Trouble in the airways...

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Université Laval, Québec City
Objectives

• Review current hypotheses on the mechanisms of development of asthma

• Describe what are the main asthma phenotypes and discuss the clinical relevance of such categorization

• Provide an update on research on knowledge translation in respiratory health
My first steps in research

Involvement of the thalamus in mechanisms of memory in the cat

Mircea Steriade

Étude ultrastructurale de la sécrétion de la cellule parafolliculaire thyroïdienne du rat

Louis-Philippe Boulet et Georges Pelletier,
Laboratoire d’Endocrinologie Moléculaire,
Centre Hospitalier de l’Université Laval, Québec

Georges Pelletier
Mentors – collaborators & friends - organisations

and many other collaborators and students

+ Pneumologues of the IUCPQ
The term asthma is referenced in the *Iliad*, book XV, line 10:

"He saw Hector lying on the plain, his companions sitting round him. Hector was gagging painfully, dazed and vomiting blood

Homer later made another reference to asthma in the *Iliad*, book XV, line 290:

"He was just starting to recover, to recognize his comrades round him. He'd stopped gasping and sweating, for aegis-bearing Zeus had revived his mind"

In this scene Homer describes Hector as just starting to catch his breath, although Homer used the word asthma (άσθμα) instead of gasping
World prevalence of asthma
Definition of Asthma

- Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation

GINA Report 2014

- Airway inflammation
- Paroxysmal or persistent respiratory symptoms
- Variable airflow limitation
- Hyperresponsiveness
Asthma phenotypes/endotypes
ASTHMA

Genetic predisposition + Environment

Genetic predisposition

Upper airways
Rhinitis

Inflammation & airway remodelling

Atopy

Lower airways
AHR

Variable Airway Obstruction

Fixed Airway Obstruction (ACOS)

Smoking ?

Symptomatic asthma

Remission ?

Bronchoconstriction
Airway Hyperresponsiveness
Airway Inflammation
Airway Remodelling

Evolving concepts
“Asthma genes”

Bossé & Hudson 2006
ASTHMA and INFLAMMATION

May-Giemsa staining

Eosinophilic

Neutrophil elastase stain

Haematoxylin and eosin stain

PAUCIGRANULOCYTOPENIC & MIXT

EOSINOPHILIC

NEUTROPHILIC

Antigens

Cellule dendritique

IL-10

Lymphocyte Th0

Selection

Expansion

IL-4

Lymphocyte Th2

Allergens

Recrutement

Activation

Mastocyte

Histamine

Prostaglandines

Leucotrienes

Enzymes

Cytokines

Chimiokines

Proteines basiques
Mechanisms of development of asthma

From Holgate 2011
Asthma and increases in nonallergic bronchial responsiveness from seasonal pollen exposure


JACI 1983
Association between clinical diagnosis and type of sensitisation to common allergens

AXIS I

- ASTHMA
- Dog
- ASTHMA + RHINITIS
- Cat
- House dust
- D. Farinae

AXIS II

- RHINITIS
- Molds
- Mixed Grasses
- Trees
- Ragweed

Variations of mean % sputum eosinophils between indoor allergens unexposed, exposed and normal control subjects during and out of the pollen season.

*Bp = 0.02 versus Exposed in season

Boulet LP et al. Magnitude of the early vs late allergen-induced response in seasonal vs perennial allergen bronchoprovocation. Submitted to ERJ 2014
What is happening in the airways of non-asthmatic atopic subjects when they are exposed to allergens?
Seasonal variation in PC$_{20}$ values of nonasthmatic rhinitic subjects

Seasonal increase in expression of IL5 in bronchial biopsies of non-asthmatic subjects with allergic rhinitis

Lower airway remodeling in allergic rhinitis

Chakir J. et al. Lower airway remodelling in non asthmatic subjects with allergic rhinitis. Lab Invest 1996
In Vitro Procollagen Synthesis and Proliferative Phenotype of Bronchial Fibroblasts from Normal and Asthmatic Subjects

