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on the cover:
photos of 2011.

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Asthma and COPD Nursing Course Information

Applications are now invited from registered nurses wanting to enrol in the Asthma New Zealand/Unitec Asthma Nursing Course for February 2012 and COPD Nursing Course for April 2012. The programmes are offered by distance learning. The primary aim of the Asthma and COPD Nursing Courses are to provide nursing health professionals with a high level of evidence-based asthma and COPD knowledge that promotes best practice and is consistent with national policy.

Since the commencement of the Asthma and COPD Nursing Courses, 890 nurses have enrolled over 35 intakes. Many applicants had not undertaken any additional study since completing their nursing training, which may have been years before. However, most find the courses to be challenging but thoroughly enjoyable learning experience that is within the grasp of any competent nurse practitioner.

Asthma New Zealand in association with Unitec New Zealand offers these courses within the Bachelor of Nursing Programme. Asthma Nursing Course is a level 7 course and attracts 24 credits. COPD Nursing Course is a level 7 course with 12 credits. **A grant towards the cost available for registered nurses.**

For an enrolment form and information for the 1st Semester please contact:

Ann/Swarna

Asthma New Zealand/The Lung Association

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Fax 09 623 0774

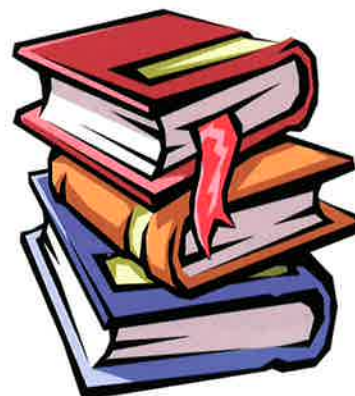
Email annw@asthma-nz.org.nz

swarnah@asthma-nz.org.nz

The closing date for 1st semester enrolment is

10th February 2012 for Asthma

10th April 2012 for COPD



Upcoming events and courses

ASTHMA NEAT COURSE

21 March 2012

20 June 2012

19 September 2012

HALF DAY COPD COURSE

18 April 2012

18 July 2012

17 October 2012



message to readers

Another year is almost over. It has been in general terms, a stressful year given all that has happened throughout the world. In general terms the world is in recession, however New Zealand has responded well to the challenges and is probably in a better position than many other countries. That does not mean however that life is any easier in New Zealand. Funding has not increased and the work load is greater than ever.

As indicated in an earlier magazine, Asthma New Zealand – The Lung Association (Inc) met with the Asthma Foundation in Wellington to discuss the likelihood of creating one body for asthma within New Zealand. A full day meeting was held in Wellington between the Board of Asthma New Zealand and the Asthma Foundation. The challenge for both Boards is to discuss the possibility of creating one organisation for people with asthma and their families across New Zealand. There are many challenges involved in this suggestion. Asthma New Zealand is the primary educator with over nine hundred nurses successfully completing the Asthma and COPD Courses through the Unitec Institute of Technology in Auckland. This will provide a tremendous educational resource throughout General Practice for people with asthma and their families. It is important that people understand how to manage their asthma in order to achieve a quality lifestyle and not be limited in any way. The Courses continue to be over-subscribed and it demonstrates the commitment of nursing personnel at GP and hospital level.

I would like to take this opportunity of thanking you all for your support over the past year and to wish you and your families every blessing

at Christmas and the hope that next year will be successful and a fulfilling one for you all.

Kind regards

Gerry A. Hanna
Secretary/Treasurer
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the health effects of tobacco smoke exposure on children

Compiled by Elaine Murray RN

Asthma Nurse Educator

The 2006 U.S. Surgeon General's Report, "The Health Consequences of Involuntary Exposure to Second hand Smoke", has concluded that there is no safe level of exposure to second hand smoke and that, on average, children are exposed to more second hand smoke than adults.



Children are significantly affected by second hand smoke. Children's bodies are still developing, and exposure to the poisons in second hand smoke puts them at risk of severe respiratory diseases and can hinder growth of their lungs. Second hand smoke is associated with low birth weight, Sudden Infant Death Syndrome (SIDS), asthma, bronchitis, pneumonia, middle ear infection, and other diseases.

It is estimated that around 1.3 billion people world-wide smoke, and the number is predicted to increase in the coming years as smoking rates continue to increase among youth, primarily among young girls.

Involuntary and avoidable exposure of children to tobacco smoke can begin before birth and continue throughout childhood. Active smoking by the mother exposes the developing foetus to agents in tobacco smoke that cross the placental barrier. Circulating nicotine from the mother's blood has been shown to reach the foetus by crossing the placenta and also reaches the foetus through the blood. This decreases the amount of oxygen and nutrients supplying the foetal tissues. This may have an effect on cell growth and development. Although foetal hypoxia and ischemia are the major contributors to developmental lung defects, nicotine has been implicated in various studies to have a direct effect on foetal development by impairing the maturation of the lung function. There is compelling evidence that maternal smoking reduces lung function in young children and that the effect is present at birth and attributable to effects of maternal smoking during pregnancy and early postnatal exposure on the child's lung development.

Prenatal smoking increases the risk of asthma, and postnatal maternal smoking is associated with increased incidence of wheezing, an increased risk of development of asthma and worsening of pre-existing asthma.

These associations of in utero exposure with respiratory outcomes are consistent with the evidence that in utero exposure adversely affects post natal pulmonary function and increases the occurrence of respiratory symptoms. Furthermore, in utero exposure may affect the development and maturation of the pulmonary immune system. Inappropriate persistence of a Th2-dominant response appears to increase allergic sensitisation upon sufficient exposure to a variety of common antigens that underlie the pathogenesis of asthma.

Babies born to mothers who smoked during pregnancy have been shown to have increased risk of wheeze (40%), increased risk of asthma (83-246%) and increased prevalence of airway hyper-responsiveness.

Children are particularly vulnerable to many environmental threats, including a contaminated and unsafe physical environment. This heightened susceptibility derives primarily from the unique biological features that characterise the various stages of development from conception to adolescence. Cell growth is very rapid in the embryo. Lung growth occurs in the foetus with the help of interactions of various chemical processes. Exposure to toxicants which enter via the umbilical cord in mothers exposed to tobacco smoke may interfere with lung growth and may also increase the risk of causing cell mutations and congenital anomalies. Although it was earlier believed that the placenta offers a barrier for the passage of these toxic chemicals, it is no longer

true, since several studies have shown the presence of various toxic chemicals circulating in the umbilical cord.

Children's metabolic pathways, especially in the first months after birth, are immature and still developing. As a consequence of this immaturity, children's ability to detoxify and excrete chemicals differs from that of adults, making them more vulnerable. Children also inhale greater volumes of air per kilogram body weight than adults, means that they inhale greater amounts of air pollutants than adults. Moreover, the size of harmful particulate matter <1µm is just right for it to enter even up to the terminal airways and alveoli.

Another factor is that children often sit closer to parents and family members, and are therefore closer to the source of pollutants than other passive smokers.

During childhood, the lung completes its development as formation of the alveoli is completed during the first few years of life and lung function grows, in parallel to the increase in height. Thus, damage to the lung during childhood may have lasting effects and compromise the lungs reserve capacity. Exposure to environmental tobacco smoke during the school years has now been shown to reduce the rate of lung function growth.

Asthma susceptibility has a large genetic component, and it has been firmly established now that lifestyle and interactions between the genetic make-up and environmental exposures, such as parental smoking, determine its development.

