COMMUNITY HEALTH COMMISSION MEETING AGENDA

Tuesday, July 9th, 2019 6:30 pm–9 pm
South Berkeley Senior Center
2939 Ellis St, Berkeley, CA 94703

Community Health Commission (CHC)

Preliminary Matters

1. Roll Call
2. Announcements & Introductions of any new members
3. Approval of Minutes from the May 23rd meeting (Attachment 1)
4. Confirm note taker
5. Public Comment (*Speakers will have up to 5 minutes each*)

Presentation Items

1. **City of Berkeley, Public Health Division—Dr. Lisa Hernandez**: Updates

Discussion and Action Items:

1. Discuss and approve cannabis ordinance language draft [Simpson]
2. Discuss and vote on Commission communication on proposed ban on natural gas infrastructure in new building (Attachment 8) [Gilman].
3. Appoint Commissioners to speak on behalf of CHC at upcoming City Council meetings [Simpson].
4. Add/remove subcommittee members (Attachment 4) [Simpson].

Subcommittee Break-Out Session

Subcommittee Reports

1. Acute Services Subcommittee
2. Basic Needs Security Subcommittee
3. Cannabis Subcommittee
4. Disease Prevention Subcommittee
5. Health Equity Subcommittee
6. Policy Tracking Subcommittee
7. Strategic Planning Subcommittee

Communication

1. Eden Environmental Citizen's Group, LLC v. Precision Technical Coatings, Inc. et al. Lawsuit (Attachment 7)

Adjournment

1947 Center Street, 2nd Floor, Berkeley, CA 94704 Tel: 510. 981.5300 TDD: 510.981.6903 Fax: 510. 981.5395
E-mail: publichealth@ci.berkeley.ca.us - - http://www.cityofberkeley.info/health/

Attachments:

1. Draft minutes of 5/23/19 CHC meeting
2. Approved minutes of 5/2/19 CHC special meeting
3. Community Health Commission Work Plan
4. Community Health Commission Subcommittee Roster 2019
5. Community Health Commission Meeting Calendar 2019
6. City Council and Community Health Commission Timeline 2019
7. Communication: Environmental Lawsuit

The next meeting of the Community Health Commission is scheduled for July 25th, 2019, with a deadline of July 15th, 2019 for the public's submission of agenda items and materials for the agenda packet. Dates are subject to change; please contact the Commission Secretary to confirm.

COMMUNICATION ACCESS INFORMATION

 "This meeting is being held in a wheelchair-accessible location. To request a disability-related accommodation(s) to participate in the meeting, including auxiliary aids or services, please contact the Disability Services Specialist at 981-6418 (V) or 981-6347 (TDD) at least three business days before the meeting date. Please refrain from wearing scented products to this meeting."

Communications to Berkeley boards, commissions or committees are public record and will become part of the City's electronic records, which are accessible through the City's website. Please note: e-mail addresses, names, addresses, and other contact information are not required, but if included in any communication to a City board, commission or committee, will become part of the public record. If you do not want your e-mail address or any other contact information to be made public, you may deliver communications via U.S. Postal Service or in person to the secretary of the relevant board, commission or committee. If you do not want your contact information included in the public record, please do not include that information in your communication. Please contact the commission secretary for further information.

Any writings or documents provided to a majority of the Commission regarding any item on this agenda will be made available for public inspection at the City of Berkeley Public Health Division located at 1947 Center Street, Second Floor, during regular business hours. The Commission Agenda and Minutes may be viewed on the City of Berkeley website: http://www.cityofberkeley.info/commissions.

Please refrain from wearing scented products to this meeting.

Secretary:
Roberto A. Terrones, MPH
Health, Housing & Community Services Department
1947 Center Street, 2nd Floor
(510) 981-5324
E-mail: RTerrones@cityofberkeley.info

Community Health Commission

Community Health Commission

South Berkeley Senior Center,
2939 Ellis St, Berkeley, CA 94703

DRAFT MINUTES

Regular Meeting, Thursday May 23rd, 2019

The meeting convened at 6:51 p.m. with Commission Chair Simpson presiding.

ROLL CALL

Present: Commissioners Engelman, Speich, Futoran, Carter, Smart, Gilman, Spigner, Rojas-Cheatham, Simpson, Imai, Katz (6:55)

Absent: Commissioner Rosales

Excused: Commissioners Webber, Le

Staff present: Dr. Lisa Hernandez, Roberto Terrones, Elizabeth Greene

Community Members: Michael Cooper, Ronni Pelley

COMMENTS FROM THE PUBLIC: None.

PRESENTATIONS: **Mary Behm-Steinberg—**Homelessness in the City of Berkeley
City of Berkeley, Planning and Development Department—
Phase II Cannabis Ordinance Items.

ACTION ITEM

1. M/S/C (Gilman/Speich): Motion to approve minutes from the May 2nd, 2019 Special Meeting.

Ayes: Commissioners Engelman, Speich, Futoran, Carter, Smart, Gilman, Spigner, Rojas-Cheatham, Simpson, Imai, Katz

Noes: None

Abstain: None

Absent from vote: Commissioner Rosales

Excused: Commissioners Webber, Le

Motion Passed.

1947 Center Street, 2nd Floor, Berkeley, CA 94704 Tel: 510. 981.5300 TDD: 510.981.6903 Fax: 510. 981.5395
E-mail: publichealth@ci.berkeley.ca.us - - http://www.cityofberkeley.info/health/

2. M/S/C (Futoran/Spigner): Motion to add Commissioner Gilman to the Cannabis Subcommittee.

Ayes: Commissioners Speich, Futoran, Carter, Smart, Gilman, Spigner, Simpson, Imai, Katz

Noes: None

Abstain: None

Absent from vote: Commissioner Rosales, Rojas-Cheatham, Engelman

Excused: Commissioners Webber, Le

Motion Passed.

NEXT MEETING

The next regular meeting will be on June 27th, 2019 at 6:30 p.m. at the South Berkeley Senior Center.

This meeting was adjourned at 9:10 p.m.

Respectfully Submitted, Roberto A. Terrones, Secretary.

Community Health Commission

Community Health Commission

South Berkeley Senior Center,
2939 Ellis St, Berkeley, CA 94703

MINUTES
Special Meeting, Thursday May 2nd, 2019

The meeting convened at 6:46 p.m. with Commission Chair Simpson presiding.

ROLL CALL

Present: Commissioners Webber, Futoran, Carter, Smart, Gilman, Spigner, Simpson, Imai, Rosales, Le, Katz (6:53)

Absent: Commissioners Engelman, Speich, Rojas-Cheatham

Excused: None

Staff present: Janice Chin, Roberto Terrones

Community Members: Mansour Id-Deen, Richie Smith, Austin Cable

COMMENTS FROM THE PUBLIC: Austin Cable

PRESENTATIONS: City of Berkeley, Babalwa Kwanele and Barbara White—African American Holistic Resource Center.

ACTION ITEM

1. M/S/C (Rosales/Futoran): Motion to approve minutes from the March 28th, 2019 meeting.

Ayes: Commissioners Webber, Futoran, Carter, Smart, Spigner, Simpson, Rosales

Noes: None

Abstain: Commissioners Gilman, Imai, Le

Absent from vote: Commissioners Engelman, Speich, Rojas-Cheatham, Katz

Excused: None.

Motion Passed.

1947 Center Street, 2nd Floor, Berkeley, CA 94704 Tel: 510. 981.5300 TDD: 510.981.6903 Fax: 510. 981.5395
E-mail: publichealth@ci.berkeley.ca.us - - http://www.cityofberkeley.info/health/

2. M/S/C (Katz/Webber)**:** Motion to appoint Commissioner Spigner as a CHC representative for the May 14th, 2019 City Council Meeting.

Ayes: Commissioners Webber, Futoran, Carter, Smart, Gilman, Spigner, Simpson, Imai, Rosales, Le, Katz

Noes: None

Abstain: None

Absent from vote: Commissioners Engelman, Speich, Rojas-Cheatham

Excused: None

Motion Passed.

3. M/S/C (Rosales/Smart): Motion to add Commissioner Imai to the Basic Needs Subcommittee.

Ayes: Commissioners Webber, Futoran, Carter, Smart, Gilman, Spigner, Simpson, Imai, Rosales, Le, Katz

Noes: None

Abstain: None

Absent from vote: Commissioners Engelman, Speich, Rojas-Cheatham

Excused: None

Motion Passed.

4. M/S/C (Rosales/Katz): Motion to add Commissioner Gilman to the Acute Services Subcommittee.

Ayes: Commissioners Webber, Futoran, Carter, Smart, Gilman, Spigner, Simpson, Imai, Rosales, Le, Katz

Noes: None

Abstain: None

Absent from vote: Commissioners Engelman, Speich, Rojas-Cheatham

Excused: None

Motion Passed.

NEXT MEETING

The next regular meeting will be on May 23rd, 2019 at 6:30 p.m. at the South Berkeley Senior Center.

This meeting was adjourned at 9:03 p.m.

Respectfully Submitted, Roberto A. Terrones, Secretary.

Minutes were approved on May 23rd, 2019.

Community Health Commission Work Plan FY2019-2020

Guiding Philosophy: To look at health through the lens of equity, and to address, ameliorate, and abolish health inequities in Berkeley through our work while advancing other public health efforts.

Mission/Purpose:

1. Collaborate with the community and the Berkeley Public Health Division, and City of Berkeley to eliminate health inequity by:
    a. Advocating for good policy to council that has the potential to improve the health of Berkeley residents that can be implemented, monitored, and evaluated.
    b. Representing the community through the diversity of this commission
    c. Increasing the public education/social marketing efforts, understanding, and awareness of issues
    d. Advocating together with the residents of Berkeley most affected by institutional, social, organizational inequities/disparities
    e. Providing a public forum for all community members to share concerns, ideas
2. Achieve general public health progress by being responsive to community needs and facilitating general health and safety.

Overall goals, issues & priorities: All issues can be addressed through a health equity lens.

