THE NZ JOURNAL OF RESPIRATORY HEALTH
August 2011

World Asthma Day 2011
– Remembering Logan

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contents

on the cover:
World Asthma Day photo by Steven Neville, ASP Photography.

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Unitec NEW ZEALAND Te Whare Wānanga o Wairaka

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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
PO Box 67-066, Mt Eden, Auckland
Phone 09 623 4777 Ann or 623 4771 Swarna
Fax 09 623 0774
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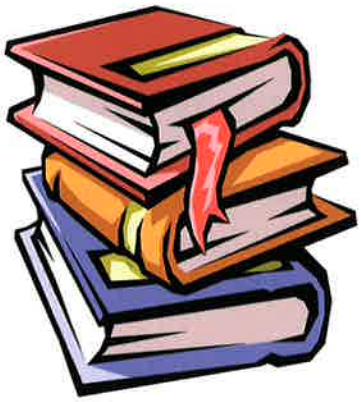
The closing date for 1st semester enrolment is
10th February 2012 for Asthma
10th April 2012 for COPD



Upcoming events and courses

ASTHMA NEAT COURSE
21 September 2011

HALF DAY COPD COURSE
19 October 2011

WORLD COPD DAY
16 November 2011
Open Day at Asthma Auckland
10am – 3pm

World COPD Day

PARENT AND CHILD SHOW
30 September – 2 October 2011
ASB Showgrounds
Auckland

Parent and Child Show

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Further enquiries for any of these events phone
09 630 2293
or
www.asthma.org.nz

message to readers

Asthma New Zealand – the Lung Association (Inc) held it's Annual General Meeting on the 24th June 2011 at 581 Mount Eden Road, Mount Eden, Auckland, commencing 7:00pm. In general terms the meeting was successful, however there was a change in terms of President. Mrs Pamela O'Brien was elected as the new President of Asthma New Zealand – the Lung Association (Inc). I would like to take this opportunity to express the Board's appreciation and thanks to Mr Kevin Walsh, who was the Acting President once Mr R. Thorogood resigned the Presidency. Kevin did a sterling job and the new Board intends to be more pro-active in order to make Asthma New Zealand better known across New Zealand.

I would have to comment that New Zealand is going through a fairly torrid period given the earthquakes in Christchurch and the run of severe weather throughout the winter period. This has been reflected in the increase in requests for our nurses to service both adults and children, either at local Societies or within their home environments. At this point staff are extremely busy and I thank them for their commitment to people with asthma and their families.

Where do we go from here? I would like to inform readers that a meeting has been held with the Asthma Foundation in an attempt to improve the relationship between the two organisations. This is an operation in progress and it will be interesting to see the final outcome of the discussions that are taking place.

I thank you all for your support over the last year and I look forward to working with you throughout the coming year and to achieving better outcomes for people with asthma and their families.

Garry A. Hanna
Secretary/Treasurer
Asthma New Zealand – the Lung Association (Inc)

Breathe Easy

asthma NEW ZEALAND THE LUNG ASSOCIATION

occupational asthma

Compiled by Elaine Murray RN
Asthma Nurse Educatior

What is occupational asthma?

The term can be used to encompass pre-existing asthma exacerbated by work environment, while other definitions refer only to asthma causally related to exposure in the work environment.

The National Occupational Health and Safety Advisory Committee of New Zealand (NOHSAC) states that Occupational Asthma is a disorder characterised by bronchial hyper-responsiveness or variable air flow limitation related to work place exposures, and that occupational asthma is probably the most common work-related respiratory disorder in industrialised countries.

Asthma can be triggered by many things both at work and away from work.

Some clues that something at work is affecting your asthma are that your asthma gets worse soon after starting a new job or while you are doing a particular part of your job, or your asthma improves when you are not at work, e.g. on holiday or at the weekend.

Asthma likely develops because of both a genetic predisposition and exposure to environmental factors. There is considerable epidemiologic evidence that occupational exposure to certain specific agents can lead to the development of asthma. The incidence and prevalence of occupational asthma in various occupational cohort studies depend on the agent(s) to which the workers are exposed and the levels of their exposure. Host susceptibility factors, such as atopy and cigarette smoking, may also play a role in at least some cases. There are convincing data to indicate that the level of exposure is a critical risk factor for sensitizer –induced occupational asthma.

It is estimated that occupational asthma represents approximately 5% of all asthma cases. Typically features of occupational asthma include a latency period of up to several years prior to onset of symptoms, worsening of symptoms shortly at the end of the shift or at night, and improvement in asthma symptoms when not at work.

Some of the most common New Zealand work place triggers are;

- Isocyanate paints
- Foams and plastics, and the fumes given off during manufacture
- Animal fur and protein from laboratories and veterinary clinics
- Flour and grain dusts from farms, granaries and bakeries
- Wood dusts
- Epoxy resins and other plastics from boat builders, mould manufacturers and plastic manufacturing processors

Are you at risk of developing asthma at work?

Sometimes people who have had asthma before can develop asthma through an allergic reaction to a substance in the work place. This may happen even after years of working safely with the substance. Sometimes the allergic reaction (and therefore symptoms) doesn't develop until some hours after the exposure. Other people develop asthma for the first time in the workplace after heavy exposure of irritants to the airways, such as welding fumes or gaseous vapours like sulphur dioxide.

The prevalence of occupational asthma is higher in smokers.

Aetiology of Occupational Asthma

There appears to be two causal pathways in the aetiology of occupational asthma.

- A sensitising agent with subsequent exposure may cause early bronchoconstrictive reactions occurring within minutes, or later reactions of longer duration. There may be a combination of dual early and late reactions.
- A heavy, or sudden exposure to an irritating agent can give rise to asthma or RADS (reactive airways dysfunction syndrome)

The late asthmatic response or the after effects of a sudden heavy exposure to an irritating agent are associated with "bronchial hyper-responsiveness" where the airways react to a variety of stimuli such as cold, exercise, smoke and dusts as well as the original provoking agent. Many of these stimuli are encountered outside the workplace, making the diagnosis of occupational asthma very difficult.

The symptoms of occupational asthma are coughing, tight chest, shortness of breath and wheezing.

Diagnosis

The best measure of the occupational origin of the worker's asthmatic symptoms is obtained by peak flows. A minimum of 4 peak flow measurements every day for 2 weeks is recommended. The best of 3 (to ensure a consistent result) peak flows is recorded. Before work, during work (mid shift), immediately after work and as the patient goes to bed are the suggested times The worker must mark the time at work and not at work and, if possible, periods when they are exposed to the suspected aetiological agent, presuming the exposure was not continuous. They should also try and record symptoms such as cough, wheeze, and shortness of breath on the chart.

Lung function (spirometry) tests (FEV1 and FEV1/FEVC) both before and after administration of a bronchodilator.