Studies show that a child with asthma has more frequent and more severe asthma attacks if exposed to cigarette smoke. In fact, children with asthma whose parents smoke at home are twice as likely to have asthma symptoms all year long as children of non-smokers. These children are more likely to attend a hospital emergency department with asthma and their recovery time is slower after being in hospital.

In New Zealand in 2006, around 22 percent of pregnant women reported they smoked while pregnant.

Quitting smoking before or during pregnancy, and avoiding exposure to second-hand smoke, will have a positive impact on the health of both the mother and the unborn baby. It will also reduce the likelihood of related health problems for the child after birth.

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dear nurse column

Introduction by Karen Little RN

The five Asthma Nurse Educators at Asthma Auckland spend most of their time in the community.

We all encourage our patients to ask any sort of questions. In fact we often ask at the beginning of our education session what their most pressing questions are so we can tailor our session around them.

Many clients have received conflicting information on their asthma or Chronic Obstructive Pulmonary Disease (COPD). We all try to reply to questions so clients understand the reasoning behind the answer. For example if we just instruct people to wash their spacer in hot soapy water without rinsing or drying there is the possibility that this may not happen. However if we demonstrate static electricity by rubbing a plastic bag on our hair (with the subsequent hair raising effect) people can start to understand that the static electricity generated by the plastic spacer rubbing on things will attract the medication to the sides of the spacer and not into the lungs where we want it. This is also the reason why we advise people not to keep their spacer in plastic bags.

There is no question about asthma or COPD that is too small or too complicated to ask us. If we are unsure about complex questions we discuss it amongst ourselves and research it if required. In fact we love questions and all enjoy expanding our knowledge and debating the different theories and urban legends that abound in our community.

We are going to start including in our magazine a question and answer column and welcome you to email us at editor@asthma-nz.org.nz or post to Dear Nurse, Asthma New Zealand, PO Box 67 066, Mt Eden, Auckland 1349 or phone us on 09 630 2293. Remember nothing is too insignificant to ask us. If you have been wondering about something the chances are that other people have been also.

To start things off these are a few questions we have been asked this week.

Dear Nurse why can't my child keep using the spacer mask?

The nose is there to filter and warm the air entering our lungs. That is what the nose hairs are there for. If a child nose breathes through the mask some medication will get stuck in the nose and not into the lungs where we want it. We start teaching children in a fun way from about the age of three to use the spacer without the mask. We always advise the parents however not to throw the mask away as it is useful in an emergency and to administer Ventolin if the child is



sleeping. We do not make an issue out of it as the most important thing is that the child has the medication as prescribed, the process of switching from a mask if they are used to it may take some time.

Dear Nurse can I stop my child's asthma medications as I am now using the Buteyko Method to control his asthma?

As explained in the last answer it is important to mouth breath when using a spacer with a mask. It is fine to use Adjunct Therapy (a drug or other substance that serves a supplemental purpose in therapy) as long as it does not interfere with the prescribed asthma medications. Relievers and preventers are essential in managing your child's asthma so do not discontinue them. Buteyko helps in controlling breathing patterns but will not assist the underlying inflammation.

Dear Nurse will my asthma go away?

Simple answer is no, symptoms of asthma may be reduced if the asthma trigger has been removed, therefore eliminating symptoms of asthma. Please see the article in this edition "Can you grow out of asthma?"

Dear Nurse what are triggers?

Triggers are substances that may trigger asthma. They can be allergy related (e.g. dust mites, mould or pets) or non-allergy related (e.g. respiratory infections, exercise second-hand smoke, chemicals and fumes. Triggers are different for everyone.

IF YOU HAVE A QUESTION PLEASE EMAIL OR POST TO:
editor@asthma-nz.org.nz or Dear Nurse, Asthma New Zealand,
PO Box 67 066, Mt Eden, Auckland 1349.

Heartiest Congratulations on successfully completing Unitec / Asthma Nursing Course 2011 – 1st semester

Megan Jane Asplin _____ Palmerston	Kylie Donna Hoskin _____ Upper Hutt
Deborah Aynsley-Hunt _____ Waikaka	Karen Little _____ Auckland
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Janet Delooze _____ Auckland	Kate Riddell _____ Masterton
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Michelle Nichola Fields _____ Tauranga	Amanda Lee Turnbridge _____ Kapiti Coast



definition of asthma

Compiled by Sharron Daniels Asthma Educator RN BN

Asthma is a common condition that affects the airways. The typical symptoms are wheeze, cough, chest tightness, and shortness of breath. Symptoms can range from mild to severe. Treatment usually works well to ease and prevent symptoms. Treatment is usually with inhalers. A typical person with asthma may take a preventer inhaler every day (to prevent symptoms developing), and use a reliever inhaler as and when required (if symptoms flare up), (Asthma Management Hand Book 2006).

Day in the life of a respiratory educator

With a background as a Practice Nurse I thought the transition to an Asthma Educator, would be quite straightforward. I had, after all, previously run nurse- led asthma clinics within a GP setting. There are, however, some interesting aspects to the Asthma Educator role. Some are very familiar and some not so.

The first challenge I faced when I began this role was actually getting to the client. Asthma Auckland covers a huge area, from Wellsford in the north to Tuakau in the south and all points in between. Not a problem. With a GPS on board providing directions what could possibly go wrong? Unfortunately, a GPS only works if you actually follow its directions. I found out very quickly that ignoring the GPS because you think you know better is a great way of getting lost. Of course, it wasn't always my fault. Sometimes the road is so new even the technology is stumped. Just a quick thank you to the person who approved the GPS updates recently.

Once I have arrived at my destination and open the gate to gain entrance on the property, there appears to be a very unfriendly looking dog showing lots of sharp teeth to greet me instead of my client. A



quick check on my appointment notes tells me A) Where to park and B) Any dogs that I should know about.... Only to be told, 'don't worry about Buster, his barks worse than his bite'. Hmm does Buster know this?

Part of the education we offer is to discuss and explain the differences between preventers and relievers and the importance of why both types of medications are required for effective management of asthma symptoms (Epton, Town, Ingham, Wickens, Fishwick, & Crane, J. (2007). It still surprises me how often I find that individuals either forget to use their preventers regularly or are so confused they aren't sure which medication is which. Note to self. Start with the basics. Don't assume.

Next step, always ask to see their medication, check expiry dates and that their metered dose inhalers are not empty. Each will have an expiry date printed on it. On occasion when asked if I could see the client's medication I have had a response such as, "I've lost them" or "I've forgotten to collect another script" or "My sister has taken it to work because she has lost hers." Some are removed from a pocket with the lid off and with the chamber full of debris. I have even seen them amongst a collection of children's toys.

Let's say, for arguments sake, that as a person who has asthma and you are experiencing shortness of breath, wheezing, tight chest and coughing. You need to use your reliever. By the time you have located it, you are becoming worse, becoming stressed now because you can't find your reliever. When you do find it, you inhale debris/dust along with

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the medication and suddenly you require the Heimlich manoeuvre! Actually this is not funny but could possibly be a reality. If you don't understand the environment your client lives in you simply can't have these conversations.

Some individuals forget the reason for using a preventer. Asthma Auckland recommends using preventer's every day, morning and night, even when well. Why? Because the inhaled corticoid-steroid is only effective for twelve hours and there is twenty four hours in one day. These preventers work quietly in the background to help with inflammation and secretion build up in someone who has asthma (Haynes, Ackloo, Sahota, McDonald, & Yao, X. 2008). By ensuring that our clients are using their preventers regularly we can achieve the following.

