THE NZ JOURNAL OF RESPIRATORY HEALTH
April 2014

O2

a commitment to our pacific community...

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THE NZ JOURNAL OF RESPIRATORY HEALTH O_2

contents

On the cover: Aorere College – a commitment to our pacific community... *see page 16-17*
Photo by Ben Campbell Photography

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Asthma/COPD Nursing Course Information

Dear Nurse

Applications are now invited from nurses wanting to enrol on the Asthma Nursing Course in July 2014 and COPD Nursing Course in April 2014. The programmes are offered by distance learning. Not everyone has the same pace of learning. Some students pick up things fast, others need time to grasp a concept. One of the biggest advantages of distance learning is that you can study at a pace that is comfortable for you. The primary aim of Asthma/COPD Nursing Courses are to provide nursing health professionals with a high level of Asthma/COPD knowledge that promotes best practice, based on available evidence, and is consistent with national policy.

Since the commencement of the Asthma and COPD Nursing Courses, over 1000 nurses have enrolled over 44 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 Nursing Course and COPD Nursing Course are accredited with 15 credits each, which can be used towards gaining your Bachelor of Nursing degree.

If possible would you be able to pin-up the following Asthma and COPD Nursing Course information on your work place notice board. Also feel free to circulate, make photocopies if you like.

Could you please phone/fax or email for an enrolment form.

Asthma Nursing Course closing date – 10th of July 2014

COPD Nursing Course closing date – 10th of April 2014

For information contact:
Ann/Swarna
Phone Ann on 09 623 4777 or Swarna on 09 623 4771
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Upcoming events and courses

ASTHMA NEAT COURSE
18 June 2014
17 September 2014

HALF DAY COPD COURSE
16 April 2014
16 July 2014
15 October 2014

SUPER 8
8 FIGHTERS, SEVEN FIGHTS, ONE CHAMPION
Wednesday 4th June 2014 – The Trusts Arena, Auckland
See back page for all the details.

WORLD ASTHMA DAY

Tuesday 6th May 2014

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

message to readers

With the second issue of O_2 during my time as CEO, I would like to introduce to you the ways in which I am addressing some of the challenges Asthma New Zealand faces.

With World Asthma Day (Tuesday, 6th May 2014) approaching, it's time to turn our attention to funding. It's common knowledge that asthma-related health problems create enormous costs for New Zealand. Asthma-induced hospital admissions can cost thousands of dollars per person. Add to that the loss of productivity in the workplace, and it's easy to see how the costs add up.

Why then, is this not being addressed? Asthma continues to affect one in five New Zealanders, yet due to lack of support, local societies are folding and tired committees are unable to secure much-needed funding. It is becoming increasingly difficult for organisations to make ends meet, let alone make any headway in the bigger issues that those with asthma face every day.

I recently wrote to Labour Health Spokesperson and Health Committee member Annette King, requesting her assistance in helping us to receive nationwide funding for Asthma NZ. Our vision is to employ more nurses to meet the demands of the community, ensure ongoing education and continue to make a difference in the lives of the fifth of the NZ population who are affected by asthma. National funding will allow us to achieve a high standard of nurse training nationwide, provide comprehensive education services to the community and support people from their own homes.

I hope to meet with Annette to discuss this further. Annette is a Patron of Asthma Wellington, and has previously stated that she would like asthma to be a government priority. With government support, Asthma New Zealand will be better equipped to address the systemic issues that exacerbate asthma-related problems, rather than just treat people's symptoms when hospitalised. We will be in a position to refer people for improvements to their homes such as insulation, as well as take a whanau-ora approach, which will provide quality healthcare and education catering to Maori and Pacifica communities.

In order to raise Asthma New Zealand's profile, draw attention to the services we provide, and encourage new members, we are increasing our social media presence. We'd love to hear from you on Facebook, and you can follow us on Twitter at @asthmanz.

I'd like to thank you all for your support in our work to improve the lives of New Zealanders with asthma. With the cooler months approaching I hope you all stay warm and healthy while enjoying New Zealand's beautiful outdoor spaces.

Linda Thompson
CEO

bronchial thermoplasty a new non-pharmacologic treatment for asthma

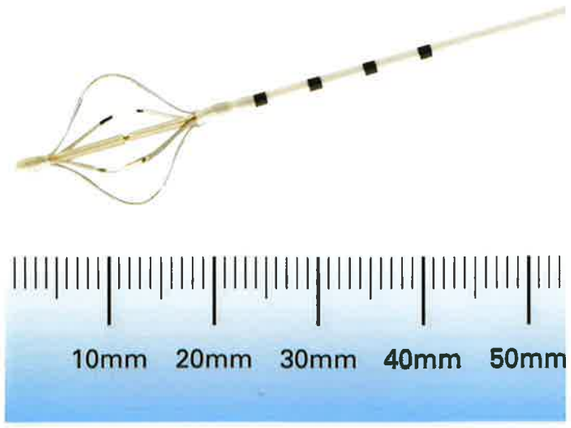
by Karen Little RN
Asthma Nurse Educator

Bronchial thermoplasty (BT) was approved by the United States Food and Drug Administration (FDA) in 2010.[1] The FDA is led by the Commissioner of food and drugs who is appointed by the President of the United States with consent from the senate. The FDA approved the Alair® Bronchial Thermoplasty System for the treatment of severe persistent asthma in patients 18 years and older whose asthma is not well controlled. They must have been taking inhaled corticosteroids and long acting beta agonists. The FDA based its approval on data from a clinical trial of 288 adult subjects; the Asthma Intervention Research 2 (AIR2) trial, with severe and persistent asthma. Of 580 subjects screened 190 subjects went into the BT group and 98 subjects in the sham group. This was the largest ever randomised, double-blind, sham-controlled clinical trial to test a new device for the treatment of asthma. The BT subjects experienced fewer severe exacerbations (33%) emergency department visits (84%) hospitalisations for respiratory symptoms (73%) and days missed from work/school (66%). The majority of respiratory adverse events occurred within one day of the bronchoscopy and resolved within 7 days.[2]

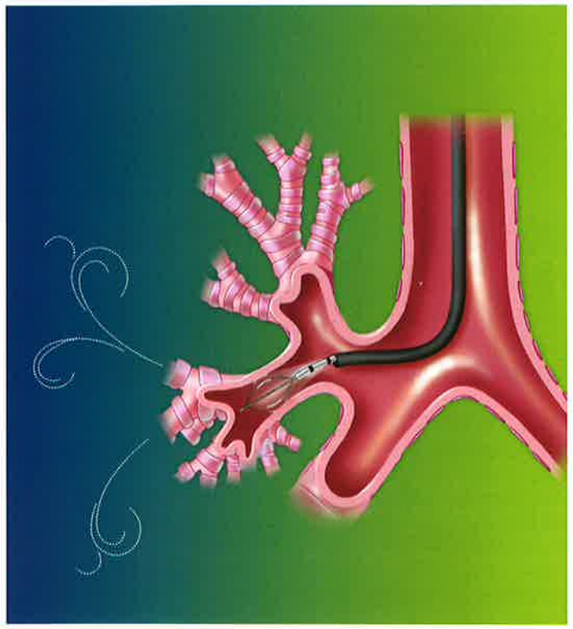
The FDA required a five-year post-approval study of the device to study its long term safety and effectiveness. One hundred and sixty-two (85.3%) of 190 BT treated subjects completed 5 years of follow up. The average 5-year reduction in exacerbations was 44% and 78% for ED visits. Prebronchodilator FEV_1 values remained stable between years 1 and five after BT, despite an 18% reduction in average inhaled corticosteroid dose. Postbronchodilator FEV1 remained higher at all times; CT scans from baseline to 5 years after BT showed no structural abnormalities that could be attributed to BT.[3] The sham control group was not followed up over five years as it was deemed to be unethical in this study after breaking the blind and requiring them to continue the same treatment regimen despite poor control.

