C.U.R.E.S.

Center for Urban Responses to Environmental Stressors

Leadership: Melissa Runge-Morris, MD, Christine Cole-Johnson, PHD

Faculty: 54 faculty in 4 interest groups; (11) environmental modulators of the immune system, (11) environmental modulators of metabolism and metabolic disease, (6) gene-environment and cancer, (16) life stages and environmental disease

Community Advisory Board: @18 separate groups

COEC - “Logic Model”
- Symposium
- Chats
- “Spin Offs”
- Newsletter
## Wayne County Health Status Rankings

<table>
<thead>
<tr>
<th>Measure</th>
<th>Description</th>
<th>Ranking</th>
</tr>
</thead>
<tbody>
<tr>
<td>SE Factors</td>
<td>HS graduation, some college, unemployment, children in poverty, income equality, social associations, violent crimes, injury deaths</td>
<td>81</td>
</tr>
<tr>
<td>Health Behaviors</td>
<td>smoking, obesity, food index, inactivity, access to exercise, excessive drinking, impaired driving STDs, teen births.</td>
<td>71</td>
</tr>
<tr>
<td>Physical Environment</td>
<td>air pollution, drinking water violations, severe housing problems, driving alone to work, long commute</td>
<td>82</td>
</tr>
<tr>
<td>Clinical Care</td>
<td>uninsured, primary care physicians, dentists, mental health providers, preventable hospital stays, diabetic monitoring, mammography screening</td>
<td>81</td>
</tr>
<tr>
<td>Health Factors</td>
<td></td>
<td>82</td>
</tr>
<tr>
<td>Quality of Life</td>
<td>poor health, poor health days, poor mental health days, low birthweight</td>
<td>80</td>
</tr>
<tr>
<td>Health Outcomes</td>
<td></td>
<td>82</td>
</tr>
</tbody>
</table>

First Speaker Dennis Onwby

Rates increased dramatically around the 60’s and 70’s, seemed to have plateaued off.

Seems to also affect those that are poor, as well as being overweight.

Rates in Detroit very high compared to most other places (must be impressive if known by a researcher from another part of the country).

Dennis Onwby, MD
Georgia Regents University
Rate per 100,000 population at ages 0-14 years

- Queensland, Australia
- Tasmania, Australia
- New Zealand
- Canada
- England and Wales
- United States
Second Speaker Kathleen Slonager
She will have her own comments, but I was most impressed with her observation, that the majority of asthma sufferers are not employing the best knowledge that we already have (neither medical management nor environmental controls), and we have asked her to share, specifically what you can do to achieve a better outcome.

Kathleen Slonager, RN, AE-C, CCH
Asthma and Allergy of America, Mi Chapter

Impact of Asthma - Michigan
- Nearly 1 million - ~25% are children
- Rate is 20% higher than national average
- Only 40.4% of children & 33.8% of adults met the national treatment guideline recommendation of at least 2 routine asthma care visits during 12 months
- Only 30% of children with persistent asthma are filling prescriptions for inhaled corticosteroids, the preferred, first-line medication

The Bottom Line
- Proper asthma management has the potential to save at least 25% of total asthma costs—over $5 billion nationwide annually
- Asthma interventions can save up to $36 in health care costs and work days lost for every $1 spent
- Targeted Asthma programs improve quality of life, save lives, and enrich community wellness
IMPACT OF ASTHMA - MICHIGAN

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- Rate is 25% higher than national average
- Only 40.4% of children & 33.8% of adults met the national treatment guideline recommendation of at least 2 routine asthma care visits during 12 months
- Only 30% of children with persistent asthma are filling prescriptions for inhaled corticosteroids; the preferred, first-line medication

Michigan Asthma Call Back Survey, Michigan Department of Health & Human Services, 2010
Third Speaker - John Cambier - mainly doing animal studies - clarifying the immunology

There seems to be two pathways in the development...