- Avoid troublesome symptoms night and day
- Minimise reliance on reliever medication
- Have productive, physically active lives
- Have (near) normal lung function
- Avoid serious attacks

GINA GUIDELINES 2010 obtained from <http://www.ginasthma.com/Committees.asp?11=7&12=2>

An asthma educator emphasises identifying possible triggers for asthma, incorporating the use of care plans and action plans. Our goal is to reduce exacerbations and distress to the client and the equally unnecessary calls on the ambulance service. By emphasising effective management we avoid our hospitals becoming over burdened with respiratory conditions as recently reported in our national newspapers.

I have worked with client's to ensure that they know what medications to use and when and I have ensured that they can access their medication as required. The next step is to review the technique and delivery of their various asthma medications. The use of a spacer and effective inhalation techniques can significantly increase the amount of medication reaching the lungs as well as decrease side effects and can make a significant contribution to their well-being.

Last but not least, when seeking asthma education from Asthma Auckland, please remember to advise the educator visiting of any hazards to assist them when they visit.




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Note to self; bring along a nice juicy bone for Buster...

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influence of obesity on the prevalence and clinical features of asthma

Compiled by Sharron Daniels
Nurse Educator, RN BN

What came first, the chicken or the egg? It could be said that asthma may promote weight gain because of the sedentary lifestyle that can occur from chronic breathing disorders. It doesn't help that some medications, such as corticosteroids are known to contribute to weight gain. Common genetic factors could also be part of the equation. There does appear to be a basis for genetic predisposition to both asthma and obesity. Is there a link? What can be verified is that obesity is more prevalent amongst those with chronic breathing disorders such as asthma and that obesity results in poorer control of the associated symptoms.

Obesity can possibly influence the development of asthma through genetic, developmental, hormonal, neurogenic or mechanical influences.

The mechanisms by which obesity influences airway function and, subsequently, makes asthma more difficult to control remains unclear. Studies have shown that having a high body mass index is linked to a strong positive association with risk of adult-onset asthma.

Studies demonstrated that individual's whose BMI is greater than 25kg/m were significantly more likely to display breathing disorder symptoms. With regard to alterations in lung function, obesity can be associated with a restrictive pattern, a mild reduction in total lung capacity (TLC), altered pattern of breathing, higher breathing frequencies, reduced tidal volumes or, most commonly, reduced expiratory reserve volume (ERV).

Obesity is known to affect the lungs and is very strongly associated with breathing disorders. This is due, in part, to the mechanical effect of central body fat on the airways. Excessive body fat restricts the free movement of air and compresses the lungs. Not only does excessive body fat restrict normal lung movement, research shows a reduction in deep breathing associated with obesity and a sedentary lifestyle that may also lead to narrowing of the airways. Obesity results in a restrictive lung pattern which limits deep breathing and results in a reduction of bronchial smooth muscle expansion.

Even modest weight loss has been universally found to improve asthma control and reduce its severity. Weight reduction from diet and exercise has been found to improve asthma symptoms, reduce peak expiratory flow variation and improve spirometric values.

Children and young adults who live with asthma are generally less active than their non-asthmatic peers, narrative findings believe this is due to lack of understanding in the management of asthma as even caregivers and educators can be confused as to what is considered an acceptable level of activity for those living with asthma. The biggest barrier to maintaining weight for those living with asthma appears to be belief. Both children and their parents simply believe that there are limitations to what they can do simply because they have asthma. This is often exacerbated by educators who do not understand how to manage asthma or do not have protocols in place to enable them to understand and manage the signs and symptoms of asthma when they occur.

The mere fact of asthma should not prevent an individual from participating in normal exercise regimes.

How can obesity contribute to the development of asthma?

The main goal of asthma is to achieve optimum wellness and adequate control of the disease. However evidence supports the fact that a person with high BMI levels hinders the response to inhaled corticosteroids (ICS) and long acting bronchodilators (ICS). This has been universally proven that weight loss however modest, may improve asthma control and reduce its severity. Weight reduction from diet and exercise has been found to improve asthma symptoms, reduce peak expiratory flow variation and improve spirometric values.

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ASp PHOTOGRAPHY

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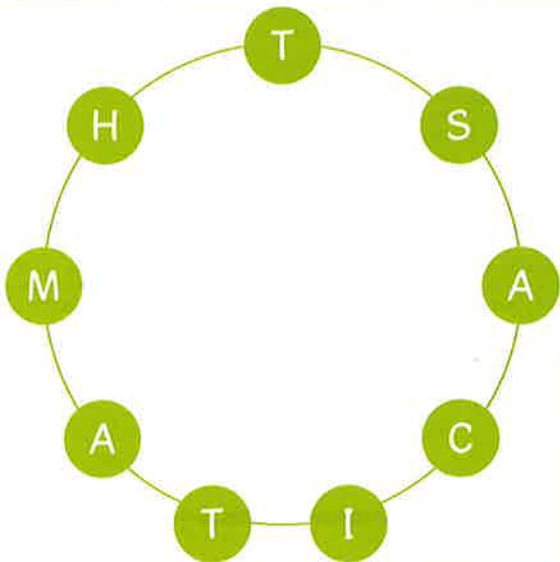
Kid's Page



Find A Word

C	L	O	X	Y	G	E	N	Y	U	R	W	E	L	L
C	H	J	R	B	L	C	X	R	M	E	Z	M	A	A
Z	L	E	B	P	E	R	U	E	A	Y	N	U	O	R
M	Z	E	M	C	L	U	L	M	L	R	O	F	C	U
R	F	A	A	I	M	O	F	A	E	D	I	S	L	R
O	S	R	A	N	C	S	N	Z	B	A	T	E	B	P
S	T	Y	E	X	H	A	U	S	T	S	S	C	O	R
N	K	O	S	P	B	W	L	Z	U	E	U	U	D	E
E	N	G	I	N	E	R	B	O	I	K	B	T	R	T
S	P	O	L	L	U	T	E	D	G	Y	M	O	N	E
O	Z	O	N	E	S	S	R	A	C	A	O	U	G	M
G	C	W	E	U	A	U	M	N	T	N	C	U	T	K
S	M	O	G	G	O	K	E	A	S	H	A	B	R	C
E	F	F	E	C	T	G	D	A	I	G	E	E	I	U
K	C	N	S	T	A	C	K	S	M	W	I	N	D	S

- | | |
|--------------|------------|
| 1 Agency | 19 Gust |
| 2 Analyze | 20 Labs |
| 3 Breathe | 21 Leak |
| 4 Chemical | 22 Measure |
| 5 Clean | 23 Meter |
| 6 Coal | 24 Mist |
| 7 Combustion | 25 Oxygen |
| 8 Data | 26 Ozone |
| 9 Diesel | 27 Pollute |
| 10 Dirt | 28 Rural |
| 11 Dryer | 29 Sample |
| 12 Effect | 30 Scour |
| 13 Engine | 31 Sensor |
| 14 Exhausts | 32 Smog |
| 15 Flux | 33 Source |
| 16 Fume | 34 Stacks |
| 17 Gaseous | 35 Trace |
| 18 Gauge | 36 Winds |



Make as many words as you can of three letters or more, I found 40, see if you can beat me! There is one word that uses all the letters...

Answers bottom of page but no cheating!