In specialist centres the measurement of non-specific bronchial responsiveness or irritability is a useful tool in assessing the patient. This involves measurement of the provocative concentration of histamine or methacholine that causes a 20 % fall in FEV1. A positive methacholine or histamine challenge supports a clinical diagnosis of asthma when baseline pulmonary function is normal.

Skin testing with common allergens (house dust mite, grass and tree pollens) can be useful in determining the atopic status of the patient. Atopy may point to an occupational exacerbation of a pre-existing asthma.

Treatment of occupational asthma

The pharmacological treatment of occupational asthma is no different from any other form of asthma. The only important difference between the treatment of occupational asthma compared with non-occupational asthma is the necessity of identifying the causative exposure and controlling it.

The Health and Safety in Employment Act (1992) requires that employers identify hazards and then control them by elimination, isolation and minimisation.

Occupational asthma is defined in the Health and Safety in Employment Act as "serious harm".

Occupational asthma is relatively uncommon, but when it does occur it has a profound impact on the individual's wellbeing. It should be considered in any adult who is suffering asthma for the first time in their lives.

Many other asthma sufferers have their condition made worse by workplace factors, and the suggestions about control measures apply to them as well as those with "occupational asthma".

Up to 50% of people who have suffered from occupational asthma are left with persisting asthma after removal from exposure and this may be severe and disabling. Occasionally asthma attacks, precipitated by occupation exposures, are fatal even though the preceding disease may have been considered quite mild.

The outcome of interventions following a confirmed diagnosis of occupational asthma may depend on several factors, including the worker's age and the causative agent.

Early diagnosis and early avoidance of further exposure, either by relocation of the worker or substitution of the hazard, offer the best chance of complete recovery.

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spacer devices

Compiled by Sharron Daniels RN BN

It has been well documented that the use of spacer devices plays an important role in the management of both asthma and COPD (Chronic Obstructive Pulmonary Disorder). The value of spacers when using metered dose inhalers (MDI's) have been shown to significantly increase the uptake of drug deposition as well as help reduce side effects such as sore throat, husky voice and oral thrush. With the number and variety of spacers available on the market choosing the best option can be a challenge. This article discusses the benefits provided by spacers as a guide to choosing the best option for you.

Spacers were first released to help alleviate issues of poor technique when using MDI's. Studies such as Keely (2006) have demonstrated that the amount of drug actually reaching the lungs using a MDI without a spacer can be as little as 10% (with correct technique). The remaining drug is deposited either in the mouth or throat causing the aforementioned side effects common to MDI use. By using a spacer in conjunction with a MDI, drug deposition to the lungs may be improved because the smaller drug particles are able to reach the lungs, while at the same time significantly reducing the amount of drug caught in the mouth or throat. The end result is lower dose requirements to achieve the same level of control along with significantly reduced possibility of side effects due to the reduced amount of drug residue in the mouth and throat. All good reasons to include spacers as part of your asthma management programme.

There are many spacers available on the market including open tube spacers, holding chambers and reverse-flow type spacers to name a few. A spacer comprises a central port which can optionally have a mask attached or is placed directly into the mouth. The central port is linked to a tube with an opening at the other end to fit the MDI. Spacers vary widely in their design, shape and size, with volume ranging from 50 ml to 750 ml.

Spacers act by trapping larger drug particles that would normally come to rest in either the nose or throat inside the spacer chamber. They also decrease the velocity of the remaining smaller particles resulting in an increase in the amount of preventer or reliever actually reaching the lungs. Large volume-spacers provide superior delivery due to the reduced speed and size achieved as spacer size increases. Although not as effective as their larger counterparts, smaller spacers provide a significant improvement in effectiveness over using MDI's alone. Their reduced size also makes them more convenient when you are out and about. When considering the type of spacer to use as part of your asthma management regime the general rule is 'bigger is better'. Larger, volumatic type spacers will generally provide an increased bronchodilator response over smaller volume spacers. They are

ideal in particular when used morning and night with your preventer as the large size is unlikely to be an issue. When convenience is a factor consider using smaller style spacers as they will still provide a significant improvement when using both preventer and reliever medications.

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

Judith Jones	Christchurch
Marianne Eliasen	Palmerston North
Ingrid Hoogenboom	Waipukurau
Marjorie Kunaka	Hamilton
Susan Walker	Kaikohe
Michele Bebich	Auckland
Elizabeth Holmes	Cambridge
Charlotte Oliva	Upper Hutt
Lucia Manene	Lower Hutt
Kerry Ellison	Napier

is there a connection between premature birth and asthma?

Compiled By Karen Little RN

Unfortunately information on long term respiratory symptoms in prematurely born children is scanty. Preterm birth is associated with asthma like – symptoms in childhood and possibly in adolescence, but the longer term risk of asthma is unknown and increasingly relevant as larger numbers of these individuals enter adulthood. About one baby in ten is born early. Only a very small number are at risk of having a severe lung condition; about one in every hundred premature babies. It's usually those with very low birth weight.

A Swedish national Cohort Study (Crump et al, 2011) found that babies who were born extremely preterm (23-27 weeks gestation) were 2.4 times more likely to be prescribed asthma medications than those who were born at term. No association was found between later preterm birth (28-32 or 33-36 weeks gestation) and asthma medications in young adulthood. Maternal smoking during pregnancy and maternal asthma are independent risk factors associated with preterm delivery.

Respiratory distress syndrome (RDS) is a breathing disorder that may affect babies born prematurely. These baby's lungs are not able to make enough surfactant, this is a liquid that coats the inside of the lungs that helps to keep the lungs open so that infants can breathe in air once they are born. Nearly all infants born before 28 weeks of pregnancy develop RDS. RDS may be an early phase of bronchopulmonary dysplasia (BPD). Some infants who have RDS recover and never get BPD. Infants who have RDS and get BPD have lungs that are less developed or more damaged than the infants who recover. Infants who develop BPD usually have fewer healthy air sacs and tiny blood vessels in their lungs. Some babies can develop complications from RDS or its treatments such as asthma. (National Heart Lung & Blood Institute, n.d).

The term bronchopulmonary dysplasia (BPD) was first described in 1967 (Northway et Al, 1967), this was a new chronic respiratory disease that developed in premature infants exposed to mechanical ventilation and oxygen supplementation. Twenty years later the same authors found that the respiratory symptoms persisted into adolescence and early adulthood. BPD is now the most common chronic lung disease of infancy in the United States. The other name for this condition is Chronic Lung Disease of the new born.

Today newborns consistently survive at gestational ages of 23 to 26 weeks – 8 to 10 weeks younger than the infants in the first study so the pathological and clinical characteristics have changed profoundly. It is only now that large populations of persons born prematurely are approaching adulthood, and they may be at increased risk for respiratory disease in adult life.