The Alair System delivers controlled therapeutic radiofrequency energy to a prespecified temperature of 650C to the airway walls during a series of three bronchoscopies. The small diameter Alair catheter is delivered into the airways through the working channel of the bronchoscope; four arms of the expanded tip of the catheter come in contact with the airway wall. The complete treatment is performed in three separate procedures each covering different regions of the lung. The tip of the catheter heats the tissue and reduces the amount of bronchial smooth muscle (BSM) present in the airway wall.

Tip of the Alair catheter



The Alair catheter in the working channel of the bronchoscope



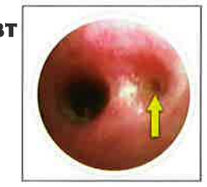
An increase in the mass of airway smooth muscle is evident in patients with asthma and this increase has been shown to correlate with asthma severity. Major anti-asthmatic treatments such as corticosteroids remain totally ineffective in decreasing BSM.[4] BT aims to reduce BSM by 30-40%. Although there are many different triggers, an acute asthma attack is always characterised by contraction of the smooth muscle in the airway wall. Consequently after this treatment there is decreased potential for bronchoconstriction and possibly decreased frequency and severity of asthma symptoms. BT treatments target the airways distal to the mainstream bronchi down to airways 3mm in diameter.[5] The epithelium (which does regenerate) sloughs off and is coughed up. There is no scarring, charring or burning. It has been suggested that the body contains many organs which no longer appear to be required such as the appendix. It has been argued that BSM may fall into this category and that its only contribution to us is its potential to cause problems.[6] Although asthma is usually defined as being an inflammatory disease the link remains unclear with airway hyper responsiveness. It has been established that airway hyper responsiveness can be separated from airway inflammation.[7]

The Therapeutic Goods Administration in Australia has conducted the evaluation to list the Alair Bronchial Thermoplasty System on the Australian Register of Therapeutic Goods, and the product has been used there. I was informed by the Product Safety Team at Medsafe that there is no pre-market approval process for medical devices in New Zealand. There is however a regulatory requirement for suppliers of medical devices to notify details of the products they supply here to a database known as the Web Assisted Notification of Devices, or WAND, database. This information is collected on behalf of the Director-General of Health about the range of medical devices supplied in New Zealand so that in the event of a post-market issue – adverse event, recall, etc., – Medsafe is able to identify whether the product is available here or not. Unfortunately there is no public access to the WAND database and Medsafe cannot release the information. Boston Scientific NZ Ltd who are the suppliers of the device did supply to me a copy of the Wand data base notification for the electrosurgical diathermy system, and the electrosurgical endotherapy electrode, single use.

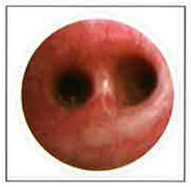
One in six New Zealand adults has asthma. The direct and indirect cost of asthma in the late 1990's was conservatively estimated to be over 800 million.[8] Imagine what new figures would tell us.

If subjects meet the criteria, BT may well be a new way to reduce the symptoms of severe persistent asthma in adults.

Airway before BT



Airway after BT



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dear nurse

Dear Nurse, I am flying overseas and am worried about taking my asthma inhalers on the flight. Will they explode in the high altitude and will they be counted as liquid?

It is best to take your puffers in your hand luggage in a plastic bag. To be certain that you will not be delayed by security have the puffers in the original pharmacy packaging labelled with your name. I would also bring along a letter from your doctor explaining the medicine's purpose and verifying that a doctor prescribed it for you. As a guide most inhalers contain approximately 10-20ml of liquid. Each container must not have more than 100mls of liquid in it. Puffers in hand luggage will not explode as the cabin is pressurised. If your puffers are in the hold they may become very cold and may not be as effective. Have your asthma well controlled before you fly and know the generic names of your medicines.

Dear Nurse, I have been told that the Volumatic® Spacer is better than the small volume spacer. Why should I use a Volumatic® Spacer?

The Volumatic® Spacer is recommended for all people over the age of 6 years. It has been shown to deliver more medication to the lungs than the small volume spacer. This means that there is more medication available in the spacer to be breathed in thus increasing the delivery of medication to the lungs to 40% and as a result if you are using a preventer medication it will help to control your asthma even more. Spacers should be used with all metered dose inhalers (MDIs) as they more than double what reaches the lungs versus using an MDI on its own. They also help to reduce the side effects that some people have from using preventer medication. Remember to wash your Volumatic® spacer weekly in warm soapy water, not to rinse or dry.

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

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Unitec NEW ZEALAND Te Whare Wānanga o Wairaka

Heartiest congratulations on successfully completing Unitec/Asthma New Zealand Asthma Nursing Course 2013 – 2nd semester

Rebecca Amy Atkinson _____ Auckland
Carolyn Margaret Duncum ____ Central North Island
Donna Lee Hesp _____ Wellington
Jayasmitha Jose Karumalil _____ Auckland
Donna Patricia Marshall _____ Christchurch

Alice Mary Paul _____ Wellington
Victoria Pizarro-Paulino _____ Whangarei
Starla Titus Pothiyil _____ Wellington
Anju Dahiya Sangwan _____ Christchurch

Heartiest congratulations on successfully completing Unitec /Asthma New Zealand COPD Nursing Course 2013 – 2nd semester

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Marianne Eliasen _____ Palmerston North
Jayasmitha Karumalil _____ Auckland
Somchit Kunraksa _____ Waitakere
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Vicki Lyford _____ Timaru
Janet McGivern _____ Bay of Island
Brigid Neilson _____ Hastings
Joan Rafols _____ New Plymouth
Julie Shaw _____ Hestings
Diane Wade _____ Blenheim

peak flow monitoring in asthma

by Elaine Murray RN
Asthma Nurse Educator

Asthma can cause narrowing of the bronchial tubes (airways) and as a result, difficulty moving air through the airways into and out of the lungs, resulting in a tight chest, shortness of breath, coughing and wheezing.

A peak flow meter for asthma is like a thermometer for a fever. Both are tools to help monitor what is going on in your body. For instance, there may be times when you feel feverish, but when you take your temperature with a thermometer it is normal. With asthma, sometimes you may feel your breathing is fine, but when you measure it with your peak flow meter your lung function is slightly decreased.[1]

A peak flow meter can help you and your doctor evaluate how severe your asthma is at any point of time. With a peak flow meter you can often see a drop in your reading even before symptoms get worse. Decreases in peak flow may show that you need to increase your medication, such as adding in the use of your reliever for a cold.

Variability in peak flow, especially at night, is another indication that perhaps the asthma is not well controlled.

The peak expiratory flow (PEF), also called peak expiratory flow rate (PEFR), measures how fast air comes out of the lungs, i.e. it measures the flow rate generated during a forceful exhalation following full lung inflation[2], therefore you need to use a good technique otherwise the result is not accurate.