S	D	N	I	N	W	M	S	K	S	A	C	S	T	A	S	N	K	C	N
E	F	E	C	T	G	D	A	I	G	E	E	I	U						
S	M	O	G	G	O	K	E	A	S	H	A	B	R	C					
G	C	W	E	U	A	U	M	N	T	N	C	U	T	K					
O	Z	O	N	E	S	S	R	A	C	A	O	U	G	M					
S	P	O	L	L	U	T	E	D	G	Y	M	O	N	E					
E	N	G	I	N	E	R	B	O	I	K	B	T	R	T					
N	K	O	S	P	B	W	L	Z	U	E	U	U	D	E					
R	F	A	A	I	M	O	F	A	E	D	I	S	L	R					
O	S	R	A	N	C	S	N	Z	B	A	T	E	B	P					
S	T	Y	E	X	H	A	U	S	T	S	S	C	O	R					
N	K	O	S	P	B	W	L	Z	U	E	U	U	D	E					
E	N	G	I	N	E	R	B	O	I	K	B	T	R	T					
S	P	O	L	L	U	T	E	D	G	Y	M	O	N	E					
O	Z	O	N	E	S	S	R	A	C	A	O	U	G	M					
G	C	W	E	U	A	U	M	N	T	N	C	U	T	K					
S	M	O	G	G	O	K	E	A	S	H	A	B	R	C					
E	F	F	E	C	T	G	D	A	I	G	E	E	I	U					
K	C	N	S	T	A	C	K	S	M	W	I	N	D	S					

Answers - How many words?
 ASTHMATIC, CHASM, MATCH, MASTIC, MASTIC, STITCH
 STATIC, ATTICS, SCAM, MATHS, TACTS, CHATS, MITTS, ATTIC, CHAT,
 AMAH, MASH, TACT, ITCH, SHAM, CHIT, CASH, STAT, MITT, MATT,
 MATS, AIMS, CAST, MAST, ACTS, HITS, HATS, THIS, THAT, HIT,
 HAT, MAT, SAT, SIT

Answers - Find a Word

can you grow out of asthma?

Compiled by Karen Little RN

This is one of the most common questions that asthma nurse educators are asked. Parents will also inform us that they only had 'childhood asthma'. Many people are unaware that years can go by without asthma symptoms and that asthma can be diagnosed at any age.

There is convincing evidence that childhood asthma constitutes a spectrum of different phenotypes with different prognoses. It has also recently become clear that children who progress to develop persistent wheezing beyond infancy and early childhood usually have a family history of asthma and allergies and present with allergic symptoms very early in life (1).

Atopy, defined as positive skin test reactivity to inhaled allergens, is present in the majority of patients tested, especially children with asthma. Atopy has not been shown to be an independent determinant of prognosis with regard to lung function, but it has been shown to be a risk factor for relapse of asthma after remission. It has been associated with current symptoms, persistence of symptoms and late onset symptoms in children (2).

Allergy and atopy while primarily genetically determined are also dependent on exposure to certain environmental factors. Although they are clearly shown as risk factors for the development of persistent asthma in infants and young children, available findings are far from conclusive that they contribute to the risk profile for developing adulthood asthma. This supports the concept that once asthma has become established in the child, inflammation appears to run its own course, irrespective of the subject's atopic status (3).

With respect to gender, available data in the literature are consistent in showing that the male predominance in asthmatic children gradually diminishes, disappearing after puberty. After the age of 20 years, asthma incidence and severity are greater in females than in males, suggesting that prognosis in girls is worse. In addition hormonal differences may potentially influence airway inflammation and airway smooth muscle function (4).

A study that included one hundred and fifteen children with asthma in Ankara, Turkey regularly followed up between 1990 and 2000 documented some interesting percentages. At the end visit 27% were in complete remission of asthma and a further 26% were in clinical remission. Approximately 30% and 28% reported asthma symptoms and use of short-acting beta-2 agonist, respectively, within the last year. Approximately 26% were on regular prophylactic medication (5). This study also showed that high blood eosinophil count, an indirect marker of airway inflammation, was predictive for the persistence of symptoms as well as for the presence of airway hyper responsiveness. There was no predictive childhood characteristic for complete remission during early adulthood. This study monitored lung function throughout the study period and demonstrated that patients with reduced airways and/or obstructive pattern during childhood predicted the presence of obstructive pattern and decreased airflows in early adulthood respectively.

The Melbourne Epidemiological Study of Childhood asthma (6) was a large longitudinal long-term study. The children were first assessed when they were 7-10 years old and were last assessed at 42 years of age. This study found that the pattern of asthma during childhood is a strong predictor of outcome. Of the children with episodic asthma (wheezing occurring only with respiratory tract infections) only 33% still had episodic asthma. Of the children with persistent asthma (wheeze not associated with infection), 89% continued to have asthma symptoms. The severity of childhood asthma has also been shown to be a prognostic marker for lung function later in adulthood.

The study showed that at the age of 42, lung function in those with episodic asthma did not differ significantly from controls. In contrast, subjects in the persistent asthma groups had significantly reduced lung function.

As asthma nurses we are reluctant to assure parents that their children will grow out of asthma. When I see year 13 students in schools, I always give them my "leaving home" speech. Even if they have had no symptoms for years I explain that if they are leaving home to go to university or going overseas there is always a possibility that they may come into contact with a new trigger that may cause asthma symptoms. Student flats, mould, dust and respiratory infections are included in these triggers.

A positive fact, however, as previously discussed, is that if your child has episodic asthma and there is no history of atopy the chances are good that they will leave asthma behind in childhood.

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deserving auckland children re ne



Sophie Hartnoll (centre) above.



Each year Variety – The Children's Charity connects with more than 10,000 Kiwi kids, their families and communities. Since 1989 Variety has given over \$13 million to help sick, disabled and disadvantaged children.

As part of their annual Bikes For Kids Tour, Variety presented a bike and helmet to deserving children throughout New Zealand.

"Bikes For Kids" is an initiative within Variety's Kids On The Move programme which provides life-enriching assistance to Kiwi kids.

Variety aims to fill the gaps that other organisations can't or won't fill. Throughout the year, they call for nominations of children who are seen by their peers as a role model or who have made a difference within their school or community environments. Other nominations have been from families who are simply not in a position to purchase a bike for their child or who have a child who has overcome adverse circumstances.

This initiative encourages young people to aspire to achieve their goals and recognises those achievements through the presentation of bikes.

Sophie Hartnoll was nominated to receive a bike by Asthma New Zealand for being an outstanding role model and for her contribution as part of "Team Logan" raising over \$20,000 for Asthma after losing her younger brother Logan, then aged 4, to Asthma in 2010. I felt so proud as I watched Sophie receive her bike and absolutely reiterate the words spoken on the day "these bikes are not a gift, you have earned them, by being an outstanding person in your community in some way" Sophie was among 450 children who received bikes nationwide as part of Variety's 21st anniversary celebrations.

Thank you Variety for the great work you do.

Linda Thompson
PR / Marketing Manager
Asthma New Zealand



north & south

NEWS FROM AROUND THE REGIONS ...

ceive
w bikes



varietyTM
the children's charity





cleancorp

Are you sleeping with the enemy?



DUST MITES AND ALLERGEN CONTROL

Common house dust is a major cause of asthma. Dust mites are prevalent in house dust and Auckland is a perfect breeding ground for dust mites where humidity is above 80% all year.

The bedroom is particularly vulnerable as we spend one third of our life in bed. During the night we perspire and shed skin scales. With every movement of the body in the bed the dust mite excrement is churned up and absorbed by inhalation or by skin contact. The mattress acts as a bellows blowing the dirt into the mouth, nose, eyes and onto the skin.

