Family Physician Airways Group of Canada

Airways Update

In this issue:

Chairperson's Report

After four years of planning the conference hosted by the FPAGC in partnership with the IPCRG in Toronto in June of last year called 'Making Every Breath Count' was an overwhelming success. Over 560 participants from 42 countries combined for a spectacular educational event with wonderful opportunities to meet colleagues from around Canada and around the world. Well over 100 new abstracts were presented. Excellent educational content sprinkled across seminars, workshops, debates and special symposia. A profound thank you is needed in this space. Dr. Robert Hauptman did an outstanding job as scientific chair. Dr. Gordon Dyck was our social chair, and even did a rousing solo during the evening soiree'. We also need to thank our sponsors, who allowed this event to occur. Astra Zeneca, Boehringer Ingelhiem, Glaxo Smith Kline, Merck Frosst, and Nycomed were our platinum sponsors. Other sponsors included Afexa, Pfizer, Novartis, Talecris, ManthaMed, Trudell, and Vitalograph. And thanks to those of you, our members, who joined us at this event.

Canada made additional international noise this past June when we hosted the GARD (Global Alliance for Respiratory Disease) meeting in Toronto as a sister meeting to our conference. GARD involved many groups, including government, to facilitate respiratory issues internationally. The theme for 2010 was the role of Primary Care in Respiratory Diseases, and the IPCRG was well represented by Dr. Niels Chavannes.

The IPCRG has also had some changes, with Dr. John Haughney of the UK stepping down from the Presidency. Dr. Miguel Roman of Spain is our new President. You can see an updated IPCRG website at www.theipcrg.org.

Nationally, the FPAGC continues to be involved in Respiratory Medicine in Canada across multiple fronts. Three of your executive have been involved in the upcoming Sinusitis guidelines. More of this to come. The CNRC (Canadian Network for Respiratory Care) is the new name for CNAC. It changed its name to better define it s role as the key organization for Respiratory Educators in Canada. Certified Respiratory Educators are currently about 900 strong in Canada and can be an excellent resource to assist your patients in your local communities. If you are looking for respiratory educators, try the website www.cnac.net.

I sit as the Family Physician on the CTS guideline dissemination and implementation committee. Guidelines have been created by the CTS for asthma, COPD, pulmonary rehabilitation, sleep apnea, and others. Personally, I feel one of the key roles of the FPAGC is that of assisting in knowledge translation of guidelines for Family Physicians. The CTS is attempting dissemination through presentations at lectures, articles in Canadian Family Physician, and websites such as ours www.fpagc.com and the CTS guideline site at www.CanadianRespiratoryGuidelines.ca.

Dr. Andrew Cave is our representative at the National Lung Health Framework, an organization to attempt to foster improvements for Respiratory issues including novel research, across Canada.

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One of the main mandates of the FPAGC is education. To that end we have fostered a relationship with multiple educational opportunities, including web-based ones. Many of you participated in www.canadianfluguide.ca last year, which was a program on H1N1 in Canada. In addition, we have created a formal relationship with MD briefcase, as their medical group for the Lung Health Portal. We have been involved in created numerous educational opportunities for you on line including Asthma, COPD, anaphylaxis, allergic rhinitis, and coming up Sinusitis and TB. Check out multiple resources including case studies, guideline reviews and newsletters (all with CFPC accreditation) at www.mdbriefcase.com.

The most exciting new Primary Care Respiratory Initiative is related to the College of Family Physicians of Canada. The College is recognizing that there are many Family Physicians who spend a significant, if not all, part of their practice in special interest parts of medicine. The College wants to be the umbrella of all Family Physicians, and as such has created a home for those Family Physicians with special interest focused practices. With this in mind, an application was made and accepted to create a special interest group in Respiratory Medicine.

Special thanks are necessary to the work of Dr. Robert Hauptman and Dr. Sam Ramtullah who worked on this proposal, and to Dr. John Maxted who facilitated this process at the College. A further update of this group will be coming to all FPAGC members. Although the groups are currently separate, their common themes and membership means that their paths shall surely intertwine. A website will be coming soon. When you get your College reapplication, there will be a number of special interest groups you can join; there is no cost. Please join up to the Respiratory group to get further information, and without any obligation! If you would like more information about this, please drop me a line at for4kids@gmail.com

There are two new respiratory products released this year, ZMax SR and Daxas, for which we will have new product updates in our newsletter.

Lastly, the major diagnostic tool we have in respiratory medicine, after history and physical examinations is that of Spirometry. The FPAGC has sponsored workshops at multiple College meetings this year. Please let us know if you want a workshop in your area, it is Mainpro C accredited.

All the best for 2011,

Alan Kaplan, MD CCFP(EM), Chair, FPAGC

New viruses cause severe asthma in children

Bizzintino J. et al. Eur Respir J. 2010 Aug 6. [Epub ahead of print]

In a study of 128 children presenting with asthma, the new rhinovirus was found in 60% of children with acute asthma and it was associated with higher asthma severity scores.

Children aged 2–16 yrs presenting to the emergency department with an asthma attack were included in the study. An asthma attack severity score (from 5 mild to 15 severe) was assigned to each child based on clinical characteristics. Nasal samples were tested for viruses causing respiratory infections.

The majority of the children studied had moderate to severe asthma (85.2%) and 98.9% were admitted to hospital. The overwhelming majority of children (92.2%) had a respiratory infection at the time of their asthma attack, and most infections (87.5%) were caused by the common cold virus, known as human rhinovirus (HRV). Other respiratory viruses were detected in 14.8% of children, most of whom also had HRV. HRVC were present in the majority of children with acute asthma (59.4%) and associated with more severe asthma.

When looking at asthma attack severity, researchers from the University of Western Australia in Perth found that the new types of common cold virus were more commonly found in asthmatics whose attacks were significantly more severe. The majority 59.4% (76 out of 128) of children with acute asthma were infected with an HRVC strain compared with 26.6% (34 out of 128) whose only HRV infection was with a strain from the HRVA or B group. The 76 children infected with HRVC not only had more severe attacks than those infected with a previously known HRV group; their attacks were also more severe than those of all other children who were not infected with a new HRVC type.

Editors note:

Human Rhinovirus is also what was found to be the most common organism in the September peak of Canadian school age asthma. This new HRVC strain may have implications for changing demographics of acute asthma attacks.

Question:

What therapy do you provide a child who does not achieve good asthma control with Low Dose ICS?

Lemanske R, Mauger D, et al Step-up Therapy for Children with Uncontrolled Asthma Receiving Inhaled Corticosteroids NEJM 2010 362; 11

OVERVIEW: In this study, called the Best Add-on Therapy Giving Effective Responses (*BADGER*) trial, the frequency of differential responses to three blinded step-up treatments in children who had uncontrolled asthma while receiving low-dose inhaled corticosteroids was assessed. They used a three-way crossover design with a composite of outcomes (asthma exacerbations, asthma-control days, and the forced expiratory volume in 1 second [FEV_1]), to determine the probability that a given treatment would produce the best response. They also determined whether specific baseline characteristics could be used to predict the response to step-up treatment.

The authors randomly assigned 182 children (6 to 17 years of age), who had uncontrolled asthma while receiving 100 µg of fluticasone twice daily, to receive each of three blinded step-up therapies in random order for 16 weeks: 250 µg of fluticasone twice daily (ICS step-up), 100 µg of fluticasone plus 50 µg of a long-acting betaagonist twice daily (LABA step-up), or 100 µg of fluticasone twice daily plus 5 or 10 mg of a leukotriene-receptor antagonist daily (LTRA step-up).

CRITERIA: Each child had to have the ability to perform reproducible spirometry, have an FEV_1 of at least 60% before bronchodilation, and an increase in the FEV_1 of at least 12% (bronchodilator reversibility) or a methacholine provocation concentration causing a 20% fall (PC_{20}) in the FEV_1 of 12.5 mg per milliliter or less.