- Increase healthy food security
- Advocate for the expansion of affordable housing
- Continue to urge that Alta Bates Berkeley Medical Campus remain open while also helping to suggest actions to address consequences of planned closure
- Be responsive in potential recommendations to help Berkeley residents, and care providers and clinics cope with potential federal disruption in healthcare policy and federal spending cuts
- Further address more social determinants of health
- Continue to be a community advocate to City Council to address structural, institutional, and health inequities impacting all underserved populations
- Work to have community health data measures documented in a timely manner and to promptly evaluate and act on novel data such as the Health Status Report
- Work to support policies and initiatives that advance UHC such as Medicare for all
- Advise the City Council as the Public Health Department develop their strategic plan

General steps and actions needed to meet priorities:

1. Better follow up with council implementations
2. Collaborate with other commissions to share resources and support recommendations
3. Focused/specialized ad-hoc subcommittees
4. Keep track of local, state, and federal policy and data flow

Specific steps and actions needed to meet priorities:

❖ Subcommittees

- **Strategic Planning subcommittee**
  - Serve as point of contact with Public Health Division for city's strategic plan and facilitate deliberation between full commission and division
  - Recommend structure of portion of agenda to educate commission on strategic plan development
- **Acute Services for Berkeley**
  - Continue to recommend actions to keep Alta Bates open
  - Consider ways to increase emergency care access in Berkeley
- **Basic Needs Security**

  Focus on healthy food security and affordable/accessible housing
  - In terms of healthy food security:
    - Identify food recovery donation systems
    - Connect communities with healthy food resources (awareness)
    - Advocate for policies to mitigate unhealthy food consumption
    - Advocate for affordability and accessibility of healthy foods
      - In supporting programs like the Berkeley Food Institute, etc.,
  - In terms of accessible/affordable housing:
    - Identify areas of stark homelessness
    - Connect homeless communities with resources (awareness)
    - Advocate for affordable housing
    - Advocate for increased rent control
- **Policy tracking**
  - Track City Council minutes, state, and national legislative actions
  - Priority areas:
    - School lunch programs
    - Affordable housing in the Adeline area
- **Health Equity Subcommittee**
  - Engage Stakeholders on LGBT health equity issues to help complement findings of the Health Status Report
  - Follow up on status of the African American Holistic Resource Center
  - Work on cultural competency for health care providers--contact county health care providers and Kaiser
  - Review the Health Status Report- dialogues with staff and community to investigate the data and inequities, and recommend program interventions for the City Public Health Division
  - Implement efforts to improve immigrant access to health care
  - Investigate how health care providers are using technology to improve health
  - Meet with the public health officer to be informed
- **Chronic Disease Prevention**
  - Recommend presenters that can educate the commission on innovative approaches to chronic disease prevention
  - Consider the use of high profile figures in media campaigns to educate the community about chronic disease prevention.

- **Cannabis**
  - Decriminalizing and destigmatizing cannabis use throughout the Berkeley area
  - Advocating for holistic education of cannabis use throughout the community
  - Assessing holistically the risks and benefits of cannabis use in terms of community health
  - Assessing holistically how cannabis should be integrated within the local economy while maintaining the health of the community
    - .i.e. nurseries, dispensaries, etc.
  - Prioritizing community health following the legalization of cannabis with emphasis on holistically understanding the risk and benefits of cannabis
- Ad-hoc subcommittees as needed to quickly address City Council referrals
- Liaisons to other commissions
  - Housing Advisory Commission
  - Homeless Commission
  - Zero Waste Commission
  - Mental Health Commission
  - Human Welfare and Community Action Commission
  - Community Environmental Advisory Commission
  - Sugar Sweetened Beverage Panel

			Community Health Commission Subcommittees 2019						
District	Last	First	Acute Services for Berkeley	Health Equity	Policy Education	Basic Needs Security	Disease Prevention	Strategic Planning	Cannabis
1	Engelman	Alina	X						
1	Webber	Sara			X	X			
2	Vacant	Vacant							
2	Speich	Pamela			X		X		X
3	Futoran	Charles					X		
3	Carter	Donna	X		X				
4	Smart	Karma		X		X			
4	Gilman	John	X						x
5	Spigner	Tora		X	X				
5	Vacant	Vacant							
6	Rojas-Cheatham	Ann						X	X
6	Vacant	Vacant							
7	Simpson	May			X		X		X
7	Imai	Derek				X			
8	Rosales	Ces		X				X	
8	Le	Carolyn						X	
M	Vacant	Vacant							
M	Katz	Andy	X	X				X	
			4	4	5	3	3	4	4

Community Health Commission 2019 Meeting Dates

Community Health Commission (CHC)

Month	Meeting Day and Date	Time
January 2019	Thursday 1/24/19	6:30-9PM
February 2019	Thursday 2/28/19	6:30-9PM
March 2019	Thursday 3/28/19	6:30-9PM
April 2019	Thursday 4/25/19	6:30-9PM
May 2019	Thursday 5/23/19	6:30-9PM
June 2019	Thursday 6/27/19	6:30-9PM
July 2019	Thursday 7/25/19	6:30-9PM
August 2019 **THE CHC DOES NOT MEET IN AUGUST**		
September 2019	Thursday 9/26/19	6:30-9PM
October 2019	Thursday 10/24/19	6:30-9PM
November 2019	Thursday 11/21/19*	6:30-9PM
*Meeting in November is scheduled on the 3rd Thursday due to the Thanksgiving Holiday		
December 2019 **THE CHC DOES NOT MEET IN DECEMBER**		

1947 Center Street, 2nd Floor, Berkeley, CA 94704 Tel: 510. 981.5300 TDD: 510.981.6903 Fax: 510. 981.5395 E-mail: publichealth@ci.berkeley.ca.us - http://www.cityofberkeley.info/health/

COUNCIL MEETING TIMELINE - COMMISSIONS
HEALTH, HOUSING & COMMUNITY SERVICES DEPARTMENT
Updated October 19, 2018

2019				Thursday 12:00 PM	Thursday 12:00 PM	Monday 2:30 PM	Wednesday 11:00 AM	Thursday 5:00 PM
COUNCIL MEETING DATE	Commission needs to take action by	Reports Due to Dept. Director	Reports Due to CAO	Dept. Reports Due to Clerk **Day 33**	Agenda Committee Packet to Print **Day 19**	Agenda Committee Meeting **Day 15**	Final Agenda Meeting - (Print Agenda on wed.) **Day 13**	Council Agenda Delivery **Day 12**
Winter Recess [December 12, 2018 – January 21, 2019]								
22-Jan	11/22	11/29	12/6	12/20	1/3	1/7	1/9	1/10
29-Jan	11/29	12/6	12/13	12/27	1/10	1/14	1/16	1/17
19-Feb	12/20	12/27	1/3	1/17	1/31	2/4	2/6	2/7
26-Feb	12/27	1/3	1/10	1/24	2/7	2/11	2/13	2/14
12-Mar	1/10	1/17	1/24	2/7	2/21	2/25	2/27	2/28
26-Mar	1/24	1/31	2/7	2/21	3/7	3/11	3/13	3/14
2-Apr	2/2	2/9	2/16	2/28	3/14	3/18	3/20	3/21
Spring Recess [April 3 through April 22]								
23-Apr	2/21	2/28	3/7	3/21	4/4	4/8	4/10	4/11
30-Apr	2/28	3/7	3/14	3/28	4/11	4/15	4/17	4/18
14-May	3/14	3/21	3/28	4/11	4/25	4/29	5/1	5/2
28-May	3/28	4/4	4/11	4/25	5/9	5/13	5/15	5/16
11-Jun	4/11	4/18	4/25	5/9	5/23	5/28 - Tue	5/29	5/30
25-Jun	4/25	5/2	5/9	5/23	6/6	6/10	6/12	6/13
9-Jul	5/9	5/16	5/23	6/6	6/20	6/24	6/26	6/27
16-Jul	5/16	5/23	5/30	6/13	6/27	7/1	7/3	7/3 - Wed
23-Jul	5/23	5/30	6/6	6/20	7/3 - Wed	7/8	7/10	7/11
Summer Recess [July 24 through September 9								
10-Sep	7/11	7/18	7/25	8/8	8/22	8/26	8/28	8/29
24-Sep	7/25	8/1	8/8	8/22	9/5	9/9	9/11	9/12
15-Oct	8/15	8/22	8/29	9/12	9/26	9/30	10/2	10/3
29-Oct	8/29	9/5	9/12	9/26	10/10	10/15 - Tue	10/16	10/17
12-Nov	9/12	9/19	9/26	10/10	10/24	10/28	10/30	10/31
19-Nov	9/19	9/26	10/3	10/17	10/31	11/4	11/6	11/7
3-Dec	10/3	10/10	10/17	10/31	11/14	11/18	11/20	11/21
10-Dec	10/10	10/17	10/24	11/7	11/21	11/25	11/27	11/27 - Wed
Winter Recess [December 11, 2019 – January 21, 2020]								

to Terrones - Secretary
unity Health Commission
Center Street, 2ND Floor
keley, CA.

SACRAMENTO CA 957
17 JUN 2019 PM 6 L

USA FOREVER

To: City of Berkeley / Roberto Terrones
c/o: Community Health Commission
2939 Ellis Street
Berkeley, CA. 94703

94703-210739

Attachment 7

Eden Environmental Citizen's Group, LLC v. Precision Technical Coatings, Inc. et al

-LAWSUIT-

Plaintiff:	Eden Environmental Citizen's Group LLC and Eden Environmental Citizen's Group, LLC
Defendant:	Precision Technical Coatings Inc., Douglas Tateoka, Michael Emmerich and Precision Technical Coatings, Inc.
Case Number:	3:2018cv05333
Filed:	August 29, 2018
Court:	California Northern District Court
Presiding Judge:	Joseph C Spero
Nature of Suit:	Environmental Matters
Cause of Action:	33:1319
Jury Demanded By:	Both

DEFENDANT:
PRECISION TECHNICAL COATINGS CORPORATION
1220 4th Street
Berkeley, CA. 94710

PLAINTIFF:
EDEN ENVIRONMENTAL CITIZEN'S GROUP

Eden Environmental Citizen's Group

Telephone: 925-732-0960
Email: edenenvcitizens@gmail.com
Offices in Richmond, Concord, San Jose, Santa Rosa and Sacramento

SOURCE: https://dockets.justia.com/docket/california/candce/3:2018cv05333/331364

Eden Environmental Citizen's Group, LLC v. Precision Technical Coatings, Inc. et al

Plaintiff:	Eden Environmental Citizen's Group LLC and Eden Environmental Citizen's Group, LLC
Defendant:	Precision Technical Coatings Inc., Douglas Tateoka, Michael Emmerich and Precision Technical Coatings, Inc.
Case Number:	3:2018cv05333
Filed:	August 29, 2018
Court:	California Northern District Court
Presiding Judge:	Joseph C Spero
Nature of Suit:	Environmental Matters
Cause of Action:	33:1319
Jury Demanded By:	Both

The city better not get stuck with clean-up bill for this toxic plant. The pollution situation at this plant is absolutely shameful. Water, soil, sewer, storm-drain pollution. It has been a problem plant!

The city better not get stuck with clean-up bill for this toxic plant. The pollution situation at this plant is absolutely shameful. Water, soil, sewer, storm-drain pollution. It has been a problem plant!

The city better not get stuck with clean-up bill for this toxic plant. The pollution situation at this plant is absolutely shameful. Water, soil, sewer, storm-drain pollution. It has been a problem plant!