Mite excrement contains “guanine” which causes an allergic reaction in humans such as inflammation of the skin, eczema, itching, inflammation of the mucosa membrane particularly in the nose, reddening of the eyes, coughing and asthma attacks.

As dust mites also live in your carpets to maximize effect carpets should be cleaned at the same time as your mattress.

3 STAGE CLEANING AND SANITISING PROCESS OF YOUR MATTRESS, CARPETS AND UPHOLSTERED ITEMS

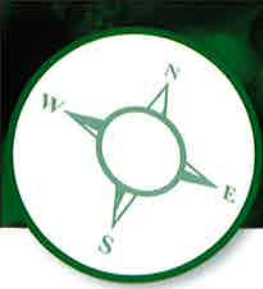
- 1 A special HEPA filter is used to ensure all the dust mites and faeces etc are trapped and not re-circulated in the room. The mattress cleaner generates high frequency waves that loosen particles of dirt in the mattress. It pulverizes them and eliminates them with a vacuum that is adjusted to suit the mattress.
- 2 Intense UVC radiation functions bombard your mattress, carpets etc exterminating bacteria, viruses, dust mites and fungal spores.
- 3 “Anti Allergen Spray” is an intervention where the feces are neutralized through contact with the extract and further protects by neutralizing allergens for up to 35 days.

ANTI ALLERGEN SPRAY

The anti allergen has grapefruit seed extract proven to be effective in killing dust mites. It works as an intervention by instantly de-activating the allergen in house dust mite droppings. The sanitizing spray limits growth of dust mites and also moulds.

This process is safe for both humans and your pets and does not damage fabrics and should be completed every 6 months or every 3 months for very serious sufferers.

“Our carpets and bedding have been treated by Cleancorp and we have had no asthma or allergy symptoms since” — Dean and Kirsten Hartnoll



north & south



sail away with koru care

Koru Care Charitable Trust always gets into the Christmas spirit!

Koru Care held its annual Christmas Cruise, which went on a cruise in the Auckland harbour and they had a party/BBQ at a local beach.

They took 250 children, aged from 6 to 14 years that suffer from a serious illness, or have suffered some sort of major trauma, or are from a disadvantaged background.

They also invite other children from the same family along for the day, as often they miss out on all the fun.

We sent 7 children from Asthma Auckland along for the day and we heard they had a fantastic time. Congratulations to the Robinson Twins, Connor and Caitlyn aged 10, who took out first prize in the Karaoke Competition!

Koru Care Charitable Trust does a fantastic job and we would like to acknowledge their good work and thank them for everything they do!



parent & child show

30 September – 2 October 2011

The parent and child show is New Zealand's most popular parenting event and it provides Asthma Auckland with a marvellous opportunity to provide advice and education to members of the public that we may not have otherwise seen.

Asthma is a chronic inflammatory condition of the airways. There is no cure, but it can be managed successfully.

Asthma Auckland's mission is to provide information, education and support to people with asthma and their families, to assist them to lead a life as free from symptoms of asthma in order that they might lead as normal a lifestyle as possible.

Over the three days we not only spoke to many people with asthma, but to lots of people who knew someone with asthma. A high number of people perceived their asthma as controlled, but after speaking with a nurse they realised that their asthma was not well controlled and they were advised to speak with their G.P.

As we are a Non-profit Organisation, we are grateful for the grants and donations which enabled us to hire the space for our stall and provide a service for the wider community.

A bad dose of hayfever nearly prevented Veronica Johnston and her husband David Ishii from attending this year's Parent and Child Show. But she went anyway and luckily stopped by the Asthma New Zealand stand to find out more about asthma as her 10-year-old nephew has just been diagnosed with it. She also handed over a gold coin donation and entered the draw to win the prize we were offering. It turned out to be a lucky gold coin as her name was drawn from around 1000 entries. Veronica and her family would like to say a big thank you to Asthma New Zealand and all of our sponsors.

Elaine Murray
Asthma Nurse Educator

Call for sponsors and exhibitors!

Email: info@adpn.org.nz

the b!g event

Health and Disability Expo 9-10 March 2012 ASB Showgrounds

Call for sponsors and exhibitors!

Email: info@adpn.org.nz

Explore the latest health and disability products, services, technology and resources. An incredible educational experience, great networking opportunity and most of all fun, fun and more fun!

Features include assistive and adaptive devices, durable medical equipment, computer software, mobility products, adaptive learning tools, disability law resources, recreation and travel, adapted bikes and exercise equipment, personal care products, communication devices, products for the visually and/or hearing impaired, local agencies and support groups, product demonstrations, entertainers, raffles and giveaways.

Free entry and free parking

Friday-Saturday 9-10 March 2012, ASB Showgrounds, Greenlane

This event is proudly bought to you by the ADPN EXPO Trust. info@adpn.org.nz





wellington region



At Asthma Wellington we are once again getting out and about making our presence felt in the community. In this last part of the year our two Asthma Nurse Educators, Adie Riddell and Patricia Sullivan have put their focus on pre-schoolers and have been busy carrying out educational sessions with PORSE and local day care centres. These have been very well received and are usually very interactive, giving both parents and teachers the opportunity to ask questions in general about asthma and in some cases very specific questions with regards to their children. One of the common issues that seem to come up repeatedly is regarding medications being locked away until needed, they are often exceeding their use by date; these need to be checked regularly and kept current.

We recently acquired a half hour slot on Samoan Pacific Radio talking about asthma, who we are and what services we offer, which we are hoping will be repeated on a regular basis. This is aimed specifically at the Pacific Islanders in the community, with different days featuring a different language and being translated into such with the help of an interpreter. We are hoping through this to reach a greater number of people we are not currently gaining access to.

We are looking to carry out some public awareness campaigns in local supermarkets and shopping malls throughout the Wellington area so look out for our big blue and purple flag and support us by buying a raffle ticket or two!

We received a fairly good response to our latest membership drive – a huge thank you to all of you who supported us by updating your membership and an even bigger thank you to those who also donated more than the \$25. As always it is greatly appreciated. We could not carry out the level of service that we do without your generosity and support.

Our nurses are available to do free home visits for asthma education. If you wish to make a referral to our service we can be contacted by phoning 04 237 4520 or by faxing a referral through to 04 237 4254.

We also have nebulisers for sale or for weekly rental. Contact Kim for more information on 04 237 4520.

Once again, Christmas is fast approaching and we will be closing for a couple of weeks, for a well deserved break from 22nd December and will be back in action 9th January. We would like to take this opportunity to wish you all a safe, joyous and happy Christmas and New Year.

Best wishes
Kim, Adie and Patricia

in a beautiful set of ecoLinen sheets!

These premium organic cotton sheets by ecoLinen are used in top resorts all around the world. They are made from high quality naturally matured 100% certified organic cotton which is spun into a thick more absorbent yarn, and then woven into beautiful thick crisp sheets that breathe and draw moisture away from the body. They are long lasting, wrinkle resistant and feel soft and smooth to the touch. Perfect for allergy sufferers and children's sensitive skin.

To enter to win send your name and address on the back of an envelope to Asthma NZ / ecoLinen Competition, PO Box 67 066, Mt Eden, Auckland 1349 before 29 February 2012.





asthma canterbury – what's new?

