiene Association, Fairfax, VA, 2010.
5. Alberta Government: *Clandestine Drug Operations, Awareness and Safety*, 2nd ed. Edmonton, Alberta, Canada: Office of the Fire Commissioner, 2006.
6. Johnson, L.L., and J.D. Miller: Consequences of large-scale production of marijuana in residential buildings. *Indoor Built Environment* 21: 595-600 (2011).
7. Canada Mortgage and Housing Corp.: *A Discussion Paper on Indoor Air Quality Investigations of Houses Used for Marijuana Grow Operations, Technical Series 07-101*. Ottawa, ON, Canada: Canada Mortgage and Housing Corp., 2007.
8. U.S. Environmental Protection Agency (EPA): *Mold Remediation in Schools and Commercial Buildings*, EPA# 402-K-01-001. Washington, D.C.: EPA, September, 2008.
9. Institute of Medicine: *Damp indoor spaces and health*. Washington, D.C.: National Academies Press, 2004.
10. World Health Organization (WHO): *WHO Guidelines for Indoor Air Quality: Dampness and Mold*. Copenhagen: WHO Regional Office for Europe, 2009.
11. Hood, L.V., M.E. Dames, and G.T. Barry.: Headspace volatiles of marijuana. *Nature* 242(5397):402-403 (1973).
12. Arizona Department of Public Safety: *Officer Safety Information: New Guidelines for Indoor Grow Operations*. Arizona Counter-Terrorism Information Center, Phoenix, AZ: Arizona Department of Public Safety, WMD Hazmat Section, SO9006. April 2007.
13. Royal Canadian Mounted Police: *National Conference: Illegal Marijuana Grow Operations: Post Conference Report*, 2004.
14. Hall, W., and N. Sotowit: Adverse effects of cannabis. *Lancet* (352):1611-1616 (1998).
15. Ashton, C.H.: Pharmacology and effects of cannabis: A brief review. *Brit. J. Psychiatry* 178:101-106 (2001).
16. Macher, J. (ed.): *Bioaerosols: Assessment and Control*. (Chapter 7, pg. 6). Cincinnati, Ohio: ACGIH® 1999.
17. Shelton, B.G., K.H. Kirkland, W.D. Flanders, and G.K. Morris: Profiles of airborne fungi in buildings and outdoor environments in the United States. *Appl. Environ. Microbiol.* 68(4): 1743-1753 (2002).
18. Codina, R., R.W. Fox, R.F. Lockey, P. DeMauro, and A. Baggi: Typical levels of airborne fungal spores in houses without obvious moisture problems during a rainy season in Florida, USA. *J. Investig. Allergol. Clin. Immunol.* 18(3): 156-162 (2008).
19. Lee, T., S.A. Grinshpun, D. Martuzevicius, A. Athakari, C.M. Crawford, and T. Reponen: Culturability and concentration of indoor and outdoor airborne fungi in six single-family homes. *Atmos. Environ.* 40(16):2902-2910 (2006).
20. Solomon, G.M., M. Hjelmsroos-Kuski, M. Rotkin-Ellman, and K.S. Hammond: Airborne mold and endotoxin concentrations in New Orleans, Louisiana after flooding, October through November 2005. *Environ. Health Perspect.* 114(9): 1381-1386.
21. Rautiainen, S., T. Reponen, A. Hyvarinen, A. Nevalainen, T. Husman, A. Vehvilainen, and P. Kalliohokski: Exposure to airborne microbes during the repair of moldy buildings. *Am. Ind. Hyg. Assoc. J.* 57(3):279-284 (1996).
22. Morey, P. and S. Hunt: Mold contamination in an earthquake damaged building, in *Proceedings of Healthy Buildings* 95:1377-1380. (1995)
23. D'Andrea, C. (ed.): *Guidelines on Assessment and Remediation of Fungi in Indoor Environments*. Bureau of Environmental and Occupational Disease Epidemiology, New York City Department of Health and Mental Hygiene, January, 2002.

# Appendix AB: Moller et al. (2011) Examining the Health and Drug Exposures Among Canadian Children Residing in Drug Producing Homes

ORIGINAL  
ARTICLES

www.jpeds.com • THE JOURNAL OF PEDIATRICS

## Examining the Health and Drug Exposures among Canadian Children Residing in Drug-Producing Homes

Monique Moller, MSc, Gideon Koren, MD, Tatyana Karaskov, MD, and Facundo Garcia-Boumissen, MD

**Objective** To examine the health and well-being of children residing in residences where drug production is occurring.

**Study design** Starting in January 2006, children identified by police and the Children's Aids Society in the York region of Ontario, Canada, were referred to the Motherisk Program at the Hospital for Sick Children for pediatric assessment of their general health and well-being, with specific focus on illicit-drug exposure. We used a standard protocol to collect all available medical and environmental history, conducted physical and neurologic examinations, and collected hair for analysis of illicit drugs.

**Results** In total, 75 children, at the mean age of 6.5 years, were referred to us after being removed from homes where marijuana was grown (80%) or other operations linked to drug production were occurring (20%). Overall, rates of health issues in this cohort fell below reference values for Canadian children. Of the hair tests, 32% were positive for illicit substances. In the majority there were no clinical symptoms related to these drugs.

**Conclusion** The majority of children removed from drug-producing homes were healthy and drug free. Comprehensive evaluations should be performed on a case-by-case basis in order to determine what is ultimately in the best interest of the child. (*J Pediatr* 2011;159:766-70).

See editorial, p 710

**P**roduction of illicit substances in residential homes poses public health concerns.<sup>1</sup> In Ontario, as many as 15 000 illegal drug-producing homes existed in 2002, a 250% increase from 2000 estimates.<sup>2</sup> In 2004, approximately 60 clandestine synthetic drug laboratories were seized in Canada.<sup>3</sup>

Children may be present in homes where illegal drug operations occur because their families are involved in the operation or because they act as "crop sitters" to conceal it by adding a thread of legitimacy to the residence. With an estimated 10 000 children residing in such homes between 2000 and 2003 in Ontario,<sup>2</sup> police and the Children's Aid Society have been concerned about the associated risks for these children. In 2006, the Motherisk clinic at the Hospital for Sick Children was asked by police and the Children's Aid Society to develop a program to follow these children and to assess their health and well-being, with a focus on the risks to children in drug-producing homes of environmental exposure to drugs.

The risks associated with a clandestine methamphetamine laboratory differ greatly from those associated with an illegal drug-producing home where the child is exposed primarily to plants.<sup>4</sup> These compounds are produced by using a variety of industrial and pharmaceutical chemicals, often in make-shift "laboratories." Risks associated with these types of facilities include the potential for explosions and for contact with or ingestion of irritating and caustic chemicals (and drugs), a serious toxicologic threat.<sup>4,5</sup>

In contrast, marijuana-growing operations usually contain, aside from the plants themselves, chemicals such as fertilizers and, more rarely, insecticides. In large quantities, these chemicals might be harmful but pose little risk for explosion or similar, more immediate dangers. These risks may not be significantly different from those affecting children residing in places in which farming or other horticultural activities take place.<sup>7,8</sup> Other risks usually associated with marijuana-growing operations are related to housing modifications, such as illegal electric connections and issues associated with living in a closed, humid environment such as mold overgrowth in the interior walls, which have the potential to cause respiratory and allergy-related illnesses.<sup>2,9</sup>

Hair testing has become a useful tool in substance-abuse monitoring and in determining exposure to environmental contaminants.<sup>10</sup> Hair can offer a chronological time frame of exposure in addition to being a stable matrix and a specimen that can be sampled noninvasively.<sup>10</sup> Use of hair testing to identify exposure to drugs of abuse is widespread in workplace testing but has also been used to study exposure to drugs present in the environment.<sup>11-13</sup>

From the Department of Pharmacology and Toxicology, Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada (M.M., G.K.) and the Motherisk Program, Division of Clinical Pharmacology and Toxicology, Hospital for Sick Children, Toronto, Ontario, Canada (M.M., G.K., T.K., F.G-B).

Supported by a grant from the Canadian Institutes for Health Research. Funding was received from the Ontario Graduate Scholarship Program and University of Toronto Fellowships (to M.M.); Lecturer, Hospital for Sick Children, Ivay Chuk in Molecular Toxicology, University of Western Ontario (to G.K.); Children Scientist Training Program, Hospital for Sick Children (to F.G-B). The authors declare no conflicts of interest.

0022-3426/\$ - see front matter. Copyright © 2011 Mosby Inc. All rights reserved. 10.1016/j.jpeds.2011.02.044

MEMA 3,4-Methylenedioxymethamphetamine

766

The objective of the present study was to evaluate the cohort of children examined by Motherisk clinicians after having been removed from drug-producing homes in the greater Toronto area and referred for evaluation by child welfare authorities.

## Methods

Since January 2006, children from drug-growing homes in the Toronto area identified by police and the Children's Aid Society were referred to the Motherisk Program at the Hospital for Sick Children for assessment of their general health and well-being, with specific focus on exposure to illicit drugs. Upon discovering children in these dwellings, police routinely called the Children's Aid Society, which removed them immediately from their homes and families. We were then asked to examine the children to assess their health and well-being. The Motherisk Program is a counseling and follow-up program for families that focuses on safety and the risk for drug and chemical exposure during pregnancy and lactation. In addition to counseling and follow-up, the program has a large analytic laboratory that measures drugs of abuse and alcohol in hair and meconium.

The children were accompanied to our clinic by child protection workers with or without their parents. A standard protocol was followed in the evaluation of these children (Appendix; available at [www.jpeds.com](http://www.jpeds.com)); it included demographic details, drug contexts, medical histories, and school histories of the children; child development and neurologic examination; toxicology-related evaluation; in-clinic examinations; and hair analysis for drugs of abuse.

Hair tests for drugs of abuse were conducted by the Motherisk laboratory, employing validated analytic tests that we use routinely.<sup>11-14</sup> Results of the hair tests were not known at the time of the clinical assessments. In adolescents, we also inquired about the possibility of their own use of recreational drugs.

Descriptive statistics were used to characterize the studied cohort. ORs were calculated when appropriate with 95% CI. The study was approved by the Hospital for Sick Children's Research Ethics Board.

## Results

The mean age was 6.5 years (range 2 months to 15 years). Among marijuana-growing homes, less than 25% (9/37) had 1 child; the majority being inhabited by families of multiple children (>75%, or 28/37). Of the children who came to the consultation, 26 (34.7%) were accompanied by somebody other than their parents because custody had been taken away from the parents, at least temporarily. Of the children, 45 (60%) were of Asian ethnicity (Chinese or Vietnamese), 28 (37.3%) were Caucasian, and 2 (2.7%) were Hispanic. These numbers are distinctly different from the ethnic makeup of Toronto (>70% Caucasian and <20% Asian). Asian children found in marijuana-growing houses (n = 41) were more likely to come to the consultation accompanied by somebody other

than their parents than were the Caucasian children (n = 18) (OR 3.3; 95% CI; 1.04-11.1; P = .04).

## Home Environment

In total, 75 children (from 46 different homes) were assessed between January 2006 and January 2010. The majority of these homes (39/46, 80%) were marijuana-growing operations or homes where large quantities of marijuana were found. The remaining homes were engaged in cocaine (1/46) or amphetamine (3,4-methylenedioxymethamphetamine [MDMA]) production (2/46) or were homes in which multiple drugs were being produced or stored, including marijuana, cocaine, MDMA, and heroin (2/46). The median number of plants found in marijuana-growing houses for which information was available (n = 38) was 326 (95% CI; 200-350; range 20-675).

One marijuana-growth operation in which 2 children resided contained unspecified weapons. In one methamphetamine lab, explosives and hazardous chemicals were in close proximity to the children's play area. In 4 homes, at least one adult (parent or step-parent) was using or abusing the drugs produced. Finally, for one home, the written report specified "horrendous living conditions," although no further details were provided. The remaining 39 homes were not reported to be associated with any significant structural problems or safety issues.

## Health of Assessed Children

Most of the children were in good health at the time of assessment. School-age children attended school at grades appropriate for their ages. In all cases, parents reported anxiety and apprehension in the children after being removed from their families. An array of mostly minor health issues were detected in some children, including respiratory, speech/language, and developmental pathologies (Table I). Of those children, 3 (4%) had mild eczema; 3 (4%) had asthma; 1 suffered mild allergies; 3 (4%) had experienced recent respiratory infections, one of whom had pneumonia. Of the children, 5 (6.6%) had developmental issues, including attention deficit disorder, speech impairment, learning problems, and minor autistic tendencies.

Four children were overweight. In the home in which conditions were described as "horrendous," the 2 children were found to be small for their age (weights 10<sup>th</sup> and below 3<sup>rd</sup> percentile, heights 25<sup>th</sup> and 5<sup>th</sup> percentiles, respectively). All other 69 children (92%) were deemed to be growing at the appropriate rates for their ages.

Fifteen children had incomplete medical histories as the result of the absence of their parents at these assessments, which prevented the obtainment of full pediatric histories and necessitated the reliance on written reports and interviews with child workers and the children themselves.

## Toxicology Hair Test Results

Of the 75 children assessed, 72 underwent hair tests and 3 did not have sufficient hair; 24 (33%) tested positive for at least 1 substance, and 4 tested positive for more than 1 (Table II). The

Table I. The occurrences of identified health issues in the cohort of assessed children apprehended from drug-producing homes compared with the values found in respective Canadian reference populations

Pathology/Issue	Present study	Canadian prevalence	Reference
Dermatologic (eczema)	4%	14.5%-22%	19
Respiratory (asthma and bronchitis)	4%	15%-20% in early years; 13% in late adolescence	20-22
Neurodevelopmental problems (ADHD, speech and learning impairments, autistic tendencies)	6.6%	14.1%-16.2% speech; 21.8% learning impaired	23
Physical development (overweight)	4% overweight; 1.3% obese	17% overweight; 9% obese	24
Premature delivery	4%	8%	25

ADHD, attention deficit hyperactivity disorder.

hair-test results showed that 7 were positive for cocaine (2 of which were also positive for benzoylecgonine, the cocaine metabolite), and concentrations ranged from 2 ng/mg to 23.26 ng/mg. Concentrations in children younger than 12 months of age tended to be higher (median = 7.51 ng/mg;  $n = 4$ ) than those in older children (median = 2 ng/mg;  $n = 3$ ). Unfortunately, the limited number of children in each group precluded statistical analysis of this trend. However, this is in line with previous observations that nonambulatory children (ie, those <18 months) who depend on their caregivers more than older children also have significantly higher exposures to drugs present in the environment.<sup>13</sup> Only the toddlers with the highest hair concentrations for cocaine were also positive for benzoylecgonine, suggesting some degree of systemic exposure to cocaine. The remaining 5 children were negative for benzoylecgonine, suggesting external contamination of the hair.

Of the hair samples, 12 were positive for cannabinoids; concentrations ranged from 0.1 to 0.6 ng/mg, and 3 samples were reported as having "trace amounts." Unlike cocaine, there did not seem to be any age-dependent differences in concentrations of cannabinoids in hair (median level for children <8 months, 0.24 ng/mg;  $n = 2$ ; median level for older children, 0.2 ng/mg;  $n = 7$ ). Two hair samples were positive for opiates. Finally, 4 hair samples were positive only for methamphetamine and 2 for MDMA. One sample was confirmed positive for both methamphetamine and MDMA.

Stratifying the test results by type of drug produced or found in the homes, children tended to test positive for the substances produced in their homes (Table II). Notably, the 4 children found in the crack-cocaine home did not have positive hair tests for drugs of abuse. The 2 children found to be living in the home storing MDMA that was described as having "horrendous living conditions" had

positive hair tests for MDMA at high concentrations. These 2 children exhibited stunted growth. Two other children found in a clandestine methamphetamine laboratory were positive for the drug itself as well as for cocaine (benzoylecgonine, the cocaine metabolite, was absent). Of the 57 children found in the 46 homes where marijuana was the concern, 15 produced a variety of positive test results (26.3% positivity rate): 2 children were positive for cocaine; 10 (17.5%) were positive for cannabinoids; 4 (7%) were positive for opiates; and 1 child (1.75%) was positive for both methamphetamine and MDMA. Children found in homes where multiple drugs were seized tested positive for such drugs accordingly; 6 of 8 children had positive test results for at least 1 substance, a 75% positivity rate: 1 child was positive for 2 drugs (cocaine and cannabinoids); 2 children tested positive for cocaine only; 1 child tested positive for cannabinoids only; and 2 children tested positive for methamphetamine. None of the children displayed signs of acute toxicity related to the drugs found in their hair, but 2 children tested for MDMA exhibited evidence of stunted growth, as described above.

## Discussion

Despite our findings that 30% of the children in our study tested positive for drugs of abuse in their hair, we found that the vast majority were in good health at the time of examination, which was within 1 to 2 weeks from their removal from their homes. The rates of the mostly minor health issues observed were well within the range expected in Canada and other developed countries (Table I). The current protocol followed by Police and Children's Aid Societies has been based on the assumption that the grow-houses and the individuals who operate them are not safe for children. It is

Table II. Hair test results for common illicit substances for assessed children, stratified by the drug found in their respective residences

Drug found in home	Children total	Cocaine-positive test	Opiate-positive test	Cannabinoid-positive test	Methamphetamine/MDMA-positive tests
Cocaine	4	0	0	0	0
Marijuana	61	2	2	8	1
Methamphetamine/MDMA	4	2	0	0	4
Multiple drugs	3	1	0	0	2



not clear whether the risk of interrupting a nurturing parent-child relationship has been adequately considered in all cases. There is a growing body of literature concerning the health and developmental outcomes of children of substance-using parents. Although often conflicting in their results, the overall concern is that these children are over-represented in a number of health problems, particularly behavioral and developmental problems and adolescent drug use.<sup>26</sup> Our study suggests that living in drug-producing homes, especially in marijuana grow-houses, cannot be automatically equated with parental use of illicit drugs. Many of the parents probably enjoy lucrative income from these operations and were not necessarily using the drugs produced, which concurs with the favorable outcome in the children. Moreover, none of these families had been under the care of child protection agencies prior to the index event. Two children assessed as being quite small for their ages and found in a clandestine MDMA laboratory in extremely undesirable living conditions were probably subject to child maltreatment and neglect, based on social worker and pediatric evaluations.

The positive drug hair-test results in the children in our study were mostly the result of passive exposure. This is probably best exemplified by the fact that only 2 children of 7 who tested positive for cocaine were also positive for its systemic metabolite, benzoylecgonine. There are a variety of ways by which children's hair may become contaminated by drugs of abuse, thereby testing positive. We have previously reported that younger children who might be handled more or who spend more time in the home environment have higher concentrations of drug in their hair samples when compared with older children.<sup>15</sup>

It is critical to review the consequences of automatic removal from their families of children found in such environments. Child apprehension may inflict fear, neglect, confusion, sadness, and similar adverse effects.<sup>16</sup> Indeed, in many cases, the reports of parents suggested such trauma among our patients. Therefore, removal of children from their parents must be based on solid evidence that the risks of staying with the parents outweigh the risks of separating the child from the core family.