The most frequent use of a peak flow rate measurement is in "home monitoring" of asthma, where it can be beneficial for both short-term and long-term monitoring. When properly performed and interpreted, peak flow rate measurement can provide the person and the doctor with objective data upon which to base therapeutic decisions.[2]

Since peak flow rate measurement depends significantly on patient effort and technique, clear instructions, demonstrations, and frequent review of technique are essential.

There is some conflicting data regarding the efficacy of peak flow monitoring, but most studies have shown a benefit when peak flow rate monitoring is linked to symptom diaries and patient education.[2]

Another study suggested that with symptom-based monitoring, some people under estimate the severity of their condition.[2]

Many people who have had asthma for a long time feel that they can judge the severity of an attack by their symptoms alone, but studies have shown that more than half the time people with asthma will be mistaken in their estimates.[3]

Another measure of asthma control is peak flow variability. For many people with asthma, symptoms are usually worse at night. By consistently monitoring night time peak flow measurements, you can tell how well your asthma is controlled. A decrease of 15% or greater from your usual measurements may be a sign of night time asthma.[1]

In 2007, an expert panel of the National Asthma Education and Prevention Program recommended periodic assessment of pulmonary function by spirometry or peak flow rate monitoring.[2] If peak flow rate monitoring is used, the person should have a written asthma management plan and know what their personal best peak flow is.

The panel recommended consideration of long-term daily peak flow rate monitoring or home peak flow rate assessment during exacerbations for people with the following;

- Moderate or severe persistent asthma
- History of severe exacerbations
- Poor perception of airflow obstruction and worsening asthma
- Preference for peak flow rate monitoring rather than the use of a symptom-based action plan

In managing chronic asthma, long-term daily peak flow rate monitoring may assist with the following;

- Detecting early changes in asthma that may require therapy
- Evaluating responsiveness to changes in therapy
- Giving a quantitative measurement for improvement

It can be very useful to assist in the diagnosis of asthma, including occupational asthma, and also to help in identifying triggers.

People with asthma who are "poor perceivers" have few symptoms when their airways narrow. People with under-treated asthma who have become used to chronically poor lung function do not recognise or under-estimate worsening asthma. There are some people who do not develop symptoms in response to a large fall in lung function.[4]

However, the use of peak flow rate during an acute asthma exacerbation is controversial. The 2007 Expert Panel Report suggested that measuring peak flow rate in acute episodes helps to determine the severity of exacerbations and assists in guiding therapeutic decisions whether at home, at school, in the GP's rooms or emergency departments[2], especially if it is known what that persons personal best peak flow is.

So, what is your personal best peak flow? Your personal best peak flow is the highest peak flow number you can get over a two or three week period when you are well and your asthma is well controlled.

Your personal best peak flow is important because it is the number to which all of your other peak flow recordings will be compared and it is the number that will be used as your 100% best on your management plan (green zone). If your peak flow drops to 85% or less (yellow zone) of your personal best your asthma is getting worse. If it continues to drop to 60% or less (orange zone) then you need to seek medical help. If it is 40% or less (red zone) it is an emergency.

Everyone with asthma should have an asthma management plan that is filled in by their GP telling them what medications they need to take to keep their asthma under control when well, what to "add in" as their asthma gets worse and more importantly, what to do in emergencies.

How do you find your personal best?

Peak flow meters are free from your GP. To do a peak flow you need to take a deep breath, as deep as you can, then put the mouth piece in your mouth, seal your lips tightly around the mouth piece and then blow out through the peak flow meter with a short sharp forceful breath. Use your chest and abdominal muscles to force the air out of your lungs as quickly as possible. Do this while standing up.

Make sure your fingers do not interfere with the indicator (i.e. stop the indicator moving up the peak flow meter). Record the level that the indicator stops at. Return the indicator to zero. Do two more blows. Record the highest that you got.

Do your peak flow recording twice a day at the same time for 2-3 weeks, when you are well!

Always use the same meter.

The expected peak flow rate will depend on height, age and gender for an adult, and height only for children. Ask your practice nurse to measure your height and then along with your age and gender she will be able to tell you what your expected peak flow rate should be, and this will give you a guideline as to what you should be blowing.

As children grow they will need to be re-measured as their expected peak flow will go up accordingly.

If your asthma is not well controlled or you are becoming unwell, your scores are likely to be lower than expected, and there may be a big difference between your morning and evening reading.

It is important to know that with worsening asthma, a low peak flow reading can occur before asthma symptoms such as shortness of breath, tight chest or wheezing. Note: wheezing may not occur in all children with worsening asthma.

As an asthma nurse educator, I encourage all adults and children from 6 years to have their own peak flow meter, know their personal best peak flow when well and to have a written asthma management plan to follow.

If you have a cold or are feeling unwell, do your peak flow and follow your care plan.

I also advocate that people should be doing a peak flow regularly two to three times a week to ensure that the peak flow is where it is expected to be.

It is also important that you see your doctor for a regular review of your asthma control and asthma medications.

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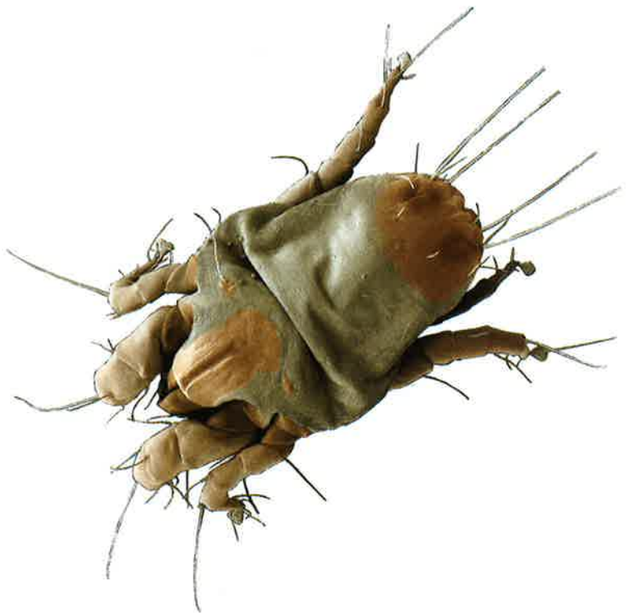
dust mite and their prevention

by Adie Riddell RN
Asthma Nurse Educator

There are many substances in household dust which can cause allergies in humans, including animal dander, insect parts, mould spores and pollen.

But the most common allergenic components of house dust are from house dust mites. They are tiny creatures related to ticks, chiggers, and spiders. Their primary food is dander (skin scales) shed from humans and pets. Most homes will probably have detectable levels of house dust mites.



House dust mites are not parasitic nor are they capable of biting or stinging or burrowing under the human skin. Symptoms of a house dust mite allergy include stuffy or runny nose, sneezing, coughing or watery eyes. Their significance as pests is due to the powerful allergens contained in the mites, their cast skins, faecal material and secretions which contain a substance called DerP1, a very potent allergen.

House dust mites go through five major life stages: egg, larva, protonymph, tritonymph and adult. Between the dust mites life stages the mites moult, shedding their outer skin. When temperature and humidity are optimum, development from egg to adult takes about one month. Adults live approximately 1-2 months, and the females lay about 50 eggs. It is not uncommon to find thousands of mites in a single gram of house dust (a gram is about the weight of a paperclip).