At Asthma Canterbury we have been very busy with new client consultations this year. Over the winter period many clients struggled to achieve good asthma control due to living in earthquake damaged homes that were damp and mouldy. The two major snowfalls during winter also made things more difficult. We hope that repairs to clients homes can be undertaken soon so that this issue can be largely resolved before next winter. At the end of October our nurses provided a stand at the Gluten Free Food & Allergy show. This proved very successful with over 160 people visiting the stand and receiving advice and information over the two days. Our nurses also provided a 30 minute talk on asthma triggers as part of the show seminar series.



Asthma Canterbury Committee

Left to right back row – Dr Simon Causer, Dr Phil Hamilton, Nigel Riley.
Front row – Teresa Chalecki (Manager), Sarah Numan (President), Pamela O'Brien (Vice President), Lesley Larkin (Secretary).

Absent – Gavin Shepherd (Treasurer).



Following the February earthquake the location of our old premises at 275 Cashel Street was no longer ideal for clients or visitors and also had sustained some damage. In late August Asthma Canterbury moved to new premises and our new location is in Unit 1, 6 Raycroft Street. The two level unit is painted red and green and is easy to find. Client and visitor parking is available at the front of the building. Our telephone, fax number and postal address remain the same.

Our new location is more accessible and we have had more clients and visitors since the move than we received in the six months post February. While approximately the same size as our old location the new premises have a more flexible lay out and we can now

accommodate small group education on site. This worked well for the Asthma Fundamentals course for Nurses we ran early in November.

The Asthma Canterbury Committee (pictured above) would like to thank Asthma New Zealand for the financial assistance they provided to us to support our move.

We are expecting another busy year in Canterbury in 2012 but hopefully without any more major upsets from mother nature.

Teresa Chalecki
Manager

world copd day



**World
COPD
Day
2011**

November 16 2011

world copd day 16 november 2011

World COPD Day is an annual event organised by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) to improve awareness and care of Chronic Obstructive Pulmonary Disease (COPD) around the world.

“COPD is highly prevalent, underdiagnosed, undertreated and under perceived”¹

So, what is COPD?

COPD is a chronic respiratory condition presenting as air flow limitation that is not fully reversible. The air flow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases.

The most common cause of COPD is cigarette smoking or exposure to tobacco smoke. This irritates the lungs and causes irreversible damage.

Many patients present to their doctor late in the disease and it not uncommon to have serious impairment of lung function at the time of prognosis.

Early detection is therefore very important.

A spirometry test is necessary to confirm diagnosis and to establish the severity of the disease.

Asthma Auckland held an open day at our office at 581 Mt Eden Rd. Asthma Nurse educators provided free COPD education, advice and spirometry tests.

We also attended a presentation by Associate Professor Robert Young on Modern Management of COPD.

There is no cure for COPD but early diagnosis and lifestyle changes, along with appropriate medication, can prevent further damage or decline in lung function and successfully manage the symptoms to improve quality of health.

- 1 Are you over the age of 40 years?
- 2 Are you getting short of breath on exertion?
- 3 Are you a smoker or ex-smoker?
- 4 Have you got a chronic productive cough, often in the morning?
- 5 Do you hear a wheeze when breathing out?
- 6 Do you have frequent episodes of bronchitis or chest infections?

If you answer yes to the above questions, please see your doctor.

If you are a smoker, please ensure your doctor is aware and consider smoking cessation.

Stopping smoking is the most important treatment as it stops further lung damage and improves oxygen levels in your blood. You will look and feel better. You will have more energy and feel less short of breath. You may have fewer chest infections and fewer hospital admissions. You may sleep better and regain your appetite, sense of smell and food will taste better.

There are many services available to you that will help you stop smoking.

Elaine Murray R.N.

Asthma Nurse Educator

¹ Bartolome R. Celli
Chest 2008; 133; 1451 - 1462

asthma and menopause

Compiled by Ann Wheat (RN)

Is there a connection?

It has been well documented that asthma can be affected by hormones especially around a women's menstrual cycle and during pregnancy but there appears to have been little research on the effects of hormones, menopause and asthma. According to Bonner et al, as cited in van den Berge, Heijink, van Oosterhout & Postma (2009), there is a peak in prevalence of adult-onset asthma in women at around the age of 50 which is the mean age of onset of menopause. Although there is this awareness that hormones do play a role in the development of asthma, it is unclear why it happens or the mechanisms involved (Real, Svanes, Macsali & Omenaas, 2008). Van den Berge et al (2009) go on to say that if asthma starts at menopause, it is often non allergic and the condition is often more severe, is present in 18% of the total number of female asthma patients (Foschino Barbaro et al, 2010) and has more episodes of uncontrolled asthma (van den Berge et al, 2009). In women with pre-existing asthma, the condition can also deteriorate at menopause (Foschino Barbaro et al, 2010). Foschino Barbaro (2010) also say that menopausal asthma is associated with recurrent sinusitis, urticarial/angioedema, aspirin sensitivity, use of oral steroids to control symptoms, frequent hospitalizations and an altered perception of asthma symptoms. Real et al (2008) said that women were more at risk of developing asthma if they were lean at the time of menopause, lean and using hormone replacement therapy, or women of fertile age with irregular menstruation.



So how does this happen?

Foschino Barbaro et al (2010), state that menopausal asthma is a different phenotype to premenopausal asthma. Premenopausal asthma is identified by a high exhalation of Leukotriene 4 (LTE-4). LTE-4 is a product that causes inflammation especially in the airways when it is over produced. There is also a decrease in exhaled pH and an increase in exhaled nitric oxide which is common to eosinophilic inflammation of the airways. Nitric oxide is produced when inflammation is present in the airways. In menopausal asthma there are also high levels of exhaled LTE-4 but urinary levels of LTE-4 are increased as well. Foschino Barbaro et al (2010) go on to say that oestradiol concentrations have been found to be lower in women with menopausal asthma than in normal menopausal women. Hatfield (2009) says that it is not the oestrogen per se that triggers the asthma but the fluctuations in the levels of the hormone. Further research by Dimitropoulou-Catras (n.d.) as stated in Hatfield (2009) says that it is the fluctuating oestrogen levels which activate proteins that produce the inflammatory response which brings on asthma symptoms. Foschino Barbaro et al (2010) also showed that there were increased levels of neutrophils in induced sputum and exhaled Interleukin-6 (IL-6). Neutrophils are associated with non-allergic asthma.

So how is Menopausal Asthma treated?

The most important factor is that if women suspect that they are having symptoms of asthma, such as coughing, wheezing, shortness of breath or chest tightness, they must see their medical practitioner as soon as possible for a diagnosis and treatment. Once diagnosed as with all asthma, menopausal asthma is treated with short acting relievers, inhaled cortico-steroids and long acting relievers as needed. Inhaled cortico-steroids (the mainstay of all asthma treatment) are

given twice daily and must be continued every day. When started it is important to remember that their effect is not rapid and will take several weeks before the full effect will become obvious.

There has also been much research done on the use of hormone replacement therapy (HRT) and how HRT affects asthma. It seems that the use of oestrogen only HRT may play a part in the development of asthma in menopause (Romieu 2009). Romieu (2009) also said that those who had allergies before the asthma diagnosis or those who have never smoked on oestrogen only HRT are at greater risk to develop asthma. It is therefore important to talk with your doctor so that you are given the correct type of medication when treating menopausal symptoms.

Finally it seems important that when reaching menopause women should have an ideal body mass index of between 23 – 28 as under 23 and over 28 means a higher risk of developing asthma.

In conclusion, menopausal asthma appears to be a new phenotype which causes the development of asthma or worsening of asthma around menopause. More research is being undertaken on this so it is important to watch this space.