DESIGN: All patients were enrolled in a run-in period of 2 to 8 weeks to determine whether their asthma was poorly controlled while they were receiving 100 µg of fluticasone twice daily. The run-in period could be shortened by up to 1 week for safety reasons in case of a sudden worsening in symptoms. Patients or their parents or guardians recorded symptoms and peak-flow determinations in a daily diary. Uncontrolled asthma was defined as the occurrence of at least one of the following for more than 2 days per week on average during a 2-week period: diary-reported symptoms (coughing rated as moderate or severe or wheezing rated as mild, moderate, or severe), rescue use of an inhaled bronchodilator with two or more puffs per day, or peak flows under 80% of the predetermined reference value.

Patients then entered a randomized, double-blind, three-treatment, three-period crossover trial for a total of 48 weeks. During each 16-week period, patients received 250 µg of fluticasone (Flovent Diskus, GlaxoSmithKline) twice daily (inhaled-corticosteroid [ICS] step-up therapy), 100 µg of fluticasone plus 50 µg of the long acting beta-agonist salmeterol (Advair Diskus, GlaxoSmithKline) twice daily (LABA step-up therapy), or 100 µg of fluticasone twice daily plus 5 or 10 mg of the leukotriene-receptor antagonist montelukast (Singulair, Merck) daily (LTRA step-up therapy). The drug assignments were masked with the use of placebo tablets and dummy disk devices that discharged powder without the active drug.

OUTCOMES MEASURED: The outcomes measured were the need for treatment with oral prednisone for acute asthma exacerbations, the number of asthma control days, and the FEV_1. One treatment period was ranked as better than another if the total amount of prednisone received during the period was at least 180 mg less, if the number of annualized asthma-control days during the final 12 weeks of the period was increased by at least 31 days, or if the FEV_1 at the end of the period was at least 5% higher. If the prednisone threshold was met, then they ignored the number of asthma control days and the FEV_1. If the threshold for asthma-control days was met, then they ignored the FEV_1. Otherwise, the order of response was determined by the FEV_1. If none of the thresholds were met, then they gave the same rank to each treatment period and the patient was considered not to have had a differential response. A patient was considered to have had a differential response if at least one treatment period was ranked as better than another.

CONFOUNDERS: The percentage of asthma-control days differed according to season in all study groups, ranging from 71% in winter months to 79% in summer months. Asthma exacerbations were most frequent during winter months. The average FEV_1 (measured as a percentage of the predicted value) varied by less than 1% across seasons. Seasonal variation, however, affected only 12% of patients for whom the number of annualized asthma-control days determined the differential response. In the analysis, seasonal differences in the FEV_1 had no significant effect on the results, whereas seasonal differences in exacerbations had a small non-significant effect.

Patients completed 90% of the study visits and provided sufficient data in the symptom diaries to determine control status on 96% of study days. The rate of adherence to study medication was 84% for study tablets (as measured by an electronic monitor in the bottle cap) and 87% for study inhalers (as measured by a disk counter).

RESULTS: Overall, a differential response occurred in 161 of 165 patients who were evaluated ($P<0.001$).

In the pair wise comparisons, the proportion of patients who had a better response to LABA step-up was higher than the proportion with a better response to LTRA step-up (52% vs. 34%, $P = 0.02$), and the proportion with a better response to LABA step up was higher than the proportion with a better response to ICS step-up (54% vs. 32%, $P = 0.004$), whereas the responses to LTRA and ICS step-up therapies were similar.

The primary outcome of the trial, a three-way comparison of step-up therapy with the use of rank-ordered logistic regression with the criteria listed above, predicted that the response to LABA step-up was significantly more likely to be the best response, as compared with the response to LTRA step-up (relative probability, 1.6;95% confidence interval [CI], 1.1 to 2.3; $P = 0.004$) and the response to ICS step-up (relative probability, 1.7; 95% CI, 1.2 to 2.4; $P = 0.002$).

PRIMARY ANALYSIS OF PREDICTORS OF DIFFERENTIAL RESPONSE: The ability of the four preselected factors to predict patterns of differential was evaluated. Patterns of differential response were not predicted by methacholine PC20 values, by the fraction of exhaled nitric oxide, or by the genotype at position 16 of the 2-adrenergic receptor. Baseline scores on the Asthma Control Test and the Childhood Asthma Control Test that were dichotomized at validated numerical scores indicative of acceptable control (>19) or unacceptable control (≤19) significantly predicted patterns of differential response ($P = 0.009$), with higher scores predicting a greater probability that the best response would be to LABA step-up.

It should be noted that baseline values for the fraction of exhaled nitric oxide and PC20 were obtained after at least 2 weeks of low-dose inhaled corticosteroids in the run in period, which may have decreased these factors from being predictive.

SECONDARY ANALYSIS OF PREDICTORS OF DIFFERENTIAL RESPONSE: Race or ethnic group significantly predicted patterns of differential response ($P = 0.005$) with Hispanic and non-Hispanic white patients most likely to have a best response to LABA step-up and least likely to have a best response to ICS step-up, and black patients equally likely to have a best response to LABA or ICS step-up therapy and

less likely to have a best response to LTRA step-up. There were no differences in the patterns of differential response according to age. Patients who did not have eczema were most likely to have a best response to LABA step-up (P = 0.006)

SUMMARY: A differential response to step up therapy occurred in 161 of 165 patients who were evaluated (P<0.001). The response to LABA step-up therapy was most likely to be the best response, as compared with responses to LTRA step-up (relative probability, 1.6; 95% confidence interval [CI], 1.1 to 2.3; P = 0.004) and ICS step-up (relative probability, 1.7; 95% CI, 1.2 to 2.4; P = 0.002). Higher scores on the Asthma Control Test before randomization (indicating better control at baseline) predicted a better response to LABA step-up (P = 0.009). White race predicted a better response to LABA step-up, whereas black patients were least likely to have a best response to LTRA step-up (P = 0.005).

Despite step-up in daily therapy, 120 exacerbations requiring the use of oral corticosteroids occurred during the treatment periods. Day-today asthma control, as reflected by the number of asthma-control days, was quite good with all three step-up therapies. However, none of the step-up therapies completely prevented asthma exacerbations.

CONCLUSIONS: Nearly all the children had a differential response to each step-up therapy. LABA step-up was significantly more likely to provide the best response than either ICS or LTRA step-up, especially in those who were very symptomatic with a high ACT score or those who did not have eczema. It is important to note that safety was not measured in this study, so risk benefit ratio of the additional medications must also be included in your decision for step up therapy. There has not been a lot of evidence for combination therapy with LABA in children up until this point. That being said, many children did have a best response to ICS or LTRA step-up therapy, highlighting the need to both individualize each child's therapy and regularly monitor and appropriately adjust each child's asthma therapy based on response.

Beta blockers may reduce the mortality and risk of AECOPD

Rutten et al. Arch Internal Med 2010; 170(10):880-887

The long term effect of beta blocker use on survival and AECOPD was assessed in an observational cohort study gathered from Dutch EMRs. This data contained standardized information about daily patient contacts, diagnoses and prescription medications prescribed. The period studied was 1996-2006 and 2.230 patients ≥ 45 years old with a diagnosis (incident or prevalent) of COPD were followed for a mean of 7.2 years; mean age of ~65 and 53% were male. 30.8% of the patients died, and 47% had at least one exacerbation.

Results:

The adjusted hazard ratio of beta blocker use for mortality was 0.68 (CI 95% of 0.56-0.83) and for exacerbation of COPD was 0.71 (CI 95% of 0.6-0.83). Interestingly sub analyses of patient with COPD but WITHOUT overt cardiovascular disease had similar results.

Conclusion:

Beta blocker treatment may reduce exacaerbations and improve survival in patients with COPD.

Editors note:

This does NOT mean that you should go out and put all your patients with COPD on beta blockers because of this purported dual cardiopulmonary protection. We do know from TORCH[1] that deaths from COPD come from CV disease and AECOPD primarily, and that other cardiovascular protection strategies such as statins[2] also improve outcomes. As we continue to understand that COPD is a systemic disease and that accompanying the chronic inflammation in the airways are inflammatory mediators responsible for many of the comorbidities[3] our attention is paid to the CV protective methods we use in practice. A COPD patient has a 'double whammy' of the effects of tobacco on both the lungs and the rest of the body. It is easy to advocate the use of statins, ECASA, ACE inhibitors but B blockers have been controversial due to the feeling that they may worsen dyspnea by worsening bronchoconstriction, and in some patients they certainly do. A recent Cochrane analysis has said that cardioselective B blockers are safe in COPD[4] .