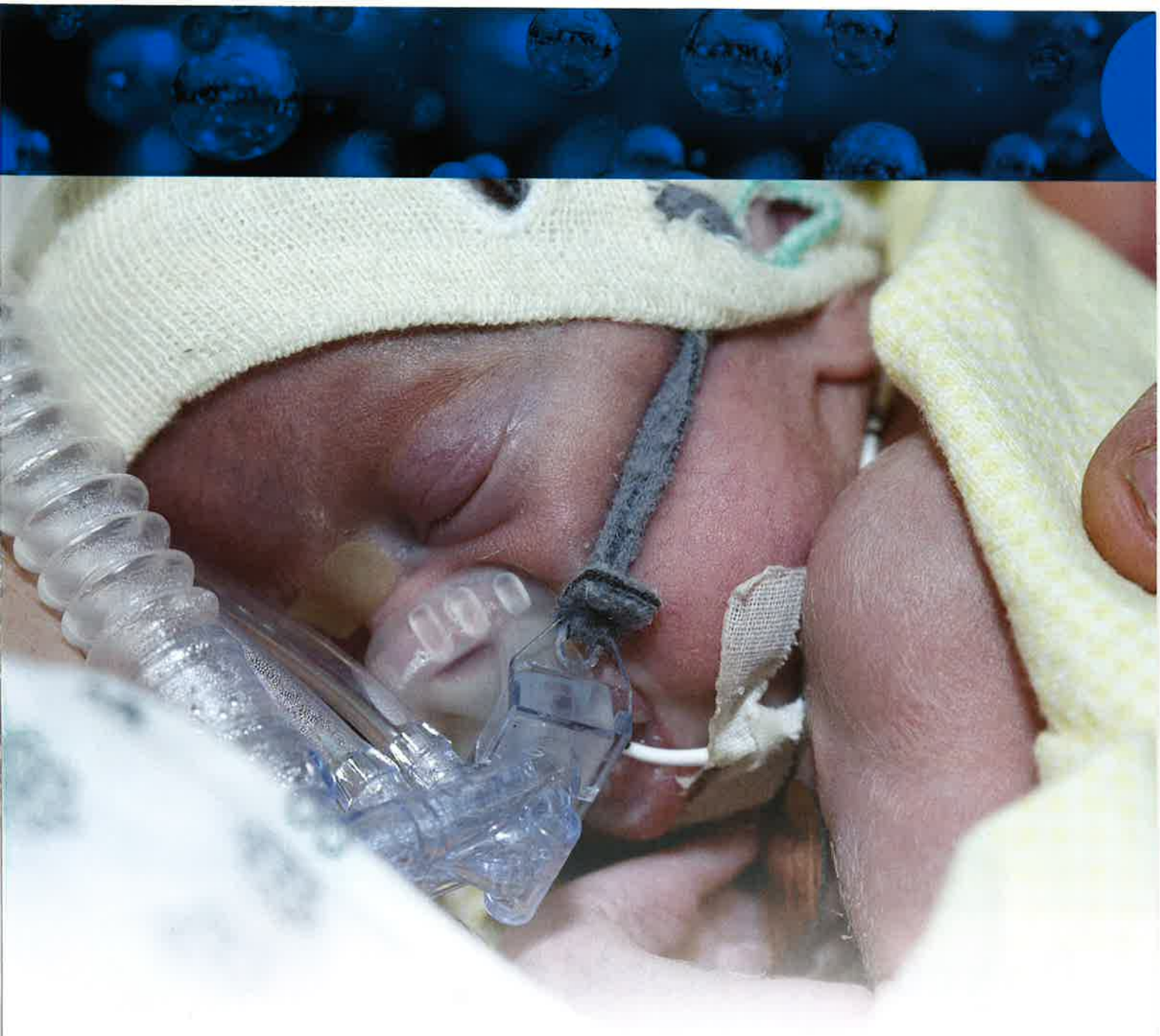
BPD is now defined as the need for supplemental oxygen for at least 28 days after birth and its severity is graded according to the respiratory support required near term, (Jobe 2001). Accurate markers of chronic lung damage in premature infants is still lacking however as many infants with BPD have a full clinical and functional recovery.

Recurrent wheezing is markedly increased in infants born before 33 weeks of gestational age as compared with those born at term and the rate of readmission to hospital with complications of respiratory tract infections is high, up to 50% in the first year of life (Lamarche-Vadel et al, 2004). Strict measures to prevent viral infection and avoid adverse environmental factors (e.g., passive smoking) are crucial. The need for immunization is crucial.

Cohort studies show a significantly greater prevalence of asthma like symptoms and the use of inhaled asthma medication among persons 8 to 19 years old who were born prematurely – regardless of whether they had BPD – than among persons born at term (Anand 2003). Spirometric values reflecting airflow are also consistently lower at any age than in controls born at term (Gross 1998).

There is currently no clear evidence of the long term beneficial effects of improved neonatal care among young children who were born preterm after the introduction of antenatal corticosteroids and surfactant replacement, the prevalence of respiratory symptoms and the need for inhaled drugs remain high (Vrijlandt 2007).

Some investigators have expressed concern that survivors of preterm birth and BPD may be more susceptible to Chronic Obstructive Pulmonary Disease (COPD) in later life (Eber, Zach, 2001). There may be an overlap in the clinical and physiological characteristics of the



two conditions, but longer follow-up and data will be needed before it can be included in the well established diagnosis of COPD.

Advances in neonatal care have increased survival after preterm birth. Because many of these survivors are now approaching adulthood, family doctors and chest physicians will be seeing more cases of PDF which begins in neonatal life.

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asthma management tips for winter

Colds and viruses are very common triggers for asthma which are much more prevalent in winter. Colds are not easily avoided. The aim is to have your asthma controlled so that you can cope with whatever triggers may come along. So try this check list for managing your asthma in winter.

✔	Get your doctor to review your asthma regularly especially before winter
✔	Talk to your doctor about the influenza vaccine
✔	At the first sign of a cold or flu follow your Asthma Action Plan given to you by your doctor. If you do not have a written Asthma Action Plan please discuss this with your doctor next time you visit.
✔	Pay close attention to your asthma symptoms. If you notice warning signs of an asthma episode- such as coughing, wheezing, chest tightness or shortness of breath-adjust your medication as directed by your doctor e.g. the use of the blue reliever 2 puffs 4 times a day from day 1 of a cold and continue for the duration of the cold. Quick action can help prevent a severe attack.
✔	Use a peak flow meter to monitor how well your lungs are working from day to day. Take your readings at the same time every day. If you notice a drop in your peak flow rate, adjust your medication as directed by your doctor.
✔	Take your preventer medication every day morning and night even when well
✔	Avoid smoke from tobacco, fireplaces and wood stoves as this can trigger symptoms
✔	Protect yourself from the cold by dressing warmly and wearing a scarf around your mouth, and try to breathe through your nose as this warms and moistens the air
✔	Try and keep the inside of your home at an even temperature throughout.