Jean Dubé, Jamila Chakir, Michel Laviolette, Sylvie Saint Martin, Michel Boutet, Charles Desrochers, François Auger, and Louis-Philippe Boulet

Unité de Recherche en Pneumologie (JD, JC, ML, SSTM, MB, CD, L-PB), Hôpital Laval, Université Laval, Saint Foy, and Laboratoire d'Organogénèse Expérimentale (FA), Hôpital du Saint Sacrement, Université Laval, Québec, Canada

$r = 0.93, p < 0.0001$
Variations in IS % eosinophils

Non-asthmatic rhinitics

Allergic Asthma

† p = 0.0005
* p = 0.01

Same pattern for ECP

Changes in Airway Responsiveness in Asymptomatic AHR (3y)

Laprise C, Boulet LP. AJRCCM 1997
Asymptomatic airway hyperresponsiveness: relationships with airway inflammation and remodelling

Changes in airway responsiveness vs changes in sub-epithelial fibrosis

Atopy
No atopy

rs = 0.78
p = 0.03

Laprise et al. ERJ 1999
Asthma with a fixed component of airway obstruction

Internal / total airway diameter

PC20 Methacholine

Boulet LP et al AJRCCM 1995

Ward et al. AJRCCM 2001

$r = 0.37, p < 0.05$
Influence of inhaled corticosteroids on airway hyperresponsiveness, inflammation and remodelling in recently diagnosed and long-standing mild asthma.

Boulet LP et al. AJRCCM 2001

But no change in Subepithelial fibrosis
Occupational Asthma

Gautrin et al. JACI 1994;93:12-22.
Bronchial Biopsies: 
Super epithelial Fibrosis (µm)
Occupational Asthma

**FIGURE 1.** Magnitude of the asthmatic response during specific inhalation challenges in a) low-molecular weight agents and b) high-molecular weight agents.

**FIGURE 2.** Mean fall in double concentrations of methacholine (MCh) according to the type of response in a) low-molecular weight agents and b) high-molecular weight agents.

<table>
<thead>
<tr>
<th></th>
<th>Before SIC</th>
<th>After SIC</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total cell count (x10⁶)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMW</td>
<td>1.9 [1.5-4.2]</td>
<td>3.8 [2.3-8.3]</td>
<td>0.004</td>
</tr>
<tr>
<td>HMW</td>
<td>1.6 [1.1-5.9]</td>
<td>2.9 [1.5-9.1]</td>
<td>0.28</td>
</tr>
<tr>
<td><strong>Eosinophils (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMW</td>
<td>2.5 [0.5-5.1]</td>
<td>8.8 [2.1-14.9]</td>
<td>0.005</td>
</tr>
<tr>
<td>HMW</td>
<td>1.0 [0.5-3.0]</td>
<td>6.0 [4.0-19.0]</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Eosinophils (x10⁶)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMW</td>
<td>0.04 [0.01-0.15]</td>
<td>0.30 [0.06-0.66]</td>
<td>0.0001</td>
</tr>
<tr>
<td>HMW</td>
<td>0.03 [0.01-0.07]</td>
<td>0.27 [0.06-0.82]</td>
<td>0.01</td>
</tr>
<tr>
<td><strong>Neutrophils (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMW</td>
<td>34.9 [21.3-58.9]</td>
<td>41.7 [21.8-66.0]</td>
<td>0.38</td>
</tr>
<tr>
<td>HMW</td>
<td>52.2 [27.0-67.8]</td>
<td>47.0 [33.3-66.8]</td>
<td>0.45</td>
</tr>
<tr>
<td><strong>Neutrophils (x10⁶)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMW</td>
<td>0.7 [0.3-1.9]</td>
<td>1.4 [0.6-5.1]</td>
<td>0.006</td>
</tr>
<tr>
<td>HMW</td>
<td>0.7 [0.2-3.6]</td>
<td>1.8 [0.5-5.5]</td>
<td>0.94</td>
</tr>
</tbody>
</table>

Dufour et al 2010

Boulet et al. 2013
Smoking and asthma

Arginase I, ODC and iNOS immunoreactivity score in the epithelium of asthmatic nonsmokers and smokers

Bergeron et al. 2008
Definition (tentative) of ACOS

- Asthma-COPD overlap syndrome (ACOS) is characterized by persistent airflow limitation with several features usually associated with asthma and several features usually associated with COPD. ACOS is therefore identified by the features that it shares with both asthma and COPD.