There are some reasons why this study's result may be flawed, however. This was observational data only. These patients may have had more attentive care and thus had better outcomes, and those who could not tolerate beta blockers may not have made it to the data.

So, while it is eminently reasonable to treat those with CV disease and COPD with B blockers if they can tolerate them, we are not yet at a place where they are used preventively. I think we should still consider CV risk in those with COPD and treat all of the other cofactors aggressively. A recent yet unpublished study in New Zealand presented at ATS last spring by Dr. Gwavava looked at patients admitted with COPD, Of those 117 patients, 32 had ischemic heart disease, but the other 85 did not and were assessed for CV risk by Framingham risk scores. 58 of the 85 had a 10 year risk above 10%, only 21of the patients were on antiplatelet agents, and even less were on statins. However, if you do treat with a beta blocker, please remember to reassess your patient for dyspnea and quality of life.

References:

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MEETING

EEACI 2009 report

I thought it would be the most useful for me to review a number of the pertinent abstracts that were presented which will highlight the newest science currently out there. I would like to thank Merck Frosst for a travel grant to help me to attend this meeting.

PET EXPOSURE IN EARLY INFANCY, ASTHMA AT SCHOOL AGE? A META-ANALYSIS OF GALEN
CARLSON K ET AL

The endpoint of Asthma at age 6-10 years was derived from a number of birth cohorts (26,500 European children) that had childhood exposure to one or more pets including cats, dogs, birds and/or rodents before the age of 2.

Bottom line: Pet exposure had no effect on the risk of development of asthma.

My conclusion: This was a large group; perhaps it would have been different if we had looked at the risk of those children of parents with asthma that had pet allergy?

PET EXPOSURE IN INFANCY; ALLERGIC RHINITIS AT SCHOOL AGE? A META-ANALYSIS OF GALEN
KEIL T, ET AL

Same group of children analysed for risk of allergic rhinitis at age 8-10 and pet exposure before the age of 2 years. In analysis of dust in the homes, those homes with the highest level of dog allergen in the dust had the lowest risk of asthma.

Bottom line: Early exposure to pets does not seem to increase the risk of allergic rhinitis

My conclusion: Again, subgroup analysis of children of allergic patients would have been nice.

VITAMIN D STATUS DURING PREGNANCY AND EARLY LIFE AND ATOPIC OUTCOMES IN CHILDHOOD
MOMMERS ET AL.

2834 children were followed measuring maternal and children's 25 OH vitamin D levels and subsequent risk of development of atopy

Bottom line: The risk of atopic outcomes was highest in the mothers with the highest quartile of vitamin D levels. In children it was lowest in the children with highest and lowest quartiles.

My conclusion: While it is tempting to just push Vitamin D for all of us Canadians who do not get enough sunlight due to proposed Vitamin D benefits, there may be a cost in pushing doses too high to pregnant mothers!

ALLERGIC RHINITIS, NASAL SYMPTOMS AND THEIR ASSOCIATION WITH ASTHMA
EKELJURG L ET AL

This study was a postal study of 30,000 people in Sweden to assess current prevalence rates of allergic rhinitis and persistent nasal symptoms, their variation with gender and age, and how these conditions co-vary with asthma and respiratory symptoms.

Results: The prevalence of allergic rhinitis was 26.9% (men 26.0%, women 27.6%, p=0.02), nasal obstruction 14.9% (men 15.3%, women 14.5, p=0.11) and rhinorrhea 13.1% (men 12.9, women 13.3, p=0.41). Of the subjects with allergic rhinitis, 27.3% also had nasal congestion and 23.9% had rhinorrhea. The prevalence of allergic rhinitis, nasal obstruction and rhinorrhea decreased with age; however, the prevalence of allergic rhinitis was higher among subjects aged 31-45 years than among those aged 16-30 years. Non-smokers had a higher prevalence of allergic rhinitis and a lower prevalence of nasal obstruction and rhinorrhea compared to smokers and ex-smokers. All investigated nasal symptoms showed a strong association with asthma, recurrent wheeze, attacks of shortness of breath, longstanding cough and chronic productive cough.

Bottom Line: Rhinitis and Asthma are linked epidemiologically.

My conclusion: Look and you shall find, rhinitis and asthma co-exist. Smoking by itself causes rhinitis and also causes/worsens asthma.

LUNG FUNCTION MEASUREMENT AND EXHALED NITRIC OXIDE IN CHILDREN WITH ALLERGIC RHINITIS MRKIC ET AL.

Allergic rhinitis can often lead to asthma. It is known that some inflammatory markers are in common for both diseases. In this study they did lung function measurements and level of exhaled NO (eNO) in patients with allergic rhinitis. In 53 children (35 male, 4-18 yrs, without initial therapy) with diagnosed allergic rhinitis without asthma, they measured lung function, eNO, evaluated allergen sensitivity (skin prick tests, specific IgE) and therapy and compared them to the 37 healthy controls (22 male, 4-18 yrs) for lung function measures.

Results: Fifty one percent of patients were allergic to pollen, 9 to mites, 2 to other perennial allergens and pollen, 23 to pollen and mites, and 15 were multisensitized. Spirometry showed lower values in 15% patients and 5% controls (FVC ‹80% predicted), while up to 19% (none in controls) had lower values in the area of small airways (MEF25 or MEF50 ‹80%), and up to 28% (21% in controls) in the area of large airways (MEF75 or PEF ‹80%). 53% of patients (27% in controls) had any of lung function measures "impaired". In 17% of patients there were elevated eNO levels (diagnostic for asthma). There was no association between type of allergen and lung function measurements or measured eNO levels.

Bottom line: It seems that allergic rhinitis in children can be related to lower values of lung function measures without clinical symptoms

My conclusion: These children do need to be followed as they are at increased risk of developing asthma. Would aggressive rhinitis management prevent asthma? Would trigger avoidance do this? Would early maintenance therapy make a difference?

ANTIBIOTIC USE IN EARLY LIFE AND THE DEVELOPMENT OF ALLERGIC DISORDERS; INFECTION AS THE EXPLANATION MAI X ET AL.

Almost 4,000 Norwegian children were followed in a birth cohort study with reference to antibiotic use, respiratory infections under the age of 1 year old and subsequent development of allergic diseases at 4 and 8 years old.

Bottom line: 44% of the children were exposed to antibiotics and this seemed to increase the risk of developing asthma or other allergic diseases. They were used for pneumonia (8%), bronchitis (15%) and otitis media (55%). When these three infections were accounted for, the antibiotic risk disappeared, indicating that the condition which was being treated may have been the issue, not the antibiotic!

My conclusion: Antibiotics may not be the confounding cause of allergic disease that we thought it was, but infection may be. So do not throw out the antibiotics; however we must use them responsibly.

Canadian Metropolitan TB Subcommittee

I am the Family Physician representative to this committee. It is sponsored by the Public Health Agency of Canada.

MANDATE:

To share information and identify key operational problems in the implementation of tuberculosis prevention and control services in metropolitan areas. Recommendations and final reports will be developed by the Subcommittee in concert with Public Health Agency of Canada's Tuberculosis Prevention and Control staff at the appropriate working level. These reports will be submitted by the Chair to the Canadian Tuberculosis Committee. The meetings are generally held annually.

It s members are made up of the following. To be provocative, it is interesting that there are no CTS representatives or people from the society of Infectious Diseases.

a. One representative from the local public health unit/TB control program of a metropolitan area in Canada with a population of at least 500,000 and typically having at least 25 cases of TB annually.
b. One Canadian Tuberculosis Committee member or designate from each province/territory with a metropolitan area represented on the Subcommittee.
c. One representative from the Association of Medical Microbiology and Infectious Disease Canada.
d. One representative from Citizenship and Immigration Canada, Medical Services Branch.
e. One representative from the College of Family Physicians of Canada.
f. One representative from Correctional Service Canada.
g. One representative from the Federal/Provincial/Territorial Heads of Corrections Working Group on Health.
h. One representative from the National Tuberculosis Program of Health Canada's First Nations and Inuit Health Branch.
i. Manager, Tuberculosis Prevention and Control, Public Health Agency of Canada.
j. Senior Epidemiologist, Tuberculosis Prevention and Control, Public Health Agency of Canada (*ex-officio*).