Some tips to help reduce the spread of colds and viruses	
✔	Keep your hands away from your eyes, nose and mouth
✔	Use tissues to wipe your nose, and then discard them
✔	Wash your hands after blowing your nose
✔	Cough or sneeze into the inside of your elbow
✔	Wash your hands before preparing or eating food
✔	Do not share cups or cutlery with other people
✔	Avoid people with coughs and colds
✔	Take good care of yourself
✔	Rest
✔	Drink plenty of fluids
✔	Always have your blue reliever with you at all times (check expiry date and ensure you have enough medication in the inhaler)

Kid's Page

Answer Yes / No

If I Have Asthma, Can I Keep My Pet?

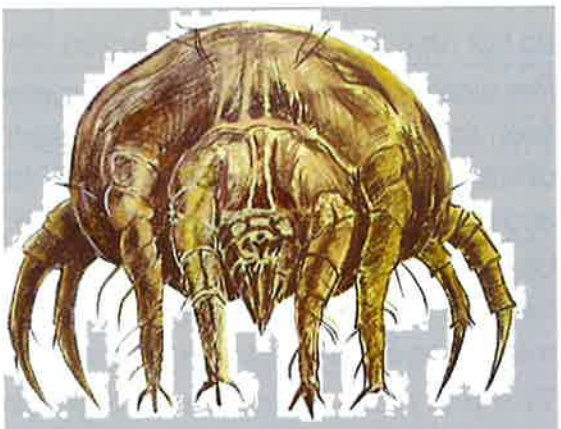
YES / NO

Can the Weather Affect My Asthma?

YES / NO

Is smoking a trigger for asthma?

YES / NO



Is this dustmite a trigger for asthma?

YES / NO

Yes for all

Yes, you can keep your pet but not in your bedroom

Yes, weather conditions and changing climate conditions may be an asthma trigger.

Yes, smoking can cause asthma flare-ups

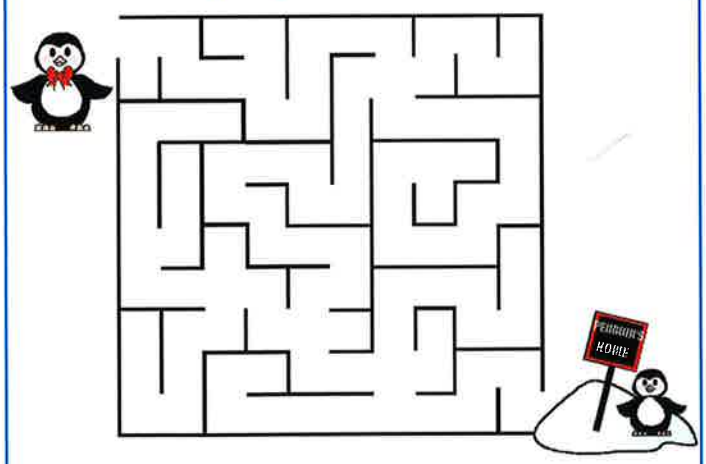
Yes, droppings from dust mites can trigger asthma

FIND the inhaler

How long did it take?

Penguin's Home

Help your penguin friend find his home



introducing...

asthma aucklands newest recruit and asthma nurse educator sharron daniels

Sharron started her nursing career as a mature student. Over the years she has worked in various areas under the umbrella of nursing. She began working at Middlemore Hospital on the Burns and Plastics Ward and also on the Respiratory Ward from there she moved into the mental health arena. For the last 8 years Sharron has been a practice nurse, this involved running nurse led clinics; on any given day she would find herself involved with asthma and diabetes patients through to immunisations for young people. Her main professional interest's are Asthma and Woman's health along with adolescent mental health.

Because Sharron herself has asthma she is able to share her personal experience of asthma which may be of some help to our newly diagnosed clients offering support and education in asthma and COPD and promoting the rights of people with asthma.

Sharron has three grown up children and enjoys quilting and accompanying her husband on the motorbike.

On the weekend of the 9th and 10th of July the Asthma Wellington staff hosted a stand at the Gluten Free Food and Allergy Show in Wellington for the second year running and once again it was a great success. With 3145 people through the shows doors we talked with a significant number of people with asthma who do not have their asthma well controlled. We were able to provide people with either on the spot advice or they were able to make a "self referral" for an educational visit in their own home; this is a free service provided by Asthma Wellingtons nurse educators to help manage and control asthma symptoms.

Throughout the day there was a range of free seminars on relevant topics and Adie Riddell, one of our asthma nurse educators, gave a presentation on "identifying and managing asthma triggers" which was well received. We also had very positive feedback on our service, the range of resources that we have available, and it was a fantastic opportunity to engage with members of the public we would not have otherwise seen.

As we are a non-profit organisation, we are always most grateful of all donations and grants that enable us to carry out our service and have a strong presence in the community.

north & south

NEWS FROM AROUND THE REGIONS ...

Asthma Auckland CEO Gerry Hanna with George Reid

Kevin Locke, Jacob Lillyman, Dylan and Dean Hartnoll

Kevin Locke, Jacob Lillyman with some of Team Logan

From left – Matt Purcell and Jay Denton (Bach Espresso)

Tracey Slako, Dan Beban and Aaron Mahon (More FM team)

Adie Riddell RN (right) providing Asthma education

Logan Hartnoll

10 April 2005 – 20 February 2010

world asthma

World Asthma Day 2011 marked another milestone in the amazing fundraising efforts of "Team Logan" with the introduction of a NIOX Analyser which was purchased with funds raised by "Team Logan" in memory and honour of Logan Hartnoll who passed away last year from an exacerbation of asthma, aged 4. His devoted family and friends raised over $20,000 towards the purchase of this machine which will be an invaluable tool for our Asthma Nurse Educators in assessing people with asthma and COPD. Vodafone Warrior Jacob Lillyman, has asthma and was the first person to have his lung function tested using the new machine. Jacob was joined by teammate Kevin Locke at Asthma New Zealand's open day and they were a real hit with everyone, they are real team players, both on and off the field. After all the official cutting of ribbons so to speak and morning tea for the 100 or so people who were in attendance we all headed outside and released helium balloons in honour of Logan!

The day of course would not have been successful without our

Kevin Locke, Liam Thompson, Isla Thompson and Jacob Lillyman waiting to release the helium balloons for Logan

ASTHMA CAN KILL

- 1 in 4 children in New Zealand have asthma
- 1 in 7 adults in New Zealand have asthma

KNOW THE FACTS!

day 2011

wonderful Breathe Easy partners – DVS, Fujitsu and Greenstuf Insulation. I would like to thank GSK who sponsored us throughout Asthma Awareness Week which enabled us to advertise and promote asthma awareness in mini events, in print and on the radio. I would also like to thank Autex Industries – Greenstuf Insulation staff, Francine McCormick and Kate Bourke and One Community Manager, Petrece Kesha assisting us on the day and for bringing Jacob and Kevin along too! Thanks also to More FM Team for the wonderful radio feeds and sausages on the day. Wonderful coffee was supplied by Bach Espresso and bouncy castle supplied by Neverland Castle. Last but not least I would like to thank all of our volunteers for their contribution throughout the year as we couldn't do it without you.