In 2006, the province of Alberta passed the Drug Endangered Child Act,<sup>17</sup> which authorized the state (child welfare authorities or the police) to seize children from drug-producing homes, even if based on suspicion alone.<sup>18</sup> Often these children, and even the parents, might not know about the drugs. More troubling is that there may not even be illicit substances present, but rather the chemicals used to create such substances, and this may be deemed sufficient for apprehension of the children. To add to the equation, the Motherisk Laboratory at the Hospital for Sick Children receives hair samples to be analyzed for drugs of abuse from thousands of parents implicated in child-protection matters each year from across the country, and they are analyzed for drugs of abuse. Based on consultations with child protection workers or the respective authorities, children are rarely removed from drug-using parents' care until substantial evidence of

child safety issues is built. Among our cohort of children presented here, however, the majority of the parents were not known to be using illicit substances themselves and, on the basis of our clinical assessments, appear to be able to parent their children adequately. It is not likely that the production of drugs, particularly marijuana, hinders effective parenting much more than actual drug use, yet the differences in the ways these cases are handled suggest that police and child protection agencies perceive the former to be of greater concern with respect to child safety than the latter.

If residential marijuana production is discovered, children and their parents should be removed from the physical location of any such hazards; however, our data suggest that in most cases there is no medical justification to remove them from their parents. Our study documents that only a small proportion of children assessed were likely to be in need of interventions by child-welfare services because of potential risks caused by parents.

Our study has several weaknesses. We assessed children's health and developmental histories partially based on parental reports, which could be biased because they could affect child custody. In some cases the parents were not available and the assessments were based on medical records and interviews and on the assessments of child workers and of the children themselves. Under the urgent clinical situation, we could not conduct neurodevelopmental assessments that could detect more discreet deficits, and in most cases information on abuse and psychological evaluations were not made available to us. This report is not population based and lacks an appropriate contrast group, so it cannot be used to calculate the prevalence of or incidence of this very troubling phenomenon.

The strengths of our study include the direct referral from authorities, which allowed us to examine a relatively large number of children in a systematic manner. Also, the children were evaluated by pediatricians with clinical pharmacology/toxicology backgrounds, and we conducted highly specific toxicologic tests to evaluate toxic exposure. Such tests are commonly missing from environmental toxicologic studies. Our study lays the groundwork for the design of much-needed prospective cohort studies in this population of children. ■

Submitted for publication Jan 24, 2011; last revision received May 16, 2011; accepted May 24, 2011.

Reprint requests: Facundo Garcia-Bourisleen, MD, Hospital for Sick Children, Division of Clinical Pharmacology and Toxicology, Motherisk Program, 555 University Ave, Toronto, Ontario, Canada M5G 1X8. E-mail: facugb@hosm.mcgill.com

## References

1. Drug situation in Canada: 2007. <http://www.rcmp-grc.gc.ca/drugs-druguey/drug-2007-eng.htm>. [accessed Sept. 15, 2010].
2. Green Tide: indoor marijuana cultivation and its impact on Ontario. <http://www.ontario.ca/library/Repository/monographs/3000/10317711.pdf> [accessed Sept. 15, 2010].
3. Crystal meth: what you need to know. <http://www.rcmp-grc.gc.ca/qc/pol/meth/meth-eng.htm>. [accessed Sept. 15, 2010].

4. Kalant H. The pharmacology and toxicology of "ecstasy" (MDMA) and related drugs. *CMAJ* 2001;165:917-28.
5. Grant P, Bell K, Stewart D, Paulson J, Rogers K. Evidence of methamphetamine exposure in children removed from clandestine methamphetamine laboratories. *Pediatr Emerg Care* 2006;26:33-4.
6. Grant P. Evaluation of children removed from a clandestine methamphetamine laboratory. *J Emerg Nurs* 2007;33:31-41.
7. Gerberich SG, Gibson RW, French LR, Bazler CM, Lee TY, Carr WP, et al. Injuries among children and youth in farm households: Regional Rural Injury Study-1. *Inj Prev* 2001;7:117-22.
8. Pickett W, Brisson RJ, Hooey JR. Fatal and hospitalized agricultural machinery injuries to children in Ontario, Canada. *Inj Prev* 1995;1:97-102.
9. Marijuana grow operations. <http://www.rcmp-grc.gc.ca/fo-pdl/grow-ops-culture-eng.htm>. [accessed Sept 15, 2010].
10. Pragst F, Balkova MA. State of the art in hair analysis for detection of drug and alcohol abuse. *Clin Chim Acta* 2006;370:17-49.
11. Klein J, Karasik T, Koren G. Clinical applications of hair testing for drugs of abuse; the Canadian experience. *Forensic Sci Int* 2000;107:281-5.
12. Garcia-Bourmissen F, Nesterenko M, Karasik T, Koren G. Passive environmental exposure to cocaine in Canadian children. *Pediatr Drugs* 2009;11:30-2.
13. Garcia-Bourmissen F, Rokach B, Karasik T, Koren G. Methamphetamine detection in maternal and neonatal hair: implications for fetal safety. *Arch Dis Child Fetal Neonatal Ed* 2007;92F:251-5.
14. Garcia-Bourmissen F, Rokach B, Karasik T, Koren G. Cocaine detection in maternal and neonatal hair: implications to fetal toxicology. *Ther Drug Monit* 2007;29:71-6.
15. Garcia-Bourmissen F, Nesterenko M, Karasik T, Koren G. Passive environmental exposure to cocaine in Canadian children. *Pediatr Drugs* 2009;11:30-2.
16. Bruckas D. Children in foster care: a vulnerable population at risk. *J Child Adolesc Psychiatr Nurs* 2008;21:70-7.
17. Government of Alberta. Drug-endangered Children Act 2006. <http://www.child.alberta.ca/home/526.cfm>. [accessed Sept 9, 2010].
18. Drug Endangered Children Regulation: Alberta Regulation 256/2006. [http://www.op.alberta.ca/574.cfm?page=2006\\_256.cfm&reg\\_type=Reg&isbncln=079751116](http://www.op.alberta.ca/574.cfm?page=2006_256.cfm&reg_type=Reg&isbncln=079751116). [accessed Sept 15, 2010].
19. Dell SD, Poy RG, Gilbert NL, Jenet M, To T, Walter SD, et al. Asthma and allergic disease prevalence in a diverse sample of Toronto school children: results from the Toronto Child Health Evaluation Questionnaire (T-CHHQ) Study. *Can Respir J* 2010;17:e1-6.
20. Sears MR, Greene JM, Willan AR, Wieck EM, Taylor DR, Hannery EM, et al. A longitudinal, population-based, cohort study of childhood asthma followed to adulthood. *N Engl J Med* 2003;349:1414-22.
21. Subbarao P, Mandhane PJ, Sears MR. Asthma: epidemiology, etiology and risk factors. *CMAJ* 2009;181:E181-90.
22. Healthy People Healthy Environment: Children's health and the environment report. Toronto Public Health. [http://www.toronto.ca/healthy/hphe/childrens\\_health\\_report.htm](http://www.toronto.ca/healthy/hphe/childrens_health_report.htm). [accessed Sept 15, 2010].
23. McDougall J, King G, de Wit D, Miller LT, Hong S, Offord DR, et al. Chronic physical health conditions and disability among Canadian school-aged children: a national profile. *Disabil Rehabil* 2004;26:33-45.
24. Body mass index (BMI) for children and youth 2007 to 2009. <http://www.statcan.gc.ca/pub/92-625-x/2010001/article/11090-eng.htm>. [accessed Sept 15, 2010].
25. Ananth CV, Liu S, Joseph KS, Kramer MS. Fetal and Infant Health Study Group of the Canadian Perinatal Surveillance System. A comparison of foetal and infant mortality in the United States and Canada. *Int J Epidemiol* 2009;38:480-5.
26. Lester BM, Lagasse LL. Children of addicted women. *J Addict Dis* 2010;29:239-76.

**Appendix****Motherisk Clinic Protocol for Children in Grow Houses and Meth Laboratories**

1. Demographics: Name, age, contact, family context; demographic details, including age, height, weight, and BMI and family structure and siblings.
2. Drug context: What was found by police (drugs involved, environment: firearm risk, electricity risk, mold, other).
3. Medical history of child: Interviews with parents, child workers; reviews of all medical charts, routine visits to physician, vaccination history.
4. School history of child: Concordance between chronologic age and education level and evidence of delays.
5. Child development: Interviews with parents, examination of child, review of school reports; achievements as expected from the chronologic age through interviews with parents and children and neurologic examination.
6. Toxicology-related symptoms: Symptoms of CNS stimulants or depressants, stunted growth, aberrant behavior, or developmental delays, addressing both acute and chronic symptoms that occurred while residing in the drug-producing home.
7. In-clinic examination: Full pediatric physical and neurologic examinations.
8. Hair analysis for drugs of abuse: Testing for drugs discovered by police (according to police report); routine test also for cocaine, opiates, and amphetamines (including methamphetamine and MDMA).

## Appendix AC: Freeman et al. (2003) Household Exposure Factors, Asthma and School Absenteeism in a Predominantly Hispanic Community

*Journal of Exposure Analysis and Environmental Epidemiology* (2003) 13, 169–176  
© 2003 Nature Publishing Group All rights reserved 1053-4245/03/131100  
www.nature.com/jea

### ORIGINAL RESEARCH

### Household exposure factors, asthma, and school absenteeism in a predominantly Hispanic community

NATALIE C.G. FREEMAN,<sup>a,b</sup> DONA SCHNEIDER,<sup>b,c</sup> AND PATRICIA MCGARVEY<sup>d</sup>

<sup>a</sup>Robert Wood Johnson Medical School, Piscataway, New Jersey USA

<sup>b</sup>Environmental and Occupational Health Sciences Institute, Piscataway, New Jersey, USA

<sup>c</sup>Rutgers University, New Brunswick, New Jersey, USA

<sup>d</sup>Passaic Beth Israel Hospital, Passaic, New Jersey, USA

The Passaic Asthma Reduction Effort (PARÉ) used an asthma symptom and household exposure factor questionnaire to screen 4634 elementary school children over a 4-year period in Passaic, New Jersey. During the first year, an additional 240 preschool children were also screened. Overall, 16% of the school children were reported by their parents to have been diagnosed with asthma. In all, 30% of responding families claimed to have at least one family member diagnosed with asthma and this was five times more likely if the target child had asthma. Exposures consistently associated with childhood asthma diagnosis included environmental tobacco smoke (ETS), presence of dampness/mold, roaches, and furry pets in the home. Diagnosis of asthma was primarily associated with all six symptoms used in the PARÉ questionnaire, and secondarily with environmental factors. Puerto Rican and black children had the highest asthma prevalence (26% and 33%), while Mexican children had the lowest (7%). Use of medications and school absenteeism among asthmatic children were associated with wheeze and night cough, but not with any specific environmental exposure. Increased school absenteeism by children undiagnosed with asthma was associated with ETS and dampness/mold in the home. Differences in asthma diagnosis and absenteeism in response to environmental factors were found across ethnic subgroups. Getting asthmatic children on medical management protocols and providing families with education about environmental risk reduction should aid in reducing morbidity in this ethnically complex population. Such coordinated efforts offer the promise of reducing school absenteeism.

*Journal of Exposure Analysis and Environmental Epidemiology* (2003) 13, 169–176. doi:10.1038/sj.jea.7500266

**Keywords:** childhood asthma, exposure, risk factors, school absenteeism, screening, hispanic.

#### Introduction

Asthma prevalence is significantly higher in African- and Hispanic-American communities than in other populations (Beckett et al., 1996; Lieu et al., 2002). The disease has increased in both morbidity and mortality (Weiss et al., 1992). Asthma is also the major cause of school absenteeism in the US (Fowler et al., 1992; Ahammer, 1996). The Passaic Asthma Reduction Effort (PARÉ) was a 4-year screening program developed by Passaic Beth Israel Hospital in coordination with all public, private, and parochial schools in this predominantly Hispanic community (Freeman et al., 2002; Schneider et al., 2002). The objective of PARÉ was to screen all elementary school children (grades 2–5) for asthma and related respiratory problems over a 4-year period. The

screening program was designed to be broad in scope, with both biomarker and survey components. The biomarker component (peak flow measures and spirometry) identified children in need of treatment for respiratory problems, including asthma. The survey component was to help physicians identify environmental triggers for children as they designed individualized asthma treatment plans. The need for this extensive screening program was prompted by increased school absenteeism because of asthma and respiratory illnesses, and asthma crises among children who had not previously been diagnosed with asthma. The study was designed so that no child would be screened more than once in the 4 years.

Passaic is a unique community in that it has a wide range of ethnic Hispanic groups coming from the Caribbean, Mexico, Central, and South America. This diversity provides an opportunity to compare asthma and exposure issues for a variety of ethnic groups within the same urban and predominantly poor community. The two end points of interest are asthma diagnosis (determined from both biomarker and parental survey data) and school absenteeism (determined from the parental survey data).

1. Abbreviations: CI, confidence intervals; df, Degrees of freedom; dx, diagnosis; ETS, environmental tobacco smoke; OR, odds ratio  
2. Address all correspondence to: Dr. Natalie C.G. Freeman, 11 Cleveland Circle, Skillman, NJ 08558, USA. Tel: +1-609-924-4921. Fax: +1-732-445-0116. E-mail: nfreeman@ehsi.rutgers.edu  
Received 27 June 2002; accepted 19 November 2002



## Methods

Each child screened was weighed and measured and given a peak-flow or spirometry test. During this procedure, the children were also asked five questions about whether anyone smoked at home and if so who, whether they had pets and if so what they were, and whether they had a rug or carpet in their bedroom. These questions were used to validate answers on the parental questionnaires.

A 25-item questionnaire was developed from existing instruments (Chikmonczyk et al., 1993; American Institute of Research, 1991; Asher et al., 1995; New York City Health and Hospitals Corp., 1995; Beckett et al., 1996; Koren, 1997; Eskenazi et al., 1999; Joseph et al., 1999) and was designed to characterize symptoms, health practices, and environmental exposure factors that have been documented to be asthma triggers or suspected asthma triggers (Chikmonczyk et al., 1993; Ingram et al., 1995; Stoddard and Miller, 1995; Beckett et al., 1996; Koren, 1997; Platts-Mills and Carter, 1997; Eskenazi et al., 1999). Parents were requested to complete the questions (in either Spanish or English) about their child's respiratory health (symptoms, diagnosis, and medications), family history of asthma, school absenteeism, potential environmental exposures in the home, insurance status, and race/ethnicity. The questionnaire data required mostly nominal (yes, no) or ordinal responses (e.g., categories of number of days absent). Six questions addressed symptoms: "Does your child experience: Frequent coughs? Shortness of breath? Noise in the chest (sometimes called wheezing)? Trouble breathing? Tightness in the chest? Cough during the night or early morning?" Positive responses were aggregated to produce an aggregate symptom score.

Environmental factors in the questionnaire included environmental tobacco smoke (ETS) exposure (number of smokers in the home; who smokes in the home; amount of ETS exposure the child has per day classified as none, less than a pack, a pack, more than a pack); mildew/mold in the bathroom, basement, closets, other areas of the home; carpeting in the child's bedroom; the presence of furry pets; pet access to the child's bedroom; use of feather pillows; presence of roaches; and the use of pesticides in the home.

Several derivative exposure factors were developed from these variables. An aggregate mold variable summed the number of places where parents reported dampness or mold. The aggregate mold variable was then used to develop two additional dichotomous variables: any mold and multiple mold. Any mold asks if there is mold anywhere in the home, while multiple mold addresses general household conditions under the assumption that mold in more than one area of the home reflects poor housing conditions. In addition to the aggregate mold variable, an aggregate exposure variable was developed based on the number of different types of exposure factors that the parent reported (e.g. any mold + furry pets + pesticide use = 3).

In year 1, children in two preschool programs and all third graders were screened. In year 2, the program targeted fifth graders. Third and fourth graders were screened in year 3, and second and third graders were screened in year 4.

Data were analyzed using SPSS, version 10.1 (2000) with Fisher's exact and  $\chi^2$  tests. Odds ratios (OR) with 95% confidence intervals (CI) were calculated on the  $\chi^2$  distributions to assess the relation between environmental exposure factors in the home and the outcome variables: asthma diagnosis and school absenteeism. Secondary predictor variables included symptoms, use of medications and family asthma. Variables significantly associated in bivariate analyses were introduced into a stepwise logistic regression with Hosmer and Lemeshow test for goodness of fit to identify independent factors contributing to asthma diagnosis and school absenteeism.

Analyses were performed for the entire study population and for major ethnic subsets. Student's *t*-test and analysis of variance were used to evaluate differences in age, aggregate symptoms, and aggregate exposure factors between and across groups.

Three Hispanic groups predominate in this community: Dominicans, Mexicans and Puerto Ricans. Most of the analyses were conducted on these three groups only, although additional analyses were conducted for black and Non-Hispanic White children, and the category "other Hispanic." The "other Hispanic" group included individuals from 14 other Central and South American countries with the largest numbers of children originally from Colombia, Ecuador, and Peru.

## Results

Of the potential 6480 elementary children to be screened, parental questionnaires were returned for 4634 children (72% response rate) and 77% of the returned questionnaires were fully completed by parents. The data presented here are from 64% of all elementary school Passaic children during the study period. A large proportion of questionnaires were completed in Spanish, indicating the strong Hispanic character of the Passaic community. In all, 57% percent of questionnaires returned by preschool parents were in Spanish, while 46% of questionnaires returned by elementary school parents were in Spanish. Among Hispanic groups, Puerto Rican parents were most likely to respond in English (76%), and Mexican parents most likely to respond in Spanish (88%).

From school rosters, we were able to ascertain that nonrespondents were similar to respondents in terms of age and gender, except during year 2 when it was found that fifth grade boys were slightly less likely to return questionnaires than fifth grade girls. We cannot make statements about the race and ethnicity of non-respondents, however, as these



variables were derived from the survey data. In order to determine if our sample was representative of the community as a whole in terms of race and ethnicity, we examined 2000 US Census data for the City of Passaic (US Department Commerce, 2002). In that year, 62.5% of the population and 68% of those aged less than 18 years of age were identified as Hispanic. The larger proportion of Hispanics in the younger age group demonstrates the young nature of the Hispanic community. Also in the year 2000, US Census data show black people as 13.8% and Asians as 5.5% of the Passaic population.

The overall questionnaire responses identified 75% of children as Hispanic, with 10.5% and 5% as black and Asian children, respectively. We conclude that our sample is likely a representative of the children of Passaic as a whole, but we cannot rule out the possibility that some non-Hispanics may be under-represented.

Table 1 lists the characteristics of children whose parents completed questionnaires by year of the screening program. The proportions of school aged children with an asthma diagnosis, on respiratory medications and missing school for respiratory problems once a month or more were significantly higher at the beginning of the study. In addition, preschoolers who were only sampled during the first year of the study had even higher rates of diagnosis, medication use, and school absenteeism than was reported for the older children.