There are many interesting topics reviewed. While TB is overall a fairly uncommon condition in Canada, there are some relevant issues for the Family Physician. The comorbidity of HIV and TB is very important. The number of native born Canadians with TB is falling, with the number of foreign born cases rising significantly. Temporary foreign-workers actually make up a large number of TB cases but aren't captured in the system because they don't apply for permanent residents until after they've been in the country for several years. The highest incidence of TB is reported within the first two years following arrival to the country, with another 1/3 within five years. There are 220-240 thousand new settlers per year in Canada. 20% of Canadians are foreign born. Government looks at these people as 'human capital' and there seems to be no slowing down of this happening. Other high risk groups are people living on reserves, crack users in some studies, in shelters/homeless and in correctional institutions.

Establishing dedicated TB clinics seems to be a priority for this group. This may be due to the fact that most of the representatives do work at TB clinics! That being said, treating TB is not simple, and with multi-drug resistance strains, it perhaps is not unreasonable.

Dr. Cowie presented much evidence demonstrating the effectiveness of having dedicated TB clinics in Alberta, particularly with respect to monitoring treatment and prescription practices, contact tracing, and management of HIV/TB coinfected patients. In Ontario, there are currently four existing dedicated TB clinics in Toronto (St. Michael's Hospital, Toronto Western Hospital, Hospital for Sick Children and West Park Hospital). In Peel Region, Dr. de Villa indicated that there is a proposal in place to develop a TB clinic in Brampton (William Osler Hospital) and a second hospital in Mississauga has also expressed interest. Ms. Marshall reported that in York Region, Markham Stouffville Hospital has approached the public health department to discuss the possibility of having a TB clinic established in the new facility.

Contact tracing was a significant point of discussion at this current meeting. A typical TB case can infect 16 others on average, so capturing contacts who seroconvert is obviously critical. There seems to be great discrepancy across the country in how this occurs, and an attempt was made to standardize how this is done. Who should be tested, when and what constitutes a positive test all are questions that are answered differently across the country. For instance a low risk person will be considered positive with a TB test at 10 mm. A high risk will be positive at 5 mm (eg. HIV, children). Most seem to agree that household contacts do need to be tested. Remember, this will diagnose people with LTBI (latent TB infection) not active disease, and thus they are not considered infective.

Genotypic tracings have allowed improvement on contact tracing to see where the cases originate, which can help guide your contact tracing and outbreak management. This is done through the public agencies, again highlighting the need for public health involvement.

While TB is uncommon, it is a disease with significant morbidity and mortality and can be a public health threat. We, as Family Physicians, should be aggressive in diagnosing this condition. I would, at least, discuss this with a TB clinic, as they are set up for proper surveillance and follow up of what is really becoming quite a complicated condition. My goal is to work with this group to create a good CME program on this topic, as while uncommon, TB should NOT be out of mind!

Zmax SR™: New azithromycin formulation provides a full course of antibacterial therapy in a single-dose treatment

Respiratory infections are the number one source of office visits to primary care. Nearly two thirds of solid antibiotics are used for respiratory infections. Antibiotic utilization is a significant precipitant of antibiotic resistance, and antibiotics should be used appropriately. Appropriate utility of antibiotics in communities can result in lower antibiotic resistance[1,2]. Adherence can be a key issue when it comes to traditional antibiotic treatment, and improper use of antibiotics is a contributing factor to antibiotic resistance. Zmax SR™, an innovative new antibiotic formulation that provides a convenient single-dose treatment, is now available in Canada. Zmax SR is introduced by Pfizer Canada.

Zmax SR™ (azithromycin sustained-release granules for oral suspension) is indicated for treatment of respiratory tract infections caused by susceptible strains of the designated microorganisms in the following diseases and specific conditions:

- Acute bacterial exacerbations of chronic bronchitis due to Haemophilus influenzae, Moraxella catarrhalis or Streptococcus pneumoniae.
- Acute bacterial sinusitis due to Haemophilus influenzae, Moraxella catarrhalis or Streptococcus pneumoniae.
- Community acquired pneumonia of mild severity due to Chlamydia pneumoniae, Haemophilus influenzae, Mycoplasma pneumoniae or Streptococcus pneumoniae.

Zmax SR™ is a modified-release formulation, which provides a full course of antibacterial therapy in a single dose[3,4,5]. The first dose of Zmax SR™ is therefore the last dose.

Zmax SR™ treats ABS, ABECB and mild CAP effectively and is generally well tolerated. The most common adverse events were diarrhea (10.9%), nausea (3.9%), abdominal pain (2.7%), headache (1.3%) and vomiting (1.1%). Most gastrointestinal events were mild-to-moderate in severity, occurred on the day of dosing and resolved within 1-2 days. Gastrointestinal side effects are minimized by giving the medication on an empty stomach. In the event that patient vomits there is often concern of whether the dose should be repeated. As a general rule, it depends on the timing of the vomiting[6].

- Within 5 minutes of administration: consider additional antibiotic treatment (minimal absorption)
- Between 5 and 60 minutes: alternative therapy to be considered (insufficient data)
- >60 minutes: neither second does, nor alternative treatment

Zmax SR™ is not recommended in pediatric patients (below 18 years of age). Zmax SR™ is contraindicated in patients with hypersensitivity to azithromycin, erythromycin, any macrolide or ketolide antibiotic or to any ingredient in the formulation or component of the container.

A single 2 g dose of Zmax SR™ is not bioequivalent and is not interchangeable with regimens employing azithromycin immediate-release oral formulations (tablets or oral suspension) due to a different pharmacokinetic profile. The microsphere formulation makes it possible to provide an entire antibiotic therapeutic course in a single dose. In addition, the ZMax SR™ formulation allows three times the normal systemic dose of antibiotic in the first day[7]. This front-loaded dose results in significantly higher peak concentrations in white blood cells when bacterial burden likely to be highest[8]. High concentrations are attained in lung and sinus tissues rapidly allowing good bacteriocidal levels (to kill the bacteria) and persistence of the antibioitic in tissue fluid helps prevent resistance from occurring[9].

Editors Note: As practitioners, we know how critical it is for patients to follow the instructions for treatment to the letter in order to avoid treatment failure and antibiotic resistance. This new formulation is an improvement on current macrolide options and is likely going to rapidly be a patience preference.

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Routine surveillance of Respiratory Disease in Canada

A report of the Public Health Agency of Canada Respiratory Surveillance Committee using data from:

- Canadian Community Health Survey (CCHS), Statistics Canada
- Vital Statistics, Statistics Canada
- Hospital Morbidity File, CIHI
- Canadian Tobacco Use Monitoring Survey (CTUMS), Health Canada

Smoking

- Overall, the prevalence rate of smoking has decreased in all age categories since 1998.
- Smoking prevalence is highest among 20–24 year-olds and lowest among individuals aged 45+ years.
- The greatest decrease in smoking prevalence was seen in youths aged 15–19 years; and, since 2003 the smoking prevalence among youth aged 15–19 years has been lower than that of the overall population.
- 16.8% of the population aged 12 years and older reported that they are current smokers(2008 data).
- Among current smokers, the prevalence of smoking was significantly different between the total (12+ years), asthmatic (12+ years), and COPD (35+ years) populations (including when COPD was compared only to those aged 35+ years). The prevalence of current smoking was lowest in the total population and highest in individuals with COPD.
- Among former smokers, there was no significant difference between the total population (12+ years) and those who reported having asthma (12+ years); but, prevalence among those with COPD (35+ years) was significantly higher than either the total or asthmatic populations. However, no significant difference was noted between prevalence of former smoking in those with COPD and those 35+ years.
- Among those who have never smoked, the difference between total (12+ years), asthmatic (12+ years), and COPD (35+ years) populations was significant (including when COPD was compared to only those aged 35+ years). The proportion of never smokers was highest for the total population and lowest for individuals with COPD.
- Overall, the trends were similar between the sexes.