Dust mites are everywhere. Hundreds of thousands of dust mites can live in bedding, mattresses, upholstered furniture, carpets or curtains of your home.

Where do dust mites come from? Dust mites occur naturally. People and pets regularly shed small flakes of skin from their bodies as the skin continually renews itself. Since the greatest fallout occurs in areas of human and pet activity, the mites tend to be most numerous in beds, overstuffed sofas and chairs, and adjacent carpeted areas.

House dust mites absorb and lose moisture through their skin, and are very vulnerable to dehydration. Humidity seems to be the determining factor whether a house has high concentrations of dust mites as they absorb moisture from the air. So in areas with low humidity (think dry deserts), dust mite struggle to survive. They like to be indoors, where they can get plenty of food like mould spores and dead skin cells from people and pets.

Do dust mites affect health? Inhalation of dust mite allergens by hypersensitive individuals can result in acute attacks of bronchial asthma, accompanied by wheezing, and shortness of breath. Diagnostic tests and clinical studies by allergists have shown house dust mite to be the most common allergy in asthmatics, and an important "root cause" for the development of asthma in young children. Recent studies suggest that at least 45 percent of young people with asthma are allergic to house dust mites. Unlike "seasonal" allergies caused by moulds and pollen, people who are allergic to dust mites often will have symptoms year round.

For those people that are 'allergic' to them, they may have asthma like symptoms, eczema or chronic sinus problems. These allergens cause an immune system response, known as allergic rhinitis which can range from mild to severe. People with asthma who are sensitive to dust mites face an increased risk of flare-ups or an asthma attack.

Who should worry about dust mites (and who shouldn't)? People with 'known' allergy to dust mites or with asthma triggered by dust mite allergies will need to reduce the levels of dust mite in their homes. Higher concentrations of dust mite are likely to be found in areas of high humidity, older homes, and homes where a musty or mildew odour is present.

Dust mite allergens do not stay airborne as they cling to particles that are too heavy to stay in the air for long. Hence they are most likely to settle within minutes into dust or fabric. This is why pillows and bedding as well as upholstered furniture tend to have large concentrations of dust mite.

Most exposure to dust mite allergens occurs while sleeping or when dust is disturbed i.e. bed making or other movement.

If you are sensitive or allergic to them, you may have:
1 Watery, red eyes
2 Runny or itchy nose and sneezing
3 Sore throat or hoarse voice
4 Coughing and other breathing problems
5 Skin rash and itching

How do i eliminate dust mites? Reducing the humidity in the house can help; air conditioners and use of dehumidifiers can help achieve this goal. Opening windows for one hour a day will help remove humidity from the house. Consider using an electric blanket, which can reduce humidity on bed surfaces.

Dust mites settle down in carpet, draperies, stuffed animals, and upholstered furniture. Mattresses, pillows, and soft bedding are favourite hangouts.

Dust mite covers for pillows and mattress really do work. Did you know, that within ten years, dead dust mites and their waste can double the weight of your mattress. It does explain why dust mite-proof pillow and mattress covers are your first line of defence against dust allergies!

In one study it was found that the amount of asthma medication was reduced by half for children who were allergic to dust mite that had mattress and protector covers. And testing of their mattresses showed that the number of dust mite colonies reduced with the protectors in place. If possible cover your pillows/mattress when new. Duvet protectors are also available. Wash bedding in hot water once a week (Cold does not kill dust mites). Wash comforters and bedspreads every one to two months. Wash and dry stuffed animals often and keep them off the bed. Alternatively non-washable bedding and soft toys can be frozen overnight to kill dust mites. Clean mattresses in late winter and early spring as this will kill any dust mites that survived the winter and reduce their numbers in the summer months.

Use a high efficiency HEPA filter vacuum cleaner and damp dust all hard surfaces at least once a week. Consider venetian blinds for bedrooms. It's a good idea to wear a mask while vacuuming to avoid inhaling the allergens or ensure the person who is allergic to the dust mite stays out of the area for at least 20 minutes after vacuuming.

In conclusion, dust mites can be a major problem for people with asthma. So if you follow the simple rules discussed in this article, it will help with your asthma control.

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tuberculosis: update on an old disease

by Janet Delooze RN
Asthma Nurse Educator

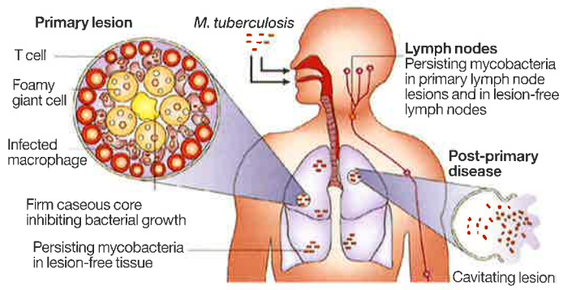
Tuberculosis, or TB, is a chronic bacterial infection that primarily affects the lungs.[1] It is a notifiable disease that must be reported to the local Ministry of Health (MOH) as soon as it is suspected or confirmed.[2]

TB is a worldwide health problem. In 2012, an estimated 8.6 million people developed TB and 1.3 million died from the disease.[3] That year, there were 279 new cases of TB in New Zealand which equates to 6.3 per 100,000 population, a slight decrease from 2011 where there were 6.9 per 100,000 population.[4] The World Health Organisation (WHO) has set targets to achieve a TB free world, for example, the Stop TB Strategy launched in 2006.[3] Worldwide, the numbers are gradually decreasing and with fewer deaths. However, there are still too many people contracting TB, particularly the young, the elderly and the vulnerable – people living in poverty, the homeless, alcoholics, people who are malnourished and those who are HIV-positive.[4]

TB is one of the oldest diseases known to man and has been found in the remains of skeletons from as far back as 4,000 BC. It has been given many names in the past: the ancient Greeks named it phthisis, and in the 18th century it was known as consumption. In the past, patients were isolated, usually in a sanatorium, and in an area where the air was 'clean'. Europeans were often sent to Switzerland, and as you can see from this illustration, they were often nursed outdoors.

TB infection is caused by Mycobacterium complex, including Mycobacterium tuberculosis and Mycobacterium bovis. The bacteria enters the body via the respiratory tract, the gastro-intestinal tract or through open wounds. Most TB infections enter via the respiratory tract and are spread by airborne bacteria through coughing, sneezing, spitting or laughing.[1]

Once in the lungs, the bacilli embed into the alveoli and over the following 3 to 4 weeks, multiply in number, producing an inflammatory reaction not unlike pneumonia. Macrophages and leucocytes engulf the bacteria but are unable to kill it completely. The alveoli become oedematous and consolidated with fluid and leucocytes. The engulfed bacteria then become encased by a protective wall known as a tubercle, or granuloma, hence the abbreviated name, TB (tubercle bacillus). The tubercle takes 2 to 10 weeks to form and keeps the bacteria from spreading throughout the body.[1]



Source: http://healthwise-everythinghealth.blogspot.co.nz201303tuberculosis-made-easy.html

Which countries have a high TB rate?