- Airway inflammation
- Persistent airflow limitation (not fully reversible)
- Frequent diagnosis of asthma in the past
- Persistent but often variable symptoms
- Contributing exacerbations & co-morbidities
Prevalence of AHR and asthma in athletes


Swimmers: 7.3 mg/ml
Skiers: 15.8 mg/ml
### Airway inflammation in athletes (Induced sputum)  
Bougault *et al.* 2009

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Healthy Controls</th>
<th>Controls with asthma</th>
<th>Swimmers</th>
<th>Cold-air athletes</th>
</tr>
</thead>
<tbody>
<tr>
<td>N = 32 per group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cell count (cells/g)</td>
<td>4.9 ± 5.0</td>
<td>3.5 ± 5.1</td>
<td>4.2 ± 3.3</td>
<td>5.4 ± 4.6</td>
</tr>
<tr>
<td>Cell viability</td>
<td>67 ± 14</td>
<td>71 ± 17</td>
<td>68 ± 19</td>
<td>69 ± 27</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>31.9 ± 25.2</td>
<td>27.5 ± 20.8</td>
<td>37.2 ± 20.1</td>
<td>38.2 ± 21.5†</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0.4 ± 0.8</td>
<td>6.9 ± 9.6§§</td>
<td>1.3 ± 1.8‡†</td>
<td>0.6 ± 1.4‡‡</td>
</tr>
<tr>
<td>Macrophages</td>
<td>63.5 ± 24.7</td>
<td>61.4 ± 20.7</td>
<td>55.6 ± 20.2</td>
<td>56.6 ± 22.0</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>1.9 ± 1.7</td>
<td>1.8 ± 1.6</td>
<td>1.5 ± 1.5</td>
<td>1.3 ± 1.1</td>
</tr>
<tr>
<td>Epithelial bronchial cells</td>
<td>2.3 ± 3.0</td>
<td>2.4 ± 3.3</td>
<td>4.6 ± 4.9‡†</td>
<td>3.3 ± 2.5</td>
</tr>
</tbody>
</table>

Data are expressed as percentages except for total cell count, and as mean ± SD.

*p* < 0.05, **p** < 0.01 between athletes (cold-air or swimmers) and healthy controls.
Airway inflammation

Bronchial biopsies in 23 elite swimmers (21±2 years) old

Fig 1

1Bougault et al. 2010
Airway remodelling

Swimmers have a similar inflammatory and remodelling process compared with mild asthmatics.

\(^1\)Bougault et al. 2010
Correlation between years of training and oxydative stress marker in swimmers

No difference was observed between swimmers with or without AHR or asthma

Reference?
AHR in athletes: a transient phenomenon?

Bougault et al. 2010
Asthma and nasal polyps inflammation

Blood Eosinophils

Induced Sputum Eosinophils

* p < 0,05

Bilodeau et al. Comparative clinical and airway inflammatory features of asthma with or without nasal polyposis. Rhinology 2010
Asthma and obesity

% patients with controlled asthma

Boulet et Franssen. Resp Med 2007
Influence of deep inspiration on airway response to methacholine

**Simard B et al. Chest 2005**

<table>
<thead>
<tr>
<th>Test</th>
<th>Time before first FEV₁</th>
<th>Doses of M inhaled</th>
<th>Period without DI before M</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>30 sec</td>
<td>Multiple</td>
<td>None</td>
</tr>
<tr>
<td>B</td>
<td>3 min</td>
<td>Multiple</td>
<td>None</td>
</tr>
<tr>
<td>C</td>
<td>3 min</td>
<td>Single</td>
<td>None</td>
</tr>
<tr>
<td>D</td>
<td>3 min</td>
<td>Single</td>
<td>20 min</td>
</tr>
</tbody>
</table>