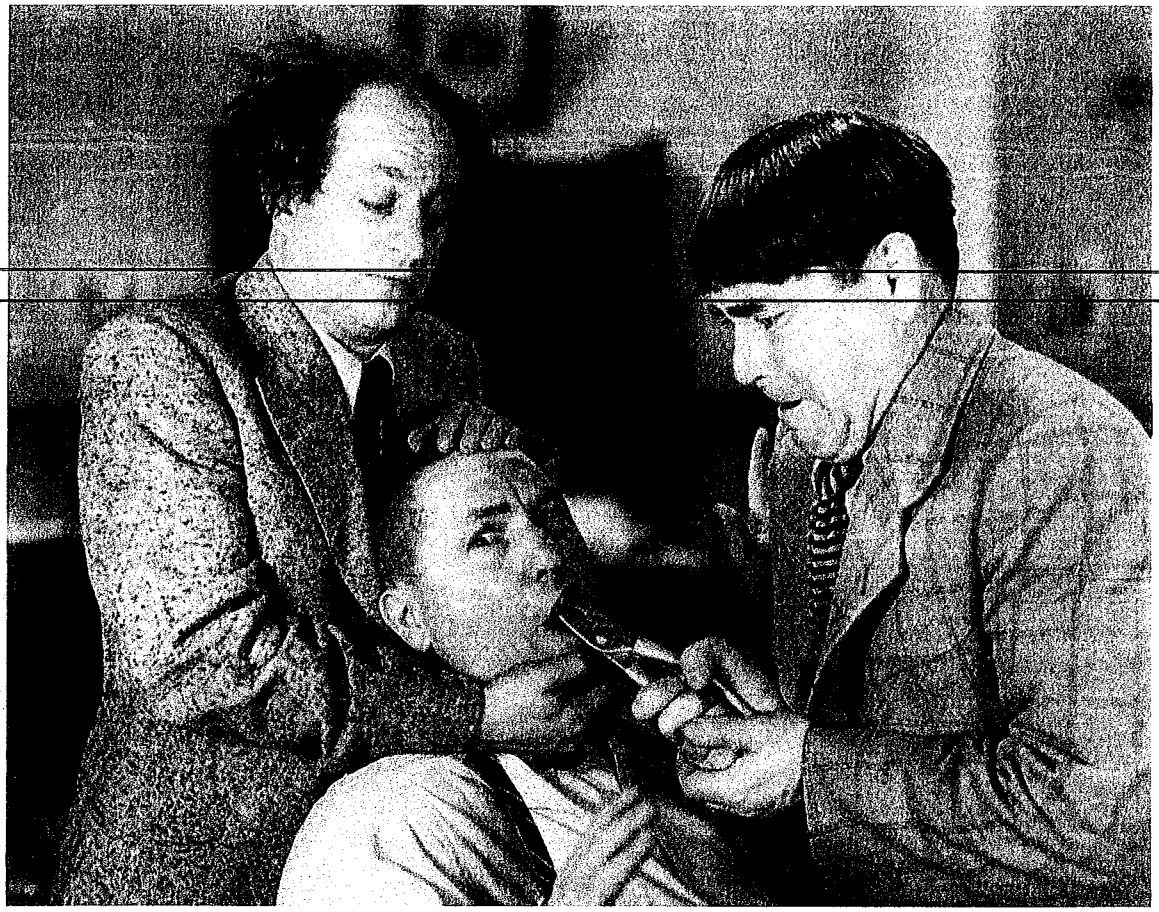
The city better not get stuck with clean-up bill for this toxic plant. The pollution situation at this plant is absolutely shameful. Water, soil, sewer, storm-drain pollution. It has been a problem plant!

The city better not get stuck with clean-up bill for this toxic plant. The pollution situation at this plant is absolutely shameful. Water, soil, sewer, storm-drain pollution. It has been a problem plant!

The city better not get stuck with clean-up bill for this toxic plant. The pollution situation at this plant is absolutely shameful. Water, soil, sewer, storm-drain pollution. It has been a problem plant!

The city better not get stuck with clean-up bill for this toxic plant. The pollution situation at this plant is absolutely shameful. Water, soil, sewer, storm-drain pollution. It has been a problem plant!

The city better not get stuck with clean-up bill for this toxic plant. The pollution situation at this plant is absolutely shameful. Water, soil, sewer, storm-drain pollution. It has been a problem plant!



"You should have taken up dentistry! Right, Moe?"

Proposed Letter from the Community Health Commission to the City Council Recommending Enactment of the Proposed Ordinance to Ban Natural Gas Infrastructure in New Buildings

The Community Health Commission recommends the adoption of the Proposed Ordinance to Ban Natural Gas Infrastructure in New Buildings because of the ordinance's positive effect on public health, specifically asthma in children.

Asthma is the most common chronic disease of childhood. According to the Centers for Disease Control and Prevention (CDC), 1 in 13 people have asthma, including 8.4% of children. The disease disproportionately causes morbidity in inner-city, minority children. According to the Institute of Medicine, several pollutants have been shown to worsen asthma including nitrogen dioxide (NO_2). Gas stoves and gas heaters are a significant source of indoor NO_2. Eliminating gas stoves and heaters in new construction will reduce the risk of asthma among children. This is especially true of minority children. The Commission would like to call the Council's attention to two relevant studies.

One study carried out by Johns Hopkins University in the City of Baltimore found that exposure to increasing concentrations of indoor NO_2 is associated with increased respiratory symptoms among a group of predominantly African-American, inner-city, preschool children with asthma. The report goes on to state:

> Understanding the effect of indoor air quality on asthma morbidity in inner-city preschool children is necessary because preschool children spend most of their time indoors and inner-city minority children suffer disproportionately from asthma and are exposed to high levels of indoor pollutants. Specifically, many inner-city households use gas stoves, an important source of indoor NO_2 concentrations, and many of these stoves are unvented. Furthermore, almost 14% of the homes in this study used gas stoves for heat. Because the use of a stove as a source of heat is seen almost exclusively in the context of profound poverty, this study also highlights the complex interaction of poverty with environmental exposures in an inner-city minority population.
>
> Our results show a consistent link between increased NO_2 concentrations and increased respiratory symptoms in preschool children with asthma. We further show that inner-city, predominantly minority children are exposed to high levels of indoor NO_2 concentrations. The burden of asthma attributable to differences in domestic NO_2 concentrations is substantial. A Longitudinal Study of Indoor Nitrogen Dioxide Levels and Respiratory Symptoms in Inner-City Children with Asthma by Hansel et. al., Environmental Health Perspectives, 116:10, October 2008.

A second more recent study from Australia, similarly found that gas stoves were associated with a considerable portion of the childhood asthma burden. Damp housing, gas stoves, and the burden of childhood asthma in Australia by Knibbs et. al., Medical Journal of Australia. 2018(7): 299-302.

Based on these findings the Community Health Commission urges the City Council to adopt the Proposed Ordinance to Ban Natural Gas Infrastructure in New Buildings.

A Longitudinal Study of Indoor Nitrogen Dioxide Levels and Respiratory Symptoms in Inner-City Children with Asthma

Nadia N. Hansel,[1] Patrick N. Breysse,[2] Meredith C. McCormack,[1] Elizabeth C. Matsui,[3] Jean Curtin-Brosnan,[3] D'Ann L. Williams,[2] Jennifer L. Moore,[1] Jennifer L. Cuhran,[1] and Gregory B. Diette[1]

[1]Department of Medicine, School of Medicine, Johns Hopkins University, Baltimore, Maryland, USA; [2]Department of Environmental Health Sciences, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, Maryland, USA; [3]Department of Pediatrics, School of Medicine, Johns Hopkins University, Baltimore, Maryland, USA

Background: The effect of indoor nitrogen dioxide concentrations on asthma morbidity among inner-city preschool children is uncertain.

Objectives: Our goal was to estimate the effect of indoor NO_2 concentrations on asthma morbidity in an inner-city population while adjusting for other indoor pollutants.

Methods: We recruited 150 children (2–6 years of age) with physician-diagnosed asthma from inner-city Baltimore, Maryland. Indoor air was monitored over a 72-hr period in the children's bedrooms at baseline and 3 and 6 months. At each visit, the child's caregiver completed a questionnaire assessing asthma symptoms over the previous 2 weeks and recent health care utilization.

Results: Children were 58% male, 91% African American, and 42% from households with annual income < $25,000; 63% had persistent asthma symptoms. The mean (± SD) in-home NO_2 concentration was 30.0 ± 33.7 (range, 2.9–394.0) ppb. The presence of a gas stove and the use of a space heater or oven/stove for heat were independently associated with higher NO_2 concentrations. Each 20-ppb increase in NO_2 exposure was associated significantly with an increase in the number of days with limited speech [incidence rate ratio (IRR) = 1.15; 95% confidence interval (CI), 1.05–1.25], cough (IRR = 1.10; 95% CI, 1.02–1.18), and nocturnal symptoms (IRR = 1.09; 95% CI, 1.02–1.16), after adjustment for potential confounders. NO_2 concentrations were not associated with increased health care utilization.

Conclusions: Higher indoor NO_2 concentrations were associated with increased asthma symptoms in preschool inner-city children. Interventions aimed at lowering NO_2 concentrations in inner-city homes may reduce asthma morbidity in this vulnerable population.

Key words: asthma, indoor pollutants, inner city, nitrogen dioxide, preschool. *Environ Health Perspect* 116:1428–1432 (2008). doi:10.1289/ehp.11349 available via *http://dx.doi.org/* [Online 23 July 2008]

Asthma is the most common chronic disease of childhood, affecting 6.5 million (8.9%) children in the United States and disproportionately causing morbidity in inner-city, minority children (Bloom et al. 2006). Many factors have been examined, and although no single explanation exists to explain such racial disparities, evidence supports a multitude of risk factors including differing exposure to environmental pollutants and differences in susceptibility (Barnes et al. 2007; Weiss et al. 1992). Several pollutants have been shown to worsen asthma including particulate matter (PM), ozone, and nitrogen dioxide (Institute of Medicine Committee on the Assessment of Asthma and Indoor Air 2000; Koren 1995). NO_2 may be a particular problem in the inner city, where gas stoves are common and proper venting of stoves may be rare. In fact, our group has previously demonstrated high indoor NO_2 concentrations in inner-city homes (Breysse et al. 2005; Diette et al. 2007). Furthermore, because preschool children spend much of their time in the home (Klepeis et al. 2001; McCormack et al. 2007a), they may be especially at risk to the adverse effects of indoor NO_2 exposure.

Although some studies have shown that NO_2 can affect asthma in children (Belanger et al. 2006; Garrett et al. 1998; Hasselblad et al. 1992; Kattan et al. 2007; Nitschke et al. 2006; Shima and Adachi 2000; Smith et al. 2000), very few have focused on African-American, inner-city children or preschool children. It is important to replicate findings from previous studies done in other populations because some results have been inconsistent (Florey et al. 1979; Hoek et al. 1984; Samet et al. 1993; Sunyer et al. 2004), and future guidelines and public policies depend on a robust evidence base. The purpose of this study was to investigate the independent longitudinal effect of indoor NO_2 concentrations on asthma morbidity, accounting for copollutants. To study this question, we conducted a prospective cohort study of predominantly African-American preschool children with asthma living in inner-city Baltimore, Maryland.