Smoking rates are down and falling, but we have still got a lot of work to do, especially with our children and young adults.

Asthma

In a previous study done in seven centers across Canada, Nova Scotia had the highest prevalence of asthma as determined by symptoms or physician; however, Nova Scotia also had the lowest confirmed rate of asthma by spirometry or by methacholine. This highlights the difficulty with asthma diagnosis by physician or symptoms.

With respect to prevalence of **self-reported physician-diagnosed asthma by recent symptom or medication use**, data from the National Public Health Survey (NPHS) and the CCHS, show that prevalence of asthma in the overall population (12+ years) was increasing between 1994 and 2000. The overall prevalence of self-reported asthma as well as prevalence of recent asthma symptom or medication use is higher among women than men, and increases for both sexes between 1994 and 2000, but appears to have levelled off since 2000. The proportion of asthmatics reporting asthma symptoms or medication use in the past 12 months has been relatively stable from 1994-2007/08, and is slightly higher in women compared to men.

Overall, there was a significant **difference between men and women** in prevalence of self-reported physician-diagnosed asthma in 2008, with the prevalence being higher in women.

Asthma remains a major cause of **hospitalization**, mostly among children. According to data from CIHI, the rate of hospitalizations has declined since 1987, which may reflect improved disease control. It may also reflect the downsizing in the hospital sector, however, and the reduction in available beds.

Authors key issues we can learn from this:

Asthma control in Canada is still suboptimal as per the TRAC study. Hospitalizations continue to be common, especially amongst children. Remember to strive for good asthma control and try to prevent exacerbations

COPD

Estimates of **self-reported physician-diagnosed COPD** (35+ years) from the CCHS were slightly higher in 2008 compared to 2005 for most provinces and for overall national rates (4.4% in 2005 and 4.8% in 2008). In 2008, none of the provinces' prevalence estimates of **self-reported physician-diagnosed** COPD (35+ years) was significantly different from national estimates.

Data from the 2008 CCHS show that COPD is now being

reported more among women than among men under age 75. Overall, however, there was no significance difference in prevalence between **men and women**. The increase in smoking among women in the past 50 years has resulted in an increased prevalence of diseases such as lung cancer and COPD among women.

There was a significant difference between **Aboriginal people** living off-reserve and the total population in prevalence of **self-reported physician-diagnosed COPD** (35+ years), with prevalence being higher in Aboriginals. Note: Aboriginal estimates of COPD were associated with high sampling variability.

Hospitalization separations due to COPD as the most responsible diagnosis continue to rise, based on data from CIHI.

Authors key issues we can learn from this:

- Length of stay for COPD multiplied by the number of admissions magnifies the impact on society because COPD patients have long hospital stays (average is 7 days).
- Patients with a mental co-morbidity will tend to stay in hospital longer and be more likely to be re-admitted.
- The average COPD admission had about 15 co-morbidities, including CVD.
- **Mortality due to COPD** among men aged 55 years and older is higher than among women of similar age, but the gap appears to be narrowing.

2008 CCHS – Self Reported Spirometry

The 2008 CCHS included a question about self-reported spirometry.

"Spirometry is a common lung function test that consists of blowing as long and hard as possible into a small tube attached to a machine. Has a health professional ever administered this test to you?

"When was the last time?

- Less than 6 months
- 6 months to less than 1 year ago
- 1 year to less than 2 years ago
- 2 years to less than 5 years ago
- 5 or more years ago"

The spirometry question was asked if someone said "yes" to having either Asthma or COPD. The sample size was approximately 65,000, aged 12+ years. Data were released this past summer.

Nearly two thirds of respondents with COPD (35+ years), over one half of those with Asthma, and nearly one quarter of Aboriginals living off-reserve reported that they had had spirometry administered by a health professional. Rates of spirometry testing were significantly lower than the national rate (21.3%) in British Columbia and the Territories, while they're significantly higher in Alberta, Manitoba, and New Brunswick. In the total Canadian population, the proportion of rural respondents who reported ever having received spirometry was significantly higher than urban respondents; provincially, this trend was only observed for Ontario.

Authors key issues we can learn from this:

- The proportion reporting ever having had spirometry are unexpectedly high overall and for the subgroups. This may be due to a bias of the people who are willing to answer surveys. This data is a bit surprising, but perhaps all of those CMEs the FPAGC have been doing on Spirometry are paying off!

MEETING

ANNUAL GENERAL MEETING

The Annual General Meeting of FPAGC will be held at the FMF meeting in November.

More details will be made available at a later date in a later issue of the newsletter and on the web site. Nearer the time you can also obtain information from the office by email at, admin@fpacg.com, on the internet at www.fpagc.com or by telephone 1-866-406-4345.

Notification that you plan to attend must be made to the office no later than seven days prior to the meeting.

Counseling your patients about air pollution and health and the Air Quality Health Index

In some parts of Canada, we are very aware of the quality of the air. On bad air days, sometimes called smog days, the air smells and tastes bad, and is hazy and dirty. It is obvious on these days that the air is bad for health. But pollutants in the air affect health significantly even when the air quality is not as noticeably poor. In this article, I will discuss the various air pollutants themselves, their health effects, and what we, as family physicians, can do to protect our patients from air pollution.

Air pollution causes a significant **burden of illness** in Canada. Studies done by Health Canada suggest that 6000 people die prematurely each year from exposure to air pollution in 8 Canadian cities (Judek). The Canadian Medical Association, in its study "The National Illness Cost of Air Pollution (ICAP) (ref CMA)", has calculated that across the entire country air pollution is responsible for a substantial burden of illness, including 21,000 deaths, 11,000 hospital admissions and 92,000 emergency department visits, at an estimated cost of over C$8 billion, attributable to air pollution. The CMA ICAP report can be used to calculate the air pollution attributable mortality and morbidity in each city across the country.

There are a number of **outdoor air pollutants** that are harmful to health, and that are monitored across the country, by Environment Canada, provincial ministries of the environment and some municipalities. The most significant criteria pollutants are Particulate Matter, ozone and Nitrogen Dioxide.

Particulate matter (PM) consists of small solid or liquid particles suspended in the air. They are derived from combustion sources mostly. The size and source is important: the smaller particles, diameter less than 2.5 microns (PM2.5), reach the alveoli when inspired, and the smallest particles of less than 1 micron (ultrafine particles), can cross into the bloodstream, reaching various organs. Different sources i.e. industry, automobile exhaust, diesel, wood smoke etc emit particles with different complex chemical constituents, some carrying metals such as zinc and manganese. Current research is focusing on these different particles and their health effects. They affect the respiratory tract, but also the cardiovascular system, causing vasoconstriction, increased coagulability, intimal inflammation, arrhythmias and promote atherosclerosis. (Brook)

Ozone is formed in the air by complex chemical reactions, under the influence of light and heat. Hence it is mostly found in summer, building in the afternoon. It causes inflammation of the airways.

Nitrogen dioxide (NO2) is found in higher concentrations near roads. It is an indicator of Traffic Related Air Pollution (TRAP), but likely not the only toxic compound emitted from traffic.

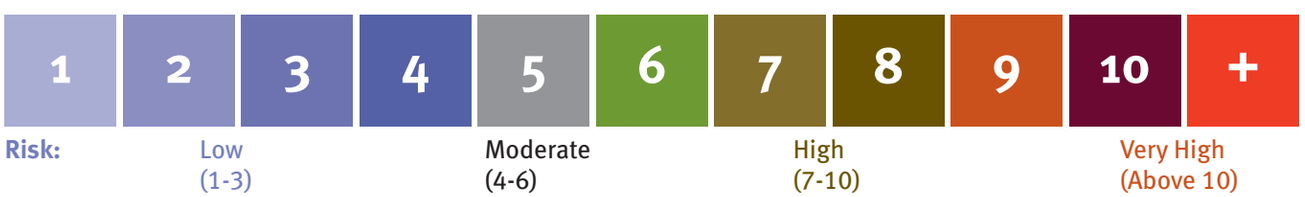
For more information on pollutants and their health effects, I recommend a CFPC accredited online course through UBC School of Environmental Health: Outdoor Air Quality and Health and the Air Quality Health Index. http://www.soeh.ubc.ca/Continuing_Education (Ref UBC)

The health effects of air pollution are categorized as related to short-term exposure or long-term exposure to pollutants. There is a large body of evidence, both epidemiological and toxicological, supporting these associated health effects.