In general, the following places are considered to have a high TB rate (≥ 40 per 100,000):

- most of Africa
- much of South America
- Russia and the former Soviet states
- Indian subcontinent
- China, including Hong Kong
- South East Asia (except Singapore)
- some Pacific nations (except Cook Islands, Fiji, Niue, Samoa, Tokelau and Tonga).[2]

Possible progression

- The tubercle can become necrosed; the bacilli can destroy lung tissue
- The bacilli are held by the body's immune system forever
- The bacilli are destroyed by anti-tuberculosis drug treatment.
- The tubercle can break down and spread to other parts of the body via the blood stream or lymphatic system, usually to oxygen rich organs such as the brain.
- The tubercle lies dormant but remains live and can be reactivated, sometimes years later – known as latent TB.[2]

Clinical features

Cough, night sweats, weight loss, increased respiration rate, increased cardiac output, increased BP, chest pain, cyanosis, blood-stained sputum, digital clubbing, and peripheral oedema.[1]

Diagnosis

Sputum or wound culture can confirm TB but this can take several weeks for the result. Chest x-ray remains a valuable diagnostic tool for suspected cases.

Intradermal tuberculin skin testing (Mantoux) often identifies TB in people who have not had symptoms. 0.1ml of purified protein derivative (PPD) is injected intradermally in the forearm to form a bleb. The result is read 48 to 72 hours later. 10mm or more is considered positive and requires follow-up; a second Mantoux is often carried out approximately a week later depending on local protocol.[5]

QuantiFERON-TB Gold test is a blood test that can also identify whether a Mantoux test is positive due to infection or because of previous immunisation, and has replaced the Mantoux test in many areas. This can be costly, however, with local charges being approximately $65.

Treatment

TB is usually treated with a combination of antibiotics and may include a combination of drugs such as Isoniazid, Rifampicin, Ethambutol, Pyrazinamide and Streptomycin.

As the tubercle bacillus can be in various body cavities, a single antibiotic may not be able to penetrate and destroy all the bacilli. Treatment is lengthy, usually 6 to 12 months duration which can create problems completing the course. If the bacilli are not completely destroyed, they can become resistant to the antibiotics thus creating Multi Drug Resistant TB (MDR-TB) TB Manual.

Directly observed therapy (DOT), where the nurse observes the patient actually taking the medication, may be required if patients are not adherent to treatment and for all cases of MDR-TB.[5]

Vaccination

The BCG (Bacille Calmette-Guérin) vaccination is offered to babies and children under 5 years of age who are at risk of contracting TB but the vaccine is not part of the general NZ immunisation schedule. In some circumstances the local Medical Officer of Health may also recommend BCG vaccination for specific populations.[3]

Contact tracing

To reduce the spread of TB, contact tracing is carried out by specialist public health nurses, and all possible contacts are given counselling. Confirmed cases are kept in isolation until treatment has rendered them non-infectious.[5]

And to the future?

According to the WHO, there are more than 50 companies working on new diagnostic tests, at least 10 new drugs in the late phases of clinical trials and 10 vaccines for TB prevention currently being developed.[3] Perhaps one day this highly infectious disease will be totally eradicated.

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Massey High School

Alfriston College

a commitment to our pacific community...

Asthma Auckland is dedicated to providing outstanding asthma education across the wider Auckland region to all ethnicities. Our work especially in south Auckland involves many people from Polynesia, Micronesia and Melanesia.

> It was very exciting to be involved in two gatherings in March that reflected our diverse range of clients and demonstrated our commitment to our pacific communities.

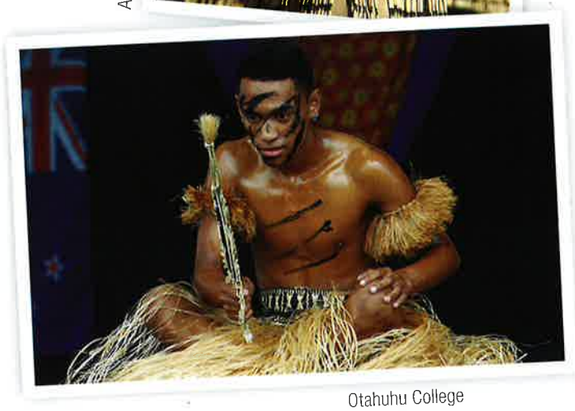
For the past five years Auckland Hospital has run a Pasifika week. The theme this year was Pasifika Navigation which is aligned to the journey of Pasifika peoples. It is also aligned to the service delivery model of their re-named service Tautai Fakatahaha which delivers health navigation services across ADHB and WDHB to Pasifika peoples. Asthma Auckland had a stall for 4 days 3-6 March 2014 providing asthma information and education to a variety of staff, students and the general public. At lunch time we were entertained by dance performances and networked with other health providers. I would like to thank Tuliana Guthrie Team Leader Pacific Health Hospital Services for facilitating our participation.

The following week 12-15 March 2014 we took our asthma education bus to ASB Polyfest which is held each year in the Manukau Sports Bowl. This is the 39th year of the ASB Polyfest which is the largest event of its kind in the world. The festival provides opportunities for over 9000 students from 60 different Auckland secondary schools to present traditional and contemporary cultural performances. As well as the students participating, the event usually attracts over 90,000 spectators each year. Usually 60% of the stalls provide food, 20% craft and the last 20% educational sevices. Students competed on five stages performing traditional items from the following cultures – Cook Islands, Maori, Niue, Samoan and Tongan. There was also a diversity stage featuring performances from other cultural groups such as Chinese, Fijian, Indian, Korean and Siri Lankan. Unfortunately the number of spectators was down this year due to the weather warnings about cyclone Lusi as well as the Saturday performances being postponed until the following Tuesday. This is the first time in the 39 year history of the event that weather has caused a postponement.

The Kura Matua Host School for the 2014 ASB Polyfest was Western Springs College. The theme they selected for this year's festival was – *"Care for our seas and our lands, so that the safety of our homes, both present and past endures forever."*

The four registered nurses from Asthma Auckland provided asthma education and information to hundreds of students and their families as well as networking with a variety of organisations dedicated to supporting our pacific communities. We were thrilled to be part of such an outstanding event and hope to be involved next year. We would also like to thank Remuera New World for providing grocery items for a raffle and Ben Campbell Photography for the use of some fantastic photos.

Karen Little
Asthma Nurse Educator



Otahuhu College

Epsom Girls Grammar School

Papakura High School

Papatoetoe High School

Epsom Girls Grammar School

Entering ASB Polyfest Powhiri

slyvia gilles

Forty-five years of continuous service as a volunteer for the Southland Asthma Society and the Southland TB and Chest Diseases Society was recognised by the Invercargill City Council at an Honour Award Ceremony held recently. Sylvia Gillies from the Southland Asthma Society was one of six recipients who were duly awarded Civic Honours.

In the late 1950's a family friend asked Sylvia if she would like to assist with visiting patients at the Sanatorium at Waipiata in South Otago. Little did she foresee that she would still have an interest in the welfare of those with respiratory diseases forty-five years later.

Hospital visiting and attending to the welfare of families affected by the hospitialisation or death of a loved one took priority over her own family matters and busy life as a hairdresser.

In latter years the added responsibility of looking after an elderly mother meant restricting the social work but she still remains an active member of the Southland Asthma Society Executive Committee.

The award was well deserved.