Methods

Participants were recruited for the Baltimore Indoor Environment Study of Asthma in Kids as previously described (Hansel et al. 2006; McCormack et al. 2007a; Sharma et al. 2007). All subjects were residents of inner-city Baltimore, defined by nine contiguous ZIP codes and encompassing a relatively small area (approximately 4 mi^2). Potential participants were identified from a random sample of children with a health care encounter for asthma in the previous 12 months at Johns Hopkins Community Physicians or Bayview Pediatrics. Eligibility criteria were as follows: *a*) age between 2 and 6 years, *b*) physician-diagnosed asthma, *c*) at least one health care encounter for asthma within the preceding 12 months (according to the *International Classification of Diseases, 9th Revision*, code 493.xx) (World Health Organization 2000), and *d*) symptoms or use of asthma medications within the preceding 6 months. All participants were told the general purpose of the study, including the study of the effects of the home environment on asthma health, but were not aware of the specific study hypotheses addressed here. The Johns Hopkins Medical Institutional Review Board approved the study protocol.

Participants underwent home visits at baseline and 3 and 6 months to complete health and environmental surveys, home inspection, and environmental sample collection.

Patient characteristics. The health questionnaire included questions assessing demographics, comorbidities, and medication use and modified questions from the International Study of Asthma and Allergies in Childhood (Asher et al. 1995) and the Children's Health Survey for Asthma (Asmussen et al. 1999) to evaluate indicators of poor asthma control. Caregivers were asked about their child's symptoms in the previous 2 weeks regarding *a*) any daytime wheezing, coughing, or tightness in the chest; *b*) need to slow down or stop activities at home or playing because of asthma, wheezing, or tightness in the chest or cough; *c*) wheezing that was so bad that he or she could speak only one or two words at a time between breaths; *d*) wheezing, coughing,

Address correspondence to G.B. Diette, Division of Pulmonary and Critical Care Medicine, Johns Hopkins University, 1830 East Monument St., Baltimore, MD 21205 USA. Telephone: (410) 502-7038. Fax: (410) 955-0036. E-mail: gdiette@mail.jhmi.edu

We thank F. Dominici and L. Pham for their review of our statistical modeling methods.

This study was supported by grants from the U.S. Environmental Protection Agency (R82672401), National Institute of Environmental Health Sciences (ES09606), and National Heart, Blood, and Lung Institute (HL04266, HL076322, HL67850).

The authors declare they have no competing financial interests.

Received 8 February 2008; accepted 23 July 2008.

or tightness in the chest when running or going up stairs; *e*) coughing that wasn't from a cold; and *f*) nocturnal awakenings because of cough, wheeze, shortness of breath, or tightness in chest. Each symptom was quantified as the number of days that the symptom was present (0–14 days). Questions also ascertained information about acute health care use for asthma in the preceding 3 months (emergency department visits, unscheduled doctor visits, and hospitalizations) and rescue medication use in the previous 2 weeks (short-acting beta agonist). Child's asthma severity was categorized as recommended by the National Asthma Education and Prevention Program guidelines (EPR-2) (National Heart, Lung, and Blood Institute 1997). Allergic sensitization status was assessed by skin prick test to 14 common aero-allergens. A child was considered atopic if he or she had at least one positive (wheal diameter ≥ 2 mm) skin test.

Environmental assessment. We used an interviewer-administered environmental questionnaire to assess housing characteristics and potential sources of indoor NO_2 concentrations. A time–activity diary was administered during the 72-hr monitoring period to track household activities that may be correlated with indoor NO_2 concentrations, including number of windows open for > 10 min/day, vacuuming, sweeping, and stove/oven, space heater, candle/incense, air purifier, and cigarette use. A home inspection was conducted to assess distance from curb to front door of the home and type of street in front of house (side street, arterial, or parking lot).

Indoor air sampling for NO_2 and $PM_{2.5}$ (PM with an aerodynamic diameter ≤ 2.5 μm) was conducted continuously over a 72-hr period, as previously described (Diette et al. 2007; McCormack et al. 2007a). The monitors were placed in the sleeping room of the child, which was ascertained by the technician before placement of the monitors. All sampling heads and passive badges were attached to the outside of a sampling frame that was placed in a convenient location in the child's bedroom. In most cases, the sampling frame was placed on the dresser or a nightstand. In some cases, when there was no available elevated surface, the sample frame was place on a portable stand constructed out of polyvinyl chloride pipe. NO_2 was measured using a passive sampler (Ogawa badge) loaded with filters coated with triethanolamine (TEA) (Palmes et al. 1976). Samplers and coated filters were purchased from Ogawa, Inc. (Pompano Beach, FL). In the presence of a color reagent, NO_2 and TEA form a highly colored azo dye that is measured spectrophotometrically at 540 nm. The median limit of detection calculated from the analysis of field blanks was 6.8 ppb. $PM_{2.5}$ samples were collected using 4 L/min MSP impactors (MSP Corp., St. Paul, MN) loaded with 37-mm, 2.0-μm pore size, PALL Teflo PTFE membrane filters with polypropylene support rings (Pall Corp., Ann Arbor, MI).

Daily ambient NO_2 levels during the study period were obtained from the U.S. Environmental Protection Agency Air Quality System database (U.S. Environmental Protection Agency 2007) All homes were within 4.8 miles of the central monitoring site.

Statistical analysis. We used descriptive statistics to characterize the patient sample and the pollutant levels. We compared summary statistics using Spearman correlations, chi-square tests for proportions, and *t*-tests for continuous data. All variables had < 10% missing data; therefore, missing values were imputed with the median or mode for continuous or categorical variables, respectively.

Adjusting for season of sampling, we used linear regression models to assess whether household characteristics and daily activities during the monitoring period predicted baseline indoor NO_2 concentrations. Variables with $p \le 0.05$ were included in a multivariate model to assess the independent effect of each predictor. To evaluate the effect of NO_2 on asthma health, we analyzed NO_2 as a continuous variable using negative binomial or logistic regression models and generalized estimating equations (Diggle et al. 2002) to estimate incidence rate ratios (IRRs) and odds ratios (ORs), respectively, and to take into account the intercorrelation arising from repeated measures over time. At each time point (baseline, 3 months, and 6 months), the mean NO_2 concentration over the 3-day monitoring period and symptom frequency reported over the previous 2-week period were used as exposure and outcome variables, respectively. We used multivariate models to adjust for potential confounders, including age, sex, race, caregiver education level, season of sampling, $PM_{2.5}$, secondhand smoke (SHS) exposure [defined as caregiver report of presence of a smoker in the home (yes vs. no)], distance from curb, and type of street in front of house. We conducted additional analyses including mean ambient NO_2 as a covariate to ensure that the effects of indoor NO_2 were independent of ambient NO_2 levels. Stratified analyses were performed separately on subjects with atopy and those using daily inhaled corticosteroids (ICS). All analyses were performed with StataSE statistical software, version 8.0 (StataCorp, College Station, TX). Statistical significance was defined as $p < 0.05$.

Results

Participant characteristics. Participants were preschool children with asthma, most of whom were atopic (69%) and had persistent asthma symptoms at baseline (Table 1). Approximately 80% of the primary caregivers had no more than a high school education. Children were predominantly African American, and many children (42%) lived in households with incomes < $25,000, representing an urban population of low socioeconomic status. Eighty-three percent of participant homes had gas stoves, and 72% were heated by natural gas fuel. As ascertained by the daily time–activity diary, when home the children spent most of their time in the room where the monitoring occurred. Specifically, children in this study, on average, spent 13 hr/day in their own home, and 7 hr/day in the bedroom where monitoring occurred.

Indoor NO_2 concentrations. Most of the homes were row homes (homes that share adjacent walls; 79%) and close to the street (within 25 feet; 71%). Sixty-two percent of homes were in front of a side street, 27% in front of an arterial street, and 11% in front of a parking lot. The overall mean (± SD) indoor NO_2 concentration was 30.0 ± 33.7 ppb (range, 2.9–394.0 ppb), and mean $PM_{2.5}$ concentration was 40.3 ± 35.4 μg/m^3 (range, 0.1–216.1 μg/m^3). There was no statistical difference in mean pollutant concentrations between baseline, visit 2, and visit 3. NO_2 concentrations were significantly lower in summer (15.9 ± 14.0 ppb) than in any other season (fall: 30.8 ± 27.5 ppb; winter: 41.4 ± 52.1 ppb; spring: 30.7 ± 23.5 ppb; $p < 0.001$). The mean ambient NO_2 concentration during

Table 1. Child and caregiver characteristics ($n = 150$).

Characteristic	Percent
Child characteristics	
Age [mean (range)]	4.4 (2–6)
Male sex	58.0
Race	
Black	91.2
White	4.7
Other	4.1
Asthma severity	
Mild intermittent	37
Mild persistent	17
Moderate persistent	21
Severe persistent	25
Atopic	69
Asthma medication use (last 2 weeks)	
Albuterol	53
ICS	34
Other (cromolyn, leukotriene modifier, theophylline, oral corticosteroids)	19
Caregiver characteristics	
Primary caregiver	
Birth mother	87.1
Grandmother	4.8
Birth father	2.7
Other	5.4
Education	
Not high school graduate	38.5
High school graduate	42.6
At least some college	19.1
Household income (annual)	
< $25,000	41.6
$25,000–$50,000	10.8
> $50,000	2.0
Not reported	20.8

the study period was 25.7 ppb. There was minimal correlation (r^2 = 0.056, p < 0.01) between ambient and indoor NO_2 concentrations (Figure 1).

NO_2 concentrations were higher in homes with a gas stove (mean, 33.1 ppb) compared with those without a gas stove (mean, 16.8 ppb). Similarly, the mean indoor NO_2 concentrations were 7.2 ppb higher in homes with a gas heater compared with those without a gas heater, and the presence of a gas heater had a greater effect on indoor NO_2 concentrations during the winter months (β = 17.8; SE 9.7). In addition, after adjusting for season, using a space heater or the stove or oven for heat during the monitoring period

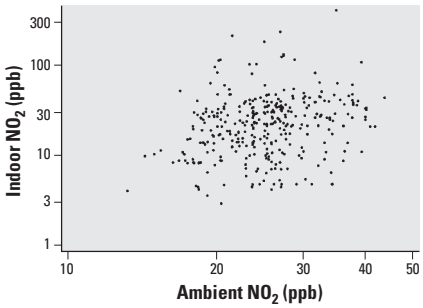


Figure 1. Correlation between indoor and ambient NO_2 concentrations. Mean ambient and indoor NO_2 concentrations during the monitoring period are represented in parts per billion on the *x*- and *y*-axes, respectively. There was minimal correlation between indoor and outdoor concentrations (Spearman's r^2 = 0.056, p < 0.01).

was associated with higher NO_2 concentrations. The independent effect of the presence of a gas stove and using a space heater or the stove or oven for heat on indoor NO_2 concentrations remained essentially unchanged after adjustment for the presence of the other predictors. Other housing characteristics and daily activities were not associated with indoor NO_2 levels (Table 2).