We are most familiar with the understanding of air pollution as a trigger for aggravating asthma, but it can also aggravate COPD, and importantly, exposure to particulate matter is associated with aggravation of cardiovascular disease, exacerbation of cardiac failure, and also precipitating arrhythmias, cardiac arrest and myocardial infarction. (Brook)

Long-term exposure is associated with increased mortality, lung cancer, pneumonia in the elderly, and development of atherosclerosis. There is some evidence that exposure to traffic related air pollution, especially at birth or early

FIGURE 1. **AQHI SCALE**



childhood, is associated with new onset of asthma (Jerret, Dell); and also evidence of delayed lung development, as seen in a cohort of children in high pollution communities in southern California. (Gauderman)

So how can primary care physicians counsel their patients about air pollution?

The Asthma Management Continuum 2009 (Lougheed), under guidelines for environmental control, states: "Exposure to air pollution has been associated with increased morbidity and mortality in individuals with asthma. Recent Canadian data demonstrated an increase in emergency department and ambulatory visits due to asthma in adults and children following exposure to environmental air pollutants, specifically ozone and particulate matter. Efforts to reduce exposure of patients with asthma to air pollution should continue". The US NIH guidelines (Ref) advise to "avoid exertion outdoors when the levels of air pollution are high", as does the American Heart Association. (Brook)

A useful new tool is the **Air Quality Health Index (AQHI).** It was developed by Health Canada and Environment Canada. The AQHI is a 1-10 scale (Figure 1) which has been shown to reflect the health risk in terms of both mortality (Stieb) and morbidity (To), associated with increases in a mixture of air pollutants commonly found in Canada. (Figure 1). Local current and forecasted readings are available for over 50 locations in Canada and can be found at airhealth.ca. Accompanying the index are health messages, which advise on how to reduce exposure. (Figure 2) The messages are targeted at the general population, and at risk groups. At risk groups include people with chronic respiratory and cardiovascular disease (including patients with asthma and COPD), diabetics, children and the elderly. But individuals vary in their sensitivity to air pollutants, so the AQHI encourages people to self calibrate their response based on the index. In general, exercise is encouraged, except in situations where exposure to pollution might be a health risk, where outdoor exercise should be reduced in intensity, rescheduled or moved indoors.

The AQHI is being rolled out across the country, and in many regions it is reported widely in the media, e.g. weather network, so that our patients will know about it. It can easily be incorporated into an asthma action plan, or asthma education.

It is also important in counseling patients, especially vulnerable groups, to advise avoiding exercising near

FIGURE 2. **AQHI CATEGORIES AND MESSAGES.**
http://www.ec.gc.ca/cas-aqhi/default.asp?lang=En&n=79A8041B-1

Health Risk	Air Quality Health Index	Health Messages	
		At Risk Population	General Population
Low	1 - 3	**Enjoy** your usual outdoor activities.	**Ideal** air quality for outdoor activities.
Moderate	4 - 6	**Consider reducing** or rescheduling strenuous activities outdoors if you are experiencing symptoms.	**No need to modify** your usual outdoor activities unless you experience symptoms such as coughing and throat irritation.
High	7 - 10	**Reduce** or reschedule strenuous activities outdoors. Children and the elderly should also take it easy.	**Consider reducing** or rescheduling strenuous activities outdoors if you experience symptoms such as coughing and throat irritation.
Very High	Above 10	**Avoid** strenuous activities outdoors. Children and the elderly should also avoid outdoor physical exertion.	**Reduce** or reschedule strenuous activities outdoors, especially if you experience symptoms such as coughing and throat irritation.

traffic, and other industrial sources. Wood smoke, from home heating or forest fires, is another "local" source of air pollution that should be avoided. Of course the indoor environment is also important in asthma and COPD, in terms of allergens, particles (from ETS, fireplaces and outdoor PM that infiltrate indoors) and gases (e.g. NO2 from gas stoves).

The long-term solution is to reduce the emission of pollution across the country, with policies that encourage healthy urban design and transportation, as well as reduced emissions from homes, automobiles and industry. In the struggle against climate change, any reduction in greenhouse gases also leads to a reduction in air pollution. But, in the meanwhile, we can help our vulnerable patients by encouraging them to regularly use the AQHI to protect their health.

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Daxas™ : A New Phosphodiesterase 4 (PDE4) Inhibitor for the Treatment of COPD — The Clinical Evidence

Chronic obstructive pulmonary disease (COPD) is characterized by progressive airway obstruction and increasing frequency and severity of exacerbations.[1] It is well recognized that COPD is characterized by persistent inflammation of the airways and lung parenchyma[1], and that this airway inflammation is present and plays an important role at all stages of the disease.[2] The inflammation is relatively insensitive to corticosteroid administration[3-5], presumably because it is associated with neutrophils, CD8+ T lymphocytes and CD68+ macrophages, cells that are minimally inhibited by corticosteroids. The airway inflammation specific to COPD differs from that in asthma[6,7], and a therapy targeting COPD-specific inflammation would be welcome.

The progressive course of COPD is often aggravated by exacerbations. Exacerbations are defined as a sustained worsening of dyspnea, cough or sputum production leading to an increase in the use of maintenance medications and/or supplementation with additional medications.[1] Exacerbations contribute to an accelerated decline in lung function[8] and have been linked to a greater mortality risk[9] and recent Canadian statistics on hospital admission show that COPD now accounts for the highest rate of admission among major chronic illnesses.[10] COPD also has a much higher readmission rate than other chronic illnesses; 18% were readmitted once and 14% twice within the year. These numbers are far greater than for angina pectoris, heart failure, diabetes, or hypertension.[10]

A reduction in exacerbations is highly desirable in order to reduce the morbidity and mortality associated with these events. Several therapeutic options are currently available to reduce exacerbations (e.g. inhaled long-acting anticholinergic (LAAC), long-acting B2-agonist (LABA), ICS (inhaled corticosteroid)/LABA combinations), and yet even with "triple therapy" (LAAC, LABA and ICS), the reduction in exacerbations is limited.[11] As such, a therapy that further reduces COPD exacerbations is desirable.

This article reviews a new oral anti-inflammatory medicine, Daxas™ (roflumilast), that is now available in Canada for the maintenance treatment of severe chronic obstructive pulmonary disease (COPD) associated with chronic bronchitis (i.e. patients with a history of chronic cough and sputum) in adult patients with a history of frequent exacerbations.

Several double-blind, placebo-controlled, randomized clinical trials on the use of Daxas™ in patients with COPD have been published. The findings for the recently published clinical trials are summarized below:

The efficacy and safety of Daxas™ was evaluated in two confirmatory replicate one-year trials (M2-124, M2-125) and two supplementary six-month trials (M2-127 and M2-128)]. All trials were randomized, double-blind, placebo-controlled, parallel-group studies. In all studies, DAXAS™ was administered once daily (500 mcg tablet), but the inclusion and exclusion criteria and concomitant medications were not identical.