Linda Thompson
PR / Marketing Manager
Asthma New Zealand

Riley Saunders

Dylan Hartnoll with some GreenStuf not the insulation just candyfloss

Vodafone Warriors Jacob Lillyman, Kevin Locke, Sophie Hartnoll and Caden Harris Wade

Sophie and Dylan Hartnoll with friend Renee Macmillan

Callum Borland waiting to release his balloon for Logan

Callum Borland swapped his balloon for a sausage

Alison Borland and Kirsten Hartnoll

what are spirometry and niox testing?

Compiled By Karen Little RN

The machine called a Spirometer has the ability to measure and record the rate and volume of air expelled from the lungs. This can demonstrate any loss of lung function before the client becomes symptomatic. The test can also differentiate between restrictive airway disease (e.g. fibrotic conditions) and obstructive airway disease (e.g. Asthma and Chronic Obstructive Pulmonary Disease [COPD]). It will also give your lung age, so a 45yr old person who is having obstructive respiratory symptoms may demonstrate an age of an 80yr old!

Vodafone Warrior Jacob Lillyman, who has asthma, with Asthma Nurse Elaine Murray being tested on the Spirometer

Asthma is a common chronic inflammatory condition, the cause of which is not fully understood. As a result of the inflammation the airways are hyper-reactive and they narrow easily in response to a wide variety of stimuli. This may result in coughing, wheezing, chest tightness and shortness of breath. A patient will be asked if possible not to have any reliever asthma medications for at least four to twelve hours before a Spirometry test. The reason for this is that we do the first test and then the results are compared to a second test after the patient has had four puffs of their blue reliever. With asthma the narrowing of the airways is usually reversible and the degree of this reversibility can be an indication of how well, or uncontrolled, their asthma is. If there is a 15% increase in the amount of air expelled after taking the reliever this is proof that the person's asthma could be better controlled. The largest increase we have seen was 50%! That person was running on "half empty".

When a person has COPD this reversibility following the administration of their reliever is non-existent, or much less. It is important to distinguish between asthma and COPD as the treatment is different. To make things more complicated a person can have elements of COPD and asthma together.

Until recently routine assessment of the level of inflammation inside the airways has not been possible. We are very grateful to the Hartnoll family for their fundraising efforts that have enabled Asthma Auckland to help purchase a new machine called the Hypair FeNO which tests for Feno (fraction of expired nitric oxide) that enables us to measure the degree of inflammation inside the airways. Nitric oxide (NO) is widely distributed throughout the body and its effects are generally benign and important to cell physiology. When the airways are inflamed such as occurs with people with asthma NO in exhaled air is increased.

Jacob Lillyman

Kevin Locke on the NIOX Analyser

Dean and Kirsten Hartnoll watching the NIOX Analyser, dedicated to son Logan, in use.

Kirsten Hartnoll with son Dylan watching the NIOX Analyser in use

Extensive research has shown that exhaled NO levels of 5 to 25 parts per billion are considered normal, whereas higher levels indicate an active inflammation in the lungs. Exhaled NO measurement represents an objective method of assessing airway inflammation and the response to anti-inflammatory therapy.

Kevin Locke

The nurses at Asthma Auckland are looking forward to having this extra tool which, in combination with the usual assessment and client history will help us to distinguish between asthma and COPD. So many people are reluctant to take their asthma preventers that control inflammation in the airways, so having this objective test can show the person to what level their asthma is controlled. Airways that are red, inflamed and swollen over a long period can develop structural changes called fixed airways obstruction. If left unchecked, remodelling of the airways results, which contributes to irreversible airflow obstruction (COPD).

Vodafone Warrior Jacob Lillyman with Asthma Nurse Ann Wheat being tested on the NIOX Analyser

We will be able to test children from about the age of five with the new FENO machine. It is an easy fun test to do with screen images that help the person to perform the test correctly. It is advisable to have no food, drink or exercise for one hour before the test.

Asthma is divided into two main classifications, allergic or non-allergic. The FENO will differentiate between the two types as allergic asthma causes more inflammation in the lungs, hence more exhaled NO.

The nurses at Asthma Auckland are looking forward to the opportunity to demonstrate to children and adults the level to which their asthma could be controlled.

Jacob Lillyman filling in the pre Spirometry Questionaire, Asthma Nurse Elaine Murray getting the Spirometer set up for use.

Vodafone Warrior Kevin Locke with Asthma Nurse Ann Wheat being tested on the NIOX Analyser

Asthma New Zealand are very grateful to Vodafone Warriors Jacob Lillyman and Kevin Locke for giving up their time to attend our open day and be the first to use our new NIOX Analyser which was purchased from the proceeds of fundraising efforts of "Team Logan." This machine is dedicated to Logan Hartnoll, aged 4, who died of asthma on 20 February 2010. We are extremely grateful to the Hartnoll family and their tireless group of volunteers!

asthma wellington has a brand new team...

(Pictured from left, Kim Jansen, Patricia Sullivan and Adie Riddell)

Meet our two new Asthma Nurse Educators both of whom started earlier this year ; Adie Riddell has many years of experience in Public Health Nursing, and Patricia Sullivan from Cardiac Diagnostic Services, Patricia is also a qualified Naturopath with a strong focus on allergies.

Kim Jansen, our Office Administrator and Fundraising Coordinator has come from an extensive administration background with an interest in health and fitness.

Together they are passionate about growing the service to the Wellington area and to raise public awareness about Asthma in New Zealand and promotion of their free education sessions they offer to people with Asthma, COPD and other respiratory problems.

copd and smoking cessation interventions

Compiled by Janet Delooze RN

As Asthma Nurse Educators, we are very aware of the impact of smoking on health particularly the detrimental effects on lung function. We all strive to offer information, advice and support to people who smoke. Even if we are not the ones to continue this support, there are smoking cessation practitioners who seek to help people to give up smoking. There are many interventions that may help smokers to succeed in their quest to live a smoke-free life. However, how effective are these interventions and strategies, and why are some more successful than others?

Many smoking cessation interventions have shown impressive short term results, particularly stopping smoking abruptly, however, the relapse rate is high with only 30% achieving long term success [1]. Smoking has been described as both a behavioural disorder and a chemical addiction therefore the most effective strategies must address both aspects: this would include counselling and support strategies together with medications; pharmacotherapies double the success rates and behavioural techniques can further increase the quit rate [2].