Slyvia Gillies with the Mayor of Invercargill Tim Shadbolt.

north & south
NEWS FROM AROUND THE REGIONS ...

asthma WELLINGTON

Dear Linda

Re: ***Thanking Asthma Wellington's Asthma Nurse Educators***

I'd like to commend the work of the Asthma Nurse Educators at Asthma Wellington. Their intervention has led to a break-through in my health during a time of health challenges. This has vastly improved my ability to function in everyday life and assisted me to continue in paid employment.

After eight or so years of deteriorating health I was diagnosed with mild asthma in 2009. I suffered chronic restricted breathing/congestion but had no acute asthma attacks. Several asthma drugs were trialed before one was found to help. Over an extended period another drug was added, then doses increased to a high level in an attempt to control my symptoms. Every few months I needed a 'top up' course of steroids to alleviate breathing problems. Taking steroids was not done lightly as they exacerbated my other health conditions. The frequency with which I needed steroids was increasing; two steroid prescriptions in 2011 became five prescriptions in the first nine months of 2013.

I had difficulty accepting the asthma diagnosis: If I had asthma why were the asthma drugs not effective? Why did 'mild asthma' require such high doses of medicine? While I placed great value on my Doctor's input, 15 minute sessions were not conducive to solving the level of complexity that my breathing difficulties presented, especially given my other health issues. And although I found some excellent asthma resources online, what I really needed was someone to answer my questions. I felt frustrated, anxious and absolutely weary.

A Titahi Bay pharmacist directed me to Asthma Wellington where I subsequently spent several hours consulting with Asthma Nurse Educator, Adie Riddell. Ms Riddell had time to listen to me closely, she discussed many things with me and answered my questions in a systematic and patient manner. She also provided me with a thorough education regarding asthma. She confirmed that unremitting breathing difficulties were not the normal pattern of asthma; usually there are periods of remission.

As a direct result of the Asthma Wellington consultations, I was referred to a Respiratory Physician at Wellington Hospital in late 2013. He suspected a reaction to an asthma drug and changed my medication. To my immense relief this immediately improved my health. Several days ago I was discharged from his care after blowing my best peak flow ever.

I no longer think about asthma much as it's not a huge issue for me: I can walk to work with ease, at work I can go into the cold storage units without getting sick, laughing no longer results in violent bouts of coughing. I am the main breadwinner in my household and I no longer struggle to keep the job I love or worry about having to go on a Sickness Benefit.

I'm not sure how I would have reached this point if it wasn't for the intervention of Asthma Wellington. Please consider this my heart-felt thank you to this wonderful and very necessary organisation.

Sincerely
Monica

CANBREATHE
Helping Cantabrians breathe easy
Asthma and COPD education and support

south island respiratory educator forum (SIREF) 2014

Canbreathe is one of the organisers of the annual South Island Respiratory Educator Forum (SIREF) which was held in Christchurch on Thursday 20th and Friday 21st February at the Copthorne Commodore Hotel. The Forum was attended by nurses, physiotherapists and other practitioners with an interest in respiratory health. We were again fortunate to have a range of very knowledgeable speakers who donated their time and shared their expertise with those present.

Joe Bennett, local author and columnist, who recently quit smoking gave the opening address and provided his most informative and entertaining perspective regarding smoking and how to help people quit.

The theme for SIREF 2014 was "The Air out There" with topics and speakers focusing on respiratory issues and services provided in the community. Presentation topics included: Home Oxygen Therapy; TB home based therapy and monitoring; New ways of looking at COPD Severity including latest GOLD guidelines; Community based Spirometry; Restless Legs; CPAP in the community; Collaborative care for long term complex patients, Research update on Asthma; Asbestos issues and a presentation on the new online COPD handbook Don't Forget to Breathe (www.dontforgettobreatheorg.nz – Authors: Sue Ward, Carole Donnelly, Carol Cooper-Taylor, Phyl Cooper-Taylor).

Feedback from attendees was very positive and it was good to also receive a range of suggested topics for 2015. Some presentations will be available to view on Canbreathe's website – www.canbreathe.org.nz

Teresa Chalecki
Manager/RN
Canbreathe

manukau indian association safety day saturday 1st february 2014

Asthma Auckland was invited to participate in this well attended event in Papatoetoe. Karen Little, Asthma Nurse Educator, attended the day and spoke to over 50 people about their asthma.

As well as the Indian community residents from Papatoetoe enjoyed a free sausage sizzle, bouncy castle, face painting and ethic food. There were spot prizes and one lucky couple won two tickets to fly to Sydney Australia.

Asthma Auckland would like to thank Joseph Liava'a Community Manager from East Tamaki Healthcare for involving so many organisations in this very worthwhile Safer Families event.

Health	Safety	Social Services/Education
Diabetes NZ	Auckland Council	Pacific Homecare
WONS (Well Women)	Police	Elections
Arthritis NZ	Watersafe Auckland	AUT
Asthma NZ	Plunket	Greenstar
Breastscreen CM	Fire	Open Waananga
ProCare	Papatoetoe Crime Patrol	Independent Living Services
Southseas	Corrections	Glowbug/Mercury
Mangere Health		TANI
East Tamaki Healthcare		ARMS
CMDHB		Send Money Pacific
		Enliven
		Fatimah Foundation
		Shanti Niwas

Joe's cold

Written by Karen Little RN
Asthma Nurse Educator

Joe started to cough at night again.

"Cough, cough," all night long. His sister Mary complained in the morning that she was tired because Joe had kept her awake.

Mum wondered if Joe was starting to get a cold. "How are you feeling Joe, are you sick?"

"How do I know" grumbled Joe, "I think I should stay home from school." Joe looked quite pleased with this idea.

"Wait a minute," Mum quickly answered, "Go and get your peak flow, this is why we got one from our doctor."

Joe found the peak flow meter (thank goodness he had put it in the asthma bag that the nurse had given him or he would never have found it).

Mum remembered that a few months ago Joe had blown into the meter, morning and night, for four weeks before any medication to see what his best blow would be.

"Right, we know that you can blow 350 when you are well and the nurse said that was great for your height. Let's see what you can blow now."

"Huff," Joe blew as hard as he could. Mum could hear a wheeze after this.

"Oh dear you have only got up to 200," mum sounded worried, "try again two more times."

"Well, it's still low. You are going to have to stay home. I think you do have a cold and I don't want all your class to catch it. Thank goodness I have the day off so I can keep an eye on you or you would have to go to your Aunty Robyn."

Mum checked the care plan that her doctor had filled in and it said to give two puffs of the blue puffer every four hours for as long as the cold lasted for. She got the spacer and watched Joe have one squirt of the puffer and take six breaths, he did this twice. Every four hours he did this again.

After two days Joe's peak flow had come back up to 300 and he had stopped coughing at night.

"Thank goodness you have been taking your orange puffer morning and night even though you are well; you used to get sick so often and for so long. Now you can go back to school much more quickly." Mum was pleased with this and Joe did want to go back to school as he was tired of mum asking him how he was!

Kid's Page

1 How many words can you make from the letters given. You can use each letter several times. (Try to find related words to our asthma magazine only).