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tai chi and copd

by Janet Delooze RN

The October meeting of the North Shore COPD Group welcomed a Tai Chi teacher who works with the ACC Accident Prevention Programme on the North Shore. She teaches Tai Chi Qigong to older adults in order to reduce the number of injuries from falls.



Tai Chi is a form of mind-body exercise that originated from the ancient Chinese martial arts. It can help improve muscle tone, flexibility and balance, and incorporates rhythmical breathing with its slow gentle movements inducing a state of meditation.

Exercise is important for everyone: bodies like movement. However, for those with COPD, the fear of becoming more breathless can lead to doing less and less. Feeling short of breath with exercise is NOT harmful but lack of exercise is: it can lead to muscle weakness, weak bones, increase in weight, depression and subsequently less activity¹. Tai Chi can be adapted to suit all age groups and abilities due to its low-impact of slow movements which makes it an ideal exercise for people with COPD.

A small study² carried out on 18 people looked at the effects of Tai Chi exercise training on stable COPD. They found that whilst there was no change in the FEV1 (Forced expiratory volume at 1 second) at the end of the 4 week training, the FVC (Forced vital capacity) had increased significantly in the participants suggesting a decrease in lung hyperinflation.

In COPD, lung hyperinflation comes about from decreased elasticity of the lung parenchyma. The alveoli and airways of normal lungs are elastic and well tethered by connective tissue, which not only helps to keep airways open during exhalation, but also provides the lung recoil to counter chest wall recoil³. In COPD, the elastic tissue is destroyed affecting the recoil balance causing the air to become trapped in the lungs.

A recent study⁴ demonstrated that a 3 month Tai Chi Qigong

programme improved respiratory function and activity capacity in the participants. The study divided the 158 participants into three groups: the Tai Chi group who practised 13 movement forms of Tai Chi Qigong twice a week for one hour; the walking group who walked for one hour daily and also practised pursed lip breathing and diaphragmatic breathing; the control group who were not requested to exercise or practise breathing exercises. The results of the study showed a significant improvement in FVC in the Tai Chi group (7%) compared to 4% in the walking group and a 4% decline in the control group. The Tai Chi group also improved their exercise capacity by 11% compared to 2% in both the walking and control group.

The social aspects of attending a group activity can also have a part to play in the overall feeling of wellbeing. Exercise and physical activity are associated with better quality of life and health outcomes⁵.

To find out more on Tai Chi in your area, check online or at your local YMCA, community or leisure centre. If you are interested in coming along to the North Shore COPD group, contact Janet Delooze on Ph: 630 2293.

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Source: J Asthma

Eosinophil Degranulation Is More Important than Eosinophilia in Identifying Asthma in Chronic Cough;

Kim CK, Callaway Z, Kim DW, Kita H; Journal of Asthma (Oct 2011)

Objective. We investigated whether eosinophil degranulation is a distinctive feature of asthma and can distinguish between chronic cough patients with asthma and those without. Methods. Thirty-seven patients, with a chronic cough for more than 1 month, and nine normal individuals (controls) were enrolled. Subjects were divided into two groups: one group with asthma and positive bronchial hyperresponsiveness (BHR) (Asthma group, n = 18) and the other group without asthma and negative BHR (Non-Asthma group, n = 19). From induced sputum, total cell counts and differentials were determined. Myeloperoxidase levels were measured by Enzyme-Linked Immunosorbent Assay (ELISA), and eosinophil-derived neurotoxin (EDN) and major basic protein (MBP) levels were measured by radioimmunoassay. Results. The percentage of sputum eosinophils was increased in the Asthma ($p < .001$) and Non-Asthma ($p < .05$) groups compared with the Control group and when comparing the Asthma and Non-Asthma ($p < .001$) groups. Sputum EDN and MBP levels were increased in the Asthma group compared with the Non-Asthma ($p < .05$ and $p < .05$, respectively) and Control groups ($p < .05$ and $p = .055$, respectively). However, EDN and MBP levels were not increased in the Non-Asthma group compared with the Control group. The percentage of sputum eosinophils in the Asthma group correlated positively with sputum EDN ($R_s = 0.921$, $p < .001$) and MBP ($R_s = 0.882$, $p < .0001$) levels and negatively with max FEV(1) ($R_s = -0.501$, $p < .05$) (FEV(1), forced expiratory volume in 1 second). Unexpectedly, the percentage of eosinophils in the Non-Asthma group did not correlate significantly with any of these markers. Increased EDN and MBP levels and significant correlations between the percentage of eosinophils and EDN and MBP were only observed in asthma patients. Conclusions. These findings suggest that eosinophil degranulation is more important than eosinophilia in identifying asthma.

greater familial correlation than either specific or general SPT response and significantly higher sibling correlation in TESAOD than in CRS, probably due to the older age of the siblings and the longer period of ascertainment. Conclusions: Significant familial aggregation of specific response to allergen after adjustment for other atopy appears to reflect a genetic propensity toward atopy, dependent on shared familial exposures. Results also suggest that inheritance of asthma is independent of atopic sensitization.

Source: Pediatr Allergy Immunol

Familial aggregation of allergen-specific sensitization and asthma;

Kurzius-Spencer M, Guerra S, Sherrill DL, Halonen M, Elston RC, Martinez FD; Pediatric Allergy and Immunology (Oct 2011)

ABSTRACT: Background: Familial aggregation of specific response to allergens and asthma adjusted for age and sensitization to multiple allergens was assessed in two large population cohorts. Methods: Allergen skin prick tests (SPTs) were administered to 1151 families in the Tucson Children's Respiratory Study (CRS) and 435 families in the Tucson Epidemiological Study of Airway Obstructive Disease (TESAOD). Sensitization was defined by wheal size ≥ 3 mm; physician-diagnosed asthma at age ≥ 8 yr was based on questionnaires. Using s.a.g.e. 6.1 software assoc and fcor, familial correlations of crude and adjusted phenotypes were evaluated. Results: Crude estimates of parent-offspring (P-O) and sibling correlations were statistically significant for most allergens, ranging from 0.03 to 0.29. After adjusting for age of assessment and 'other atopy' (SPT-positive response to additional allergens), correlations were reduced by 14-71%. Sibling correlations for specific response to allergens were consistently higher than P-O correlations, but this difference was significant only for dust mite and weed mix in the TESAOD population. Familial correlation for atopic status (any positive SPTs vs. none) tended to be higher than for specific allergens. Asthma, with and without adjustment, showed

Source: J Allergy Clin Immunol I

Quantifying the proportion of severe asthma exacerbations attributable to inhaled corticosteroid nonadherence;

Williams LK, Peterson EL, Wells K, Ahmedani BK, Kumar R, Burchard EG, Chowdhry VK, Favro D, Lanfear DE, Pladevall M; Journal of Allergy and Clinical Immunology (JACI) (Oct 2011)

BACKGROUND: Asthma is an inflammatory condition often punctuated by episodic symptomatic worsening, and accordingly, patients with asthma might have waxing and waning adherence to controller therapy. OBJECTIVE: We sought to measure changes in inhaled corticosteroid (ICS) adherence over time and to estimate the effect of this changing pattern of use on asthma exacerbations. METHODS: ICS adherence was estimated from electronic prescription and fill information for 298 participants in the Study of Asthma Phenotypes and Pharmacogenomic Interactions by Race-Ethnicity. For each patient, we calculated a moving average of ICS adherence for each day of follow-up. Asthma exacerbations were defined as the need for oral corticosteroids, an asthma-related emergency department visit, or an asthma-related hospitalization. Proportional hazard models were used to assess the relationship between ICS medication adherence and asthma exacerbations. RESULTS: Adherence to ICS medications began to increase before the first asthma exacerbation and continued afterward. Adherence was associated with a reduction in exacerbations but was only statistically significant among patients whose adherence was greater than 75% of the prescribed dose (hazard ratio, 0.61; 95% CI, 0.41-0.90) when compared with patients whose adherence was 25% or less. This pattern was largely confined to patients whose

asthma was not well controlled initially. An estimated 24% of asthma exacerbations were attributable to ICS medication nonadherence. CONCLUSIONS: ICS adherence varies in the time period leading up to and after an asthma exacerbation, and nonadherence likely contributes to a large number of these exacerbations. High levels of adherence are likely required to prevent these events.