**Change in FEV₁ (Test D - test C)**

- **Control**
  - Green dots
  - p = 0.0096
  - 20.3 et 40.0 %
  - 18.5 et 23.6 %

- **Asthma**
  - Pink dots
  - p = 0.0065
  - IMC < 30
  - IMC ≥30
Improvement in FEV1, ERV and airway responsiveness during the year following bariatric surgery

Boulet et al. Respiration 2012
INDUCED SPUTUM VS ASTHMA SEVERITY

**Mean ± SEM**

* *p < 0.05 vs the two other severity categories*

Ducharme et al. 2011
Urgent visits for asthma

**Group C**

- First 6-month: 25
- Last 6-month: 25

**Group LE**

- First 6-month: 14
- Last 6-month: 14

**Group SE**

- First 6-month: 4
- Last 6-month: 4

- *p < 0.0002
- †p = 0.01

- SE: structured education
- LE: limited education
- C: control group

- Basic notions
- Inhaler technique
- Action Plan
- Reference to an Asthma educator

Côté et al.  
AJRCCM 001
Regional differences in ED visits for asthma

Quebec Asthma Cartography
<table>
<thead>
<tr>
<th>Question Asked</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>(What percent of your Asthma patients do you currently....)</td>
<td></td>
</tr>
<tr>
<td>1 Confirm diagnosis by pulmonary function tests (either spirometry and bronchodilator reversibility or bronchoprovocation)</td>
<td>48</td>
</tr>
<tr>
<td>2 Refer to asthma educator for patient education</td>
<td>23</td>
</tr>
<tr>
<td>3 Provide a written action plan for exacerbation management</td>
<td>18</td>
</tr>
<tr>
<td>4 Assess inhaler technique (or refer to Asthma Educator) at each visit</td>
<td>29</td>
</tr>
<tr>
<td>5 Prescribe inhaled corticosteroids (ICS) alone</td>
<td>26</td>
</tr>
<tr>
<td>6 Use the Canadian Thoracic Society (CTS) control criteria to assess your patient’s asthma control</td>
<td>38</td>
</tr>
<tr>
<td>7 Refer to specialist for management of difficult asthma</td>
<td>39</td>
</tr>
<tr>
<td>8 Schedule regular follow-up appointments</td>
<td>42</td>
</tr>
</tbody>
</table>
Playing cards on asthma management: A new interactive method for knowledge transfer to primary care physicians

Louis-Philippe Boulet MD FRCPC\textsuperscript{1}, Francine Borduas MD\textsuperscript{2}, Jacques Bouchard MD\textsuperscript{2}, Johanne Blais MD\textsuperscript{2}, Frederick E Hargreave MD FRCPC\textsuperscript{3}, Michel Rouleau MD\textsuperscript{2}

Recognized by the majority of patients as a useful mean of updating their knowledge on asthma management...
### Addressing care gaps in asthma management

<table>
<thead>
<tr>
<th>CARE GAPS</th>
<th>Spirometry</th>
<th>Facilitated visit</th>
<th>Adult emergencies</th>
<th>Pediatric emergencies</th>
<th>Pharmacists</th>
<th>Getting rid of pets</th>
<th>Info-Santé nurse</th>
<th>Integrated care</th>
<th>Chronologues</th>
<th>Support for decision-making</th>
<th>Family environment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disease under or over diagnosed</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Poor compliance with environmental measures</td>
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<tr>
<td>Inadequate understanding of asthma and its severity</td>
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<td></td>
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<tr>
<td>Therapeutic guidelines not applied</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
<td></td>
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<tr>
<td>Inadequate patient education</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
<td></td>
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<tr>
<td>Inappropriate therapy</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Lack of targeting for high risk groups</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Inadequacy of objective measurements</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
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<td></td>
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<tr>
<td>Inconsistent messages by caregivers</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td>X</td>
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<tr>
<td>Poor therapeutic compliance</td>
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<td></td>
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<tr>
<td>Inadequate patient self-management abilities</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>X</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Boulet et al. Can Resp J 2008
Patterns of adherence of ICS in asthma (ratio usage/prescription)