W	h	e	r	k
J	p	g	m	n
L	s	u	d	f
Y	a	o	t	h

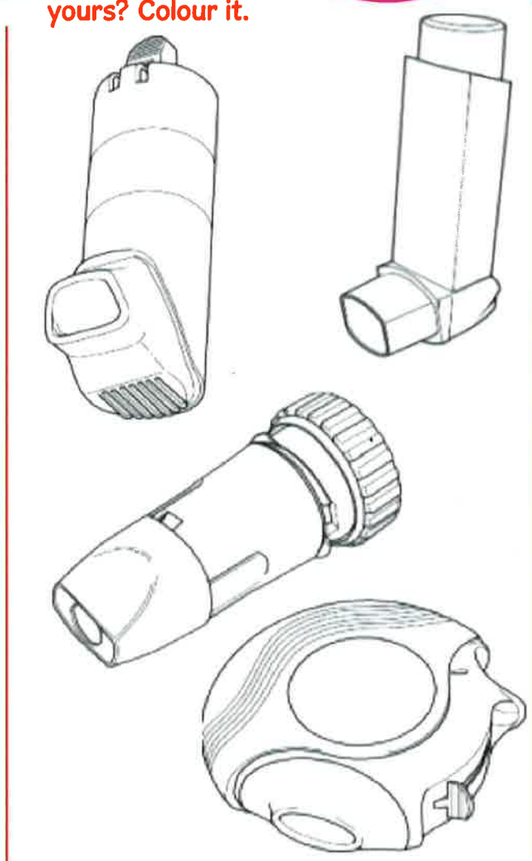
2 **Each 2x2 box, as well as each row and each column, must contain each of the four numbers (1,2,3,4) in the grid. Work out the numbers.**

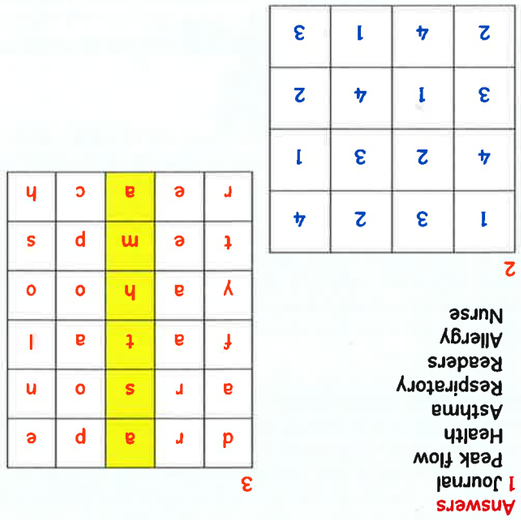
1		2	
3		4	
	4		3

3 Each of the words in the grid below has lost its middle letter. Write them in, and the letters in the yellow squares will spell a word.

d	r		p	e
a	r		o	n
f	a		a	l
y	a		o	o
t	e		p	s
r	e		c	h

4 **Which inhaler is yours? Colour it.**





Answers
1 Journal
Peak flow
Health
Asthma
Respiratory
Readers
Allergy
Nurse

2

3	1	4	2
2	4	1	3
1	3	2	4
4	2	3	1

3

h	c	a	e	r
s	p	m	e	t
o	o	h	a	y
l	a	t	e	f
n	o	s	r	a
e	p	a	r	d

is it too soon to diagnose COPD?

by Ann Wheat RN BN
Asthma Nurse Educator

Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of morbidity and mortality worldwide. The economic and social cost to countries is substantial and increasing year by year. The main causes of COPD are cigarette smoking, biomass fuels, noxious particles and a hereditary factor. The definition of COPD is "a common preventable and treatable disease, characterised by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and comorbidities contribute to the overall severity in individual patients".[1]

In New Zealand it is estimated that COPD affects an estimated 15% of all New Zealanders over the age of 45 years and is the fourth leading cause of death.[2] It is also recognised that the prevalence of COPD increases with age and intensity of smoking and can vary from 25% in a general smoking population up to approximately 50% in elderly smoking population.[3] Over 85% of cases of COPD are caused by inhalation of cigarette smoke.[2] In Maori COPD affects individuals up to 20 years earlier than non-Maori and Maori are more likely to die from COPD related causes. Pacific peoples also have similar rates to Maori.[2] Evidence shows that COPD is under-diagnosed plus often inaccurately diagnosed both here in New Zealand and worldwide.[4]

So why would this be?

In the early stages of COPD the symptoms include coughing and breathlessness with exercise. They think that they are just getting older and therefore cannot exercise as well as they could before. In many cases people put up with these symptoms until they become much worse and then they seek medical attention. Sputum production increases with many people coughing up sputum most days. Chest infections become more prevalent as the condition worsens but often this is not associated with COPD until much later. In fact in one study 27% of people presenting with a chest infection had not previously been diagnosed.[3] Of those, 45% were in stage 1 – mild COPD, 53% – moderate COPD, 3% in stage 3 – severe COPD and 0 had stage 4 or very severe COPD.[3] These results seem to be mimicked in most studies undertaken when looking at undiagnosed COPD.

COPD often starts in people around the age of 40 years who are current or former smokers but often the symptoms do not show themselves until the mid-50s.[5] Symptoms then become progressively worse and breathlessness can be present even with the slightest of activity. As a result a person's whole life is affected and early death can become a reality

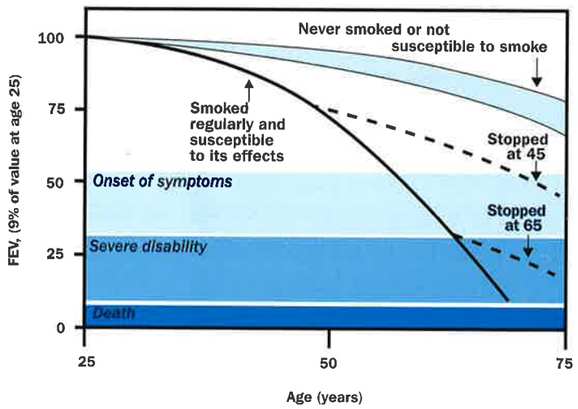
What can be done to help?

Any person who has recurrent chest infections, who are over the age of 40 years and in some cases even 35 years of age, should have a smoking history assessment taken. This should include whether they are a current or former smoker and in fact even a passive smoker. It is necessary to identify the amount of cigarettes smoked per day and for how long. People that meet the above criteria should be offered spirometry testing to ascertain their current lung function.[3] A spirometry will identify those with underlying COPD that have not previously been diagnosed as well as identifying what stage of COPD a person may be in.

Why is this important?

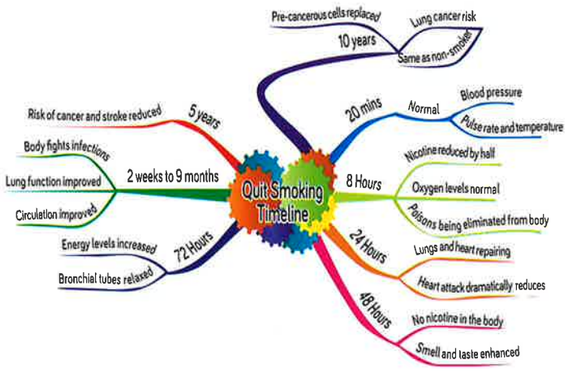
The diagnosis of COPD and Asthma are often confused. Asthma too can frequently be underdiagnosed as well as being misdiagnosed.[6] In fact it is now becoming obvious that both conditions can and do co-exist. It is therefore very important to accurately diagnose each condition as they both have different treatments.[6]

COPD as it progresses becomes a very disabling condition. It is therefore very important to encourage all smokers to stop. It has been shown that 31-45% of cigarette smokers who have never had a diagnosis of COPD do in fact have the disease.[7] Smoking cessation is the most valuable and far reaching option therefore in the management of COPD. People who stop smoking can help themselves to reduce the long lasting effects and slow the progression of COPD. It is known that if a person stops smoking at the age of 45 years, then the rate of lung function decline returns to almost the same level as a non-smoker but is still below that of a never smoker. Even stopping smoking at 65 will have some benefits for the lungs.