Questionnaire responses about the characteristics of the children and the primary exposure factors are listed by ethnic group in Table 2. There were significant differences across groups for all variables except for gender distribution. Non-Hispanic white children were younger than children in the other ethnic groups, but there was no difference between the ages of the various Hispanic subgroups.

#### Asthma Diagnosis in School-age Children

The proportion of children diagnosed with asthma varied across ethnic groups. Puerto Rican and Black children had consistently high rates of asthma diagnosis (26% for the 4 years, range 22–28% for Puerto Ricans and 33% for the 4 years, range 30–42% for black children). Consistently lower rates of asthma diagnosis were observed for Dominican children (14%, range 12–17%) and Mexican children (7%, range 4–9%). Reporting of asthma for non-Hispanic white children was somewhat variable across the 4 years (14%, range 11–27%) (Table 2).

Among children diagnosed with asthma, medication use was low (Tables 1 and 2). Not reflected in these tables are the differences by ethnic group by year. For the first 3 years of the study, medication use was reported for less than half of the children diagnosed with asthma. Mexican and non-Hispanic white children (30% and 31%, respectively) were less likely to use medications than Dominican, black and Puerto Rican children (42%, 44%, and 47%, respectively) ( $P < 0.05$ ). In year 4, parents reported a significant increase in medication use compared to the previous years, to 48% for Mexican, 66% for non-Hispanic white, 62% for Dominican, 51% for black and 57% for Puerto Rican children.

#### Exposure Factors Related to Reported Diagnosis

Across all 4 years, the major household exposure factors associated with asthma diagnosis were ETS ( $\chi^2 = 77.03$ ,  $df = 3$ ,  $P < 0.001$ , 31% of asthmatics exposed compared to 17% of nonasthmatics) and dampness/mildew in the bathroom ( $\chi^2 = 27.6$ ,  $df = 3$ ,  $P < 0.001$ , 24% of asthmatics exposed compared to 15% of nonasthmatics). Two additional environmental factors were reported more frequently by parents of asthmatic children compared to

Table 1. Characteristics of children whose parents completed questionnaires (percentage calculated by excluding cases with missing data).

Year	1998	1999	2000	2001	
Grade	Preschool	3	5	3-4	2-3
Hispanic country of origin (N %)					
Dominican Republic	96 (40)	201 (25)	129 (24)	320 (25)	294 (18)
Mexico	31 (13)	134 (17)	94 (17)	338 (26)	428 (26)
Puerto Rico	16 (7)	182 (23)	86 (16)	238 (20)	289 (18)
Other Hispanic*	84 (35)	84 (10)	90 (16)	61 (5)	192 (12)
Black	13 (5)	98 (12)	61 (11)	131 (10)	139 (9)
Non-Hispanic White	0 (0)	68 (8)	41 (7)	139 (11)	194 (12)
Non-Hispanic other (Asian)	0 (0)	41 (5)	47 (9)	51 (4)	80 (5)
Asthma dx (%)	24.4	20.2	12.3	14.0	12.3
Respiratory meds (%)	17.9	11.0	7.0	7.3	7.7
Absent > 1/month (%)	15.5	10.1	4.3	5.7	5.9
Responses/total screening pool	240/350	1029/1300	548/790	1441/2087	1616/2303
Questionnaires returned (%)	69	79	69	69	70
Age of children (SD)	—	8.7 (0.7)	10.9 (0.7)	8.9 (0.8)	8.1 (0.9)

\*Includes children from Argentina, Bolivia, Chile, Colombia, Costa Rica, Cuba, El Salvador, Guatemala, Honduras, Nicaragua, Peru, Ecuador, Uruguay, and Venezuela.



Table 2. Children's characteristics and household exposure factors by ethnic group.

Characteristics	Black	Non-Hispanic White	Mexican	Puerto Rican	Dominican	Other Hispanic
N	489	479	979	765	999	349
Gender (% male)	48.3	46.9	47.1	44.3	46.2	42.5
Mean age (SD)	8.7 (1.2)	8.2 (1.0)	8.7 (1.1)	8.8 (1.0)	8.9 (1.2)	8.7 (1.1)
Asthma dx (%) <sup>a</sup>	33.1	14.6	6.5	25.8	13.5	15.1
Use meds (%) <sup>a</sup>	18.0	9.2	4.2	15.7	9.3	8.3
Absent > 1/month (%) <sup>a</sup>	12.3	3.0	4.7	11.4	6.9	8.3
Family dx (%) <sup>a</sup>	57.6	28.1	12.1	64.3	30.4	28.1
Insured (%) <sup>a</sup>	90.9	91.1	55.5	84.9	72.1	72.5
Mean symptoms <sup>a</sup> (SD)**	1.7 (1.6)	0.7 (1.3)	0.6 (1.2)	1.4 (1.8)	1.0 (1.6)	1.1 (1.5)
Household Exposure Factor						
Smoking in home (%) <sup>a</sup>	43.8	12.7	10.3	35.5	10.8	16.4
Bathroom damp/mold (%) <sup>a</sup>	15.6	15.5	21.4	21.2	12.3	13.2
Carpet in child's room (%) <sup>a</sup>	63.7	79.2	40.1	60.2	50.3	56.9
Furry pets (%) <sup>a</sup>	24.0	19.8	9.1	31.1	13.9	24.7
Pets in child's room (%) <sup>a</sup>	33.8	35.5	12.9	37.7	13.7	18.9
Feather pillows (%) <sup>a</sup>	28.0	19.9	5.4	20.2	11.9	12.9
Roaches (%) <sup>a</sup>	21.8	6.0	28.6	28.9	23.2	21.2
Pesticide use (%) <sup>a</sup>	37.1	14.2	43.7	43.1	39.5	34.0
Mean factors (SD)**	2.2 (1.3)	1.6 (1.0)	1.5 (1.1)	2.1 (1.3)	1.5 (1.1)	1.6 (1.1)

<sup>a</sup>Derived from six survey questions: "Does your child have: Frequent coughs? Shortness of breath? Noise in the chest (sometimes called wheezing)? Trouble breathing? Tightness in the chest? Cough during the night or early morning?"

\*\* $\chi^2$ -test, \*\*ANOVA  $P < 0.001$

Table 3. Odds ratios of household exposure factors and school-aged target child asthma diagnosis, medication use, and school absenteeism (with 99% CI).

Household exposure factors	dx	med use	absent
ETS (none/home)	2.27 (1.89, 2.74)***	1.66 (1.31, 2.10)***	2.29 (1.77, 2.98)***
Multiple mold sites (> 1)	1.90 (1.35, 2.69)***	1.79 (1.18, 2.72)**	1.89 (1.17, 2.03)**
Any mold (none/some)	1.82 (1.49, 2.23)***	1.97 (1.56, 2.50)***	1.65 (1.25, 2.17)***
Damp bathroom (yes/no)	1.72 (1.40, 2.11)***	1.88 (1.47, 2.40)***	1.81 (1.35, 2.41)***
Furry pets (yes/no)	1.45 (1.16, 1.82)***	1.09 (0.83, 1.46) NS	1.26 (0.81, 1.74) NS
Roaches (yes/no)	1.40 (1.17, 1.69)***	1.36 (1.08, 1.71)**	2.26 (1.76, 2.90)***
Pets in bedroom (yes/no)	1.09 (0.91, 1.24) NS	1.23 (0.83, 1.82) NS	0.79 (0.49, 1.27) NS
Pesticide use (yes/no)	1.17 (0.99, 1.39) NS	1.01 (0.82, 1.25) NS	1.46 (1.15, 1.85)**
Feather pillow (yes/no)	0.95 (0.74, 1.23) NS	0.86 (0.62, 1.20) NS	1.19 (0.84, 1.69) NS
Carpet in room (yes/no)	0.91 (0.77, 1.07) NS	0.74 (0.60, 0.91)**	0.68 (0.53, 0.86)***
Mean factors (SD)	2.0 (1.3) versus 1.7 (1.3)***	1.9(1.3) versus 1.7 (1.2)*	2.1 (1.3) versus 1.7 (1.1)***
N	4058	4107	4022

$\chi^2$ -tests: \*\*\* $P < 0.001$ , \*\* $P < 0.01$ , \* $P < 0.05$ , NS  $P > 0.05$ .

t-Test mean factors: \*\*\* $P < 0.001$ , \* $P < 0.05$ .

parents of nonasthmatic children: the presence of furry pets (predominantly dogs, 30% versus 22%,  $P < 0.001$ ) and the presence of roaches in the home (28% versus 22%,  $P < 0.001$ ).

Table 3 presents ORs for the major exposure factors for asthma diagnosis, use of medication for respiratory problems, and school absenteeism for the entire study population obtained from bivariate analyses. Significant differences in aggregate exposure were found between diagnosed and nondiagnosed children (2.0 exposure factors versus 1.7), between those using respiratory medications and those without (1.9 versus 1.7), and between children absent at

least once a month and those less frequently absent (2.1 versus 1.7).

Nearly all the exposure factors had OR in the expected direction (Table 3). Several potential exposure factors were not associated with the outcome variables. Furry pets in the house were more associated with a diagnosis of asthma than was pet access to the child's bedroom. This is likely due to the fact that only a subset of furry pets are allowed access to the child's bedroom, whereas the pet is likely allowed near the television and kitchen, places where children spend a great deal of time. When dampness/mildew in individual areas of the home were used as independent variables, they were not



Table 4. Environmental factors associated with asthma diagnosis and school absenteeism by ethnic group.

Group	Factor	Asthma diagnosis		Absenteeism	
		Odds ratio (95% CI)	P-value*	Odds ratio (95% CI)	P-value
Mexican n=868	Bathroom mold	1.55 (0.81-2.54)	0.181	2.49 (1.27-4.88)	0.006
	Roaches	1.16 (0.65-2.07)	0.612	2.22 (1.20-4.10)	0.009
Puerto Rican n=721	Bathroom mold	1.95 (1.33-2.87)	0.001	2.20 (1.20-3.71)	0.003
	Roaches	1.45 (1.01-2.08)	0.042	1.87 (1.16-3.00)	0.003
	ETS	1.38 (0.98-1.95)	0.052	1.61 (1.01-2.56)	0.044
	Pesticide	1.27 (0.91-1.79)	0.157	1.57 (0.99-2.49)	0.054
Dominican n=885	ETS	1.69 (0.98-2.91)	0.056	1.93 (0.97-3.85)	0.058
	Bathroom mold	1.67 (0.99-2.85)	0.058	1.27 (0.59-2.78)	0.538
Black n=458	Bathroom mold	2.17 (1.30-3.62)	0.003	1.67 (0.83-3.35)	0.150
	Roaches	1.82 (1.16-2.86)	0.009	2.78 (1.54-5.03)	0.001
	ETS	1.60 (1.09-2.37)	0.017	1.27 (0.72-2.23)	0.406
Non-Hispanic White n=456	ETS	2.22 (1.16-4.26)	0.014	13.17 (4.14-41.87)	0.001
	Bathroom mold	2.27 (1.23-4.17)	0.007	1.51 (0.48-5.56)	0.406
	Roaches	1.38 (0.51-3.84)	0.513	8.00 (2.34-27.63)	0.001

\*P-value for  $\chi^2$ -test.

consistently associated with asthma diagnosis, except for bathroom dampness/mold. Although the "any mold" and "multiple mold sites" variables were statistically significant, they had little effect beyond what could be obtained using the variable "damp bathroom" by itself. The other exposure variables elicited in the questionnaire, feather pillows and carpeting in the child's room, had no statistical association with asthma diagnosis for the target children in this study. However, carpeting in the child's room was associated with lower rates of medication use and school absenteeism ( $P < 0.01$ ).

When the significant exposure variables were included in a stepwise logistic regression model, three exposure variables were found to contribute to diagnosis: ETS (OR 2.103, 95% CI 1.72-2.56,  $P < 0.001$ ), mold (OR 1.539, 95% CI 1.27-1.87,  $P < 0.001$ ), and the presence of furry pets (OR 1.371, 95% CI 1.12-1.67,  $P = 0.002$ ). However this model only explained 10% of asthma diagnosis. When symptoms were included in the model either as individual symptoms or aggregate symptoms, the explanatory value of the model increased to 47%, and mold dropped out of the model, leaving ETS and the presence of furry pets as the significant exposure factors.

For preschool children, damp bathrooms (OR 4.78, 95% CI 2.04-11.18,  $P < 0.001$ ) or the composite variable "any mold" (OR 3.30, 95% CI 1.57-6.97,  $P = 0.001$ ) were the only household exposures associated with asthma diagnosis in the target child.

Comparison of household exposure factors and target child asthma diagnosis for specific subgroups found that damp/moldy conditions were associated with diagnosis for Mexican and Puerto Rican children, but not for Dominican children (Table 4). ETS was associated with asthma diagnosis for all

Table 5. Significant odds ratios of environmental factors and family asthma diagnosis (95% CI) by ethnic group.

Ethnic group	Environmental factors	Odds ratio (CI)
Mexican	Multiple mold sites	5.18 (2.49-10.81)***
	Bathroom damp/mold	2.27 (1.45-3.54)***
	Any mold	2.21 (1.41-3.45)***
	Roaches	1.83 (1.20-2.77)**
	Furry pets	1.76 (1.01-2.78)*
Puerto Rican	Multiple mold sites	4.16 (1.60-10.79)**
	Roaches	2.46 (1.69-3.58)***
	Any mold	2.07 (1.41-3.05)***
	ETS	1.98 (1.41-2.78)**
	Bathroom damp/mold	1.96 (1.30-2.95)*
	Pesticides	1.67 (1.21-2.30)**
Dominican	Furry pets	1.57 (1.07-2.31)*
	Any mold	2.09 (1.41-3.08)***
	Furry Pets	1.96 (1.24-3.10)*
	ETS	1.98 (1.29-3.04)**
	Roaches	1.86 (1.35-2.57)***
	Multiple mold sites	1.88 (1.35-2.62)*
Other Hispanic	Bathroom damp/mold	1.78 (1.17-2.72)**
	Any mold	2.75 (1.49-5.09)***
Black	ETS	2.52 (1.37-4.63)**
	Any mold	1.95 (1.26-3.02)**
Non-Hispanic White	Any mold	1.95 (1.26-3.02)**
	ETS	1.90 (1.30-2.77)***
	Roaches	1.76 (1.11-3.79)*
Non-Hispanic White	Pesticides	1.56 (1.07-2.29)*
	Any mold	1.95 (1.26-3.02)**

 $\chi^2$ -tests, \*\*\* $P < 0.001$ , \*\* $P < 0.01$ , \* $P < 0.05$ .

three Hispanic groups. ETS and damp/moldy conditions were associated with a target child asthma diagnosis for non-Hispanic White and black groups, while exposure to roaches





in the home was associated with asthma diagnosis for black children. Mexican children who had low rates of asthma diagnosis showed significant associations between exposure to mold and roaches and school absenteeism, even though these were not associated with asthma diagnosis.

Non-exposure factors associated with school absenteeism included asthma diagnosis (OR 9.093, 95% CI 7.06–11.70,  $P < 0.001$ ) and use of medications for respiratory problems (OR 7.912, 95% CI 6.06–10.34,  $P < 0.001$ ) for all school-aged children, and significant factors for black and all Hispanic subgroups.

#### *Familial Asthma*

Asthma in members of the family other than the target child was reported by 33% of families and was more often reported by those families where the target child was asthmatic (OR 5.38, CI 4.52–6.42,  $P < 0.001$ ). Evaluation of environmental factors and family asthma found that the same variables that were found for the target school-age child (ETS, furry pets, roaches, and damp bathrooms) were significantly associated with family asthma diagnosis.

For families with preschool children, family asthma was associated with ETS (OR 5.21, 95% CI 2.29–11.87,  $P < 0.001$ ) and damp bathroom (OR 2.41, 95% CI 1.07–5.41,  $P = 0.03$ ).

For all Hispanic groups, damp/mold conditions were associated with family asthma. For Puerto Rican, Dominican, and other Hispanics, ETS was significantly associated with family asthma (Table 5). In contrast, ETS and presence of cockroach were associated with family asthma for black groups. Any mold was implicated for non-Hispanic white families.

#### *Household Exposure Factors Associated with Respiratory Symptoms*

As previously reported in the literature, symptom reporting (aggregate symptom score) was associated with prior diagnosis of asthma (Asher et al., 1995). The mean number of symptoms reported for asthmatic children was 3.3 (SD 2.1, median 3) compared to 0.6 (SD 1.1, median 0) for children without diagnosed asthma ( $P < 0.001$ ). Among children with reported asthma, symptoms were more likely to be reported if the children were on medications than if they were not using medications (4.3 symptoms compared to 2.3 symptoms,  $P < 0.001$ ). At the same time, children without asthma showed a similar pattern, although with fewer symptoms. Nonasthmatic children on respiratory medications reported 1.6 symptoms compared to 0.6 for children not on medicines ( $P < 0.001$ ).

Symptoms were not only associated with asthma diagnosis but also, among school-aged children, with a variety of household exposures and the aggregate exposure factors. Aggregate symptom was significantly rank-correlated with aggregated exposure ( $r = 0.208$ ,  $P < 0.001$ ). In pairwise comparisons, exposure to ETS and presence of mold in the

home were associated with a variety of symptoms in school-aged children. In contrast, no household exposure factors were associated with symptoms among preschool children in pair-wise comparisons.

For most of the ethnic subgroups, mold was the primary exposure factor associated with nearly all symptoms. The aggregated symptoms were significantly greater for all groups except black children when there was mold in the house. Only black children and non-Hispanic white children showed significant associations between ETS and specific symptoms ( $P = 0.043$  for wheeze and  $P = 0.007$  for trouble breathing, respectively).

#### *Conditions in the Home associated with School Absenteeism*

For preschool children, absenteeism was driven by symptoms and, in particular, cough at night or in the morning (cough pm/am). For this group, household exposure factors *per se* were not associated with school absenteeism.

This was not the case for school-age children. While the strongest associations were between symptoms and absenteeism, a number of household exposure factors were also associated with this outcome: ETS, dampness and mildew, and presence of roaches. Stepwise logistic regression analysis across all 4 years found that asthma diagnosis and symptoms were the driving forces behind absenteeism (asthma dx, OR 2.25, 95% CI 1.35–3.76,  $P = 0.002$ , symptoms OR 1.54, 95% CI 1.38–1.72,  $P = 0.001$ ), followed by ETS (OR 1.67, 95% CI 1.12–2.49,  $P = 0.012$ ), and presence of roaches (OR 1.52, 95% CI 1.02–2.28,  $P = 0.042$ ). These four variables explained 25% of absenteeism. Replacing aggregate symptoms by each of the six symptoms in the stepwise analysis found that three symptoms contributed to the model: frequent cough, tightness in chest, and night cough. Replacing aggregate symptoms by the individual symptoms did not increase the explanatory value of the model.