Larger Particles (standard) Pollens/Dander, that involves a memory

Chemicals may have a different pathway - can directly stimulate the inflammatory response of asthma with a memory

Genetics / Epigenetics also important as for these pathways to work (or not work), requires a lot of communication between the players, and this can differ between people and genetics may play a role (perhaps only 10% of the story)

John Cambier, PhD
University of Colorado-Denver
The Immunological Orchestra at Work in Allergy

Epithelial Barrier

Allergen

Mast-cell stimulation

Mast cell

FcεRI

Release of mediators of acute and chronic inflammation

IL-4, IL-5, and IL-10

Precuror T helper cell

TCR

MHC class II

Expansion of TH2-type cells

TH2 cell

TH1 cell

B cell

Production of IgE

Dendritic cell
Third Speaker - mainly doing animal studies. Clarifying the immunology.

There seems to be two pathways in the development...

Larger Particles (standard) Pollens/Dander, that involves a memory. Chemicals may have a different pathway - can directly stimulate the inflammatory response of asthma with a memory.

Genetics / Epigenetics also important as for these pathways to work (or not work), requires a lot of communication between the players, and this can differ between people and genetics may play a role (perhaps only 10% of the story).
Fourth Speaker - Jack Harkema at MSU
Looking at the effects of a Ozone a particular type of exposure
Does seem to effect the cellular response - mucus and inflammation over 9 days. This seems to be a change in gene expression
When you take out T and B cells, you can still see a response, suggesting that there it is more than one pathway that can lead to the Asthmatic response
So a pollutant can also lead to an asthmatic response and act as an allergen and the changes can go away but it can take some time.

Jack Harkema, PhD
Michigan State University
Looking at the effects of ozone, a particular type of exposure, does seem to effect the cellular response - mucus and inflammation over 9 days. This seems to be a change in gene expression. When you take out T and B cells, you can still see a response, suggesting that there are more than one pathway that can lead to the asthmatic response. So a pollutant can also lead to an asthmatic response and act as an allergen, and the changes can go away but it can take some time.

Jack Harkema, PhD
Michigan State University
exposure alone in 2010

- Air pollutant exposure exacerbates pre-existing allergic rhinitis and asthma
- Ozone and airway allergy are predicted to increase with climate change
- Do air pollutants contribute to the onset of airway allergy?

Influx of eosinophils
Mucous cell metaplasia
Epithelial hyperplasia
Ym1/2 proteins
Airway Hyper-responsiveness

Initiation and propagation of type 2 immune responses

Nasal epithelial protein and mRNA expressions with increasing days of exposure

A

Mucosubstances

YM1/YM2 proteins

1 d ozone (24 h)

9 d ozone (24 h)

B

Days of ozone exposure (h post-exposure)

(1) 1 (2) 2 (4) 4 (9) 9

(1) 1 (2) 2 (4) 4 (9) 9

Log fold change

Air

Ozone

(2 & 24) (24) (24) (24) (24)

Days of exposure (h post-exposure)

(2 & 24) (24) (24) (24) (24)

Mucosubstances

YM1/YM2 proteins

C

Days of ozone exposure (h post-exposure)