Association of NO_2 levels with asthma outcomes. Higher NO_2 concentrations were associated with statistically significant increases in respiratory symptoms (Table 3). After adjusting for potential confounders, increasing NO_2 concentrations remained significantly associated with increasing frequency of limited speech due to wheezing, coughing without a cold, and nocturnal awakenings due to cough, wheeze, and shortness of breath or chest tightness during the daytime and while running. There was no significant relationship between NO_2 concentration and rescue medication use or health care utilization [rescue medication use OR = 1.00 (95% confidence interval [CI], 0.93–1.07); unscheduled doctor's visit OR = 0.99 (95% CI, 0.92–1.35); asthma-related hospital visit OR = 1.13 (95% CI, 0.69–1.09); emergency department visit for asthma OR = 0.88 (95% CI, 0.83–1.17)].

In general, the presence of atopy did not modify the effect of NO_2 exposure on asthma symptoms, except that individuals with atopy were more likely to experience nocturnal symptoms with increasing NO_2 concentration (IRR = 1.13 per 20-ppb increase in NO_2) compared with nonatopic individuals (IRR = 1.03). In addition, daily use of ICS did not modify the association of NO_2 concentrations and asthma symptoms, and mean ambient NO_2 concentrations were not significantly associated with any respiratory symptoms (data not shown).

Discussion

Our study shows that exposure to increasing concentrations of indoor NO_2 is associated with increased respiratory symptoms among a group of predominantly African-American, inner-city, preschool children with asthma. Understanding the effect of indoor air quality on asthma morbidity in inner-city preschool children is necessary because preschool children spend most of their time indoors (Klepeis et al. 2001), and inner-city minority children suffer disproportionately from asthma and are exposed to high levels of indoor pollutants (Bloom et al. 2006; Breysse et al. 2005). Specifically, many inner-city households use gas stoves, an important source of indoor NO_2 concentrations, and many of these stoves are unvented (Breysse et al. 2005; Diette et al. 2007). Furthermore, almost 14% of the homes in this study used gas stoves for heat. Because the use of a stove as a source of heat is seen almost exclusively in the context of profound poverty, this study also highlights the complex interaction of poverty with environmental exposures in an inner-city minority population.

Table 2. Predictors of indoor NO_2 concentrations (adjusted for season).

Characteristic	Percent reporting characteristic or activity[a]	β	95% CI	*p*-Value	Multivariate models[b] β	Multivariate models[b] 95% CI	Multivariate models[b] *p*-Value
Housing characteristics							
Gas stove	83	15.0	6.2 to 23.8	< 0.001	15.7	6.9 to 24.6	0.001
Gas heater	72	7.2	–0.1 to 14.6	0.05	4.4	–2.8 to 11.6	0.23
Daily activities over the monitoring period							
Space heater use	5	16.40	1.19 to 31.61	0.04	14.4	0.8 to 28.8	0.05
Stove/oven for heat	12	12.49	2.44 to 22.53	0.02	12.4	2.6 to 22.2	0.01
Sweeping (per sweeping event)	85	1.00	–0.12 to 2.11	0.08	—	—	—
Cigarettes (per cigarette)	56	0.04	–1.0 to 0.17	0.59	—	—	—
Open windows (per open window)	85	–0.38	–1.09 to 0.33	0.29	—	—	—
Candles/incense	32	–2.37	–9.66 to 4.93	0.52	—	—	—
Air purifier use	1	–9.17	–49.38 to 31.03	0.65	—	—	—

—, no data are available for multivariate models for these variables because only variables with p < 0.05 on bivariate analyses were included in the multivariate model.
[a]All characteristics or activities are reported as present (yes vs. no) during the monitoring period, except for the number of cigarettes smoked, number of sweeping events, and number of open windows (for > 10 min) during the monitoring period, which were analyzed as continuous variables. [b]Multivariate models are adjusted for presence of gas stove, presence of gas heater, use of space heater, use of oven/stove for heat and season.

Table 3. Risk of asthma symptoms per 20-ppb increase in NO_2 exposure.

Symptom	Unadjusted IRR	Unadjusted 95% CI	Adjusted[a] IRR	Adjusted[a] 95% CI	Adjusted[b] IRR	Adjusted[b] 95% CI
Daytime wheezing, coughing, or chest tightness	1.05	0.99–1.12	1.03	0.96–1.11	1.04	0.97–1.12
Slowing activity due to asthma, wheeze, chest tightness, or cough	1.07	1.00–1.14*	1.06	0.99–1.14	1.08	0.94–1.15
Limited speech due to wheeze	1.12	1.04–1.21**	1.15	1.05–1.25**	1.17	1.08–1.27**
Wheeze, cough, or chest tightness while running	1.08	1.01–1.15*	1.07	0.99–1.14	1.09	1.01–1.17*
Coughing without a cold	1.13	1.06–1.20#	1.10	1.02–1.18**	1.15	1.07–1.23#
Nocturnal awakenings due to cough, wheeze, shortness of breath, or chest tightness	1.11	1.04–1.18#	1.09	1.02–1.16*	1.12	1.04–1.19**

[a]Models are adjusted for $PM_{2.5}$; SHS; season of sampling; age, sex, and race of the child; and mother's education level. [b]Models are adjusted for $PM_{2.5}$; SHS; distance from the curb, type of street in front of home, season of sampling; age, sex, and race of the child; and mother's education level. *p < 0.05. **p < 0.01. #p < 0.001.

Current evidence has not yet convincingly demonstrated that high indoor NO_2 concentrations contribute to the risk of developing asthma, because NO_2 concentrations are similar in homes of children with and without asthma (Diette et al. 2007; Hoek et al. 1984; Institute of Medicine Committee on the Assessment of Asthma and Indoor Air 2000). Studies done in subjects with asthma have suggested that higher indoor NO_2 concentrations lead to increased asthma symptoms; however, results have not been consistent across subpopulations (Belanger et al. 2006; Garrett et al. 1998; Hasselblad et al. 1992; Kattan et al. 2007; Nitschke et al. 2006; Shima and Adachi 2000; Smith et al. 2000). Young children and those with lower socioeconomic status (SES) may be at particular risk. For example, Smith et al. (2000) identified an association between NO_2 concentrations and increased risk of asthma symptoms in individuals < 14 years of age, but not in older individuals, except for a marginal increased risk of cough in subjects 35–49 years of age. In a cross-sectional analysis, Belanger et al. (2006) found that indoor NO_2 exposure was associated with increased incidence and frequency of chest tightness and wheezing, but only in individuals living in multifamily housing units, which was an indicator of lower SES. Our longitudinal study with repeated measures of NO_2 concentrations and respiratory symptoms improves on our ability to directly model individual response to changing NO_2 concentrations accounting for within-person correlations of asthma severity. Our results show a consistent link between increased NO_2 concentrations and increased respiratory symptoms in preschool children with asthma. We further show that inner-city, predominantly minority children are exposed to high levels of indoor NO_2 concentrations. The burden of asthma attributable to differences in domestic NO_2 concentrations is substantial. For example, a child would experience 10% more days of cough symptoms or 15% more days with limited speech due to wheeze with each 20-ppb increase in NO_2 exposure. A child experiencing limited speech once a week would experience an additional 13 days of limited speech per year if living in a home where NO_2 levels increased by 33.6 ppb (1 SD). Importantly, NO_2 was consistently associated with coughing, nocturnal symptoms, and limited speech, even after adjusting for potential confounders and other pollutants.

The indoor NO_2 concentrations observed in this study are high compared with levels found in some other studies, with mean indoor NO_2 concentrations in most studies ranging between 6 and 30 ppb (Nitschke et al. 1999). However, the levels we found are consistent with levels measured in other studies that have focused on inner-city homes (Breysse et al. 2005; Kattan et al. 2007; Zota et al. 2005). Because the presence of a gas stove or gas heater and the use of a space heater or gas or oven for heat were associated with higher NO_2 concentrations, and ambient NO_2 concentrations were only minimally correlated with indoor levels, it appears that changes to home heating and cooking devices may be a feasible means to reduce the burden of asthma. To our knowledge, there has been only one previous trial specifically targeting NO_2 (Pilotto et al. 2004). In that study, conducted in schools, unflued gas heaters were replaced with either electric heaters or flued gas heaters. The intervention led to reduced NO_2 concentrations and respiratory symptoms in children with asthma (Pilotto et al. 2004). We believe that clinical trials are still needed to assess the effectiveness of reducing NO_2 concentrations in inner-city homes on improving asthma morbidity.

The link between indoor NO_2 concentrations and asthma symptoms appears to be robust, because the associations were not significantly affected by the potential confounders studied. Indeed, our study was strengthened by its ability to adjust for other relevant copollutants. Although $PM_{2.5}$ was associated with increased asthma symptoms (McCormack et al. 2007b) (data not shown), adjusting for other copollutants did not meaningfully alter the association between indoor NO_2 concentrations and asthma symptoms. However, because ambient NO_2 concentrations can vary within a community based on traffic-related exposure (Clougherty et al. 2008; Gauderman et al. 2005; Wheeler et al. 2007), residual confounding of traffic-related exposure may lead to misclassification of individual ambient NO_2 exposure. Additionally, stratified analyses showed that the associations between NO_2 exposure and asthma symptoms were not significantly different in atopic subjects and those with daily ICS use. These findings are in contrast to the results of the National Cooperative Inner-City Asthma Study in which an association of higher levels of indoor NO_2 with increased asthma symptoms was found only in nonatopic children (Kattan et al. 2007). Indoor NO_2 concentrations were not associated with health care utilization. Thus, it is possible that indoor NO_2 exposure was related to increased respiratory symptoms but not sufficient to precipitate severe asthma attacks requiring unscheduled doctor visits, emergency department visits, or hospitalizations. In addition, the link between indoor NO_2 concentrations and respiratory symptoms is not corroborated with objective data on pulmonary function, given the difficulty in obtaining reliable measures of lung function in this young age group. It is also possible that our study under- or overestimated the true effect of indoor NO_2 concentrations on asthma symptoms, because we did not include personal monitoring of exposure. However, we are reassured that children in the present study spent more than half their time in their own home, most of which was in the bedroom. Furthermore, studies have shown that indoor NO_2 concentrations remain relatively stable in a given home over a month-long period (Brunekreef et al. 1987; Spengler et al. 1996), suggesting that short-term monitoring is a reasonable reflection of recent exposure levels.