Source: Respir Med |
Effect of budesonide/formoterol pMDI on COPD exacerbations:

A double-blind, randomized study; Sharafkhaneh A, Southard JG, Goldman M, Uryniak T, Martin UJ; Respiratory Medicine (Oct 2011)
 BACKGROUND: Treatment with an inhaled corticosteroid (ICS) and long-acting bronchodilator is recommended for severe/very severe chronic obstructive pulmonary disease (COPD) patients with repeated exacerbations. This randomized, double-blind, double-dummy, parallel-group, 12-month multicenter study evaluated the effect of budesonide/formoterol pressurized metered-dose inhaler (pMDI) on COPD exacerbations. METHODS: Following a 2-week run-in during which COPD patients aged ≥ 40 years with an exacerbation history discontinued medications except ICSs, 1219 patients were randomized 1:1:1 to twice-daily budesonide/formoterol pMDI 320/9 μg , budesonide/formoterol pMDI 160/9 μg , or formoterol dry powder inhaler 9 μg . An exacerbation was defined as COPD worsening requiring oral corticosteroids and/or hospitalization. A post hoc analysis, with antibiotic treatment added to the exacerbation definition, was also performed. RESULTS: Budesonide/formoterol 320/9 and 160/9 reduced exacerbation rates (number per patient-treatment year) by 34.6% and 25.9%, respectively, versus formoterol ($p \leq 0.002$). Budesonide/formoterol 320/9 prolonged time to first exacerbation versus formoterol, corresponding to a 21.2% reduction in hazard ratio (0.788 [95% CI: 0.639, 0.972]; $p = 0.026$). Exacerbation rates (number per patient-treatment year) including antibiotic treatment (post hoc analysis) were reduced by 25.9% and 18.7% with budesonide/formoterol 320/9 and 160/9, respectively, versus formoterol ($p \leq 0.023$). Both budesonide/formoterol doses were well tolerated with safety profiles similar to formoterol. Pneumonia adverse events occurred in 6.4%, 4.7%, and 2.7% of patients in the budesonide/formoterol 320/9, 160/9, and formoterol groups. CONCLUSIONS: Over 12 months, both budesonide/formoterol doses reduced the exacerbation rate (defined with or without antibiotic treatment) versus formoterol. Budesonide/formoterol pMDI is an appropriate treatment for reducing exacerbations in COPD patients with a history of exacerbations. (NCT00419744)

Source: Respir Med | Posted 5 hours ago
Pulmonary function, oxidative stress and inflammatory markers in severe COPD exacerbation;

Stanojkovic I, Kotur-Stevuljevic J, Milenkovic B, Spasic S, Vujic T, Stefanovic A, Llic A, Ivanisevic J; Respiratory Medicine 105 Suppl 1 S31-7 (Oct 2011)
 BACKGROUND Oxidative stress and inflammation play an important role in the pathogenesis of chronic obstructive pulmonary disease (COPD). OBJECTIVE Pulmonary function, oxidative stress parameters and inflammatory markers were measured in 74 patients with severe

COPD exacerbation and 41 healthy subjects. In patients all parameters were assessed at two time points: Firstly, one day after admission and secondly, after 7-10 days when they were clinically stable enough to be discharged. Patients were divided in two groups according to the presence of ischemic heart disease (IHD): IHD positive (IHD+) patients and IHD negative (IHD-) patients. METHODS AND RESULTS During hospitalisation 0(2)(\bullet -), malondialdehyde (MDA), advanced oxidation protein products (AOPP) and total oxidant status (TOS) increased and were higher at discharge compared with admission and the control group. Superoxide dismutase (SOD) activity was significantly lower in COPD patients at both time points compared with the control group. Total antioxidant status (TAS) was significantly lower and the prooxidant-antioxidant balance (PAB) was higher at both time points in COPD patients compared with the control group. High sensitive C-reactive protein (hsCRP) and also the neutrophil count were significantly higher at admission compared with discharge. Paraoxonase 1 (PON1) enzymatic activities in COPD patients did not differ compared with the control group. IHD+ COPD patients had significantly lower PON1 activity but higher PAB levels and hsCRP concentrations, compared with IHD COPD patients. CONCLUSION The oxidant/antioxidant imbalance was significantly pronounced in patients with COPD exacerbation for at least 24 hours following their admission and when they were clinically stable enough to be discharged. Increased oxidative stress, elevated systemic inflammation and decreased antioxidant defence were common in end-stage disease and particularly COPD patients with ischemic heart disease.

Source: Respir Med | Posted 12 hours ago
Benefits of adding fluticasone propionate/salmeterol to tiotropium in moderate to severe COPD;

Hanania NA, Crater GD, Morris AN, Emmett AH, O'Dell DM, Niewoehner DE; Respiratory Medicine (Oct 2011)
 BACKGROUND: Combining maintenance medications with different mechanisms of action may improve outcomes in COPD. In this study we evaluated the efficacy and safety of fluticasone/salmeterol (FSC) (250/50 mcg twice daily) when added to tiotropium (18 mcg once daily) (TIO) in subjects with symptomatic moderate to severe COPD. METHODS: This was a 24-week, randomized, double-blind, parallel group, multi-center study. Subjects 40 years or older with cigarette smoking history ≥ 10 pack-years and with the diagnosis of COPD and post-bronchodilator FEV(1) ≥ 40 to $\leq 80\%$ of predicted normal and FEV(1)/FVC of ≤ 0.70 were enrolled. Following a 4-week treatment with open-label TIO 18 mcg once daily, subjects were randomized in a double-blind fashion to either the addition of FSC 250/50 DISKUS twice daily or matching placebo. The primary efficacy endpoint was AM pre-dose FEV(1) and secondary endpoints included other measures of lung function, rescue albuterol use, health status and exacerbations. RESULTS: The addition of FSC to TIO significantly improved lung function indices including AM pre-dose FEV(1), 2 h post-dose FEV(1), AM pre-dose FVC, 2 h post-dose FVC and AM pre-dose IC compared with TIO alone. Furthermore, this combination was superior to TIO alone in reducing rescue albuterol use. However, there were no significant differences between the treatment groups in health status or COPD exacerbations. The incidence of adverse events was similar in both groups. CONCLUSIONS: The addition of FSC to subjects with COPD treated with TIO significantly improves lung function without increasing the risk of adverse events. NCT00784550

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References: 1. Global Initiative for Asthma; *Global Strategy for Asthma Management and Prevention*. Updated 2009. 2. Woodcock AA et al. *Prim Care Respir J*. 2007;16(3):155-161. 3. Bateman ED et al. *Am J Respir Crit Care Med*. 2004;170:836-844

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