(1) 1 (2) 2 (4) 4 (9) 9

Fold change mRNA

II1b

II13

Cica1

II10

Hmox

Muc5ac

Ccl11

Ccr2

Ccl2

Tnf

Neutrophil*

II1b

II10

II5

YM1/YM2*

Arg1

Ccl8

Eosinophil*

Chil4

Cica1

Mucosubstance*

Saa3

Chil3
Fifth Speaker Larry Lemke at WSU who placed over a hundred monitors across the city and Canada to measure selected pollutants and look at the relationship to asthma exacerbations. Looking at Environmental Considerations, Exposures are not equal across the city. Some were dropped from analysis as they were not found in all communities. There was a relationship between exposure and exacerbations with much scatter. Rates of exacerbations much higher in Detroit compared to Windsor, which may be due to access to care. There was a lot of scatter meaning that it's not just the air out-doors that causes a problem. An this make sense because in some communities that have heavy pollution burdens, not everyone has asthma. Something seems to be protecting them or rather not allowing them to experience symptoms of asthma.
300 x 300 m resolution!
<table>
<thead>
<tr>
<th>VOC Species Exclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Criteria: Exclude analytes with fewer than 80% of sample sites registered values above variable MDL.</td>
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<table>
<thead>
<tr>
<th>VOC Analytes</th>
<th>% Count &gt;MDL</th>
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</thead>
<tbody>
<tr>
<td>1 Toluene</td>
<td>100</td>
</tr>
<tr>
<td>2 (m+p)-Xylene</td>
<td>100</td>
</tr>
<tr>
<td>3 Dichloromethane (DCM)</td>
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</tr>
<tr>
<td>4 Benzene</td>
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</tr>
<tr>
<td>5 Ethylbenzene</td>
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<tr>
<td>9 o-Xylene</td>
<td>99</td>
</tr>
<tr>
<td>6 Hexane</td>
<td>99</td>
</tr>
<tr>
<td>7 1,2,4-Trimethylbenzene</td>
<td>99</td>
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<tr>
<td>8 n-Decane</td>
<td>98</td>
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<tr>
<td>10 Trichloroethylene</td>
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<td>12 1,3,5-Trimethylbenzene</td>
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<td>11 Chloroform</td>
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<td>13 Tetrachloroethylene</td>
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<td>15 1,4-Dichlorobenzene</td>
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<td>17 a-Pinene</td>
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<td>16 d-Limonene</td>
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<td>18 1,2-Dichloroethane</td>
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<td>19 p-Cymene</td>
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<td>20 Cumene</td>
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<td>21 Styrene</td>
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<td>22 1,1,2,2-Tetrachloroethane</td>
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<td>23 Pentachloroethane</td>
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<td>24 1,3-Dichlorobenzene</td>
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<td>25 Hexachloroethane</td>
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<td>26 1,2,4-Trichlorobenzene</td>
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| Excluded Species |

<table>
<thead>
<tr>
<th>PAH Species Exclusion</th>
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<td>Criteria: Exclude analytes with fewer than 80% of sample sites registered values above variable MDL.</td>
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<table>
<thead>
<tr>
<th>PAH Analytes</th>
<th>% &gt;MDL</th>
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</thead>
<tbody>
<tr>
<td>1 Anthracene</td>
<td>100</td>
</tr>
<tr>
<td>2 Fluoranthene</td>
<td>100</td>
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<tr>
<td>3 Fluorene</td>
<td>100</td>
</tr>
<tr>
<td>4 Phenanthrene</td>
<td>100</td>
</tr>
<tr>
<td>5 Pyrene</td>
<td>100</td>
</tr>
<tr>
<td>6 Acenaphthene</td>
<td>97</td>
</tr>
<tr>
<td>7 ortho-Phenylnaphthalene*</td>
<td>84</td>
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<tr>
<td>8 Diazinon*</td>
<td>66</td>
</tr>
<tr>
<td>9 Chrysene</td>
<td>58</td>
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<tr>
<td>10 Acenaphthylene</td>
<td>50</td>
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<tr>
<td>11 Benz(a)anthracene</td>
<td>21</td>
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<tr>
<td>12 Chlorpyrifos (Dursban)*</td>
<td>21</td>
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<tr>
<td>13 Benz(k)fluoranthene</td>
<td>3</td>
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<tr>
<td>14 Benzo(b)fluoranthene</td>
<td>3</td>
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<td>15 Benzo(a)pyrene</td>
<td>0</td>
</tr>
<tr>
<td>16 Benzo(ghi)pyrene</td>
<td>0</td>
</tr>
<tr>
<td>17 cis-Permethrin*</td>
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<tr>
<td>18 Dibenzo(a,h)anthracene</td>
<td>0</td>
</tr>
<tr>
<td>19 Indeno(123-cd)pyrene</td>
<td>0</td>
</tr>
<tr>
<td>20 Piperonyl butoxide*</td>
<td>0</td>
</tr>
<tr>
<td>21 Propoxur (Baygon)*</td>
<td>0</td>
</tr>
<tr>
<td>22 trans-Permethrin*</td>
<td>0</td>
</tr>
<tr>
<td>23 Naphthalene**</td>
<td>0</td>
</tr>
</tbody>
</table>