The C.O.P.D. Board suggest that brief counselling is effective and every smoker should be offered at least this intervention at every visit: interventions by health professionals at any stage are helpful [3]. Cessation of smoking is a process rather than a single event and Siafakas et al [1] suggest that patients need to be encouraged along the cycle of contemplation of stopping smoking, positive action and relapse many times before they achieve success. However, West [4] challenges the usefulness of the Stages of Change model so subsequently all references to this has been removed from the current New Zealand Smoking Cessation Guidelines [5].

Current best practice guidelines on the 5-A strategy, recommended by Pauwels et al [6], suggest the following:

*"**A**sk and identify smokers; **A**dvise smokers about the risks and benefits of quitting and discuss options; **A**ssess the degree of nicotine dependence and motivation or readiness to quit; **A**ssist cessation – this may include specific advice about pharmacological interventions or referral to a formal cessation program if available; **A**rrange follow-up to reinforce message."*

However, the New Zealand Smoking Cessation Guidelines (Ministry of Health, 2007) promote the Smoking Cessation ABC's which stand for **A**sk (about and document smoking status for all people), **B**rief advice (give clear advice that is personalised and document this) and **C**essation (either provide support or referring on to people who are specialised in smoking cessation).

Siafakas et al [1] advocate that firstly, there needs to be an explanation of the effects of smoking and the benefits of stopping, advice on helpful strategies and encouragement of healthy lifestyle changes. Secondly, they suggest that more intensive support is needed, including Nicotine Replacement Therapy [NRT], behavioural interventions and group or individual programmes as these have all been shown to increase success rates.

Support is most needed in the early days and engaging the support of friends and family in this endeavour will increase the chances of success [7]. Sources outside of friends and family that may offer face-to-face or telephone support include GP surgeries, respiratory professionals and Quitline. The family doctor can prescribe medications to support smoking cessation including NRT, some antidepressants,

such as Bupropion and Nortriptyline, and Varenicline which works by binding to the nicotine receptors in the brain [5]. There are also Smoking Cessation Practitioners who can provide NRT after they have completed appropriate smoking cessation training.

Interventions on a wider scale are also helpful: discouragement of smoking in public places and by media advertisement are useful ways to discourage smoking [1]. A successful tobacco control strategy involves integration of public policy, information dissemination programmes and health education through the media and schools[7].

The Ministry of Health [MOH] [5] have identified certain priority population groups, for example, Maori, because of their high smoking prevalence rates: interventions that have been proven to be effective should also be used for these groups, however, they must be acceptable, accessible and appropriate for each particular group.

So why aren't some people's attempts at smoking cessation successful? One of the reasons may be that smoking involves an addiction to nicotine and also, the habit of smoking when certain cues are present. This behavioural disorder and chemical addiction has been described as a dual reinforcement model [9]. Therefore, it could be deduced that if both aspects are not addressed then smoking cessation is unlikely to be achieved. Nicotine affects the mood of the smoker in a complex way as it can either stimulate or relax the smoker, and addiction is reinforced by a cycle of high and low levels of nicotine [10]. Nicotine withdrawal can result in restlessness, increased appetite, inability to concentrate, irritability, dizziness and nicotine craving [11].

According to Percival [11], around 70% of smokers actually want to quit, although only about 45% make a serious attempt in each year [12], and as mentioned previously, 70% of smokers who give up abruptly without preparation are not successful. Therefore, it is helpful to have strategies and support to help deal with withdrawal symptoms, and a quit date determined in advance.

Many people find behavioural change like smoking cessation difficult, particularly those who are socially or economically disadvantaged, those living in stressful family situations or those who have little or no social support [13]. Some people may find that their personal circumstances influence their motivation to quit smoking. Decramer et al [14] suggest that an individualised approach and close support will improve smoking cessation outcomes.

Other factors that can influence people's success with smoking cessation are their beliefs, attitudes and personal attributes. Self-efficacy, an individual's belief that he or she is capable of achieving certain goals, generally gives people more of a positive attitude towards a difficult challenge and greatly influences their determination to quit smoking [15]. Donatelle [15] adds that people with an external locus of control feel that they have limited control over their lives and lack the confidence to succeed in a particular behaviour, whereas those with an internal locus of control are more apt to take action because they think it's important and feel they are in charge of the situation.

Most smokers who attempt to quit do not use cessation aids and as a result they are usually unsuccessful with two-thirds relapsing in the first 48 hours [16]. Studies have shown that pharmacotherapies are twice as likely to be effective than placebos [17]. Some smokers try to cut down in the belief that this will help them to quit. Gradual withdrawal may be successful in reducing tobacco consumption but is generally unsuccessful in achieving smoking cessation [1].

In conclusion, C.O.P.D. is a preventable and treatable, though not currently curable, disease that becomes progressively worse over time. It is a complex disease state which involves mechanisms of inflammatory changes and airflow limitation, impacting on gaseous exchange. Exposure to long term irritants can predispose people to the development of C.O.P.D., the main irritant being cigarette smoking. As smoking is both an addiction and a behavioural disorder, smoking cessation interventions need to address both aspects. The use of pharmacotherapy in combination with behavioural support achieves higher cessation rates than either component alone and is the most effective way of helping smokers to stop.

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what conditions make up chronic obstructive pulmonary disease (COPD) and how do they differ?

compiled by Ann Wheat RN BN

Chronic Obstructive Pulmonary Disease (COPD) is defined by The World Health Organization (2011) as a lung disease characterized by chronic obstruction of lung airflow that interferes with normal breathing and is not fully reversible. The condition is usually progressive and is associated with an abnormal inflammatory response in the lungs to noxious particles or gases (National Heart, Lung & Blood Institute (NHLBI), 2010). COPD is an umbrella term for at least three conditions such as Chronic Bronchitis, Emphysema and Chronic uncontrolled Asthma. Other conditions such as Bronchiectasis and Cystic Fibrosis have also sometimes been discussed under the heading of COPD.

For this article, I will focus on Emphysema, Chronic Bronchitis and Chronic uncontrolled asthma. For any of these conditions if a patient has symptoms of cough, sputum production or dyspneoa plus a history of exposure to risk factors such as cigarette smoking, dust particles, air pollution, chemicals or fumes, then a diagnosis of COPD should be considered (NHLBI, 2010). The diagnosis of COPD is usually confirmed following a spirometry test which would show the presence of irreversible airflow limitation.

Emphysema

Emphysema affects the air sacs (alveoli) in our lungs. The condition is not only triggered by the various factors that can cause COPD but can also be caused by a hereditary condition. This heredity condition is caused by a reduction in a protein called Alpha 1 Antitrypsin which has a protective effect on the alveoli in the lungs by protecting them from an enzyme called Neutrophil Elastase which can cause lung damage. In Emphysema, the alveoli are progressively destroyed and simplistically put instead of looking like a bunch of grapes they look like one large grape. This destruction reduces the surface area of the alveoli which in turn reduces the amount of oxygen that can be absorbed into the body. The loss of the alveolar walls results in a decrease in elastic recoil causing airflow limitation (Mosenifar, 2011). He goes on to say that airflow limitation is further affected by the loss of the alveolar supporting structures which causes airway narrowing.