#### **Discussion**

The significant environmental factors associated with asthma in this study have also been found in other studies (Rylander and Etzel, 1999; Lanphear et al., 2001; Finkelstein et al., 2002). Finkelstein et al. (2002) reported lower rates of household pests and smokers in their pool of 638 asthmatics than we found across all children in Passaic. In contrast, many more of their families had carpeting and pets. The differences may lie in SES differences between the study populations. The Finkelstein et al. study population contained 89% college-educated families. The possible protective influence of bedroom carpeting found in this study is contrary to findings in other studies. These were statistically weaker outcomes and may be the result of multiple analyses. However, since the direction was similar for asthma, medication use, and absenteeism, we cannot



exclude the possibility that carpeting in this community is indicative of some aspect of these families lives on which we did not collect information. We lacked direct information about family education and income, but US Census data for Passaic and the insurance coverage information we elicited from the survey data allow us to conclude that the Passaic population is a less affluent and less educated one than in the Finkelstein et al. study. The differences in exposure factors between these studies may reflect differences in SES between the study populations.

Subset ethnic analyses found that a variety of household exposure factors were associated with asthma diagnosis, and along with diagnosis and symptoms contribute to absenteeism. For Puerto Rican and 'Hispanic other' children the factors that were associated with asthma diagnosis were also associated with school absenteeism. In contrast, a number of environmental factors were associated with asthma diagnosis in black, non-Hispanic white, and Dominican children, but either fewer or different factors were associated with school absenteeism for these groups.

We documented that not only asthmatic children suffer from exposure to environmental triggers. Less than 20% of all families reported presence of dampness/mold in their homes, yet this household exposure factor was strongly associated with asthma diagnosis, absenteeism, and use of respiratory medications in children who were and were not diagnosed with asthma. We found that more than 63% of nonasthmatic children and 73% of asthmatic children had at least one exposure factor in the home, and 11% of nonasthmatic children and 20% of asthmatic children had at least three exposure factors that are known to be asthma triggers. These values are substantially greater than those found by Lanphear et al. (2001) and contribute to the morbidity of asthmatic and nonasthmatic children in Passaic.

The associations between symptoms and exposure factors differed across the years of the study. During 1999 when the least number of relations were observed, the children were the oldest, in fifth grade. It is possible that these older children were outgrowing respiratory symptoms, or that their parents were paying less attention to them. On the other hand, the proportion of children diagnosed with asthma declined between years 1 and 4 of the study, as did the proportion of children on asthma medications and the proportion missing more than one day of school per month due to respiratory symptoms. As children in years 1 and 4 were close in age, the argument that simply growing older reduces respiratory symptoms in school children cannot be used to explain this change in outcomes. One explanation is that the weather changed in this region, with high mold conditions at the beginning of the study followed by a series of dry years with decreased mold. As mold was a significant trigger for respiratory symptoms, this is quite plausible. Another explanation is that the PARÉ program worked, reducing children's exposure to ETS through an active health

education campaign to get parents not to smoke inside the home or around their children. As ETS was another significant environmental trigger for respiratory symptoms, this is also plausible.

Ethnic differences were found in the prevalence of childhood asthma and in the types of household exposure factors that were associated with symptoms. Asthma was epidemic in Puerto Rican and black children. In contrast, Mexican children were less likely to have an asthma diagnosis and the rates for these children were nearly at the national norm. This finding is consistent with observations from other studies (Mendoza et al., 1991; Flores et al., 1999). Our study found that the Mexican children were reported to have both fewer symptoms and lower rates of ETS and pets than other groups. One of the most striking features was that while no environmental factors were associated with asthma diagnosis for Mexican children, both roaches and mold were associated with school absenteeism for this group. Environmental factors associated with asthma among the subgroups differed. Based on parental reports, Puerto Rican asthmatic children were exposed to more environmental triggers than were children in other Hispanic subgroups.

Absenteeism was associated with the number of respiratory symptoms reported. Absentee rates for children with three or more symptoms ranged from 23% to 35%, depending on ethnic group. Health-care coverage also varied across ethnic groups, with the newest immigrant group (Mexicans) less likely to have health insurance than others. Some of the differences in asthma diagnosis may be due to lack of access to health care.

In previous studies, a mix of ethnic groups have been lumped together as Hispanic. The differences observed in Hispanic subgroups in this community over a 4-year period suggest that treating these groups as homogeneous may not meet the health needs of the specific ethnically identified families. Yet the constant theme for most of the children in this study who are asthmatic or frequently absent from school because of respiratory problems was the presence of mold and tobacco smoke in the home. These triggers do not discriminate by ethnicity. ETS is a risk factor that can be managed with family education and cooperation. Mold in New Jersey is ubiquitous during much of the year and concerted efforts must be applied to reduce indoor sources such as water damage in bathrooms, basements, and from leaky roofs and gutters. Targeting these environmental triggers with a massive health education campaign holds promise for reducing school absenteeism from asthma and undiagnosed respiratory symptoms, regardless of ethnicity.

#### Acknowledgments

This project was funded by the Robert Wood Johnson Foundation–New Jersey Health Initiatives (RWJ/NJHI)



and approved by Passaic Beth Israel Hospital and the Passaic Board of Education. We acknowledge the enormous effort the Passaic public and private school systems put into this project, thank the RWJF/NJHI and the members of the Passaic Advisory Council, St. Mary's Hospital, The Passaic Board of Education, the Passaic Parochial Schools, Yeshiva K'tana, Passaic Prep School, Guidance Guild, Children's Day Nursery, Our Lady of Fatima Day Care, New Balm School, Passaic Head Start, Passaic Health Department, Hispanic Information Center, Rutgers University School of Urban Studies and Community Health, Felician College Department of Professional Nursing, Ellen Ziff, Erik and Lisha Ramos, and the project interns, Carrie Bogert and Lenora Roth.

#### References

- Aiteneier W.A. Children with asthma: we can do better. *Pediatr Ann* 1996; 25: 124-125.
- American Institute of Research. *Was Wisconsin Asthma Education Program*. 1991: 15pp.
- Asher M.L., et al. International Study of Asthma and Allergies in Childhood (ISAAC): rationale and methods. *Eur Respir J* 1995; 8: 483-491.
- Beckett W.S., et al. Asthma among Puerto Rican Hispanics: a multi-ethnic comparison study of risk factors. *Am J Respir Crit Care Med* 1996; 154: 894-899.
- Chimanczyk B.A., et al. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. *N Engl J Med* 1993; 328 (23): 1665-1669.
- Eisenazi B., et al. Exposures of children to organophosphate pesticides and their potential adverse health effects. *Environ Health Perspect* 1999; 107 (Suppl. 3): 409-419.
- Finkelstein J.A., et al. Parent-reported environmental exposures and environmental control measures for children with asthma. *Arch Pediatr Adolesc Med* 2002; 156: 258-264.
- Flores G., et al. The impact of ethnicity, family income, and parental education on children's health and use of health services. *Am J Public Health* 1999; 89: 1066-1071.
- Fowler M.G., et al. School functioning of US children with asthma. *Pediatrics* 1992; 90: 939-944.
- Freeman N.C.G., et al. School-based screening for asthma in third-grade urban children: results from the Passaic Asthma Reduction Effort. *Am J Public Health* 2002; 92: 45-46.
- Ingram J.M., et al. Quantitative assessment of exposure to dog and cat allergens relating to sensitization and asthma among children. *J Allergy Clin Immunol* 1995; 96: 440-456.
- Joseph C.L., et al. Sensitivity and specificity of asthma definitions and symptoms used in a survey of childhood asthma. *J Asthma* 1999; 26: 565-573.
- Koren H.S. Environmental risk factors in atopic asthma. *Int Arch Allergy Immunol* 1997; 113: 65-68.
- Lamphear B.P., et al. Residential exposures associated with asthma in US children. *Pediatrics* 2001; 107: 505-511.
- Lieu T., et al. Racial/ethnic variation in asthma status and management practices among children in managed Medicaid. *Pediatrics* 2002; 109: 857-865.
- Mendoza F., et al. Selected measures of health status for Mexican-American, mainland Puerto Rican, and Cuban-American children. *JAMA* 1991; 265: 227-232.
- New York City Health and Hospitals Corp. Child Health Clinic. Asthma Screening Questionnaire. 1995: p. 1.
- Platts-Mills T.A., and Carter M.C. Asthma and indoor exposure to allergens. *N Engl J Med* 1997; 336: 1382-1384.
- Rylander R., and Ertel R. Introduction and summary: workshop on children's health and indoor mold exposure. *Environ Health Perspect* 1999; 107 (Suppl. 3): 465-468.
- Stoddard J.J., and Müller T. Impact of parental smoking on the prevalence of wheezing respiratory illness in children. *Am J Epidemiol* 1995; 141: 96-102.
- Schneider D., et al. Respiratory dysfunction among a cohort of mostly Hispanic urban primary school children. 2003 (In review).
- SPSS Inc. 2000. SPSS version 10.1, Chicago, IL.
- U.S. Department of Commerce, Bureau of the Census. Census 2000 (American FactFinder). www.census.gov. Nov 2002.
- Weiss K.B., et al. Inner City asthma: the epidemiology of an emerging U.S. public health concern. *Chest* 1992; 101: 3625-3675.

## Appendix AD: Garrett et al. (1998) Indoor Airborne Fungal Spores, House Dampness and Associations With Environmental Factors and Respiratory Health in Children

*Clinical and Experimental Allergy*, 1998, Volume 28, pages 459–467

### Indoor airborne fungal spores, house dampness and associations with environmental factors and respiratory health in children

M. H. GARRETT, P. R. RAYMENT, M. A. HOOPER, M. J. ABRAMSON\* and B. M. HOOPER

*School of Applied Sciences, Monash University, Churchill, Victoria and \*Department of Epidemiology & Preventive Medicine, Monash Medical School, Alfred Hospital, Prahran, Australia*

#### Summary

**Background** Children living in a damp house are more likely to suffer from respiratory symptoms and it has been suggested that exposure to fungi is an important contributing factor. However, more knowledge about underlying mechanisms for the association are needed.

**Objective** To identify associations between measures of house dampness, levels of airborne fungal spores, housing factors and health outcomes in children.

**Methods** Eighty households with 148 children between 7 and 14 years of age were recruited in the Latrobe Valley, Victoria, Australia. Some 36% of participating children were asthmatic. Six sampling visits were made to each house between March 1994 and February 1995 on a 2-monthly cycle. Samples for airborne total and viable fungal spores were collected from bedrooms, living rooms, kitchens and outdoors. A detailed dwelling characterization, using a questionnaire and inspection surveys, was carried out. Skin-prick tests were performed with extracts of common aeroallergens and a respiratory questionnaire was completed for each child.

**Results** Large airborne fungal spore concentrations were recorded in association with: musty odour, water intrusion, high indoor humidity, limited ventilation through open windows, few extractor fans and failure to remove indoor mould growth. Visible mould growth or condensation evidence was associated with large concentrations of *Cladosporium* spores, but not with large total spore concentrations. *Penicillium* exposure was a risk factor for asthma, while *Aspergillus* exposure was a risk factor for atopy. Fungal allergies were more common among children exposed to *Cladosporium* or *Penicillium* in winter or to musty odour. Respiratory symptoms were marginally more common with exposure to *Cladosporium* or total spores in winter.

**Conclusion** Indoor exposure to certain fungal genera in winter was a risk factor for asthma, atopy and respiratory symptoms in children. On the other hand, no significant associations were seen between average viable or total spore concentrations and child health. Actual measurements of fungal spores predict health outcomes better than reported dampness.

**Keywords:** allergy, asthma, dampness, fungal spores, housing factors, indoor air, moulds, respiratory symptoms

*Clinical and Experimental Allergy*, Vol. 28, pp. 459–467. Submitted 3 April 1997; revised 3 November 1997; accepted 10 November 1997.

Correspondence: M. A. Hooper, School of Applied Sciences, Monash University, Churchill, Victoria 3842, Australia.

© 1998 Blackwell Science Ltd

459

## Introduction

Several large epidemiological studies have identified damp living conditions as a major risk factor for respiratory symptoms in children [1–4]. While these epidemiological studies were able to show that the respiratory health of children was adversely affected by damp living conditions, they provided little detail about the underlying mechanism for the association. Since both fungi and house dust mites thrive in damp conditions, an allergic reaction to either or both of these allergens is suggested as an important contributing factor in causing more respiratory symptoms among children residing in damp houses [5]. However, other mechanisms may also be important, as suggested by the results of a Canadian study where such allergies did not appear to account for more than part of the increase in disease [1]. An increased exposure to fungal metabolites such as  $\beta$ -glucans and mycotoxins has been suggested as such an alternative mechanism, as these compounds can have general immuno-suppressant or irritant properties and may therefore adversely influence the respiratory system [6].

While the health risks associated with exposure to dampness are now recognized, there is no reliable objective method of measuring exposure to dampness in a house which is suitable for epidemiology. In most studies, a simple subjective questionnaire has been used to classify houses as damp or affected by fungal contamination. While such classification has been useful in identifying the problem, more specific methods are needed in order to quantify the specific fungal exposure which is relevant to health. Air sampling for spore concentrations has been used in many previous studies to measure fungal exposure, but most studies using these methods have not included both a health assessment of the exposed subjects as well as environmental measurements. This paper presents data from a study where a detailed assessment of indoor environmental factors, including exposure to airborne fungi, was combined with an assessment of health outcomes in children. Consequently, a comparison of different ways of classifying exposure to indoor dampness and fungal exposure could be made and specific factors associated with child health could be identified. The purposes of this paper were to identify indoor environmental factors associated with airborne fungal spore concentrations and, to identify relevant exposure measures associated with respiratory illness in children.

## Materials and methods

Eighty households with children between 7 and 14 years of age, residing in the Latrobe Valley, Victoria, Australia, were recruited as volunteers from local schools and doctor's

surgeries. Approval was obtained from the Standing Committee on Ethics in Research on Humans at Monash University (project number 73/93). Forty-three of the households had at least one asthmatic child between 7 and 14 years, diagnosed by a doctor, while the remaining 37 households had only non-asthmatic children. All children in the age-group living in the 80 houses were included as participants, making a total of 148 study children. Their mean age was 10.2 years at the start of the study, and equal numbers of girls and boys were included, i.e. 74 of each gender. Six visits were made to each house over a period of 1 year, with the first in March–April 1994, and the following on a 2-monthly cycle, finishing in January–February 1995.

### *Determination of airborne fungal spore levels*

Air samples were collected for determination of both viable (culturable) and total fungal spore levels after some activity in the room. All samples were collected between 08.00h and 18.00 h. Viable fungal spore samples were collected from bedrooms of study children, living rooms, kitchens, and outside each house during each of the six sampling periods. A one-stage Andersen sampler (Andersen Instruments Inc., Atlanta, Georgia, USA) was operated at a flow rate of 24 L/min for 2 min and malt extract (20 g Oxoid CM057B/L) solidified with technical agar no. 3 (14 g Oxoid LP013B/L) was used as the collection media. No additions for prevention of bacterial growth were used. Samples were incubated for 4–5 days prior to counting the colonies. These counts were converted using the positive hole conversion method [7], and results are expressed as concentrations of colony forming units per cubic meter (CFU/m<sup>3</sup>). Identification of viable colonies to generic level was performed for the winter (July) and late spring (November) sampling periods.

Total fungal spore samples were collected from bedrooms, living rooms and outside the house during the six sampling periods using a Burkard personal spore trap (Burkard Manufacturing Co., Rickmansworth, UK), operated at a flow rate of 10 L/min for 9 min. Particles in the sampled air were trapped on a thin layer of silicone grease on a microscope slide. Fungal spores were counted on 5% of the total deposition area using a magnification of  $\times 1000$  [8]. Results are expressed as concentrations of spores per cubic meter of air (S/m<sup>3</sup>).

### *Environmental factors*

A detailed characterization was made of each dwelling. During the first sampling visit, both a dwelling questionnaire and a survey were completed. Collected data included the resident's opinion about house dampness, house age, foundation type, cladding type, extent of garden beds,

shading of house, frequency of ventilation through open windows, presence of air conditioners, fans and insulation. In addition, a detailed house survey looking for evidence of water intrusion, condensation, visible mould growth, and musty odour was performed in each sampled room during every sampling visit. Temperature and humidity were measured in all sampled rooms at the time of sample collection.

#### Health outcomes

A respiratory questionnaire was completed for each child during an interview with a parent during the last visit to each household. The questionnaire was based on the Monash Respiratory Questionnaire for which the reliability and validity has been established in adults [9]. The frequency of eight respiratory symptoms was recorded: cough, shortness of breath, waking due to shortness of breath, wheeze, asthma attacks, chest tightness, cough in the morning, and chest tightness in the morning. A child was considered symptomatic if at least one of these respiratory symptoms was present. In addition, the questionnaire included items about parental asthma, parental allergy, and presence of pets or smokers in the house.

Skin-prick tests were performed for 145 study children (three children were not tested) using 12 common aero-allergens (Hollister Stier, Spokane, WA, USA) including: cat, dog, grass mix no. 7, Bermuda grass, house dust, and house dust mites (*D. pteronyssinus* and *D. farinae*). In addition five fungal extracts were included: *Alternaria alternata* (*A. tenuis*), *Cladosporium* (*Hormodendrum*) *cladosporioides*, *Penicillium* mix, *Aspergillus* mix (for details see Hollister Stier catalogue), and Mould mix A (mixture of *Aspergillus* mix, *Botrytis cinerea*, *Chaetomium indicum*, *Epicoccum nigrum*, *Fusarium vasinfectum*, *Geotrichum candidum*, *Helminthosporium interseminatum*, *Monilia sitophila*, *Mucor racemosus*, *Phoma* spp., *Penicillium* mix, *Aureobasidium* (*Pullularia*) *pullulans*, *Rhizopus stolonifer* (*R. nigricans*), *Rhodotorula glutinis* and *Saccharomyces cerevisiae*). A saline solution was used as a negative control, while a histamine solution (10 mg/mL) was used as a positive control. Tests were performed by a trained technician between August and October 1994. The largest weal diameter was measured 15 min after pricking, and the ratio of the size of the allergen weal divided by the histamine weal was calculated. Tests were considered positive if this ratio was equal to or greater than 0.5 [10].

#### Statistical methods

Statistical analyses of data were performed using SPSS for Windows version 6.0 (SPSS Inc., Chicago, USA, 1993), with Genstat 5, release 3.1 (Oxford University Press Inc., New York, USA, 1994) being used for logistic regression

modelling [11]. Fungal spore concentration data were positively skewed and the non-parametric Mann-Whitney and Kruskal-Wallis tests were therefore used for comparisons. A  $\log_{10}$ -transformation was performed in order to gain a near-normal distribution for further analyses. Multiple linear regression was used to identify risk factors for indoor fungal spore contamination, using forward selection of variables with an entry criterion of  $P < 0.05$ . Logistic regression was applied to calculate adjusted odds ratios for asthma, allergies and respiratory symptoms with exposure to indoor spores and evidence of dampness. For the logistic regression models the proportion of children with asthma (or allergy or respiratory symptoms) in each house was used as the dependent variable since more than one child was included from some houses and independence between observations could not be assumed for data concerning the individual children. Many associations were tested and there is therefore a risk of finding false significant associations by chance. By reporting both significant and nonsignificant associations and (in some instances) indicating  $P$ -values for significant associations, and also by relating the results to previous literature, it is believed that this potential problem has been addressed. If not otherwise stated, associations and correlations were considered significant if  $P < 0.05$ .