Adherent

Non-adherence

Overuse

Intermittent non-adherence

Lacasse Y. Can Respir J. 2005;12:211
ASTHMA CONTROL SCORING SYSTEM (ACSS)

### Clinical Evaluation

<table>
<thead>
<tr>
<th></th>
<th>25%</th>
<th>20%</th>
<th>15%</th>
<th>10%</th>
<th>5%</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Daytime sx</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Days/week</td>
<td>0</td>
<td>1-3</td>
<td>4-6</td>
<td>7</td>
<td></td>
<td>Severe</td>
</tr>
<tr>
<td><strong>Night time sx</strong></td>
<td>none</td>
<td>rare</td>
<td>1-3</td>
<td>4-7</td>
<td></td>
<td>Severe</td>
</tr>
<tr>
<td>Nights/week</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

**Clinical Score (A)**: 60

### Physiological Evaluation

<table>
<thead>
<tr>
<th></th>
<th>100%</th>
<th>80%</th>
<th>60%</th>
<th>40%</th>
<th>20%</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FEV&lt;sub&gt;1&lt;/sub&gt;</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% predicted or optimal</td>
<td>≥ 90</td>
<td>80-89</td>
<td>70-79</td>
<td>61-69</td>
<td>≤ 60</td>
<td>80</td>
</tr>
<tr>
<td><strong>PEF&lt;sup&gt;1&lt;/sup&gt;</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% predicted or optimal</td>
<td>≥ 90</td>
<td>80-89</td>
<td>70-79</td>
<td>61-69</td>
<td>≤ 60</td>
<td>80</td>
</tr>
<tr>
<td><strong>ΔPEF</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 10</td>
<td>11-15</td>
<td>16-20</td>
<td>21-24</td>
<td>≥ 25</td>
<td></td>
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</tr>
</tbody>
</table>

**Physiological Score (B)<sup>2</sup>**: 80

### Inflammatory Evaluation

<table>
<thead>
<tr>
<th>% eosinophils</th>
<th>100%</th>
<th>80%</th>
<th>60%</th>
<th>40%</th>
<th>20%</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td></td>
<td>≤ 2</td>
<td>&gt; 2-5</td>
<td>&gt; 5-8</td>
<td>≥ 8</td>
<td>60</td>
</tr>
</tbody>
</table>

**Inflammatory Score (C)**: 60

### Global Result

\[
\text{GLOBAL RESULT} = \frac{\text{Score A} (60) + \text{B} (80) + \text{C} (60)}{n \text{ Scores}} \]

\[
\text{GLOBAL RESULT} = 67
\]
Guidelines in respiratory diseases*

- Despite major advances in the understanding of respiratory diseases, treatment is often sub-optimal = high morbidity & unacceptable mortality
- Priority is to undertake research that leads to improvements in the use of existing treatments through public health and primary care initiatives
- **Guidelines represent an important component of this approach**, with recommendations imbedded within respiratory guidelines that can be implemented
- This approach offers the best opportunity to close the gap between what is currently achieved in disease management and that which is potentially achievable

*Adapted from Hancox et al. Respiration 2011*
Canadian Respiratory Guidelines

Anne Van Dam
Samir Gupta
Chris Licskai
Janet Sutherland
Kisten Curren
Annual CRGC Cycle

Guideline Evaluation
*(Oct - Dec)*
- Revise new evidence
- Select topics to review
- Methodology training workshop

Guideline Production
*(Jan - April)*
- Generate updates or new guidelines
- Report at Annual Guidelines Meeting
- Document approval by CTS

Dissemination & Implementation
*(Aug - September)*
- Engage in D&I activities
- Publish an update in the CRJ
- Post documents and KT tools to the CTS website
- Concurrent research to measure

Post-Production Planning
*(May - July)*
- Finalize all new documents
- Plan dissemination and implementation (D&I)
- Prepare budget
REVIEW