In summary, in this study we found a link between higher NO_2 concentrations and increased respiratory symptoms in preschool, inner-city, primarily African-American children with asthma. Furthermore, individuals in the inner city appear to be at particularly high risk for the adverse effects of indoor NO_2 concentrations, given their high indoor exposure levels. The presence of a gas stove, as well as use of stove/oven and space heater for heat, was independently associated with higher indoor NO_2 levels, which suggests that there are modifiable sources of exposure. Our study has major implications for health care providers and asthma patients. The next step should be to conduct research studies that show whether changing from natural gas to other fuel sources (e.g., electricity) can reduce the asthma burden. In the meantime, we would recommend that families who have children with asthma, if afforded the opportunity to choose housing, choose homes without gas stoves and heaters. Those who already have these appliances should be cautioned at least about the need for proper venting of the exhaust gases. Interventions aimed at lowering NO_2 concentrations in inner-city homes may reduce asthma morbidity in this vulnerable population.

References

Asher MI, Keil U, Anderson HR, Beasley R, Crane J, Martinez F, et al. 1995. International Study of Asthma and Allergies in Childhood (ISAAC): rationale and methods. Eur Respir J 8:483–491.

Asmussen L, Olson LM, Grant EN, Fagan J, Weiss KB. 1999. Reliability and validity of the Children's Health Survey for Asthma. Pediatrics 104:e71.

Barnes KC, Grant AV, Hansel NN, Gao P, Dunston GM. 2007. African Americans with asthma: genetic insights. Proc Am Thorac Soc 4:58–68.

Belanger K, Gent JF, Triche EW, Bracken MB, Leaderer BP. 2006. Association of indoor nitrogen dioxide exposure with respiratory symptoms in children with asthma. Am J Respir Crit Care Med 173:297–303.

Bloom B, Dey AN, Freeman G. 2006. Summary health statistics for U.S. children: National Health Interview Survey, 2005. Vital Health Stat 10:1–84.

Breysse PN, Buckley TJ, Williams D, Beck CM, Jo SJ, Merriman B, et al. 2005. Indoor exposures to air pollutants and allergens in the homes of asthmatic children in inner-city Baltimore. Environ Res 98:167–176.

Brunekreef B, Noy D, Clausing P. 1987. Variability of exposure measurements in environmental epidemiology. Am J Epidemiol 125:892–898.

Clougherty JE, Wright RJ, Baxter LK, Levy JI. 2008. Land use

regression modeling of intra-urban residential variability in multiple traffic-related air pollutants. Environ Health 17:17.

Diette GB, Hansel NN, Buckley TJ, Curtin-Brosnan J, Eggleston PA, Matsui EC, et al. 2007. Home indoor pollutant exposures among inner-city children with and without asthma. Environ Health Perspect 115:1665–1669.

Diggle PJ, Heagerty P, Liang KY, Zeger S. 2002. The Analysis of Longitudinal Data. 2nd ed. Oxford, UK:Oxford University Press.

Florey CV, Melia RJ, Chinn S, Goldstein BD, Brooks AG, John HH, et al. 1979. The relation between respiratory illness in primary schoolchildren and the use of gas for cooking. III. Nitrogen dioxide, respiratory illness and lung infection. Int J Epidemiol 8:347–353.

Garrett MH, Hooper MA, Hooper BM, Abramson MJ. 1998. Respiratory symptoms in children and indoor exposure to nitrogen dioxide and gas stoves. Am J Respir Crit Care Med 158:891–895.

Gauderman WJ, Avol E, Lurmann F, Kuenzili N, Gilliland F, Peters J, et al. 2005. Childhood asthma and exposure to traffic and nitrogen dioxide. Epidemiology 16(6):737–743.

Hansel NN, Eggleston PA, Krishnan JA, Curtin-Brosnan J, Rand CS, Patino CM, et al. 2006. Asthma-related health status determinants of environmental control practices for inner-city preschool children. Ann Allergy Asthma Immunol 97:409–417.

Hasselblad V, Eddy DM, Kotchmar DJ. 1992. Synthesis of environmental evidence: nitrogen dioxide epidemiology studies. J Air Waste Manage Assoc 42:662–671.

Hoek G, Brunekreef B, Meijer R, Scholten A, Boleij J. 1984. Indoor nitrogen dioxide pollution and respiratory symptoms of schoolchildren. Int Arch Occup Environ Health 55:79–86.

Institute of Medicine Committee on the Assessment of Asthma and Indoor Air. 2000. Clearing the Air: Asthma and Indoor Air Exposures. Washington, DC:National Academy Press..

Kattan M, Gergen PJ, Eggleston P, Visness CM, Mitchell HE. 2007. Health effects of indoor nitrogen dioxide and passive smoking on urban asthmatic children. J Allergy Clin Immunol 120:618–624.

Klepeis NE, Nelson WC, Ott WR, Robinson JP, Tsang AM, Switzer P, et al. 2001. The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. J Expo Anal Environ Epidemiol 11:231–252.

Koren HS. 1995. Associations between criteria air pollutants and asthma. Environ Health Perspect 103(suppl 6):235–242.

McCormack MC, Breysse PN, Hansel NN, Matsui EC, Tonorezos ES, Curtin-Brosnan J, et al. 2007a. Common household activities are associated with elevated particulate matter concentrations in bedrooms of inner-city Baltimore pre-school children. Environ Res 106(2):148–155.

McCormack MC, Hansel NN, Matsui EC, Breysse PN, Tonorezos ES, Diette GB. 2007b. Domestic Particulate Matter Exposure and Asthma Morbidity in Inner-City preschool Children. AJRCCM 175:A272.

National Heart, Lung, and Blood Institute. 1997. Guidelines for the Diagnosis and Management of Asthma: Expert Panel Report 2. Bethesda, MD:National Heart, Lung, and Blood Institute.

Nitschke M, Pilotto LS, Attewell RG, Smith BJ, Pisaniello D, Martin J, et al. 2006. A cohort study of indoor nitrogen dioxide and house dust mite exposure in asthmatic children. J Occup Environ Med 48:462–469.

Nitschke M, Smith BJ, Pilotto LS, Pisaniello D, Abramson MJ, Ruffin RE. 1999. Respiratory health effects of nitrogen dioxide exposure and current guidelines. Int J Environ Health Res 9:39–53.

Palmes ED, Gunnison AF, DiMattio J, Tomczyk C. 1976. Personal sampler for nitrogen dioxide. Am Ind Hyg Assoc J 37:570–577.

Pilotto LS, Nitschke M, Smith BJ, Pisaniello D, Ruffin RE, McElroy HJ, et al. 2004. Randomized controlled trial of unflued gas heater replacement on respiratory health of asthmatic schoolchildren. Int J Epidemiol 33:208–214.

Samet JM, Lambert WE, Skipper BJ, Cushing AH, Hunt WC, Young SA, et al. 1993. Nitrogen dioxide and respiratory illnesses in infants. Am Rev Respir Dis 148:1258–1265.

Sharma HP, Matsui EC, Eggleston PA, Hansel NN, Curtin-Brosnan J, Diette GB. 2007. Does current asthma control predict future health care use among black preschool-aged inner-city children? Pediatrics 120:e1174–e1181

Shima M, Adachi M. 2000. Effect of outdoor and indoor nitrogen dioxide on respiratory symptoms in schoolchildren. Int J Epidemiol 29:862–870.

Smith BJ, Nitschke M, Pilotto LS, Ruffin RE, Pisaniello DL, Willson KJ. 2000. Health effects of daily indoor nitrogen dioxide exposure in people with asthma. Eur Respir J 16:879–885.

Spengler JD, Schwab M, McDermott A, Lambert WE, Samet JM. 1996. Nitrogen dioxide and respiratory illness in children. Part IV: Effects of housing and meteorologic factors on indoor nitrogen dioxide concentrations. Res Rep Health Eff Inst 58:1–29.

Sunyer J, Puig C, Torrent M, Garcia-Algar O, Calico I, Munoz-Ortiz L, et al. 2004. Nitrogen dioxide is not associated with respiratory infection during the first year of life. Int J Epidemiol 33:116–120.

U.S. Environmental Protection Agency. 2007. Air Quality System Data Mart. Available: http://www.epa.gov/ttn/airs/aqsdatamart [accessed 2 November 2007].

Weiss KB, Gergen PJ, Crain EF. 1992. Inner-city asthma. The epidemiology of an emerging US public health concern. Chest 101:362S–367S.

Wheeler AJ, Smith-Doiron M, Xu X, Gilbert NL, Brook JR. 2007. Intra-urban variability of air pollution in Windsor, Ontario—measurement and modeling for human exposure assessment. Environ Res 106(1):7–16.

World Health Organization. 2000. International Classification of Diseases, 9th Revision, Clinical Modification, 6th ed. Salt Lake City:Ingenix Publishing Group

Zota A, Adamkiewicz G, Levy JI, Spengler JD. 2005. Ventilation in public housing: implications for indoor nitrogen dioxide concentrations. Indoor Air 15:393–401.

Damp housing, gas stoves, and the burden of childhood asthma in Australia

Luke D Knibbs[1], Solomon Woldeyohannes[1], Guy B Marks[2,3], Christine T Cowie[4]

The known Australian studies have identified adverse respiratory effects in children of indoor environmental exposures, but their contribution to the national asthma burden has not been quantified.

The new Exposure of Australian children to damp housing and gas stove emissions is common, and is respectively associated with 7.9% and 12.3% of the total asthma burden in children aged 14 years or under.

The implications A coordinated national strategy is needed to increase awareness of indoor environmental exposures in the context of asthma prevention, and of interventions for reducing exposure to gas stove emissions and dampness.

Abstract

Objective: To determine the proportion of the national childhood asthma burden associated with exposure to dampness and gas stoves in Australian homes.

Design: Comparative risk assessment modelling study.

Setting, participants: Australian children aged 14 years or less, 2011.

Main outcome measures: The population attributable fractions (PAFs) and number of disability-adjusted life years (DALYs) for childhood asthma associated with exposure to damp housing and gas stoves.

Results: 26.1% of Australian homes have dampness problems and 38.2% have natural gas as the main energy source for cooktop stoves. The PAF for childhood asthma attributable to damp housing was 7.9% (95% CI, 3.2–12.6%), causing 1760 disability-adjusted life years (DALYs; 95% CI, 416–3104 DALYs), or 42 DALYs/100 000 children. The PAF associated with gas stoves was 12.3% (95% CI, 8.9–15.8%), corresponding to 2756 DALYs (95% CI, 1271–4242), or 67 DALYs/100 000 children. If all homes with gas stoves were fitted with high efficiency range hoods to vent gas combustion products outdoors, the PAF and burden estimates were reduced to 3.4% (95% CI, 2.2–4.6%) and 761 DALYs (95% CI, 322–1199).

Conclusions: Exposure to damp housing and gas stoves is common in Australia, and is associated with a considerable proportion of the childhood asthma burden. Strategies for reducing exposure to indoor dampness and gas combustion products should be communicated to parents of children with or at risk of asthma.