## Results and discussion

#### Fungal spore concentrations

The median indoor viable fungal spore concentration for all samples over the year of study was 812 CFU/m<sup>3</sup>, with a range from <20–54 749 CFU/m<sup>3</sup>. The median total spore concentration was 7778 S/m<sup>3</sup>, ranging from 667 to 118 222 S/m<sup>3</sup>. Seasonal variation was apparent with an approximate tripling of concentrations in summer compared with winter for both viable and total spore concentrations indoors as well as outdoors. Spore concentrations indoors were significantly smaller than those outdoors for every sampling period, with the exception of total spore concentrations in early spring (September). *Cladosporium*, *Penicillium*, and yeasts were the three most common colony types, together accounting for 73% of colonies in winter and 86% in late spring. In addition, the genera *Aspergillus* (2.6%), *Cephalosporium* (2.2%), and *Botrytis* (1.1%) were relatively common in winter [8].

#### Health outcomes

Some 36% of the 148 study children were diagnosed as asthmatic by a doctor. Of the asthmatic children, 83% were atopic (had at least one positive skin prick test), while 48% of non-asthmatics were atopic. The most common skin test

**Table 1.** Percentage of asthmatic ( $n=52$ ) and non-asthmatic children ( $n=93$ ) with a positive reaction to each of the fungal extracts, with 95% confidence intervals (CI)

Extract	Asthmatic children		Non-asthmatic children	
	%	95% CI	%	95% CI
<i>Cladosporium cladosporioides</i>	23*	12-35	8	2-13
<i>Penicillium</i> mix	15*	6-25	5	1-10
<i>Aspergillus</i> mix	12	3-20	5	1-10
<i>Alternaria alternata</i>	21	10-32	17	10-25
Mould mix A	19	8-30	10	4-16

\* Significantly ( $\chi^2$  test,  $P < 0.05$ ) higher proportion compared with non-asthmatics.

reactivity in both groups was to the house dust mite, *D. pteronyssinus*, (81% of asthmatics and 39% of nonasthmatics). Some 31% of asthmatic children gave a positive reaction to at least one fungal extract, compared to 23% of non-asthmatic children. A positive reaction to *Cladosporium cladosporioides* was most common among asthmatics, while reactions to *Alternaria alternata* were most prevalent among non-asthmatics (Table 1). A positive reaction to a fungal extract in the absence of a reaction to house dust mite extracts was seen for five non-asthmatic children, but not among asthmatic children. Respiratory symptoms were experienced by 94% of asthmatic children and by 48% of non-asthmatic children.

#### House characteristics

Evidence of dampness was common in the study houses with visible mould growth present in every house at some time during the study, and evidence of condensation in 92% of houses. Water intrusion was observed in 40% of houses and the investigator experienced musty odour in 67% of houses. The common problem of indoor mould growth is manifested by the fact that 60% of households removed mould growth from the house on a regular basis, yet only 23% of residents considered their house damp (including structural dampness and dampness from condensation). Consequently, while most residents know that mould growth is occurring in the house, they do not necessarily regard this as a sign of a damp house or a problem which should be rectified.

Significant differences between rooms were seen for severity of visible mould growth ( $\chi^2 = 67.6$ , d.f. = 6,  $P < 0.001$ ), condensation ( $\chi^2 = 80.8$ , d.f. = 6,  $P < 0.001$ ), and for severity of musty odour ( $\chi^2 = 16.3$ , d.f. = 4,

**Table 2.** Median viable (CFU/m<sup>3</sup>) and total spore concentrations (S/m<sup>3</sup>) by house variables in 80 houses

Variable	<i>n</i>	Viable spores	Total spores	
Open windows, months of the year	2-6	11	854	12 611*
	6-10	19	1055	11 206*
	>10	50	1056 <sup>#</sup>	8951
Foundation	Stumps	50	1024	10 052*
	Slab	30	962	8511
Cracks in cladding	yes	13	1108	12 611*
	no	66	970	9125
Remove mould growth	yes	31	965	9128
	no	47	1036	11 206*
Insulation score†	1	38	1110*	9882
	2	12	994	9298
	3	24	933	8951
	4	6	933	8951
Extractor fans	1	9	1238	9128
	2	44	1019	9326
	3	26	985	8960

\*  $P < 0.05$  <sup>#</sup>  $P < 0.10$ . † Number of locations with insulation (floor, ceiling, walls).

$P < 0.002$ ). Bedrooms were consistently associated with the most severe dampness problems and winter was the season when most problems occurred ( $P < 0.001$ ). It can also be noted that bedrooms were cooler ( $P < 0.001$ ) and the relative humidity was higher ( $P = 0.006$ ) compared to other rooms. These results suggest that heating of the bedrooms as well as the living areas could prevent much of the demonstrated problems with indoor mould growth by lowering indoor humidity and the risk of condensation in winter.

#### Environmental factors associated with fungal spore concentrations

Environmental factors were tested for an association with airborne fungal spore concentrations. Outdoor concentrations of fungal spores were significantly correlated ( $P < 0.001$ ) with indoor concentrations for both viable ( $r = 0.41$ ) and total spores ( $r = 0.52$ ) [8]. Over the year of sample collection, the mean indoor relative humidity (%) was significantly, but weakly correlated with both viable ( $r = 0.28$ ), and total ( $r = 0.22$ ) spore concentrations. However, the correlation was not significant for all sampling periods and was in some cases reversed in direction (data not shown) [8]. In the present study, humidity was measured at the time of sample collection and this may not estimate well the humidity in the microclimate which determines the possibility of fungal growth [12]. This may explain the lack of a strong consistent correlation between humidity and fungal spore concentrations.

Viable and total spore concentrations are related to some housing factors in Table 2. For viable spores, a significant association was seen with insulation score only, suggesting larger concentrations in houses with little insulation. It is possible that houses with less insulation have a more open structure allowing outdoor air to enter the indoor environment, thereby increasing the indoor viable spore concentration. Another possibility is that lack of insulation increases the risk of condensation on indoor surfaces and therefore allows moisture accumulation and fungal growth. Total spore concentrations were significantly larger in association with: stump type foundation, cracks in the cladding, limited ventilation through open windows and failure to remove mould growth from the house. These results suggest that uncontrolled ventilation through cracks in the cladding and through the foundation of the house may increase indoor total spore concentrations, while controlled ventilation through open windows may reduce spore concentrations. No significant associations were seen between spore concentrations and: air conditioning, gas stove presence, damp house (resident's opinion), blocked subventilation, cladding type, soil type, drainage, garden beds around house or house age.

During the house surveys, any evidence of dampness in sampled rooms or presence of an open window at the time of sample collection was recorded. Associations with viable and total airborne spore concentrations were tested separately for each sampling period. Evidence of condensation was significantly associated with larger total spore concentrations in spring only, while no significant association with viable spore concentrations was evident. Visible mould growth was associated with smaller concentrations of viable spores, but was not significantly associated with total spore concentrations. It is very difficult to explain why significantly smaller viable spore concentrations would be found in rooms with visible mould growth. However, one possibility is that rooms without visible mould were more likely to have an open window at the time of sample collection, thus allowing the more concentrated air spora from outdoors to enter the indoor environment. Musty odour, water intrusion and presence of an open window at the time of sample collection were all significantly associated with larger concentrations of viable and total spore levels in at least one season. Previously, large concentrations of airborne fungal spores in association with visible mould growth and in complaint homes have been reported [13,14], but some studies have found no difference in spore concentrations between houses with mould problems and reference houses [15,16]. Suggested reasons for a lack of an association in some studies have included the variable nature of airborne spore concentrations and a variable release of spores from indoor sources [16]. In the present study, data on house dampness were available from six

sampling periods and associations with spore concentrations could therefore be tested for each period. The results were not consistent between sampling periods, confirming the variability of the association and suggesting a substantial seasonal component. However, presence of musty odour or water intrusion in a room were consistently associated with larger indoor fungal spore concentrations.

The two common fungal genera *Cladosporium* and *Penicillium* were tested for associations with environmental factors. Houses with ventilation through open windows for 2–6 months of the year had significantly larger *Cladosporium* concentrations (median 335 CFU/m<sup>3</sup>) compared with houses with ventilation through open windows for more than 10 months of the year, (median 187 CFU/m<sup>3</sup>). This suggests that regular ventilation through open windows can significantly reduce indoor *Cladosporium* concentrations, despite outdoor concentrations being larger. A possible explanation for this is a reduction in indoor humidity with more ventilation at suitable times of the day. *Penicillium* concentrations were significantly larger in houses where only the ceiling was insulated (75 CFU/m<sup>3</sup>), compared with houses with insulation in ceilings, walls and floors (38 CFU/m<sup>3</sup>). The larger *Penicillium* concentrations in houses lacking wall and floor insulation may point to greater condensation risk and a resulting fungal growth potential in such houses.

Table 3 summarizes associations for *Cladosporium* and *Penicillium* concentrations with measures of dampness in sampled rooms. *Cladosporium* concentrations were significantly larger in rooms with: substantial visible mould growth, substantial condensation evidence, a mean relative humidity <60%, and open windows on most sampling occasions. The larger *Cladosporium* concentration in rooms with visible mould growth or condensation suggests

**Table 3.** Median *Cladosporium* and *Penicillium* concentrations (CFU/m<sup>3</sup>) in association with measures of dampness and presence of open windows in rooms (n = 293)

Variable		n	Cladosporium	Penicillium
Condensation	substantial	72	984*	144
	none-slight	221	744	144
Visible mould	substantial	67	998*	144
	none-slight	222	458	144
Musty odour	yes	176	816	168
	no	112	768	144
Open windows	yes	164	936*	144
	> 50% of visits	129	720	144
Relative humidity	yes	58	600	216*
	> 60%	235	912*	144

\* Significantly ( $P < 0.05$ ) larger concentration.



Table 4. Multiple linear regression results using mean indoor total spore concentrations as dependent variable ( $\log_{10}$ -transformed,  $S/m^3$ )

Variable	Estimate (B)	SE (B)	T-statistic	P
Mean outdoor spore conc., $\log_{10}$ -transformed	0.391	0.078	5.03	<0.001
Months of the year with open windows*	-0.047	0.020	-2.32	0.02
Remove mould growth †	-0.070	0.029	-2.42	0.02
Substructure, stumps †	0.085	0.030	2.84	0.006
Extractor fans ‡	-0.039	0.022	-1.79	0.08
(Intercept)	2.55	0.355	7.18	<0.001
			R = 0.67	
			R <sup>2</sup> = 0.45	

\* Coded as 1 = 2–6 months, 2 = 6–10 months, and 3 = > 10 months. † Dichotomous variables (1 = yes, 0 = no).

‡ Number of sites with a fan (bath, kitchen, toilet).

that a substantial proportion of indoor moulds are members of the *Cladosporium* genus. This is consistent with the finding that damp homes had significant indoor sources of *Cladosporium* in Finland [17], and with evidence of *Cladosporium* growth on damp surfaces in the United Kingdom [14]. The higher *Cladosporium* concentration in rooms with a mean humidity <60% is surprising. A possible explanation for this would be that drier rooms have more ventilation through open windows during sample collection which would lead to a reduced humidity ( $P=0.04$ ), and also allow entry of outdoor *Cladosporium* spores to the indoor environment (Table 3). It is also possible that dry conditions are more conducive to the dispersal of *Cladosporium* spores. *Penicillium* concentrations were significantly larger in association with a mean humidity above 60% only. In winter, musty odour was associated with larger concentrations of *Penicillium* ( $P=0.03$ ) and marginally with larger *Cladosporium* concentrations ( $P=0.10$ ).

A multiple linear regression model predicting mean total spore concentrations in the house was developed (Table 4). After controlling for outdoor spore concentrations, significant risk factors for large total spore concentrations were: a lack of regular ventilation through open windows for much of the year, failure to remove mould growth, and stump type rather than concrete slab foundation. Few extractor fans in wet areas was a marginally significant risk factor. The associations with indoor relative humidity and cracks in the cladding seen in bivariate analyses were no longer significant after adjustments. A linear regression model predicting viable spore concentrations is not presented due to paucity of significant predictors. These results would suggest that removal of mould growth may be an effective way of decreasing indoor reserves of dead spores, thus decreasing airborne total spore concentrations. Extractor fans and ventilation through open windows are likely to decrease the indoor moisture load and thereby lead to a

decreased risk of fungal contamination. The association with stump type foundation was also seen in a previous study in the Latrobe Valley, but in that case it did not remain significant after adjustment for the age of the house [18], while in the present study there was no significant association between house age and total spore levels.

Suggestions for remedial action in houses with fungal problems would include installing more insulation (ceilings, walls and floors), adequate ventilation (via open windows and extractor fans), removal of any visible mould growth and heating of the whole house to avoid high relative humidity and condensation in winter.

#### Associations with health outcomes

Asthma in children was significantly associated with exposure to *Penicillium* in winter. The odds ratio for asthma with an increase in the exposure to *Penicillium* by 100 CFU/m<sup>3</sup> was 1.43 (95% CI 1.03–2.00), adjusted for parental asthma and parental allergy. This estimate was strongly influenced by three cases and if these were excluded, the association was no longer significant. Thus, the association could be a chance finding. However, there are previous reports of an association between *Penicillium* exposure and asthma [19,20] so despite the weak association found in this study, *Penicillium* is likely to be clinically important. No other measure of spore exposure or dampness (musty odour, damp house — resident, damp house — investigator, visible mould growth, condensation evidence) showed a significant association with asthma.

Atopy was significantly associated with exposure to *Aspergillus* spores. The adjusted odds ratio for atopy with an increase by 10 CFU/m<sup>3</sup> was 1.48 (95% CI 1.10–1.99), adjusted for gender and parental asthma. No other significant associations between atopy and fungal exposure measures or dampness were seen.

Table 5. Odds ratios (OR) with 95% confidence intervals (95% CI) for fungal allergies with winter exposure to *Cladosporium* and *Penicillium* spores for 145 children, adjusted for parental asthma and gender

Extract	Proportion with positive reaction	Cladosporium + 100 CFU/m <sup>3</sup>		Penicillium + 100 CFU/m <sup>3</sup>	
		OR	95% CI	OR	95% CI
<i>Cladosporium cladosporioides</i>	13%	1.24	1.00–1.54	1.24	0.92–1.67
<i>Penicillium</i> mix	9%	1.29	1.02–1.62	1.60	1.13–2.18
<i>Aspergillus</i> mix	8%	1.37	1.07–1.76	1.42	1.04–1.95
<i>Alternaria alternata</i>	19%	1.12	0.91–1.36	1.18	0.88–1.57
Mould mix A	13%	1.16	0.95–1.43	1.19	0.88–1.62
<i>D. pteronyssinus</i>	49%	1.02	0.93–1.13	1.22	0.89–1.67
<i>D. farinae</i>	46%	1.03	0.95–1.11	1.32	0.95–1.85
House dust	34%	1.06	0.96–1.17	1.49	1.05–3.17
Dog	16%	1.02	0.91–1.15	1.46	1.09–1.96
Cat	24%	1.08	0.98–1.19	1.21	0.92–1.59
Bermuda grass	31%	1.04	0.94–1.15	1.12	0.85–1.47
Grass mix no. 7	43%	1.03	0.95–1.11	1.12	0.85–1.47

Positive reactions to some extracts included for skin-prick testing were more common with greater exposure to *Cladosporium* or *Penicillium* in winter (Table 5). Associations with spore exposure in late spring were much weaker and never significant (data not shown). As one might expect, a positive reaction to *Cladosporium cladosporioides* was more likely with exposure to *Cladosporium* spores, and a reaction to the *Penicillium* mix extract was more likely with exposure to *Penicillium* spores. However, exposure to these genera was also a risk factor for a positive reaction to other fungal extracts, and surprisingly, *Penicillium* exposure was a significant risk factor for house dust and dog allergies. These latter two associations are likely to be chance findings. The significant associations between allergy and exposure to differing fungal genera may be explained by crossreactivity which is known to occur between species and even genera of moulds [21], so that for example a positive reaction to *Aspergillus* can also occur among those sensitized to *Penicillium*. Furthermore, it should be recognized that the sensitization to fungi for most study children would have occurred well before the exposure was measured in this study which may therefore not closely reflect the exposure at the time of sensitization. Another possibility is that exposure to any fungi indoors could have the potential to increase the risk of allergic sensitization to any allergen. General immunosuppressant properties of some fungal metabolites, such as mycotoxins and volatile organic compounds [22], demonstrate the possibility of a nonspecific effect on the immune system of fungal exposure. Such a general effect of fungal exposure on the

immune system can also explain the significantly increased risk of atopy with exposure to *Aspergillus* spores since several *Aspergillus* species are known to produce mycotoxins [6].

Exposure to *Aspergillus* spores was not a significant risk factor for a positive reaction to the *Aspergillus* mix, and exposure to *Alternaria* spores was not a significant risk factor for a reaction to the *Alternaria alternata* extract (data not shown). A possible reason for this lack of association for *Alternaria* and *Aspergillus* is that exposure to those genera was not as common as to *Cladosporium* and *Penicillium* and the sample size may therefore have been too small to detect an association.

There was some evidence for a greater risk of fungal allergies with exposure to musty odour. The odds ratio for a positive reaction to mould mix A was 4.79 (95% CI 1.00–22.92), adjusted for gender and parental asthma, but a positive reaction to the other fungal extracts was not significantly more likely with musty odour. Furthermore, children exposed to musty odour had a significantly greater number of positive skin-prick tests compared with children not exposed to musty odour ( $P = 0.03$ ), suggesting a more severe allergy with exposure to musty odour. This association between allergy and exposure to musty odour could be attributed to the general immunosuppressant effect of an increased exposure to fungal products (discussed above) in houses with musty odour since this is a non-specific sign of fungal contamination [23].

A marginally significant odds ratio for the presence of respiratory symptoms was seen (adjusted for parental

asthma) with an increase in the indoor *Cladosporium* exposure by 500 CFU/m<sup>3</sup> (OR = 1.92; 95% CI 0.96–3.80). Similarly, the specific respiratory symptoms of cough (OR = 2.11; 95% CI 1.11–4.03) and wheeze (OR = 1.58; 95% CI 1.00–2.50) were significantly more common with *Cladosporium* exposure (+ 500 CFU/m<sup>3</sup>). These findings lend further support to previous reports of *Cladosporium* exposure being associated with respiratory illnesses [24–26]. Total spore concentrations in May (late autumn) were a marginal risk factor for respiratory symptoms (adjusted for parental asthma) with the odds ratio for an increase in late autumn total spores by 5000 S/m<sup>3</sup> 1.76 (95% CI 0.97–3.19). On the other hand, mean total spore concentrations were not significantly associated with respiratory symptoms. Interestingly, one previous study has found that total spore exposure in winter is a risk factor for respiratory symptoms [24], suggesting that fungal exposure in this season is important for health. Previous findings of respiratory symptoms such as wheeze being associated with visible mould growth in houses [27] were not supported by this study.