| Excluded Species |

*pesticide
b) VOC

\[ y = 4.99x - 58.73 \]
\[ R^2 = 0.26 \]
TABLE I. Sample characteristics (n = 1187)

<table>
<thead>
<tr>
<th></th>
<th>Indoor pet at PD (n = 420)</th>
<th>No indoor pet at PD (n = 727)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother’s age (y), mean (SD)</td>
<td>29.7 (5.1)</td>
<td>29.4 (5.3)</td>
<td>.30</td>
</tr>
<tr>
<td>African American race, no. (%)</td>
<td>186 (44.3)</td>
<td>552 (72.0)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mother’s allergic history, no. (%)</td>
<td>127 (30.5)</td>
<td>213 (27.8)</td>
<td>.34</td>
</tr>
<tr>
<td>Mother smoked, no. (%)</td>
<td>67 (16.0)</td>
<td>72 (9.4)</td>
<td>.001</td>
</tr>
<tr>
<td>ETS during pregnancy, no. (%)</td>
<td>131 (31.2)</td>
<td>196 (25.6)</td>
<td>.038</td>
</tr>
<tr>
<td>Delivery by means of cesarean section, † no. (%)</td>
<td>144 (34.6)</td>
<td>292 (38.5)</td>
<td>.19</td>
</tr>
<tr>
<td>Male sex (baby), no. (%)</td>
<td>217 (51.7)</td>
<td>375 (48.9)</td>
<td>.36</td>
</tr>
<tr>
<td>Firstborn, no. (%)</td>
<td>175 (41.7)</td>
<td>258 (33.6)</td>
<td>.006</td>
</tr>
</tbody>
</table>

ETS, Environmental tobacco smoke; PD, predelivery.
*Four with missing information.
†Thirteen with missing information.

increase changed (sometimes called “change point”), but we also tested to see whether using the 1-year time point was able to explain more of the variability (ie, better fit); it did not.

A 2-segment linear (piecewise) model was found to have the best fit (see the Methods section in this article’s Online Repository at www.jacionline.org). Indoor pet exposure was included as a main effect and was also tested for an interaction with the age of the child. Indoor pet exposure was evaluated as a baseline exposure variable, as well as a time-varying exposure variable (one that can change over time), because we had reports about pet exposure at each time that IgE levels were measured.

Effect modification for a priori selected variables was tested by means of inclusion of an interaction term (P < .10) and inspection of stratified models. Maternal race, smoke exposure during pregnancy, mode of delivery, firstborn status, first pregnancy age, and maternal history of allergies was used.

FIG 2. Predicted mean plot of the trajectory from the 2-piece random effects model.