A guide to the translation of the Global Initiative for Asthma (GINA) strategy into improved care

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ABSTRACT: In 1995, the Global Initiative for Asthma (GINA) published an evidence-based workshop report as a guide to clinicians managing asthma patients, and has updated it annually to ensure that recommendations remain current. Although the report has been widely disseminated and influenced clinical practice and research, its major objective, of forming the basis for local and national initiatives to improve services for asthma patients, remains to be achieved. Over recent years, the science of guideline implementation has progressed, and encouraging examples of successful asthma programmes have been published. This report is intended to draw on this experience and assist with the translation of asthma guideline recommendations into quality programmes for patients with asthma using current knowledge translation principles. It also provides examples of successful initiatives in various socioeconomic settings.

KEYWORDS: Asthma, education, Global Initiative for Asthma, implementation, knowledge translation, practice guidelines
Laval University Chair in Knowledge translation, Education and Prevention in Respiratory and Cardiovascular Health

- Shared-decision tools
- Summary of Canadian Guidelines
- Video clips
- Electronic Tools
- Virtual Lab
- Cardiorespiratory tests « bank »
- Etc.

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Mean decisional conflict score is >2/5 In the asthmatic population, decisional conflict (DC) is high which means asthmatics did not totally agree with recommendation for their asthma control.

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Decisional Conflict</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (♀ : ♂)</td>
<td>2.08 : 2.48</td>
<td>0.11</td>
</tr>
<tr>
<td>Severity (mild vs moderate)</td>
<td>2.52 : 1.97</td>
<td>0.03</td>
</tr>
<tr>
<td>Followed by physician (no: yes)</td>
<td>2.70 : 2.20</td>
<td>0.04</td>
</tr>
<tr>
<td>Recruitment (research vs clinics)</td>
<td>2.42 : 2.16</td>
<td>0.33</td>
</tr>
<tr>
<td>Asthma education (yes vs no)</td>
<td>2.40 : 2.05</td>
<td>0.28</td>
</tr>
</tbody>
</table>
Some tools produced and evaluated

• Shared-decision tools
• Summary of Canadian Guidelines
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• Etc.

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The future ... remaining questions

• What is the contribution of airway remodelling to airway function changes?
• What are the mechanisms of transition between inflammation and remodelling and how can we to block it?
• What is the role of new therapeutic agents and modalities?
• How can we best use biomarkers in optimal targeting of asthma treatment?
• What about management of asthma in ACOS/Elderly/obese patients/co-morbidities?
• How can we best support medical practice/guidelines implementation?
• How can we increase the availability of asthma education and its effectiveness?
The future ... How can we prevent/cure asthma?

How can we prevent:
- the persistence of airway inflammation
- the development of airway remodelling
- airway epithelial damage

Means...
- Genotyping/genomodulation
- Environmental measures
- Prevention of irespiratory infections in childhood
- Diet/lifestyle?
- Early immunomodulation
- Preventative pharmacology? Others...

All this will require KT + Education !!!
New therapies for asthma
Contribution of AllerGen CIC

More than 20 studies, including:

• Nebulized Heparin-Derived Oligosaccharides
• Anti-sense therapy (CCR3 and the common β chain of IL-3, IL-5, GMCSF)
• Anti-Interleukin-9
• Anti-IL13
• Anti-IgE
• Immunomodulators
• nicotinic/muscarinic agonist
• etc.
BRONCHIAL THERMOPLASTY: The ALAIR® SYSTEM

Alair Bronchial Thermoplasty System

The Alair Radiofrequency Controller supplies energy to the catheter to heat (65°C) the airway.

Alair Catheter is a flexible tube with 4 expandable wires at the tip.

CONCLUSIONS

• Asthma is still a major health problem
• It can be controlled in the majority but many are not
• Increasing understanding of physiopathology
• Many management difficulties
• Many news therapeutic avenues available soon
• Phenotyping/targetting therapy
• The best treatment is prevention
Will there be a cure for asthma?