Chronic Bronchitis

Chronic Bronchitis affects the bronchioles in the lungs. It is caused by the constant inhalation of irritants such as cigarette smoke or pollutants. The first indication for the condition in most people is increased mucous production and in fact Chronic Bronchitis is diagnosed when there is daily sputum production for 3 months of a year for two consecutive years. This mucous comes mainly from the trachea (windpipe) and the main bronchi from the mucous cells and in the lower airways from the goblet cells. The constant inhalation of cigarette smoke in particularly, causes repetitive injury to the airways, which in turn causes inflammation, ciliary abnormalities, muscle hyperplasia and bronchial wall thickening or scarring (Mosenifar, 2011). The cilia or tiny hair like projections in the lungs that help to clear the mucous or other foreign particles that get into the lungs are damaged to such an extent that they no longer work. As a result of this the excessive amount of mucous that is produced cannot be cleared from the lungs and can therefore go on to cause an increased risk of infections from the pooling of the mucous. The excessive amount of mucous can also occlude the small airways again being a trigger for infection in the lung. The constant inflammatory response can cause vasodilation, congestion and mucosal oedema. All of these issues can cause airway narrowing which will result in a reduction of airflow. Further to this bronchospasm can develop in a patient with chronic bronchitis which increases the work of breathing and further impairs gas exchange (Lindell & Van Sciver, 2000)

Chronic Uncontrolled Asthma

Asthma is a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role, in particular, mast cells, eosinophils, T lymphocytes, macrophages, neutrophils, and epithelial cells. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment. The inflammation also causes an associated increase in the existing bronchial hyper-responsiveness to a variety of stimuli (National Asthma Council of Australia, 2006).

Asthma is a condition that can normally be well controlled with medication when used as prescribed. If asthma is left uncontrolled for long periods, it can cause continual symptoms of coughing, shortness of breath, wheezing and chest tightness which in turn causes the airways to become permanently damaged from the untreated inflammation. The muscles hypertrophy due to hyper-responsiveness of the airways. These cause the airways to narrow thus causing the loss of reversibility. Reversibility is the hallmark for well controlled asthma.

Occupational asthma can also cause Chronic Obstructive Pulmonary Disease. Occupational asthma is triggered by prolonged exposure to inhaled irritants such as chemicals and this can happen even after the person has been removed from the situation.

It is known that 1 in 10 people who develop asthma when young will go on to have a degree of fixed obstructive airflow and for those who develop asthma in adulthood the rate is as high as 1 in 4. Cigarette smoking exacerbates the situation (Bellamy & Booker 2008).

In conclusion, there are three main conditions covered by Chronic Obstructive Pulmonary Disease namely chronic bronchitis, asthma and emphysema. It is important to ensure that the condition is recognized as early as possible and treated effectively. Smoking is a major confounder and it is essential that people stop smoking at soon as the diagnosis is made.

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Source: J Asthma

Gender Differences in Perception of Dyspnea, Assessment of Control, and Quality of Life in Asthma;

Chhabra SK, Chhabra P; Journal of Asthma (Jun 2011)

Background. There is limited information on the inter-relationship between gender, perception of dyspnoea and health-related quality of life (HRQoL) in asthma. Methods. In a cross-sectional study in an out-patient setting, 85 patients with bronchial asthma, 41 males and 44 females, underwent spirometry and were administered the following instruments to measure asthma control, HRQoL and dyspnoea : (a) Asthma control questionnaire (ACQ), (b) Asthma Quality of Life questionnaire (AQLQ), (c) Baseline dyspnoea index (BDI) questionnaire and Oxygen Cost Diagram (OCD). Results. Overall, male patients had greater airways obstruction but reported similar level of asthma control as females. Among patients with mild persistent asthma, females had a poorer level of control. The BDI and the OCD scores were significantly lower in female patients indicating greater dyspnoea and they also had a poorer quality of life especially in the symptoms and emotional domains of the AQLQ. After adjusting for the severity of airways obstruction in multivariate analysis, female gender and a poorer quality of life were independent predictors of increased perception of dyspnoea. Conclusions. Female patients with asthma are likely to have a greater perception of dyspnoea, report a poorer control and have a poorer quality of life as compared to males. Female gender and a poorer quality of life are independent predictors of increased perception of dyspnoea in asthmatics.

Source: Pediatr

Folic Acid Use in Pregnancy and the Development of Atopy, Asthma, and Lung Function in Childhood;

Magdelijns FJ, Mommers M, Penders J, Smits L, Thijs C; Pediatrics (Jun 2011)

Background: Recently, folic acid supplementation during pregnancy was implicated as a potential risk factor for atopic diseases in childhood. Objective: To investigate whether folic acid supplementation and higher intracellular folic acid (ICF) levels during pregnancy increase the risk of childhood atopic diseases. Methods: In the KOALA Birth Cohort Study (N = 2834), data on eczema and wheeze were collected by using repeated questionnaires at 3, 7, 12, and 24 months, 4 to 5 years, and 6 to 7 years after delivery. Atopic dermatitis and total and specific immunoglobulin E levels were determined at age 2 years and asthma and lung function at age 6 to 7 years. We defined folic acid use as stand-alone and/or multivitamin supplements according to the period of use before and/or during pregnancy. ICF levels were determined in blood samples taken at ~35 weeks of pregnancy (n = 837). Multivariable logistic and linear regression analyses were conducted, with generalized estimating equation models for repeated outcomes. Results: Maternal folic acid supplement use during pregnancy was not associated with increased risk of wheeze, lung function, asthma, or related atopic outcomes in the offspring. Maternal ICF level in late pregnancy was inversely associated with asthma risk at age 6 to 7 years in a dose-dependent manner (P for trend = .05). Conclusions: Our results do not confirm any meaningful association between folic acid supplement use during pregnancy and atopic diseases in the offspring. Higher ICF levels in pregnancy tended, at most, toward a small decreased risk for developing asthma.