In conclusion, results presented in this paper suggest a large overall effect of fungal exposure on child health (especially in winter). Asthma, atopy and respiratory symptoms were all significantly associated with exposure to one or more genera of fungal spores. On the other hand, average concentrations of viable or total fungal spores were not significantly associated with health outcomes. These results are in agreement with those reported by Strachan *et al.* [27] in that no significant association between total viable mould concentrations and health outcomes were seen despite significant associations with specific genera. This implies that measurements of specific fungal spore genera concentrations predict health outcomes better than total spore concentrations, reported dampness or observed dampness.

#### Acknowledgements

Participating families are thanked for their valuable contribution to the study. Ms Mary Cole, Charles Sturt University, Wagga Wagga assisted in the identification of fungi and provided mycological knowledge. Drs Thad and Diana Godish, Ball State University, Muncie, Indiana, USA were helpful in providing information about the methods for airborne fungal spore sampling. Ms Maria Garrett was the recipient of a Monash University postgraduate publications award while preparing this manuscript.

#### References

- 1 Dales RE, Zwanenburg H, Burnett R, Franklin CA. Respiratory health effects of home dampness and molds among Canadian children. *Am J Epidemiol* 1991; 134:196–203.
- 2 Spengler J, Neas L, Nakai S *et al.* Respiratory symptoms and housing characteristics. *Indoor Air* 1994; 4:72–82.
- 3 Brunekreef B, Dockery DW, Speizer FE *et al.* Home dampness and respiratory morbidity in children. *Am Rev Resp Dis* 1989; 140:1363–7.
- 4 Li C-Y, Hsu L-Y. Home dampness and childhood respiratory symptoms in a subtropical climate. *Arch Environ Health* 1996; 51:42–6.
- 5 Brunekreef B, Verhoeff AP, van Strien RT, van Wijnen JH. Home dampness and childhood respiratory symptoms: the role of sensitisation to dust mites and moulds. In: Samson RA, Flannigan B, Flannigan ME, Verhoeff AP, Adan OCG and Hoekstra ES, eds *Health implications of fungi in the indoor environment*. Amsterdam, Netherlands: Elsevier Science, 1994: 189–99.
- 6 Jarvis BB. Mycotoxins and indoor air quality. In: Morey PR, Feeley JC, Otten JA, eds *Biological contaminants in indoor environments*, ASTM STP 1071, Philadelphia, American Society for Testing and Materials, 1990.
- 7 Andersen AA. New sampler for the collection, sizing and enumeration of viable airborne particles. *J Bacteriol* 1958; 76:471–84.
- 8 Garrett MH, Hooper BM, Cole FM, Hooper MA. Airborne fungal spores in 80 homes in the Latrobe Valley, Australia: levels, seasonality and indoor-outdoor relationship. *Aerobiologia* 1997; 17:121–6.
- 9 Abramson M, Kutin J, Bowes G. The prevalence of asthma in Victorian adults. *Aust NZ J Med* 1992; 22:358–63.
- 10 Meinert R, Frischer T, Karmaus W, Kuehr J. Influence of skin prick tests criteria on estimation of prevalence and incidence of allergic disease in children. *Allergy* 1994; 49:526–32.
- 11 Hosmer DW, Lemeshow S. *Applied logistic regression*. New York, John Wiley & Sons, 1989.
- 12 Flannigan B, Morey PR. Control of moisture problems affecting biological indoor air quality. Ottawa: International Society of Indoor Air Quality and Climate, ISIAQ guideline: Task force 1, 1996.
- 13 Li CS, Hsu LY. Airborne fungus allergen in association with residential characteristics in atopic and control children in a subtropical region. Proceedings of the 7th international conference on indoor air quality and climate. Nagoya, Japan, 1996; 3:411–6.
- 14 Hunter CA, Grant C, Flannigan B, Bravery AF. Mould in buildings: the air spora of domestic dwellings. *Int Biodeter* 1988; 24:81–101.
- 15 DeKoster JA, Thorne PS. Bioaerosol concentrations in non-complaint, complaint, and intervention homes in the Midwest. *Am Ind Hyg Assoc J* 1995; 56:573–80.
- 16 Nevalainen A, Pasanen A-L, Niininen M, Reponen T, Kallioikoski P, Jantunen MJ. The indoor air quality in Finnish homes with mold problems. *Environ Int* 1991; 17:299–302.
- 17 Pasanen A-L, Niininen M, Kallioikoski P, Nevalainen A, Jantunen MJ. Airborne *Cladosporium* and other fungi in damp vs. reference residences. *Atm Environ* 1992; 26B:121–4.
- 18 Godish D, Godish T, Hooper B, Hooper M, Cole M. Airborne

- mould levels and related environmental factors in Australian houses. *Indoor Built Environ* 1996; 5:148-54.
- 19 Tariq SM, Matthews SM, Stevens M, Hakim EA. Sensitisation to *Alternaria* and *Cladosporium* at the age of 4 years. *Clin Exp Allergy* 1996; 26:794-8.
- 20 Licorish K, Novoy HS, Kozak P, Fairshier RD, Wilson AF. Role of *Alternaria* and *Penicillium* spores in the pathogenesis of asthma. *J Allergy Clin Immunol* 1985; 76:819-25.
- 21 Salvaggio J, Aukrust L. Mold-induced asthma. *J Allergy Clin Immunol* 1981; 68:327-46.
- 22 Flannigan B, Miller JD. Health implications of fungi in indoor environments - an overview. In: Samson RA, Flannigan B, Flannigan ME, Verhoeff AP, Adan OCG and Hoekstra ES, eds *Health implications of fungi in the indoor environment*. Amsterdam, Netherlands: Elsevier Science, 1994: 3-28.
- 23 Gravesen S, Frisvald JC, Samson RA. *Microfungi*. Copenhagen: Munksgaard, 1994.
- 24 Godish TJ, Godish DR, Hooper BM, Hooper MA. Latrobe Valley indoor air quality study: health and house variables. In: Morawska L, Bofinger ND and Maroni M, eds *Oxford, Elsevier Science Ltd, 1995: 211-4*.
- 25 Stout C, Kotses H, Carlson BW, Creer TL. Predicting asthma in individual patients. *J Asthma* 1991; 28:41-7.
- 26 Targonski PV, Persky VW, Ramekrishnan V. Effect of environmental molds on risk of death from asthma during the pollen season. *J Allergy Clin Immunol* 1995; 95:955-61.
- 27 Strachan DP, Flannigan B, McCabe EM, McGarry F. Quantification of airborne moulds in the homes of children with and without wheeze. *Thorax* 1990; 45:382-7.

## Appendix AE: Pettigrew et al. (2004) Association of Early-Onset Otitis Media in Infants and Exposure to Household Mould

441

### Association of early-onset otitis media in infants and exposure to household mould

Melinda M. Pettigrew, Janneane F. Gent, Elizabeth W. Triche, Kathleen D. Belanger, Michael B. Bracken and Brian P. Leaderer  
Yale Center for Perinatal, Pediatric and Environmental Epidemiology, Department of Epidemiology and Public Health, Yale University School of Medicine, New Haven, CT, USA

#### Summary

**Correspondence:**  
Melinda M. Pettigrew, Yale University School of Medicine, Department of Epidemiology and Public Health, 60 College Street, PO Box 208034, New Haven, CT 06520-8034, USA.  
E-mail: melinda.pettigrew@yale.edu

Otitis media is one of the most common infections of early childhood. Children who first experience acute otitis media at an early age (before 6 months) are at increased risk for recurrent otitis media. This prospective study investigated exposure to measured levels of airborne household mould and the risk of early otitis media in the first 6 months of life among a cohort of infants at high risk for asthma. Between September 1996 and December 1998, women were invited to participate if they had at least one other child with physician-diagnosed asthma. Mothers were given a standardised questionnaire within 4 months of their infant's birth. Airborne mould samples were also taken at this time, and culturable fungi were categorised into four levels according to the report of the Commission of European Communities: 0 (undetectable), 1–499 colony forming units (CFU)/m<sup>3</sup> (low), 500–999 CFU/m<sup>3</sup> (medium), ≥1000 CFU/m<sup>3</sup> (high). Infant respiratory symptoms were collected during quarterly telephone interviews at 6, 9 and 12 months of age.

Of the 806 children in the study, 27.8% experienced otitis media before six months of age. Household levels of *Penicillium* and *Cladosporium* were modestly associated with the number of otitis media episodes ( $P = 0.056$  and  $0.081$  respectively). After controlling for potential confounders, *Penicillium* and *Cladosporium* were not associated with early otitis media. High levels of 'other' mould (defined as total spore count minus counts for *Penicillium*, *Cladosporium*, and yeast) were associated with early otitis media (OR 3.49; 95% CI [1.38, 8.79]). We also found associations between day-care outside of the home and birth during the summer or fall season with early otitis media. This study is suggestive of a relationship between otitis media and mould that warrants further study.

#### Introduction

Otitis media is one of the most common infections of early childhood. During their first year of life, 60–80% of children will have at least one episode of otitis media and 17% will experience three or more episodes.<sup>1,2</sup> Otitis media is the most frequent reason for physician visits due to illness among infants and young children and is the leading diagnosis for the dispensing of antibiotics.<sup>3,4</sup> Rates of early onset otitis media are increasing.<sup>5</sup> This increase is especially troubling since children who first experience acute otitis media at an early age are at increased risk for recurrent otitis media. Although controversial, some studies

have found that recurrent otitis media may increase a child's risk for developing hearing loss and delays in speech development.<sup>3,6,7</sup>

Higher rates of otitis media have repeatedly been linked to attendance in day-care.<sup>1,8,9</sup> Additional risk factors, less consistently associated with otitis media, include race, lack of breast feeding, and exposure to environmental tobacco smoke (ETS).<sup>10–11</sup> Otitis media is often associated with, or preceded by, a respiratory infection.<sup>12–15</sup> With the exception of ETS, few studies have examined the link between indoor air contaminants and otitis media, although ear infections have been associated with dampness and mould in the

home.<sup>26,27</sup> Other studies have reported a link between indoor fungal exposures and respiratory symptoms in children.<sup>18–21</sup> We hypothesised that early exposure to airborne fungi in the home would be associated with an increased risk of otitis media. The purpose of this study was to examine the relationship between levels of household mould and otitis media among a cohort of infants at high risk for asthma.

## Methods

### Cohort

Data used in this report were obtained from 1002 infants enrolled in a longitudinal cohort to study asthma development and morbidity. Between September 1996 and December 1998, women delivering babies in five Connecticut hospitals and one south central Massachusetts hospital were invited to participate if they had at least one other child at home younger than 12 years of age with physician-diagnosed asthma. Because of the asthmatic sibling, these infants were considered high risk for this disease. A full description of the methods is published elsewhere.<sup>28,29</sup> The Yale Human Investigations Committee as well as institutional review boards at each participating hospital reviewed and approved the study.

### Data collection

Trained research assistants administered standardised questionnaires to study participants during home interviews within 4 months of birth. We collected household demographic data such as maternal race, education, and number of children as well as detailed information regarding infant care (e.g. breast feeding and day-care use) and health status of the mother (e.g. self-reported history of allergies or physician-diagnosed asthma). Because race has been inconsistently associated with otitis media, we evaluated the impact of this variable on the risk of early onset otitis media and number of otitis media episodes.<sup>1,2</sup> We classified infants according to race based on the race of the mother. Mothers were asked 'In which of these groups would you place yourself (White, Black or African-American, Hispanic, Asian or other)?' Additional questions included household characteristics, self-reported presence of mould or mildew in the home, and whether the infant experienced exposure to environmental tobacco smoke for at least 2 h per week.

Infant respiratory symptoms were collected during quarterly telephone interviews at 6, 9 and 12 months of age. During these telephone interviews, we asked mothers to report their infant's respiratory symptoms and doctor or clinic visits (month and year of visit, reason for visit, and mother's report of the diagnosis). When the infant reached 1 year of age, mothers completed an extensive phone questionnaire covering household characteristics and health status specific to the previous year.

Mould sample collection has been described previously.<sup>28,29</sup> Briefly, airborne mould samples were collected from the main living area of the home using a Burkard portable air sampler (Burkard Manufacturing Co., Rickmansworth, UK) in combination with dichloran 18% glycerol agar (DG-18). The air was sampled for 1 min at an airflow rate of 20 L/min. Fungi were identified to the genus level and recorded in colony forming units (CFU) per cubic metre. Culturable fungi were categorised according to the four levels described in the report of the Commission of European Communities: 0 (undetectable), 1–499 CFU/m<sup>3</sup> (low), 500–999 CFU/m<sup>3</sup> (medium),  $\geq 1000$  CFU/m<sup>3</sup> (high).

Otitis media was determined by mother's report at each quarterly telephone interview as described above. As only the month and year of diagnosis were known, additional physician diagnoses of otitis media occurring in the same month or next consecutive month were counted as a single episode.

### Data analysis

These analyses include 806 of the original 1002 infants for whom otitis media data were available and who remained in the home where the mould samples were taken for the first 6 months of life. We excluded 128 infants for whom otitis media information was missing. Of the remaining 874 infants, 68 were excluded because of missing information on measured mould ( $n = 29$ ) or because the families moved at some unknown date ( $n = 39$ ).

Unadjusted associations between early onset otitis media (otitis media during the first 6 months of life) or number of otitis media episodes during the first year with selected study characteristics were evaluated by  $\chi^2$  tests. A logistic regression model was used to evaluate the association between household mould levels and early onset otitis media while controlling for selected characteristics. All data analyses used SAS version 8.0.<sup>28</sup>

Table 1. Unadjusted associations between measured mould, personal, demographic, and socio-economic characteristics and early otitis media (&lt;6 months of age) among 806 infants at risk for developing asthma from Connecticut and western Massachusetts 1998-2000

Characteristic	First episode of otitis media <6 months of age			P-value
	n	None (%)	Any (%)	
Infant's sex				
Male	398	70.8	29.2	0.40
Female	408	73.5	26.5	
Ethnicity				
White or other	533	68.7	31.3	0.007
Black	103	79.6	20.4	
Hispanic	170	78.8	21.2	
Education level <sup>a</sup>				
< High School diploma	98	76.5	23.5	0.24
High School Diploma some college	419	73.5	26.5	
College degree or more	289	68.9	31.1	
Day-care outside the home (first 6 months)				
No	634	74.3	25.7	0.007
Yes	162	63.6	36.4	
Breast feeding <sup>b</sup>				
No	248	71.0	29.0	0.60
Yes	558	72.8	27.2	
Maternal allergies				
No	337	72.4	27.6	0.92
Yes	469	72.1	27.9	
Maternal asthma				
No	561	72.5	27.5	0.74
Yes	245	71.4	28.6	
Smoke exposure since birth <sup>c</sup>				
No	701	72.2	27.8	0.89
Yes	103	72.8	27.2	
Season of birth				
Spring	227	81.9	18.1	<0.0001
Summer	211	65.4	34.6	
Fall	191	61.8	38.2	
Winter	177	79.1	20.9	
Reported mould				
No	622	73.8	26.2	0.04
Yes	173	65.9	34.1	
Penicillium <sup>d</sup>				
Undetectable	484	71.0	28.9	0.20
Low	274	75.9	24.1	
Medium	17	58.8	41.2	
High	31	64.5	35.5	
Cladosporium <sup>d</sup>				
Undetectable	306	73.2	26.8	0.82
Low	362	70.7	29.3	
Medium	81	75.3	24.7	
High	57	71.9	28.1	
'Other' mould <sup>de</sup>				
Undetectable	332	74.1	25.9	0.006
Low	417	71.7	28.3	
Medium	35	80.0	20.0	
High	22	40.9	59.1	

<sup>a</sup>Breast feeding defined as any breast feeding since birth, ascertained during the home interview.

<sup>b</sup>Smoke exposure for more than two hours per week since birth, ascertained during the home interview.

<sup>c</sup>U (undetectable), 1-499 CFU/m<sup>3</sup> (low), 500-999 CFU/m<sup>3</sup> (medium), ≥1000 CFU/m<sup>3</sup> (high).

<sup>d</sup>'other' defined as the total spore count minus counts for *Penicillium*, *Cladosporium* and yeast.

## Results

Of the 806 children in the study, 27.8% experienced otitis media before 6 months of age. During the first year of life, only 34% of the infants remained free of otitis media, 31.0% had at least one otitis media episode and 35% had two or more episodes. Table 1 shows the characteristics of the study participants and their distribution by early otitis media. Similar numbers of male and female infants were included in the analysis (49.4% male and 50.6% female). Sixty-six per cent of participants were white or Asian (10 Asians) and well educated, 52% had a high school diploma or some college. The majority of the infants in the study were cared for at home during the first 6 months of life (79.6%). Participants tended to breast feed their children (69.2%). Maternal allergies were common (58.1%) and 30.4% of the mothers reported a history of asthma. Reported exposure to environmental tobacco smoke was low (12.8%). Twenty-eight per cent of the households reported visible mould or mildew. The percentages of households with detectable mould levels were 40.0%, 62.0% and 58.9% for *Penicillium*, *Cladosporium* and 'other' mould respectively ('other' defined as the total spore count minus counts for *Penicillium*, *Cladosporium* and yeast).

Early-onset otitis media was associated with ethnicity, day-care outside of the home, reported mould, season of birth and levels of 'other' mould in unadjusted models (Table 1). Table 2 contains unadjusted associations between zero, one, or two or more otitis media episodes during the first year of life and season of birth and selected mould variables. While season of birth was associated with early otitis media in unadjusted models, it was not associated with the number of otitis media episodes. Reported mould was weakly associated with number of otitis media episodes and 'other' mould was not associated with number of otitis media episodes. Household levels of *Penicillium* and *Cladosporium* were modestly associated with the number of otitis media episodes ( $P=0.056$  and  $0.081$  respectively). For example, 51.8% of infants in homes with high levels of *Penicillium* experienced two or more otitis media episodes during the first year of life compared with 36.4% of infants in homes with undetectable levels of *Penicillium*. We also compared selected personal, demographic and socio-economic characteristics to zero, one, or two or more otitis media episodes. In contrast to early otitis media, male sex was associated with the number of episodes in unadjusted

Table 2. Unadjusted associations between season of birth, reported mould, measured mould, and otitis media episodes<sup>a</sup> during their first year of life among 806 infants at risk for developing asthma from Connecticut and western Massachusetts 1998–2000

Characteristic	n	Episodes of otitis media			P-value
		None (%)	One (%)	Two+ (%)	
<b>Season of birth</b>					
Spring	227	30.8	34.4	34.8	0.58
Summer	211	32.2	33.7	34.1	
Fall	191	37.2	27.2	35.6	
Winter	177	36.7	27.7	35.6	
<b>Reported mould</b>					
No	622	35.5	30.4	34.1	0.08
Yes	173	36.6	33.5	39.9	
<b><i>Penicillium</i><sup>b</sup></b>					
Undetectable	484	33.9	29.8	36.4	0.056
Low	274	35.4	35.0	29.6	
Medium	17	29.4	29.4	41.2	
High	31	25.8	13.1	58.1	
<b><i>Cladosporium</i><sup>b</sup></b>					
Undetectable	306	40.5	26.1	33.3	0.081
Low	362	30.9	32.9	36.2	
Medium	81	27.2	37.0	35.8	
High	57	28.1	36.8	35.1	
<b>'Other' mould<sup>c</sup></b>					
Undetectable	332	36.4	28.3	35.2	0.34
Low	417	32.8	32.6	34.5	
Medium	35	37.1	34.3	28.6	
High	22	13.6	36.4	50.0	

<sup>a</sup>Episodes of otitis media were based on a maternal report of a doctor's diagnosis of otitis media. Additional episodes were considered unique if they occurred at least 1 month after the previous occurrence.