Pivotal Trials

The pivotal one-year studies (M2-124 and M2-125) were performed and reported by Calverley and colleagues.[12] The results were combined into a single publication and included patients with a history of COPD associated with chronic bronchitis for at least 12 months prior to baseline, with symptoms at baseline as determined by cough and sputum score, non-reversible airway obstruction, an FEV_1 ≤ 50% of predicted and at least one documented COPD exacerbation requiring corticosteroids or hospitalization in the previous year. In these one-year trials, long-acting beta-2 agonists (LABA) were allowed and used in approximately 50% of the study population. The use of inhaled corticosteroids was terminated at randomization. Lung function (pre-bronchodilator forced expiratory volume in one second, FEV1) and the rate of moderate exacerbations (requiring intervention with systemic glucocorticosteroids) or severe exacerbations (resulting in hospitalization and/or leading to death) were co-primary endpoints. In the individual studies and the pooled analysis, Daxas™ 500 mcg once daily statistically significantly improved lung function compared to placebo, and in the pooled analysis, Daxas™ significantly improved pre-bronchodilator FEV1 by 48 mL ($p<0.0001$). Daxas™ 500 mcg increased mean pre-bronchodilator FEV1 by 46 mL ($p<0.0001$), as compared to placebo, in patients with concomitant LABA treatment.[12]

The rate of moderate or severe acute exacerbations (primary endpoint) was significantly reduced in the Daxas™ 500 mcg group compared to the placebo arm in each of the individual studies, the pooled result being 1.14 vs. 1.37 exacerbations/patient/year, respectively, a statistically significant reduction of 17% ($p=0.0003$).[12] In addition, in patients taking concomitant LABA treatment, Daxas™ significantly decreased moderate or severe exacerbations by 21% compared to LABA alone.[13]

6-month Supportive Trials

Two additional efficacy and safety studies (M2-127 and M2-128) have recently been reported in a single publication by Fabbri et al in 2009 investigating the effect of Daxas™ in patients who were concomitantly treated with long-acting bronchodilators: salmeterol and tiotropium.[14] The protocols, which were similar to each other as well as to the previously discussed long-term Daxas™ studies, enrolled patients with moderate-to-severe COPD, and had a 4-week run-in period prior to randomization to either Daxas™ 500 mcg plus salmeterol inhalation b.i.d. or matching placebo plus salmeterol inhalation b.i.d. in one study. In the other, subjects received Daxas™ 500 mcg plus tiotropium inhalation once daily or placebo plus tiotropium inhalation once daily. In this study, subjects were also required to have a high reliever use and symptoms of chronic bronchitis prior to enrolment. At baseline, the mean post-bronchodilator FEV1 was 55% of predicted in the roflumilast plus salmeterol study and 56% in the roflumilast plus tiotropium study (moderate to severe COPD). The studies concluded after 24 weeks and the primary outcome was the change in pre-bronchodilator FEV_1 from the original baseline. In the salmeterol study, roflumilast significantly improved the pre-bronchodilator FEV_1 by a mean of 49 mL over subjects that received only salmeterol plus placebo; in the tiotropium study, the corresponding mean increase was 80 mL, both results being significant at $p<0.0001$.[14] Both studies showed that patients treated with Daxas™ 500 mcg had lower exacerbation rates than those treated with placebo but these reductions were not statistically significant at the 0.05 level. These six-month studies were neither designed nor powered to show a statistically significant effect on exacerbations.[13]

In the one-year and six-month studies, the improvement in lung function was sustained over the treatment period. Smoking status did not influence the improvement in lung function or reduction in exacerbations and the effects were similar and independent of previous treatment with inhaled corticosteroids.[13]

How it Works

Daxas™ is a highly selective PDE4 inhibitor, an anti-inflammatory that is non-steroidal and targets COPD-specific inflammation. The mechanism of anti-inflammatory action of Daxas™ is the inhibition of PDE4, a major cyclic adenosine monophosphate (cAMP) metabolizing enzyme found in structural and inflammatory cells important to the pathogenesis of COPD. The inhibition leads to elevated intracellular cAMP levels, which, in turn, reduces the inflammatory action of cells known to cause the inflammation characteristic of COPD. This includes macrophages, neutrophils and CD8+ T-lymphocytes.[13,15]

Safety and Side Effects

The overall incidence of adverse events was slightly higher in the roflumilast 500 mcg QD group than in the placebo group. Treatment-related adverse events and adverse events leading to study discontinuation were more frequent with roflumilast 500 mcg QD than with placebo. Adverse events leading to deaths were infrequent with no relevant observed differences between treatment arms.[13]

The most commonly reported adverse events among patients taking roflumilast 500 µg were: diarrhea (11.6%), weight decrease (6.8 %), nausea (5.2%), headache (4.6%) and abdominal pain (4.2%). When diarrhea, nausea, and headache were reported, they usually began within the first 4 weeks of treatment and usually resolved within 4 weeks while still on continued treatment. Approximately 70% of the events of "weight decrease" occurred within the first six months of therapy. Overall, approximately 80% of adverse events with roflumilast were mild or moderate and resolved on continued treatment.[12,13]

In the 1-year studies, body weight decreased more frequently in patients treated with Daxas™ than in those treated with placebo. Most of the weight loss (averaging 2.1 kg) occurred in the first 6 months of roflumilast treatment, and after discontinuation of roflumilast, the majority of patients had regained body weight after 3 months.[12,16] In the 6-month supportive trials (M2-127 and M2-128) a similar magnitude of weight reduction (i.e. approximately 2 kg) was observed over the 6-month period.[14]

It should be noted that more than half of the patients in the studies were overweight or obese, with the largest absolute weight loss with Daxas™ occurring in obese patients with a BMI above 30 kg/m^2. Although the mechanism underlying the weight loss is unknown, it appears that that the weight loss is not due to the gastrointestinal side effects and is primarily due to a loss of fat mass rather than a loss of muscle content. In fact, weight loss happened in those with or without gastrointestinal side effects.[12,17]

Drug Interactions

Daxas™ should not be used on its own, but as add-on therapy to bronchodilator therapy.[13]

Daxas™ can be administered without regard to time of day or food intake and may be taken with most other medicines used in the treatment of COPD such as inhaled bronchodilators, inhaled corticosteroids or short-term oral corticosteroids. There are no known clinically relevant drug-drug interactions with medications used commonly in the treatment of COPD (e.g., salbutamol, formoterol and budesonide), or with many drugs used to treat other common comorbid conditions (e.g., antacids, montelukast, midazolam, digoxin, warfarin, sildenafil).[13]

Daxas™ metabolism is not affected by renal status. Daxas™ is, however, contraindicated in patients who are hypersensitive to roflumilast or to any ingredient in the formulation or component of the container and for patients who have moderate or severe hepatic impairment (Child-Pugh B or C).[13]

Where does Daxas™ fit in our treatment algorithm?

Daxas™ is a highly selective PDE4 inhibitor, an anti-inflammatory that is non-steroidal and with a mechanism of action that is different from other available agents used in the treatment of COPD. It adds clinical benefit by reducing moderate and severe exacerbations and improving lung function independent of concomitant therapy with long-acting bronchodilators.[12-14] Daxas™ is a generally well-tolerated and safe medication and although no head-to-head studies have been completed and indirect comparisons between different studies are difficult given the different study populations, the Daxas™ results favourably compare with other COPD treatments in reducing exacerbations.

So where does this new anti-inflammatory that reduces exacerbations fit in? The clinical data needed for physicians to endorse its use in addition to long-acting bronchodilators in patients with severe COPD, chronic cough and sputum and who are prone to exacerbations is evident. Daxas™ may therefore find its role as an alternative to inhaled corticosteroids in more symptomatic COPD patients with frequent exacerbations. In addition, it is also expected that Daxas™ will be added in patients with very severe COPD who are not managing well despite "triple therapy". Although studies have not been done to evaluate the benefit of Daxas™ when added to "triple therapy", Daxas™ has demonstrated additional benefit when added to patients who are taking either concomitant salmeterol or tiotoprium therapy.[14] In addition, in some earlier studies with Daxas™, ICS has been allowed as a concomitant therapy and significant lung function improvements and reduction in exacerbations have been seen irrespective of whether or not patients were taking ICS.[18] This would suggest that the effect of Daxas™ is independent of, and additive to the effect of ICS. However, given there is no published trial demonstrating that Daxas™ has added benefit to "triple therapy", clearly further investigation is needed. In conclusion, the available studies suggest that Daxas™ is beneficial for maintenance treatment of patients with severe COPD with chronic cough and sputum production and a history of frequent exacerbations as add-on to treatment with long-acting bronchodilators. It is likely to be particularly effective in combination with other pharmacological agents used in the treatment of COPD.