The prevalence of asthma in Australia is among the highest in the world; 10% of the population has current asthma,[1] and it is the leading contributor to disease burden in Australian children aged 14 years or less.[1] Children spend 60–75% of their time indoors at home, which can expose them to several risk factors for asthma,[2] including damp housing and gas stove emissions. Although several Australian studies have reported negative associations between indoor exposures and respiratory health in children,[3-5] there have been no systematic attempts to quantify their contribution to the national childhood asthma burden.

In this study, we sought to estimate this contribution, with the aims of informing clinicians about the magnitude of the burden and equipping policy makers with recommendations for improving population health.

Methods

We selected damp housing and gas stoves as the two indoor residential exposures to investigate because exposure to gas stoves has been associated with childhood asthma, and damp housing has been causally linked with childhood asthma. Both are common in Australia, and are amenable to interventions for reducing exposure.[6,7] Although gas heaters are also common in many parts of Australia and have been associated with respiratory irritation and asthma symptoms,[8,9] we did not include them in our analysis because our preliminary search found no recent meta-analyses of their association with childhood asthma.

We applied a comparative risk assessment approach,[10] which requires data on the proportion of the population exposed, effect estimates for asthma among those exposed, and the total asthma burden in children aged 14 years or under expressed as disability-adjusted life years (DALYs). We selected 2011 as our reference year because it was the year with the greatest overlap of input data.

Dampness definition and exposure estimation

We defined dampness as the presence of visible mould in a dwelling (apart from the bathroom) and assumed, as have similar studies, that visible mould was entirely attributable to excessive dampness.[11] As there are no national estimates of household dampness in Australia, we searched PubMed, Web of Science, Scopus, and Google Scholar for peer-reviewed studies reporting the prevalence of dampness in Australian homes (online Appendix). The nine studies we identified, undertaken in four of six Australian states and published during 1998–2014, included a total of 7538 homes. We weighted the results from each study by the number of homes sampled and the 2011 census population of the greater metropolitan area of the city in which the studies were performed to estimate the proportion of children exposed to dampness at home.

Gas stove definition and exposure estimation

We defined gas stove exposure as the presence of a gas stove in the home. To estimate the proportion of children exposed to gas stoves, we used data from the 2011 Energy Use and Conservation Survey of the Australian Bureau of Statistics, which collected information on the main energy source used for stoves, including piped natural

[1]University of Queensland, Brisbane, QLD. [2]Woolcock Institute of Medical Research, Sydney, NSW. [3]Liverpool Hospital, Sydney, NSW. [4]South Western Sydney Clinical School, University of New South Wales, Sydney, NSW. l.knibbs@uq.edu.au
▪ doi: 10.5694/mja17.00469 ▪ See Editorials, p. 296, 297
Podcast with Luke Knibbs available at https://www.mja.com.au/podcasts

gas (ie, mains gas).[12] These data were employed because of the large sample size of the survey (12 841 households), its national coverage, and the well documented quality assurance information on sampling error.[12] We focused on piped gas stoves as a proxy measure of exposure to gas combustion products because they are more common than piped gas ovens. We did not include homes using liquid petroleum gas cylinders for stovetop cooking (fewer than 10% of all homes[12]) because detailed data were not available. The Energy Use survey did not collect data on range hoods, which can reduce exposure to gas combustion products. We therefore performed an additional analysis that assessed their effect, based on data from a recent experimental study which found that about 75% of gas and particle emissions are captured by high flow range hoods.[13]

Effect estimates for dampness and gas stoves

We used pooled effect estimates from recent meta-analyses, rather than individual studies, to quantify associations between the exposures of interest and asthma to ensure the robustness and generalisability of associations. We searched PubMed for peer-reviewed meta-analyses published in the past 5 years (ie, since 2011) of the association between household dampness or gas stoves and asthma in children (online Appendix). We sought meta-analyses that reported pooled effect estimates and confidence intervals from studies identified by a systematic search, assessed associations with prevalent or incident asthma, reported fixed and random effect estimates (odds ratios or relative risks) adjusted for confounders, and assessed publication bias. While we were interested in cross-sectional (effects on prevalent asthma) and longitudinal associations (effects on incident asthma), we favoured meta-analyses of incident asthma when available.

Asthma burden

The 2011 national census counted 4 144 024 children aged 14 years or under.[14] Data from the 2010 Global Burden of Disease (GBD) study (obtained using the GBD results tool: http://ghdx.healthdata.org/gbd-results-tool) were used to estimate the burden of asthma in this age group in Australia, expressed as a point estimate of DALYs — the sum of years of life lost (YLLs) and years lived with disability (YLDs) because of asthma — with 95% uncertainty intervals, without discounting or weighting for age. The data sources and methods for quantifying the burden of asthma in Australia have been described in detail elsewhere.[15] The GBD data were used because the study applied a well documented, systematic methodology that exhaustively identified input data.[15]

Estimating the contribution of dampness and gas stoves to childhood asthma

We estimated the population attributable fraction (PAF) and the asthma burden (DALYs) associated with each indoor risk factor. The PAF is the theoretical proportion of asthma burden that could be averted were the risk factor removed. Because estimating the PAF can introduce uncertainty at each step, particularly if point estimates are used, our approach was to incorporate all available data on the spread and precision of the variables used to calculate PAF and the burden for each risk factor. However, summary statistics rather than raw data for exposure, effect size, and disease burden variables were available for our analysis; we therefore used these descriptive statistics to generate normal distributions for each variable, from which we randomly drew 10 000 values. Our calculations began with a randomly selected value from the distribution of the proportion of children exposed, which was then combined with a randomly selected value from the distribution of the effect estimate to calculate an estimate of the PAF for each risk factor:[10]

$$PAF = \frac{p \times (RR - 1)}{p \times (RR - 1) + 1}$$

where p = proportion of children exposed to the risk factor; RR = relative risk (or odds ratio) of asthma for children exposed to the risk factor.[16] The PAF estimate was multiplied by a randomly selected value from the asthma burden distribution to calculate the estimated disease burden in DALYs:[10]

$$BoD_{\text{INDOOR}} = PAF \times BoD_{\text{TOTAL}}$$

where BoD_{TOTAL} = the total burden of asthma among children, and BoD_{INDOOR} = burden attributable to the risk factor. This calculation was repeated 10 000 times, generating PAF and disease burden distributions with 10 000 values each, from which we derived the mean values and 95% confidence intervals (CIs). We explored alternative distribution shapes and the sensitivity to the number of iterations before settling on these parameters. All analyses were conducted in R 3.2.2 (R Foundation for Statistical Computing).

Sensitivity analyses

We undertook additional analyses to assess the sensitivity of our PAF results to plausible changes in input parameters: the proportion of children exposed (including and excluding certain studies used to estimate dampness; assessing the effects of range hoods on gas stove exposure), effects (using different effect estimates reported by the meta-analyses), and changing the age range for childhood to include those aged 19 years or under when calculating asthma-associated DALYs.

Ethics approval

Our modelling study was based on exposure prevalence and effect estimates identified in literature searches and publicly available, aggregated estimates of national asthma burden. Further, national data were drawn from Australian Bureau of Statistics surveys collected under the *Census and Statistics Act 1905 (Cwlth)*. Formal ethics approval for our investigation was therefore not required.

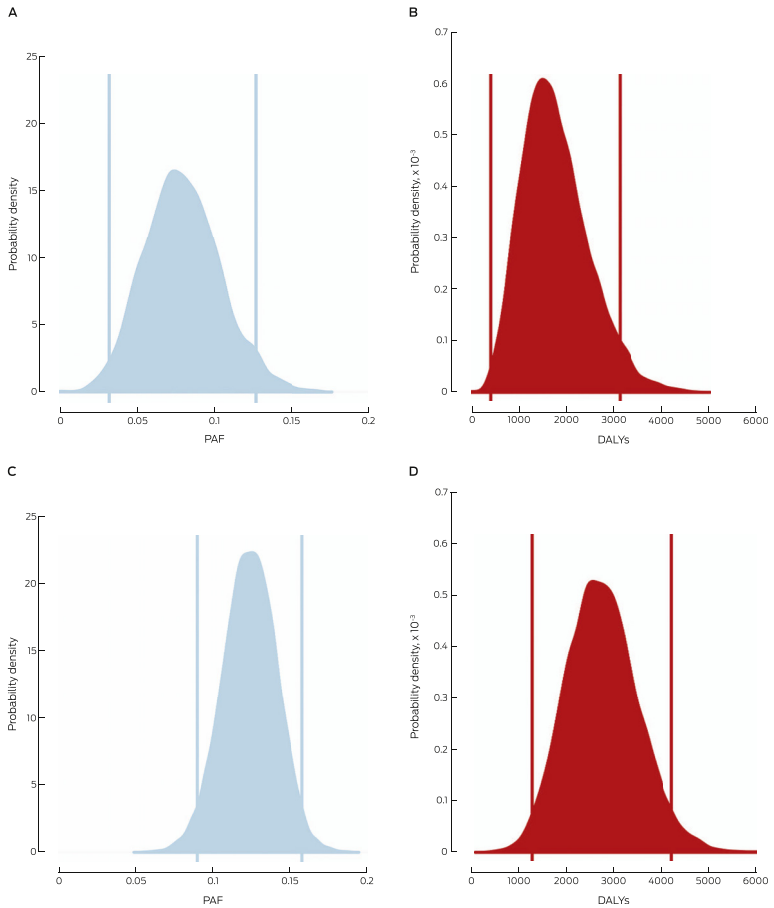
Results

We estimated that the population-weighted prevalence of dampness in Australian homes is 26.1% (standard deviation, 9.4%), based on data in the identified studies for a total of 7538 dwellings (online Appendix). There was substantial variation between states in the proportions of households using natural gas for stovetop cooking, ranging from 2% in Tasmania to 56% in Western Australia and 67% in Victoria.[12] Overall, 38.2% of Australian households used piped natural gas for stovetop cooking.[12]

Three meta-analyses that examined the association between exposure to dampness and asthma met our inclusion criteria.[7,17,18] We selected the analysis by Quansah and colleagues[7] for our study because it was the most recent (2012), included studies from a wide geographic range of locations, and focused on new asthma diagnoses in cohort and incident case–control studies. The single eligible meta-analysis that examined gas stove exposure and asthma[6] was based primarily on cross-sectional studies and cross-sectional analyses within cohort studies (38 of 41 included studies).