Source: Rev Mal Respir

Patients' illness perceptions and adherence to treatment with inhaled corticosteroids in asthma;

Charles C, Ninot G, Sultan S; Revue des Maladies Respiratoires 28 (5), 626-635 (May 2011)

Introduction: Regular use of inhaled corticosteroids as preventive treatment of asthma is an integral part of management but remains inadequate among adults. Studying the perceptions of illness and treatment beliefs is one way to understand the patient's adherence to medication. Method: A systematic review was performed of empirical studies in adults published between 1999 and 2009, and indexed in the Pubmed, PsycInfo and Scopus databases. We investigated the associations between (1) perceptions of asthma and treatment beliefs and (2) adherence to inhaled corticosteroids. Eighteen articles meet these criteria. Results: Perception of the chronicity of asthma and its consequences on daily life, as well as the concept that it is necessary to continue treatment in the absence of symptoms, are associated with better adherence. On the contrary, fear of side effects and the belief that treatment is ineffective in controlling symptoms, are associated with poor adherence. Conclusion: Patients' perceptions of asthma and inhaled corticosteroids are predictors of adherence to treatment. The identification and discussion of these issues is an essential part of building a therapeutic relationship that facilitates adherence

Source: Thorax

Genome-wide association study of smoking behaviours in patients with COPD;

Siedlinski M, Cho MH, Bakke P, Gulsvik A, Lomas DA, Anderson W, Kong X, Rennard SI, Beaty TH, Hokanson JE, Crapo JD, Silverman EK, the COPDGene Investigators and ECLIPSE Investigators; Thorax (Jun 2011)

Background: Cigarette smoking is a major risk factor for chronic obstructive pulmonary disease (COPD) and COPD severity. Previous genome-wide association studies (GWAS) have identified numerous single nucleotide polymorphisms (SNPs) associated with the number of cigarettes smoked per day (CPD) and a dopamine beta-hydroxylase (DBH) locus associated with smoking cessation in multiple populations. Objective To identify SNPs associated with lifetime average and current CPD, age at smoking initiation, and smoking cessation in patients with COPD. Methods GWAS were conducted in four independent cohorts encompassing 3441 ever-smoking patients with COPD (Global Initiative for Obstructive Lung Disease stage II or higher). Untyped SNPs were imputed using the HapMap (phase II) panel. Results from all cohorts were meta-analysed. Results Several SNPs near the HLA region on chromosome 6p21 and in an intergenic region on chromosome 2q21 showed associations with age at smoking initiation, both with the lowest $p=2\times10(-7)$. No SNPs were associated with lifetime average CPD, current CPD or smoking cessation with $p<10(-6)$. Nominally significant associations with candidate SNPs within cholinergic receptors, nicotinic, alpha 3/5 (CHRNA3/CHRNA5; eg, p=0.00011 for SNP rs1051730) and cytochrome P450, family 2, subfamily A, polypeptide 6 (CYP2A6; eg, $p=2.78\times10(-5)$ for a non-synonymous SNP rs1801272) regions were observed for lifetime average CPD, however only CYP2A6 showed evidence of significant association with current CPD. A candidate SNP (rs3025343) in DBH was significantly (p=0.015) associated with smoking cessation. Conclusion The authors identified two candidate regions associated with age at smoking initiation in patients with COPD. Associations of CHRNA3/CHRNA5 and CYP2A6 loci with CPD and DBH with smoking cessation are also likely of importance in the smoking behaviours of patients with COPD.

Source: Lung

The Relationship of the BODE Index to Oxygen Saturation During Daily Activities in Patients with Chronic Obstructive Pulmonary Disease;

Cutaia M, Brehm R, Cohen M; Lung (Jun 2011)

Background: The frequency of oxygen desaturation during daily activities in chronic obstructive pulmonary disease (COPD) is poorly

defined. The BODE index predicts survival in COPD. The purpose of this study was to determine the relationship between BODE scores and oxygen saturation during daily activities. Methods: Seventy-eight patients with COPD (FEV(1) = 37%) underwent ambulatory oximetry and activity monitoring. We defined four activity categories: Walking, Slow-Intermittent-Walking (SIW), Active-Not-Walking (ANW), and Rest. We quantified oxygen desaturation during activity using a desaturation index (DSI = % time oxygen saturation<90%). BODE scores were categorized into three groups: group I (0-3), II (4-6), and III (7-10). Results: The percentage of patients demonstrating oxygen desaturation (DSI ≥ 10%) during each activity was 55% for Walking, 35% for SIW, 15% for ANW, and 28% for Rest. There was a strong association between BODE score and desaturation for Walking and SIW. During Walking, 21, 44, and 86% of patients in BODE groups I, II, and III, respectively, demonstrated desaturation. The DSI for Walking and SIW was increased in patients in BODE groups II and III compared to group I ($P < 0.006$, $P < 0.007$, respectively). BODE score was also linked to long-term oxygen therapy (LTOT) usage; the majority of patients not on LTOT (89%) had a BODE score<7. The majority of patients on LTOT (84%) demonstrated desaturation during Walking, but 42% of patients not on LTOT also demonstrated desaturation. In this subgroup of patients not on LTOT, all patients with a BODE score ≥7 demonstrated desaturation during Walking. Conclusions: The link between the BODE index and oxygen desaturation during daily activities suggests that desaturation is linked to disease severity. Our data suggest that patients with a BODE score ≥7 should be evaluated for desaturation during daily activities. Use of the BODE index to screen for exertional desaturation may have value as a tool that can lead to the earlier identification of patients who may be candidates for LTOT.

Source: Respir Med

Clinical and economic burden of patients diagnosed with COPD with comorbid cardiovascular disease;

Dalal AA, Shah M, Lunacsek O, Hanania NA; Respiratory Medicine (Jun 2011)

Background: Cardiovascular disease (CVD) and chronic obstructive pulmonary disease (COPD) commonly coexist, increasing the risk of hospitalization and mortality compared to either condition alone. The purpose of this study was to evaluate the impact of comorbid CVD on healthcare utilization and costs in a COPD population. METHODS: A retrospective cohort study of COPD patients CVD ± ≥40 years of age using administrative claims data was conducted. COPD-CVD patients were matched to COPD patients without CVD (COPD-Only cohort) using propensity scores. Multivariate analyses were conducted to assess the 1-year risk of COPD exacerbations (hospitalization and/or emergency room [ER] visits), along with differences in 1-year and 2-year all-cause and COPD-related utilization and costs (2008 USD) among COPD-CVD and COPD-Only cohorts. Results: Each cohort included 4594 patients. Compared to COPD-Only cohort, the COPD-CVD cohort was almost 2 times more likely to require COPD-related hospitalization (odds ratio [OR], 1.95; $p < 0.001$), 47% more likely to have an ER visit (OR, 1.47; $p < 0.001$) and 62% more likely to require hospitalization and/or ER visit (OR, 1.62; $p < 0.001$). Average annual all-cause medical costs per patient were $22,755 for COPD-CVD vs $8036 for COPD-Only ($p < 0.001$), and total costs were $27,032 vs $11,506 ($p < 0.001$), respectively; corresponding average COPD-related annual medical costs were $1891 vs $1060 ($p < 0.001$) and total costs were $3295 vs $2379 ($p < 0.001$). Conclusions: COPD patients with CVD have significantly higher risk of COPD exacerbations and increased costs than COPD patients without CVD. This suggests a close association between COPD and CVD that warrants further exploration.

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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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