Editors Note:

It is nice to have another agent to add to our toolbox to help our patients with COPD. Preventing exacerbations with Roflumilast will help our patients. Perhaps a single daily dosage with a tablet can help adherence, which may be an issue in COPD. Don't forget the other basics of AECOPD prevention, however. Smoking cessation, vaccination, pulmonary rehabilitation, appropriate pharmacotherapy, and aggressive appropriate treatment of exacerbations all combine to help us prevent this costly destructive complication of COPD.

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STOP-BANG

How about a quick office screen for Obstructive Sleep apnea! Use the **STOP-BANG** tool

Snoring loudly

Tired during daytime

Observed Apnea during sleep Having of being treated for high blood

Pressure

BMI >35

Age >50 years

Neck circumference > 40 cm

Gender = Male

3 of 7 criteria are considered high risk

Reference: STOP Questionnaire. *Anesthesiology* 2008; 108: 812-21. Q *Chung* F. Yegnesviaran B. Herrera F. Shenderev A. Shapiro CM.

The FPAGC supports the CMA's 2009 Policy Resolution regarding Asbestos.

The health hazards relating to asbestos exposure have been the subject of media attention recently. The FPAGC supports the CMA resolution below.

CMA Policy Resolution, General Council 2009 re Chrysotile Asbestos: The Canadian Medical Association calls upon the federal government to:

a) support the international designation of chrysotile asbestos as a hazardous chemical;

b) eliminate the use and exportation of asbestos; and

c) support the proper management of asbestos that has been used, including remediation.

Background *(adapted from CMA document on Asbestos)*

Asbestos is the generic name for a variety of fibrous minerals found naturally in rock formations. The three most common varieties are chrysotile, amosite, and crocidolite. It has been widely used in home construction materials, such as floor and ceiling tiles, insulating boards, roofing shingles, water supply lines, plastic fillers, and pipe covers. Asbestos is rarely used alone, and it is generally safe when combined with other materials with strong bonding agents. As long as the material remains bonded it poses no health risk. Asbestos fibres can become loose and airborne, most often when contained in soft, easily crumbled materials or when such well-bonded materials as floor tiles and painted surfaces are cut, scraped, filed, sanded, or removed. Remodelling or demolitions can lead to the release of asbestos fibres.

Although Canada has strict restrictions on the domestic use of asbestos under the *Hazardous Products Act* and the *Canadian Environmental Protection Act*, it is the world's fifth-largest exporter of Chrysotile asbestos to developing countries (*CMAJ* October 21, 2008). Ninety six per cent of the output from the country's two remaining mines, both in Quebec, is for export, primarily to developing countries such as India and Indonesia. (Amir et al, 2008) These countries either do not have or do not enforce health and safety regulations. More than 40 countries, including all member states of the European Union, have banned the use of all forms of asbestos, including Chrysotile.

Health implications

Asbestos poses health risks when fibres are present in the air. When inhaled in significant quantities, asbestos fibres can cause asbestosis (a scarring of the lungs which makes breathing difficult), mesothelioma (a rare cancer of the lining of the chest or abdominal cavity) and lung cancer. Smoking, combined with inhaled asbestos, greatly increases the risk of lung cancer. Because asbestos fibres remain in the body, each exposure increases the likelihood of developing an asbestos-related disease. Asbestos related diseases may not appear until years after exposure. Asbestos has been declared a proven human carcinogen by the US Environmental Protection Agency (EPA) and by the International Agency for Research on Cancer of the World Health Organization. Early indications that chrysotile might be less dangerous than other forms of asbestos have not been supported. The preponderance of scientific evidence to date demonstrates that chrysotile also causes cancer, including lung cancer and mesothelioma. (LaDou et al, 2001)

Exposure to asbestos is the number one cause of workplace-related deaths for Quebec workers and amounts to about 60 per cent of all such fatalities in 2009, according to statistics gathered by Quebec's workers compensation board. (Montreal Gazette, 2009) According to global estimates at least 90 000 people die each year from asbestos related lung cancer, mesothelioma and asbestosis resulting from occupational exposure. (WHO, 2006)

The Politics

In 2002, Canada became a Party to the Rotterdam Convention (RC) whose objectives are:

a) to promote shared responsibility and cooperative efforts among Parties in the international trade of certain hazardous chemicals in order to protect human health and the environment from potential harm; and

b) to contribute to the environmentally sound use of those hazardous chemicals, by facilitating information exchange about their characteristics, by providing for a national decision-making process on their import and export and by disseminating these decisions to Parties.

The RC also creates legally binding obligations for the implementation of the Prior Informed Consent (PIC)procedure.

In 2006, and again in 2008, efforts to make the PIC procedure of the RC apply to Chrysotile asbestos was blocked by a handful of countries, including Canada. The addition of Chrysotile asbestos to the list of Hazardous Chemicals and Pesticides of the PIC procedure would not ban sales or exports of these products but would ensure that importing nations are knowledgeable about the health hazards related to the product they are importing and would require their prior informed consent to receive them.

In 2008, Canada convened an international committee of scientific experts to study the risks of Chrysotile exposure. The committee delivered its report in March and Health Canada has yet to publish it. The Canadian Public Health Association has sent a letter to Prime Minister Stephen Harper recommending that Canada support the listing of Chrysotile as part of the Prior Informed Consent of the Rotterdam Convention. The Canadian Association of Physicians for the Environment wrote to Health Minister Aglukkaq in December 2009 on behalf of numerous organizations, physicians and scientists calling for the end to the mining, use and export of asbestos.

Summary

Asbestos fibre inhalation, especially when combined with smoking, purports a significant health risk and should be avoided. The FPAGC supports the further legislation of this to add to the other health groups with similar concerns.

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You need to know this for your patients!

Canadian Airlines change their Allergy Policies with regards to Peanuts

On December 2nd, 2010 both Air Canada and WestJet made significant changes to their allergy policies based on a recent Canadian Transportation Agency (CTA) decision about nuts on planes.

Air Canada has instituted the minimum required "buffer zone" on their planes. In economy class, this is the row where an allergic passenger sits, plus the row in front of them, and the row behind them.

In business class, it is only the row in which the passenger sits. Within this zone, Air Canada will not be serving any snacks which contain peanut or nut products, and the surrounding passengers will be personally asked to voluntarily avoid eating any peanut/nut products they brought on board.

To qualify for this policy, a passenger must fill out a medical approval form and file it at least 48 hours prior to the flight.

To ask for this buffer zone, you must:

- Get medical approval and fill out a Fitness for Travel Form filled out by your doctor within 10 days of your departure
- Book at least 48 hours in advance by phone, and notify Air Canada that you have a 'Fitness for Travel' form ready to fax in.

Unfortunately, however, Air Canada makes clear that on international flights, even within a 3-row buffer zone, it can't be certain that meals served are nut- or peanut-free, which is something to be aware of! In addition, in its new online policy, Air Canada is adamant that it "cannot offer a meal that is nut-/peanut-free". That being said allergic persons need to bring their *own* food any way as the potential for accidental cross-contamination is too high.

At **WestJet**, which already does not serve snacks or meals containing peanuts or nuts directly (though some snacks "may contain" or may have come into contact with nuts), the buffer zone is expanded to 2 rows in front, and 2 rows behind the allergic passenger. WestJet will personally ask passengers in these rows to avoid eating nut products on the flight. At any point before departure, if the allergic passenger is further concerned about their safety on that flight, they will be allowed to depart the plane and take the next available flight at no cost, or receive a refund.

WestJet has also formed a significant partnership with the manufacturers of Epi-Pens, and Epi-Pen Jr, to put these devices into the first aid kits aboard all aircraft, to ensure that Adrenalin is rapidly available in case of a reaction on board.

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Mission Statement

The Family Physician Airways Group of Canada is committed to helping those with airway diseases lead a full life. The group is dedicated to helping all family physicians maintain and increase their skill in assisting those with asthma and COPD. The strategy of the Group is to maintain a speaker bank, a data base, and practical tools to help physicians attain in these skills.

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