level of 0.30 IU/mL at birth and 7.74 IU/mL at the 6-month time point, corresponding to a 25.8-fold increase (7.74/0.30). If there were no missing data and the timing of study contacts did not differ among subjects, this estimate of a 25.8-fold increase (equivalent to a 2580% increase) would directly correspond to the ratio of the (cross-sectional) geometric means at birth and the 6-month visit (see Table E1 in this article’s Online Repository at www.jacionline.org); a geometric means of 0.31 IU/mL at birth and 7.4 IU/mL at the 6-month visit is a (cross-sectional) 23.9-fold (7.4/0.31) increase. In contrast to the esti-
<table>
<thead>
<tr>
<th>Sub Pathway</th>
<th>Biochemical Name</th>
<th>PBS</th>
<th>L. johnsonii</th>
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<tbody>
<tr>
<td><strong>Leucine, Isoleucine and Valine Metabolism</strong></td>
<td></td>
<td></td>
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<tr>
<td>alpha-hydroxyisovalerate</td>
<td>1.0514</td>
<td>1.0514</td>
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<tr>
<td>isoleucine</td>
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<td>0.7585</td>
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<tr>
<td>3-methyl-2-oxovalerate</td>
<td>0.7198</td>
<td>1.8776</td>
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<tr>
<td>2-hydroxy-3-methylvalerate</td>
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<tr>
<td>valine</td>
<td>0.8468</td>
<td>1.36</td>
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<td>3-hydroxybutyrate</td>
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<td>alpha-hydroxyisocaproate</td>
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<tr>
<td>myristoleate (14:1n5)</td>
<td>1.0516</td>
<td>1.0516</td>
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<tr>
<td>palmitate (16:0)</td>
<td>0.6770</td>
<td>1.0210</td>
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<td>palmitoleate (16:1n7)</td>
<td>0.9217</td>
<td>1.0210</td>
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<tr>
<td>10-heptadecenoate (17:1n7)</td>
<td>0.9145</td>
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<tr>
<td>oleate (18:1n9)</td>
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<td>cis-vaccenate (18:1n7)</td>
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<tr>
<td>10-nonadecenoate (19:1n9)</td>
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<td>1.7200</td>
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<td>stearidionate (18:4n3)</td>
<td>0.5902</td>
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<td>elicosapentanoic acid (EPA; 20:5n3)</td>
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<td>docosapentenoic acid (n3 DPA; 22:5n3)</td>
<td>1.0010</td>
<td>2.1898</td>
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<tr>
<td>docosahexaenoic acid (DHA; 22:6n3)</td>
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<td>1.8130</td>
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<tr>
<td>linolenate (alpha or gamma; 18:3n3 or 6)</td>
<td>0.9729</td>
<td>1.8130</td>
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<td>docosapentenoic acid (n6 DPA; 22:5n6)</td>
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<td>1.36</td>
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<td>dihomolinoleate (20:2n6)</td>
<td>0.8407</td>
<td>1.36</td>
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<td>mead acid (20:3n9)</td>
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<td><strong>Fatty acid Metabolism</strong></td>
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<tr>
<td>myristoylcarmitine</td>
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<td>palmitoylcarmitine</td>
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<td>stearoylcarmitine</td>
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<td>2-hydroxyoctanole</td>
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<td>2-hydroxydecanole</td>
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<td>3-hydroxydecanate</td>
<td>1.1701</td>
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<td>5-hydroxylaurate</td>
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<td>3-hydroxymyristate</td>
<td>0.171</td>
<td>1.8776</td>
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<td>5-hydroxyhexanoate</td>
<td>1.574</td>
<td>0.9646</td>
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<td>13-ODHE + 9-ODHE</td>
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<td><strong>LysoLipids</strong></td>
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<tr>
<td>2-palmitoylglycerophosphocholine (16:1n1)*</td>
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<td>1-palmitoleoylglycerophosphocholine (16:1n1)*</td>
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Karma Meattle
Paharpur Business Centre
Areca Palm
Chrysalidocarpus Lutescens

Mother-in-law’s Tongue
Sansevieria trifasciata

Karma Meattle

Money Plant
Epipremnum aureum
Questions...

• what are we doing that cause the rates to rise everywhere? could it be from ingestion vs inhalation?

• what are the barriers to best practices?

• do chemicals really lead to allergy, or does the damage allow us to develop allergy to biologics that are always present? Do chemicals upset the microbiome?

• is is really dogs or just being outside on the grass?

• What do those do that don't have problems?