<sup>b</sup>0 (undetectable), 1–499 CFU/m<sup>3</sup> (low), 500–999 CFU/m<sup>3</sup> (medium), ≥1000 CFU/m<sup>3</sup> (high) smoke exposure for more than 2 h per week since birth, ascertained during the home interview. <sup>c</sup>'other' defined as the total spore count minus counts for *Penicillium*, *Cladosporium* and yeast.

models ( $P=0.02$ ). Ethnicity and day-care outside of the home were associated with number of otitis media episodes ( $P=0.0006$  and  $0.001$  respectively).

The adjusted relationship between household mould levels and early onset otitis media was examined in a logistic regression model (Table 3). After controlling for potential confounders, reported mould, *Penicillium* and *Cladosporium* were not associated with early otitis media. 'Other' mould was associated with early onset otitis media at the highest level (OR 3.49; 95% CI [1.38, 8.79]). Day-care outside of the home and



Table 3. Adjusted logistic regression models of selected characteristics and early otitis media (<6 months of age) among 806 infants at risk for developing asthma from Connecticut and western Massachusetts 1998–2000

	First episode of otitis media <6 months of age	
	OR	[95% CI]
Infant's sex		
Male	1.00	Reference
Female	0.87	[0.63, 1.21]
Ethnicity		
White or other	1.00	Reference
Black	0.60	[0.35, 1.03]
Hispanic	0.71	[0.46, 1.09]
Day-care outside the home (first 6 months)		
No	1.00	Reference
Yes	1.69	[1.15, 2.48]
Season of birth		
Spring	1.00	Reference
Summer	2.42	[1.53, 3.82]
Fall	2.86	[1.74, 4.70]
Winter	1.24	[0.72, 2.14]
Reported mould		
No	1.00	Reference
Yes	1.57	[0.94, 2.62]
Penicillium <sup>a</sup>		
Undetectable	1.00	Reference
Low	0.75	[0.52, 1.08]
Medium	1.89	[0.67, 5.30]
High	1.27	[0.56, 2.86]
Cladosporium <sup>a</sup>		
Undetectable	1.00	Reference
Low	1.04	[0.70, 1.56]
Medium	0.92	[0.48, 1.79]
High	1.09	[0.52, 2.29]
'Other' mould <sup>b</sup>		
Undetectable	1.00	Reference
Low	1.21	[0.84, 1.74]
Medium	0.72	[0.29, 1.80]
High	3.45	[1.26, 8.76]

<sup>a</sup> 0 (undetectable), 1–499 CFU/m<sup>3</sup> (low), 500–999 CFU/m<sup>3</sup> (medium), <sup>3</sup> ≥1000 CFU/m<sup>3</sup> (high).

<sup>b</sup> 'other' defined as the total spore count minus counts for *Penicillium*, *Cladosporium* and yeast.

birth during the summer or fall season was associated with an increase in early-onset otitis media in adjusted models.

## Discussion

We found that exposure to high levels of moulds other than *Penicillium* and *Cladosporium* was associated with

early-onset otitis media. Over half (59%) of the 22 infants living in homes with high levels of 'other' mould experienced their first episode of otitis media before 6 months of age. Levels measured in these homes included the sum of counts for between 1 and 4 of the following moulds: *Aspergillus* (found in 17 of the 22 homes), *Wallemia* (6), *Alternaria* (5), *Episaccium* (3), *Botrytis* (2), *Aureobasidium* (1), and/or unidentified (10).

High levels of *Penicillium* were associated with increased rates of wheeze (RR 2.15; 95% CI [1.34, 3.46]) and persistent cough (RR 2.06; 95% CI [1.31, 3.24]) within this cohort of infants.<sup>23</sup> In contrast, levels of other mould were not associated with either cough or rates of wheeze. Given this and the strong link between otitis and respiratory illness, we were surprised that neither *Penicillium* and *Cladosporium* were associated with early onset otitis media in our cohort. *Penicillium* and *Cladosporium* were modestly associated with number of otitis media episodes in unadjusted analyses ( $P = 0.056$  and  $0.081$  respectively).

The results of this study are consistent with findings from other studies. Ear infections were associated with dampness in the home and mould in the home among 4–5 year olds in Geneva, Switzerland after adjusting for maternal age and allergy.<sup>16</sup> A case control study of children aged 6–12 living in Taiwan explored the relationship between indoor environmental factors (tobacco smoke, carpet, gas stoves, pets, and mould) and acute otitis media.<sup>17</sup> Results indicated that mould (adjusted OR 1.64 95% CI [1.08, 2.47]), dampness in the home, and flooding were associated with otitis media. Mould was defined as visible mould or mildew growth inside the home. Neither of these studies quantified levels of mould exposure or specified the type of mould. In addition, the children in these studies were older and the influence of mould may be different in these age groups.

Infants born during the summer and fall months were more likely to experience early-onset otitis media. Otitis media has been shown to undergo seasonal variation in parallel with respiratory virus infections,<sup>13</sup> with rates highest in the fall and winter months. We also found an association between early-onset otitis media and attendance in day-care outside the home. Day-care attendance or exposure to a large number of siblings at home has been consistently identified as an important risk factor for otitis media.<sup>14,22,25</sup> This is presumably due to children coming into contact with more infectious agents at an earlier age. While

black and Hispanic race were associated with early otitis media in unadjusted models, these variables were not associated with early otitis media in our final model. The association between race and otitis media has been inconsistent in the literature. Langhear *et al.* reported that black race and Hispanic ethnicity were protective for recurrent otitis media.<sup>4</sup> In contrast, a large cohort of children less than 2 years of age indicated that black children of lower socio-economic status had a higher prevalence of otitis media than white children.<sup>5</sup>

One limitation of this study was that the cohort examined was not specifically recruited to study otitis media. We collected data by asking mothers whether their infant had a clinician-diagnosed medical condition. Mothers reported a diagnosis of an ear infection but were not asked to distinguish between the different types of otitis media, thus leading to an inability to distinguish between acute otitis media and otitis media with effusion. Since the risk factors for acute otitis media and otitis media with effusion may differ,<sup>28</sup> some of our findings may have been diluted by grouping all types of otitis media together. Problems may also have occurred with relying on maternal report to count the number of otitis media episodes. We attempted to limit recall bias by asking mothers about their child's medical conditions every 3 months. One study has shown that when 3 months elapsed between study visits, parents were able to accurately report the number of otitis media episodes ( $\kappa = 0.94$ ).<sup>27</sup> Furthermore, this study used a conservative count of otitis media episodes. Two episodes occurring within the same month or consecutive months counted as one episode.

One airborne sample was used to represent mould exposure during the first 6 months of life. The combination of air sampling followed by growth on agar is considered the best of sampling methods in terms of precision, yield and the number of species identified.<sup>29</sup> Nevertheless, moulds that are not airborne, or do not grow well on the chosen medium may have been missed. Furthermore, levels of some household moulds undergo seasonal variation<sup>30</sup> and our samples were not all collected at the same time of year. Within this cohort, measured levels of *Penicillium* did not vary by season while *Cladosporium* and 'other' mould had the greatest variability in measurements collected during the summer and fall months.<sup>31</sup> The inclusion of season of birth in our adjusted regression model should control for season of mould sampling since all

mould measurements were made at the time of the home interview within 2–4 months of the infant's birth. Nevertheless, measured mould levels, which we sampled on one occasion, may not accurately represent early lifetime exposure concentrations, particularly for those moulds that vary by season.

Strengths of this study include the large sample size and prospective study design. This is a well-characterised population with extensive information regarding demographic and household information, and quantitative measurement of household fungal levels. Despite potential sampling limitations, we were able to examine the relationship between otitis media and two of the most common types of mould (*Penicillium* and *Cladosporium*).

This cohort provides valuable information regarding otitis media in a susceptible population at high risk for asthma. The results of this study are interesting given that these children are at increased risk for asthma and in turn may be more susceptible to other respiratory diseases. The risk of asthma at 4 years of age was higher among infants who experienced otitis media (OR 1.80, [95% CI 1.20, 2.60]) during the first year of life.<sup>20</sup> While the findings of this study are intriguing, the relationship between otitis media and household fungi requires further study.

#### Acknowledgements

We thank the original 1002 original families who participated in this study. We also thank the following hospitals from which our study population was selected: Yale-New Haven, Danbury, Bridgeport, Hartford (CT), and Bay State (MA). This work was supported by grants ES07456 and ES05410 from the National Institute of Environmental Health Sciences.

#### References

- 1 Paradise JL, Rockette HE, Colborn DK, Bernard BS, Smith CG, Kurs-Lasky M, *et al.* Otitis media in 2253 Pittsburgh-area infants: prevalence and risk factors during the first two years of life. *Pediatrics* 1997; 99:318–333.
- 2 Teale DW, Klein JO, Keener B. Epidemiology of otitis media during the first seven years of life in children in greater Boston: a prospective, cohort study. *Journal of Infectious Diseases* 1989; 160:83–94.
- 3 McCaig L, Hughes J. Trends in antimicrobial drug prescribing among office-based physicians in the United States. *JAMA* 1995; 273:214–219.
- 4 Schappert SM. Office visits for otitis media: United States, 1975–80. *Vital and Health Statistics of the Centers for Disease*

- Centrodlogical* (National Center for Health Statistics 1992; 214:1-18).
- 5 Arvinger P, Lamphear BP, Kalkwarf HJ, Mansour ME. Trends in otitis media among children in the United States. *Pediatrics* 2003; 112:514-520.
  - 6 Lamphear BP, Byrd RS, Arvinger P, Hall CB. Increasing prevalence of recurrent otitis media among children in the United States. *Pediatrics* 1997; 99:E1.
  - 7 Daly KA, Giebink GS. Clinical epidemiology of otitis media. *Pediatric Infectious Disease Journal* 2000; 19(S Suppl.): S31-S36.
  - 8 Uhari M, Mantysaari K, Niemela M. A meta-analytic review of the risk factors for acute otitis media. *Clinical Infectious Diseases* 1996; 22:1079-1083.
  - 9 Eitzel RA, Pattishall EN, Haley NJ, Fletcher RH, Henderson FW. Passive smoking and middle ear effusion among children in daycare. *Pediatrics* 1992; 90:228-232.
  - 10 Alho OP, Laara E, Oja H. Public health impact of various risk factors for acute otitis media in northern Finland. *American Journal of Epidemiology* 1996; 143:1149-1156.
  - 11 Lieu JE, Feinstein AR. Effect of gestational and passive smoke exposure on ear infections in children. *Archives of Pediatrics and Adolescent Medicine* 2002; 156:147-154.
  - 12 Daly KA, Brown JE, Lindgren BR, Meland MH, Le CT, Giebink GS. Epidemiology of otitis media onset by six months of age. *Pediatrics* 1999; 103:1158-1166.
  - 13 Vesa S, Kleemola M, Blomqvist S, Takala A, Kilpi T, Hovi T. Epidemiology of documented viral respiratory infections and acute otitis media in a cohort of children followed from two to twenty-four months of age. *Pediatric Infectious Disease Journal* 2001; 20:574-581.
  - 14 Heikkinen T, Chonmaitree T. Importance of respiratory viruses in acute otitis media. *Clinical Microbiology Reviews* 2003; 16:230-241.
  - 15 Cassellbrant ML, Mandel DM, Fall PA, Rockette HE, Kurs-Lasky M, Bluestone CD, et al. The heritability of otitis media: a twin and triplet study. *JAMA* 1999; 282:2125-2130.
  - 16 Rylander R, Møgevang Y. Environmental risk factors for respiratory infections. *Archives of Environmental Health* 2000; 55:200-203.
  - 17 Yang CY, Cheng MF, Tsai SS, Hung CF, Lai TC, Hwang KC. Effects of indoor environmental factors on risk for acute otitis media in a subtropical area. *Journal of Toxicology and Environmental Health Part A* 1999; 56:111-119.
  - 18 Garrett MH, Rayment P, Hooper M, Abramson MJ, Hooper BM. Indoor airborne fungal spores, house dampness and associations with environmental factors and respiratory health in children. *Clinical and Experimental Allergy* 1998; 28:459-467.
  - 19 Platz S, Martin C, Hunt S, Lewis C. Damp housing, mould growth, and symptomatic health state. *British Medical Journal* 1989; 298:1673-1678.
  - 20 Waegemasters M, van Wageningen N, Brunekreef B, Boleij J. Respiratory symptoms in damp homes. *Allergy* 1989; 44:192-198.
  - 21 Gent JF, Ren F, Belanger K, Triche E, Bracken MB, Holford TR, et al. Levels of household mold associated with respiratory symptoms in the first year of life in a cohort at risk for asthma. *Environmental Health Perspectives* 2002; 110:A781-A786.
  - 22 Leaderer BP, Belanger K, Triche E, Holford T, Gold DR, Kim Y, et al. Dust mite, cockroach, cat, and dog allergen concentrations in homes of asthmatic children in the northeastern United States: impact of socioeconomic factors and population density. *Environmental Health Perspectives* 2002; 110:419-425.
  - 23 Ren F, Jankun TM, Belanger K, Bracken MB, Leaderer BP. The relation between fungal propagules in indoor air and home characteristics. *Allergy* 2001; 56:419-424.
  - 24 SAS System for Windows, 8th ed. Cary, NC: SAS Institute, 2000.
  - 25 Hardy A, Fowler M. Child care arrangements and repeated ear infections in young children. *American Journal of Public Health* 1993; 83:1321-1325.
  - 26 Faden H, Duffy L, Boeve M. Otitis media: back to basics. *Pediatric Infectious Disease Journal* 1998; 17:1105-1113.
  - 27 Daly KA, Lindgren B, Giebink GS. Validity of parental report of a child's medical history in otitis media research. *American Journal of Epidemiology* 1994; 139:1116-1121.
  - 28 Verhoeff AP, van Wijnen JH, Boleij J, Brunekreef B, van Raemden-Hockstra ES, Samson R. Enumeration and identification of airborne viable mould propagules in houses: a field comparison of selected techniques. *Allergy* 1990; 45:275-284.
  - 29 Ren F, Jankun TM, Leaderer BP. Comparisons of seasonal fungal prevalence in indoor and outdoor air and in house dusts of dwellings in one Northeast American county. *Journal of Exposure Analysis and Environmental Epidemiology* 1999; 9:560-568.
  - 30 Nafstad P, Magnus P, Jaakkola JJ. Early respiratory infections and childhood asthma. *Pediatrics* 2000; 106:E38.

# Appendix AF: Douglas & Sullivan (2013) The Role of Child Protection in Cannabis Grow-Operations

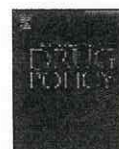
International Journal of Drug Policy 24(2013)445–448



Contents lists available at ScienceDirect

International Journal of Drug Policy

journal homepage: [www.elsevier.com/locate/drugpo](http://www.elsevier.com/locate/drugpo)



Research paper

## The role of child protection in cannabis grow-operations

Janet Douglas\*, Richard Sullivan

School of Social Work, University of British Columbia, 2080 West Mall, Vancouver, BC, Canada V1T 1Z2

### ARTICLE INFO

**Article history:**  
Received 25 August 2012  
Received in revised form 8 January 2013  
Accepted 14 January 2013

**Keywords:**  
Cannabis  
Grow-operation  
Child health  
Child welfare

### ABSTRACT

**Background:** This unique social work research examined the rationale for child protection interventions with families found living in illegal cannabis grow operations, based on the assumption of risk in the presence of probable medical harm.

**Methods:** The study examined the household, family and individual characteristics of 181 children found living in cannabis grow operations in two regions in British Columbia, Canada. Data was collected on-site on the physical characteristics of the homes, the health characteristics of the children, and their prescription drug history. Comparison of prescription drug use was also made with a group of children from the same geographic areas.

**Results:** This study found that there was no significant difference between the health of the children living in cannabis grow operations and the comparison group of children, based on their prescription history and their reported health at the time.

**Conclusion:** The findings of this study challenge contemporary child welfare approaches and have implications for both child protection social workers and the policymakers who develop frameworks for practice.

© 2013 Elsevier B.V. All rights reserved.

### Introduction

Illegal indoor cannabis grow operations have become an increasing problem in Canada, receiving significant attention from the media, policing agencies, and government (CTV News, 2011; Plecas, Malm, & Kinney, 2005; RCMP, 2002; Stop the Violence, 2012). Aside from the criminal justice perspective, police and child protection social workers are concerned about the presence of children living in homes where cannabis is being grown. For over a decade, child protection social workers have been faced with the task of assessing the risk to children found living in cannabis grow operations (Douglas, 2010). The physical hazards that exist in these homes, as well as the environmental conditions potentially pose a threat to the health of the children living there (Canadian Institute of Child Health, 2003; Dales, Zwanenburg, Burnett, & Franklin, 1991; Gustin, 2010). Illegal wiring, hydro bypasses, chemicals and pesticides, mold, and compromised air quality are all factors that contribute to these concerns. However, these home safety concerns that bring grow ops to the attention of police, fire departments, hydroelectric providers, landlords and insurance companies are arguably a consequence of the prohibition against cannabis production that has driven it underground.

\* Corresponding author. Tel.: +1 604 323 5770; fax: +1 604 323 5655.  
E-mail address: [jdouglas@langara.bc.ca](mailto:jdouglas@langara.bc.ca) (J. Douglas).

0955-3954/\$ – see front matter © 2013 Elsevier B.V. All rights reserved.  
<http://dx.doi.org/10.1016/j.drugpo.2013.01.003>

This research examined the rationale for child protection interventions with families found living in cannabis grow operations, based on the assumption of risk in the presence of probable medical harm.