We estimated that the PAF for childhood asthma associated with damp housing was 7.9% (95% CI, 3.2–12.6%; Box, A), causing 1760

Population attributable fractions (PAFs) of asthma in children aged 14 years or under and disability-adjusted life years (DALYs) associated with exposure to dampness (A, B) and gas cooktop stoves (C, D) at home, with 95% confidence intervals



CI = confidence interval. ◆

Exposure of Australian children aged 15 years or younger to indoor second-hand smoke declined from 31.3% in 1995 to 3.7% in 2013.[19] This improvement is impressive, but our results suggest that the respiratory health of children is still affected by common but less easily recognised indoor risk factors. Dampness in housing promotes the proliferation of fungi, bacteria and dust mites, and their metabolites and spores have inflammatory, cytotoxic, and immunosuppressive effects.[20,21] Gas combustion causes the emission of nitrogen dioxide, formaldehyde, nitrous acid, and ultrafine particles (under 100 nm diameter), and is associated with airway inflammation.[22]

We estimated that 26.1% of Australian homes have dampness problems, consistent with findings by studies in the United States, Canada and Europe, where at least 20% of homes had indications of dampness.[20] Eliminating dampness in dwellings would avert almost 8% of the childhood asthma burden, but the extent to which this is practicable is unclear. Indoor dampness occurs when moisture-sensitive materials, such as plasterboard, are not adequately protected from water, or indoor humidity is excessive.[20,21] Parts of most homes are transiently damp, but problems arise when dampness is persistent and supports continuous microbial growth in and near living areas. Controlling dampness requires a multifaceted approach that includes minimising the exposure of interior walls to water and nutrients, as well as using mould-resistant building products and installing vapour barriers in new buildings.[20,21] Dampness in existing building stock is more challenging, but all homes can benefit from increasing natural ventilation (eg, open windows), avoiding drying laundry in living areas, and using dehumidifiers.[21] Heavily contaminated dwellings may require professional remediation and repair.[20]

DALYs (95% CI, 416–3104 DALYs; Box, B), or 42 DALYs per 100 000 children aged 14 years or younger. The results were robust to changing values of the input parameters (online Appendix, table 1).

Our PAF estimate for childhood asthma associated with gas stoves was 12.3% (95% CI, 8.9–15.8%; Box, C), corresponding to 2756 DALYs (95% CI, 1271–4242; Box, D), or 67 DALYs per 100 000 children aged 14 years or less. These estimates did not change markedly when then input parameters changed (online Appendix, table 2). Our most conservative scenario for the effects of range hoods assumed that all gas stoves were vented outdoors with 75% efficiency (ie, reduced exposure to combustion products by 75%). Our PAF and burden estimates for exposure to gas stoves in this scenario were 3.4% (95% CI, 2.2–4.6%) and 761 DALYs (95% CI, 322–1199).

Discussion

We estimated that damp housing and gas stoves respectively account for 7.9% and 12.3% of the childhood asthma burden in Australia. Although the risks at the level of the individual are relatively small, the proportion of the population exposed is relatively large, so that the contribution to the population asthma burden is considerable.

The prevalence of gas cooking in Australia was relatively low compared with that of many western European countries (mean, 63%).[23] While our results suggest that 12.3% of the childhood asthma burden could be averted were gas replaced by a different energy source for cooking, this assumes that the new energy source would not produce indoor emissions; this is unlikely, as cooking itself can be a major source of emissions.[13] More important is our finding that the burden of asthma associated with gas stoves could be reduced from 12.3% to 3.4% were all homes with gas stoves fitted with high efficiency range hoods that vented outdoors. Particles and gases can be captured with greater than 75% efficiency by such hoods, while lower flow rates and recirculating range hoods offer less protection.[13] Range hoods also remove cooking-related water vapour, reducing indoor dampness. However, 44% of people in Melbourne with range hoods reported that they did not use them regularly,[24] indicating that their potential health benefits should be communicated to the Australian public. We acknowledge that fitting all homes with range hoods would be impractical, but improving natural ventilation in all homes should be recommended to reduce exposure to gas combustion products.

Public education with the aim of reducing the burden of asthma should include information about how indoor exposures can affect health and how to recognise problem areas, as well as offering a range of suggestions for reducing exposure, from the

simple and inexpensive to more complex and expensive but more effective options. Relevant information is disseminated by the New South Wales Ministry of Health[25] and other state health departments as general factsheets; these should be reviewed to ensure that they are up to date and easily accessible. Given the degree of exposure to dampness and gas stoves in Australia, a national approach in the context of asthma prevention is required.

Like most modelling studies, the main limitation of our investigation is the validity of the input data. We used standard methods, were selective in what we included, and performed sensitivity analyses, but we may have under- or overestimated the burden of asthma attributable to each risk factor. Further, we assessed children to 14 years of age as a single group with respect to the effects of indoor exposures on asthma. Asthma in younger children, however, may be exacerbated to a greater extent than in older children by other factors, such as respiratory infections. A more comprehensive investigation could examine further indoor risk factors and assess the feasibility of intervention-based studies for reducing the asthma burden. Clinicians treating children should be aware of the potential significance of indoor exposures for children with or at increased risk of asthma.

Acknowledgements: This work was supported by the National Health and Medical Research Council (Luke Knibbs: Early Career Fellowship [APP1036620]; Guy Marks: Centres of Research Excellence grant [APP1030259]), the Centre for Air Quality and Health Research and Evaluation (Luke Knibbs, Christine Cowie: postdoctoral fellowships), and the New South Wales Ministry of Health (funding to Christine Cowie and Guy Marks).

Competing interests: No relevant disclosures.

Received 16 May 2017, accepted 21 Sept 2017. ■

1 Australian Centre for Asthma Monitoring. Asthma in Australia 2011: with a focus chapter on chronic obstructive pulmonary disease (AIHW Cat. No. ACM 22; Asthma Series no. 4). Canberra: Australian Institute of Health and Welfare, 2011. https://www.aihw.gov.au/reports/asthma-other-chronic-respiratory-conditions/asthma-in-australia-2011-with-chapter-on-copd/contents/table-of-contents (viewed Apr 2017).

2 Institute of Medicine. Climate change, the indoor environment, and health. Washington (DC): The National Academies Press, 2011. https://www.nap.edu/catalog/13115/climate-change-the-indoor-environment-and-health (viewed Apr 2017).

3 Garrett MH, Hooper MA, Hooper BM, Abramson MJ. Respiratory symptoms in children and indoor exposure to nitrogen dioxide and gas stoves. *Am J Resp Crit Care Med* 1998; 158: 891-895.

4 Franklin P, Dingle P, Stick S. Raised exhaled nitric oxide in health children is associated with domestic formaldehyde levels. *Am J Resp Crit Care Med* 2000; 161: 1747-1749.

5 Ponsonby AL, Couper D, Dwyer T, et al. The relation between infant indoor environment and subsequent asthma. *Epidemiology* 2000; 11: 128-135.

6 Lin W, Brunekreef B, Gehring U. Meta-analysis of the effects of indoor nitrogen dioxide and gas cooking on asthma and wheeze in children. *Int J Epidemiol* 2013; 42: 1724-1737.

7 Quansah R, Jaakkola MS, Hugg TT, et al. Residential dampness and molds and the risk of developing asthma: a systematic review and meta-analysis. *PLoS One* 2012; 7: e47526.

8 Pilotto LS, Nitschke M, Smith BJ, et al. Randomized controlled trial of unflued gas heater replacement on respiratory health of asthmatic schoolchildren. *Int J Epidemiol* 2003; 33: 208-214.

9 Marks GB, Ezz W, Aust N, et al. Respiratory health effects of exposure to low-NO_x unflued gas heaters in the classroom: a double-blind, cluster-randomized, crossover study. *Environ Health Perspect* 2010; 118: 1476-1482.

10 Hänninen O, Knol A (editors). European perspectives on environmental burden of disease: estimates for nine stressors in six European countries. Helsinki: National Institute for Health and Welfare, 2011. https://www.julkari.fi/bitstream/handle/10024/79910/b75f6999-e7c4-4550-a939-3bccb19e41c1.pdf (viewed Apr 2017).

11 Fisk WJ, Lei-Gomex Q, Mendell MJ. Meta-analyses of the associations of respiratory health effects with dampness and mould in homes. *Indoor Air* 2007; 17: 284-296.

12 Australian Bureau of Statistics. 4602.0.55.001. Energy use and conservation survey, 2011. Energy use and conservation additional tables (data cube). http://www.abs.gov.au/AUSSTATS/subscriber.nsf/log?openagent&4602055001do002_201103.xls&4602.0.55.001&Data%20Cubes&E32C173D338FE75FCA257953000EE41A&0&Mar%202011&28.11.2011&Previous (viewed Apr 2017).

13 Lunden MM, Delp WW, Singer BC. Capture efficiency of cooking-related fine and ultrafine particles by residential exhaust hoods. *Indoor Air* 2015; 25: 45-58.

14 Australian Bureau of Statistics 2013, 2011 Census community profiles. Basic community profile, table B04. http://www.censusdata.abs.gov.au/census_services/getproduct/census/2011/communityprofile/0 (viewed Feb 2018).

15 Australia Institute of Health and Welfare. Assessment of global burden of disease 2010 methods for the Australian context (Working paper no. 1). Canberra: AIHW, 2014. http://content.webarchive.nla.gov.au/gov/wayback/20170421063605/http://www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=60129547710 (viewed Apr 2017).

16 Zhang J, Yu KF. What's the relative risk? A method of correcting the odds ratio in cohort studies of common outcomes. *JAMA* 1998; 280: 1690-1691.

17 Tischer C, Chen C-M, Heinrich J. Association between domestic mould and mould components, and asthma and allergy in children: a systematic review. *Eur Respir J* 2011; 38: 812-824.

18 Tischer CG, Hohmann C, Thiering E, et al. Meta-analysis of mould and dampness exposure on asthma and allergy in eight European birth cohorts: an ENRIECO initiative. *Allergy* 2011; 66: 1570-1579.

19 Australian Institute for Health and Welfare. National drug household survey: detailed report 2013 (AIHW Cat. No. PHE 183; Drug Statistics Series No. 28). http://www.aihw.gov.au/publication-detail/?id=60129549469 (viewed Apr 2017).

20 Institute of Medicine. Damp indoor spaces and health. Washington (DC): The National A cademies Press, 2004. https://www.nap.edu/catalog/11011/damp-indoor-spaces-and-health (viewed Apr 2017).

21 World Health Organization. WHO guidelines for indoor air quality: dampness and mould. Copenhagen: WHO Regional Office for Europe, 2009. http://www.who.int/indoorair/publications/7989289041683/en/ (viewed Apr 2017).

22 Heinrich J. Influence of indoor factors in dwellings on the development of childhood asthma. *Int J Hyg Environ Health* 2011; 214: 1-25.

23 Jarvis D, Chinn S, Sterne J, et al. The association of respiratory symptoms and lung function with the use of gas for cooking. *Eur Respir J* 1998; 11: 651-658.

24 Dharmage S, Bailey M, Raven J, et al. Prevalence and residential determinants of fungi within homes in Melbourne, Australia. *Clin Exp Allergy* 1999; 29: 1481-1489.

25 New South Wales Ministry of Health. Environmental health fact sheets [website]. Updated Sept 2017. http://www.health.nsw.gov.au/environment/factsheets/Pages/default.aspx (viewed Sept 2017). ■