### Background

The current response to child maltreatment has involved the creation of regulatory agencies: child protective services that exercise the power of the state to intervene with families where children have been abused or neglected, or are at risk of being abused or neglected (Dingwall & Eekelaar, 1988). It is the allegation or evidence of maltreatment that allows for the government's intrusion into the affairs of the family (Larner, Stevenson, & Behrman, 1998). However, the role of child protection in the cannabis grow operation phenomenon has become entangled with political debate and prevailing attitudes over the use and/or legalization of cannabis. Moral contagion, public safety and the social construction of cannabis grow operators as dangerous persons all contribute to this confusion (Carter, 2009). Although moralization was an important motive in the early days of social work (Danzelot, 1979), most Western child welfare policies have moved away from making decisions about children's alleged needs on the basis of social or moral judgements. During the 1970s and 1980s, interventions became grounded in the notion of "tangible risk of significant harms to children" (Carney, 1999, p. 58), and with this movement came the

gradual end of the inclusion of explicit morality considerations in child maltreatment practice.

Social work ideology no longer considers parental morality in child welfare assessments, and those involving cannabis grow operations are no exception. However, it is not the crime of growing cannabis that is the child welfare problem; it is the associated dangers that this activity presents for children. The former can be seen as a moral problem; the latter should not. It is this disconnect that partly contributes to the ambivalence that child protection practitioners face when dealing with cannabis grow operation families. The question that confronts the practitioner is whether parents' exposing their children to potentially harmful toxic environments constitutes a form of neglect, specifically the neglect of the children's need for a safe, healthy environment.

### Neglect

By most definitions, child neglect involves an omission on the part of a parent, a failure to provide or protect, that leads to a child's harm or endangerment. Parental responsibility and blame are at least implicit in most legislation. In British Columbia, the Child, Family and Community Service Act speaks directly to the problem of neglect, but child protection action requires that there be physical harm or likelihood due to the neglectful acts of the parents (Child, Family and Community Service Act, 1996). It is the introduction of potential harm as criteria for intervention that raises controversy (Dubowitz, Black, Starr, & Zuravin, 1993). In the absence of confirmed medical evidence with grow operation children, it is the 'likelihood of harm' clause that provides the justification for social work involvement and investigation, and not a determination of intent to harm.

Chronic neglect is unlikely to be mono-causal, however the experience of grow operation children may mark an exception. Although there is a trigger event that brings these children to the attention of child protection services (and law enforcement), except in rare circumstances the physical state of the grow operation as the site of parenting is the only contributing factor to the neglect of the children.

There is ample evidence in the literature to support concerns about child health in cannabis grow-operations, in addition to the observations of the lead author and other professionals who frequent grow-operations in the course of their work. The presence of chemicals and pesticides, mold, and compromised air quality all contribute to concerns of risk, and the children living in these homes often present with respiratory problems and dermatological disorders. The warm, moist environment created to produce optimal growing conditions for cannabis also creates optimal growing conditions for mold. The relationship between indoor air quality, including the presence of mold and fungal spores, and respiratory problems in children has been well documented in research (Freeman, Schneider, & McCarvey, 2003; Garrett, Kayment, Hooper, Abramson, & Hooper, 1998; Pettigrew et al., 2004). In addition, fertilizers, chemicals and pesticides are applied to the cannabis plants to encourage growth and to eliminate the spider mites that are a threat to the crop. Carbon dioxide emitted from gas furnaces and hot water tanks is often vented back into the grow rooms to enhance plant growth, and can make its way throughout the house.

Based on research about environments with conditions similar to cannabis grow operations (Canadian Institute of Child Health, 2003; Fassa, 2003; Kim et al., 2002; Peterman, Jalongo, & Qinyun Lin, 2002; United States Environmental Protection Agency, 2009), it was anticipated that children living in cannabis grow operations would have higher rates of respiratory and dermatological disorders than children who did not live in a grow operation.

### Methods

This research involved the collection of data through the use of a survey instrument and direct observation by the child protection workers who attended the grow operation homes as part of their investigation. Child protection workers were assisted in the data collection by other first responders, notably police, fire officials, and hydro employees.

The data set consists of only the children and families found living in cannabis grow operations in two regions within the Greater Vancouver Regional District, British Columbia and who were reported to the Ministry of Children and Family Development (MCFD), the government authority delegated to conduct child welfare investigations in British Columbia. Data was collected over a 26-month period between 2004 and 2006. Ninety-five grow operations, involving 181 children, were reported to the study (Douglas, 2010). PharmaNet (the provincial prescription database) provided prescription information on these children, as well as prescription information for a randomly sampled comparison group of 500 children in the same age groupings and geographic areas.

### Data analysis

The quantitative data was analysed using frequencies and logistic regression. The variable categories reported below were included in the analysis, and are part of the larger data set. All data management and analysis was conducted using SPSS version 16.0. Frequencies were calculated on all variables to provide a descriptive representation of the families and children included in the study, as well as the conditions of the cannabis grow operations where they were living. Only the data related to child health is reported in this article.

### Limitations of dataset

Children and families living in cannabis grow operations in Greater Vancouver Regional District, British Columbia who did not come to the attention of police or child welfare authorities are not included in this study. In addition, information gathered was at the 'moment in time' when there was intervention by police and child protection workers. Children lived in these homes for varying degrees of time that were difficult to ascertain, or they may have lived in multiple dwellings. Some of these homes were not their primary residences. For some of these families this was their first grow operation; other families had lived in them for years.

### Results

#### Sample characteristics

There were 95 families included in the study. The number of children per family ranged from one to five, with a little over half (52.6%) of the families having two children. The children ranged in age from newborn to 18 years old, with 68 of the 181 children (37.6%) being under five years of age.

English was spoken by 54 (57%) of the families, although 68 (72%) of the families reported that Vietnamese was their primary language.

One hundred and thirty-one (76%) of the children had lived in grow operations for twelve months or less; however, this was often an estimate provided to child protection workers by hydro employees.

Table 1  
Drug frequency by drug type.

	None	1	2	3 or more
<b>Number of respiratory/asthma prescriptions</b>				
Grow operation	45%	28%	12%	15%
Comparison	44%	33%	12%	13%
P value	.84	.28	1.0	.56
<b>Number of antibiotic prescriptions (not cream)</b>				
Grow operation	30%	28%	22%	20%
Comparison	18%	22%	23%	37%
P value	.004	.15	.81	.0004 <sup>a</sup>
<b>Number of topical corticosteroid/antibiotic prescriptions</b>				
Grow operation	47%	33%	0%	18%
Comparison	46%	26%	15%	13%
P value	.92	.119	.08	.65
<b>Number of Topical antihistamine/allergy prescriptions</b>				
Grow operation	60%	27%	0%	4%
Comparison	70%	21%	6%	3%
P value	.04	.094	.023	.38

<sup>a</sup> P < .05.

#### Household characteristics

Pesticides and chemicals were present in 96% (81/84) of the grow operation homes, indicating little variance. Re-venting of the gases from the furnace and/or hot water tank was present in 59% (46/78) of the homes, and mold was located in 77% (56/73) of the homes. The number of cannabis plants located in the homes ranged from 0 to 1900, with the mean number of plants calculated at 362.

#### Child health characteristics

Based on the child's initial presentation, parents' or child protection worker's reporting, and/or the presence of prescription medication in the home, 21% (32/152) of the children were reportedly unwell. As not all children were seen by the child protection worker, reporting of symptoms varied case by case. Respiratory concerns (coughing/breathing difficulties, nasal congestion) were noted in 17.7% (27/152) of the children; 11.2% (17/152) had dermatological symptoms (rashes, itchy skin); and 1.2% (2/162) had ear infections. Some of the children had multiple symptoms.

In 81% (123/152) of the homes, workers were unable to locate any prescription medication for the children. In the remaining homes, 10% (16/152) of the medications were for cortisone creams; 11% (17/152) were for respiratory ailments; and 7% (12/152) were antibiotics.

#### PharmaNet

Aggregate data (by age groupings) of the history of prescription medication for each child was gathered from PharmaNet. Of the 181 children located in cannabis grow operations, 176 were registered on the PharmaNet database, and prescriptions were located for 133 of these children (during the two-year period leading up to them being found in the grow operation). In addition, PharmaNet provided aggregate drug information for a comparison group of 500 children, from the same geographic areas. Of those 500 children, prescriptions were found for 412 over a similar two-year period. A total of 5435 prescription details were received: 1027 for the grow operation children, and 4408 for the comparison group.

Prescriptions for respiratory illnesses and skin disorders were taken as key dependent variables. Each child was logged for the number of different prescriptions they received within and across variables, and the number of repeat prescriptions they received. The grow operation and comparison group children were examined separately and in combination. As the PharmaNet data was aggregate only, we could only compare the children living in grow

operations with those believed to be not similarly housed in relation to their overall prescription patterns.

Contrary to expectations, results indicated no significant difference in the number and frequency of the identified prescriptions between children in cannabis grow operations and the comparison group (see Table 1).

#### Discussion

The presence of mold, re-venting of gases, and the chemicals often found in grow operation homes suggested that the children living there might well suffer from the ill effects of these environments, and could be expected to exhibit respiratory and/or dermatological ailments (Freeman et al., 2003; Garrett et al., 1998; Kim et al., 2002; Pettigrew et al., 2004). Indeed, 21% of the children were found to be unwell at the time of child welfare intervention. The examination of prescriptions focused on the medications linked to the ailments expected to be more prevalent in children living in a grow operation environment (Repchinsky, 2008). The results showed no significant difference between the grow operation children and the comparison children, with 65% of grow operation children having three or more prescriptions, as compared to 72% of the comparison children who had three or more of the same categories of prescriptions. An examination of each of the medications separately and by frequency revealed similar non-significant findings.

It was perplexing that these findings were contrary both to the professionals' beliefs about these environments, and the significant body of literature presented earlier in this paper that documents the consequences that mold and toxic environments can have on health.

These findings give rise to a number of possible explanations, all of which deserve consideration:

1. The overwhelming majority of the grow operation families are immigrants of Vietnamese and other Asian descents who may in fact choose alternate methods of health care rather than traditional Western medicine to treat their children. However, this argument is not supported by the data. The PharmaNet data indicates that 73% (133/181) of the grow operation children had received traditional medications in the two-year period leading up to them being found in a grow operation. This number is not significantly different from the comparison group, where 82% (412/500) of those children had received prescriptions during the same time period. The grow operation families may indeed choose

- alternate health care methods, but it does not appear to be in lieu of Western medicine.
- The findings are limited due to the size of sample and length of time that health was measured, resulting in exposure misclassification. As exposure to mold and toxins are considered low-to-moderate risk for health outcomes, a larger sample followed for a longer period of time may provide a more accurate representation of the health of these children. Indeed, previous studies involving much greater scientific rigour had findings more consistent with the literature regarding exposure to environmental toxins (Antova et al., 2008; Bornehag et al., 2005; Dales et al., 1991; Fisk, Lei-Gomez, & Mendell, 2007; Iossifova et al., 2009; Jaakkola, Bing-Fang, & Jaakkola, 2005).
  - The children are indeed resilient and not suffering the ill effects of these toxic environments. Children may have better recuperative capacities than adults for many toxic agents (Brent, Tanski, & Weitzman, 2004) and the restricted location of the mold within specific areas of the child's home may reduce risk to their overall health (Karvonen et al., 2009).

Finally, a recent study in Ontario conducted toxicology hair testing on 75 children found living in cannabis grow-operations (Moller, Koren, Karasikov, & Garcia-Bourissien, 2011). They found that although 30% of the children tested positive for illicit drugs (in their hair), the majority of these children had no clinical symptoms related to these drugs.

### Conclusion

Child protection and the related legislation have become tools for addressing the illegal cannabis grow operation problem, as to date no system has been successful in bringing it under control. Although there is little argument that the physical hazards found in cannabis grow-operations pose a risk to children and adults living in the homes, the associated health risks are not as clear. Policymakers involved in establishing frameworks and protocols for responding to these unique child welfare cases must consider the absence of clinical evidence to indicate these children are unwell and whether there are grounds for child welfare intervention. As provincial authorities, law enforcement agencies and other stakeholders continue discussions to create new laws and increase enforcement, care must be taken to be explicit about which risks are forming the foundation for these actions, and whether in fact they actually exist.

With recent legislative changes in neighbouring Washington state, regulatory authorities in British Columbia may be able to observe the process of bringing this industry under regulation. In the interim, police, child welfare and health care professionals are left in a quandary about to proceed with families whose children appear to be well cared for apart from their exposition to the risks associated with indoor cannabis production.

### Role of the funding source

Funding for this research was received from MITACS, BC Mental Health and Addictions Research Network and the BC Child and Youth Health Research Network. The BC Ministry for Children and Family Development also provided assistance in-kind. These study sponsors were not involved in any aspects of the study design, collection, analysis, interpretation of data, report writing, or in the decision to submit the paper for publication.

### References

- Antova, T., Patzenden, S., Brunekreef, B., Heinrich, J., Rudnai, P., Forastiere, F., et al. (2008). Exposure to indoor mould and children's respiratory health in the PATY study? *Journal of Epidemiology and Community Health*, 62(8), 708–714.

- Bornehag, C. G., Sundell, J., Hagerhed-Engman, L., Slegggard, T., Janson, S., & Åberg, M. (2005). 'Dampness' at home and its association with airway, nose, and skin symptoms among 10,851 preschool children in Sweden: A cross-sectional study. *Indoor Air*, 15, 48–55.
- Brent, R. L., Tanski, S., & Weitzman, M. (2004). A pediatric perspective on the unique vulnerability and resilience of the embryo and the child to environmental toxicants: The importance of rigorous research concerning age and agent. *Pediatrics*, 113, 935–944.
- Canadian Institute of Child Health. (2003). *Pesticides and child health*. Ottawa: Canadian Institute of Child Health. Retrieved from <http://www.cich.ca/Environment/Health/pesticides.html>
- Carney, T. (1999). Liberalism or distributional justice? The morality of child welfare laws. In M. King (Ed.), *Moral agenda for children's welfare* (pp. 53–73). London: Routledge Publishing.
- Carter, C. (2009). Making residential cannabis growing operations actionable: A critical policy analysis. *International Journal of Drug Policy*, 20(4), 371–376.
- Child, Family and Community Service Act (RSBC 1996) Chapter 46. (1996).
- CTV News. (2011, October 27). B.C. police and academics call for pot legalization. Retrieved from <http://ctvnews.ca/bc-police-and-academics-call-for-pot-legalization-1.107315>
- Dales, R. E., Zwanenburg, H., Burnett, R., & Franklin, C. A. (1991). Respiratory health effects of home dampness and molds among Canadian children. *American Journal of Epidemiology*, 134(2), 196–203.
- Dingwall, R., & Eekelaar, J. (1988). Families and the state: An historical perspective on the public regulation of private conduct. *Law and Policy*, 10(4), 341–361.
- Danzon, J. (1979). *The policing of families* (1st American ed.). New York: Pantheon Books.
- Douglas, J. (2010). *The health and safety of children living in marijuana grow operations: A child welfare perspective*. Retrieved from <http://www.cicbc.ca>
- Dubowitz, H., Black, M., Starr, R. H., Jr., & Zuravin, S. (1993). A conceptual definition of child neglect. *Criminal Justice and Behavior*, 20(1), 8–26.
- Fassa, A. G., & International Labour Organisation. & International Programme on the Elimination of Child Labour. (2003). *Health benefits of eliminating child labour: Research paper in conjunction with the ILO-IPEC study on the costs and benefits of the elimination of child labour*. Geneva: International Labour Organization, International Programme on the Elimination of Child Labour.
- Fisk, W. J., Lei-Gomez, Q., & Mendell, M. J. (2007). Meta-analyses of the associations of respiratory health effects with dampness and mold in homes. *Indoor Air*, 17(4), 284–296.
- Freeman, N. C. G., Schneider, D., & McGarvey, P. (2003). Household exposure factors, asthma, and school absenteeism in a predominantly Hispanic community. *Journal of Exposure Analysis & Environmental Epidemiology*, 13(3), 160.
- Garrett, M. H., Rayment, P. K., Hooper, M. A., Abramson, M. J., & Hooper, B. M. (1998). Indoor airborne fungal spores, house dampness and associations with environmental factors and respiratory health in children. *Clinical & Experimental Allergy*, 28(4), 459–467.
- Geslin, B. (2010). The hazards of grow houses. *Fire Engineering*, 163(6), 69–72.
- Iossifova, V., Reponen, T., Ryan, P., Levin, L., Bernstein, D., Luckey, J., et al. (2009). Mold exposure during infancy as a predictor of potential asthma development. *Annals of Allergy, Asthma and Immunology*, 102(2), 131–137.
- Jaakkola, J. J. K., Bing-Fang, H., & Jaakkola, M. (2005). Home dampness and molds, parental atopy, and asthma in childhood: A six year population-based cohort study. *Environmental Health Perspectives*, 113(3), 357.
- Karvonen, A. M., Hyvärinen, A., Roponen, M., Hoffmann, M., Korppi, M., Remes, S., et al. (2009). Confirmed moisture damage at home, respiratory symptoms and atopy in early life: A birth-cohort study. *Pediatrics*, 124(2), 329–338.
- Kim, Y. K., Chang, Y. S., Lee, M. H., Hong, S., Bae, J. M., Jee, Y. K., et al. (2002). Role of environmental exposure to spider mites in the sensitization and the clinical manifestation of asthma and rhinitis in children and adolescents living in rural and urban areas. *Clinical & Experimental Allergy*, 32, 1305–1308.
- Larner, M. B., Stevenson, C. S., & Behrman, R. E. (1998). Protecting children from abuse and neglect: Analysis and recommendations. *The Future of Children*, 8(1), 4–22.
- Moller, M., Koren, G., Karasikov, T., & Garcia-Bourissien, F. (2011). Examining the health and drug exposures among Canadian children residing in drug-producing homes. *Journal of Pediatrics*, 159(5), 766–770.
- Peterman, T. K., Jalongo, M., & Qiyuan Lin, M. (2002). The effects of molds and fungi on young children's health: Families' and educators' roles in maintaining indoor air quality. *Early Childhood Education Journal*, 30(1), 21–26.
- Pettigrew, M. M., Gent, J. F., Triche, E. W., Belanger, K. D., Bracken, M. B., & Leaderer, B. P. (2004). Association of early-onset otitis media in infants and exposure to household mold. *Pediatric & Perinatal Epidemiology*, 18(5), 441–447.
- Piezas, D., Malin, A., & Kinney, B. (2005). Marijuana growing operations in British Columbia revisited 1997–2003. Abbotsford, B.C.: International Centre for Urban Research Studies (ICURS).
- Repechinsky, C. (Ed.). (2008). *Compendium of pharmaceuticals and specialties*. Ottawa: Canadian Pharmacists Association.
- Royal Canadian Mounted Police. (2002). *Marijuana cultivation in Canada: Evolution and current trends*. Retrieved from [http://www.rcmp-grc.gc.ca/crimin/cultivation\\_e.htm#Safety](http://www.rcmp-grc.gc.ca/crimin/cultivation_e.htm#Safety)
- Stop The Violence B.C. (2012). Retrieved from <http://stoptheviolencebc.org/about-us/>
- United States Environmental Protection Agency. (2009). *An introduction to indoor air quality*. Retrieved from <http://www.epa.gov/iaq/co2.html#Sources2009>