Retrieved from:
http://www.thinkcopdifferently.com/en/About-COPD/Risk-factors-for-COPD/Smoking

Stopping smoking also has many other important health benefits apart from the improvement of lung function as per the diagram below.



Retrieved from: http://www.you-quit-smoking.com

Another reason why it is important to diagnose COPD early is to assist with the effects of the condition such as breathlessness and fatigue, to maintain physical activity and improve exercise tolerance. Many people with COPD become depressed by not being able to do what they could previously do and feel that they cannot go out and meet with their friends as they feel embarrassed by the symptoms that they have. This can be overcome by attending Pulmonary Rehab. Pulmonary Rehabilitation is a structured series of exercise and education sessions to give people the skills to improve and maintain their lifestyle where possible. Pulmonary Rehab allows people with COPD to meet regularly and so helps both mentally and physically. During these sessions diet can also be discussed to assist those who may be under or over weight, which can help with the ability to maintain their lifestyle.

Medication can also be commenced to alleviate the symptoms of COPD. The correct diagnosis allows for the appropriate medication to be prescribed which again helps with the maintenance of lifestyle.

In conclusion

It is never too soon to diagnose COPD. Early diagnosis by spirometry allows the correct diagnosis, treatment and support that a person needs who has developed this condition. The sooner smoking cessation is undertaken the better the outcome may be. So we should be supporting our clients with best practice and giving them the benefit of our expertise for this condition that if left undiagnosed and untreated leads to premature death.

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When Smokers Quit

Just 20 minutes after you've smoked that last cigarette, your body begins an ongoing series of beneficial changes.

20 Minutes
- Blood pressure drops to normal
- Pulse rate drops to normal
- Temperature of hands and feet decreases to normal

8-12 hours
- Carbon-monoxide level in blood drops to normal
- Oxygen level in blood increases to normal
- Nicotine levels fall considerably

24 hours
- Chance of heart attack decreases

48 hours
- Nerve endings start re-growing
- Ability to smell and taste is enhanced
- Walking becomes easier

2 weeks to 3 months
- Circulation improves
- Lung function increases up to 30 percent

1-9 months
- Coughing, sinus congestion, fatigue and shortness of breath decreases
- Cilia regrow in lungs, increasing ability to handle mucus, clean the lungs and reduce infection
- Exercise capacity increases
- Blood pressure level should be returning to normal

1 year
- Risk of coronary heart disease is half that of a smoker

5 years
- Risk of cancer, heart disease and stroke reduced

10 years
- Pre-cancerous cells replaced
- Lung cancer risk is the same as a non-smoker
- Reduced risk of many other cancers in the body e.g. bladder cancer

15 years
- Risk of dying from any cause will be almost the same as a person who has never smoked

the history of chronic obstructive pulmonary disease (COPD)

by Vicki Lyford RN
Asthma Nurse Educator

Today, COPD is an umbrella term that encompasses chronic bronchitis, emphysema and asthma, however, its origins began many years ago.

The word asthma is thought to originate from the Greek verb Aazein meaning to pant/to exhale with the open mouth/sharp breath, and asthma was first mentioned in Homers 'Iliad' about 300 BC.[1] However it was not until much later that the term COPD was used Hippocrates (460-357 BC) and Galen (201-130 BC) both recognised that asthma is caused by bronchial obstruction.[6]

For many years the Chinese have used herbs containing ephedrine and people would inhale the beta-agonist qualities of these herbs.[1] Ancient Egyptians used hot bricks to heat a mixture of herbs, then inhale their perfume (as shown in the 'George Ebers Papyrus', found in Egypt in the 1870's).[1]

It was in 1679 that Bonet gave a description of "voluminous lungs". This was endorsed in 1769 by Morgagni, who reported on 19 cases where lungs were 'turgid', specifically from air.[2]

In 1721, Ruysh illustrated and described the enlarged airspaces.[5]

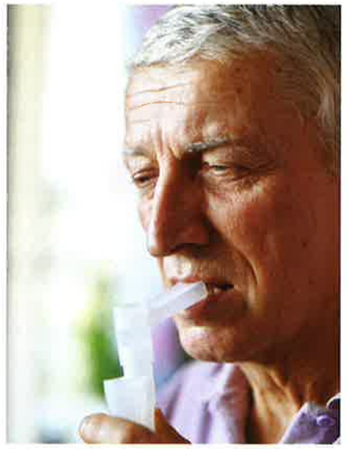
Matthew Baillie illustrated an emphysematous lung in 1789, showing the pathology of the disease and from this, emphysema was shown to be part of the COPD umbrella.[2] The word 'catarrh' was used by Badham in 1814, referring to increased mucous secretion and a chronic cough as being symptoms of bronchiolitis. He inferred that chronic bronchitis and bronchiolitis were debilitating health disorders that could also come under the COPD umbrella.[3, 4]

Rene Laennec was the physician who invented the stethoscope in 1816 and was the first man to classify the medical terms rales, rhonchi, crepitance (are the clicking, rattling, or crackling noises that may be made by one or both lungs of a human with a respiratory disease during inhalation)[7] and egophony (an increased resonance of voice sounds heard when auscultating the lungs, often caused by lung consolidation and fibrosis).[7] Rales are usually known as crackles today. He also identified emphysema as part of the COPD umbrella. In 1821 he described in his *'Treatise of Diseases of the Chest'*, lungs that were 'grossly inflated and did not empty well'. As smoking at that time was not common, he attributed the causative factors as being environmental and genetic.[2, 3, 4]

John Hutchinson invented the spirometer in 1846, but this instrument only measured vital capacity, measuring volume and not airflow. It was not until 1947 that Tiffeneau and Gaensler in 1950/51 described the principles of airflow measurement, thus adding the notion of timed vital capacity to measure airflow. Hence, spirometry became a complete diagnostic instrument. Spirometry is, to this day, still used as a diagnostic tool for diagnosis and assessment of response to therapy in COPD.[2, 4]

In 1892, Einthoven theorized the signature expiratory flow resistance seen in COPD; however it was Dayman in 1951 who became the first to accurately explain expiratory airflow collapse of the lungs.[6]

It was not until 1959 that the Ciba Guest Symposium and the American Thoracic Society Committee on Diagnostic Standards in 1962 first detailed a definition of COPD.[2,3] In 1944, Ronald Christie recognised that there were individual components of COPD and obtained a patient history and physical examination to give his diagnosis.[2]



At the Aspen Emphysema Conference in 1965, William Briscoe was the first person to use the term COPD.[3]

Charles Fletcher, the author of 'The Natural History of chronic bronchitis and emphysema', linked COPD and smoking in 1976. Along with his colleagues, he found that smoking cessation slowed the progress of COPD whilst continuing smoking hastened the disease progression. His work provides the basis for the smoking cessation education that we use today.[4]

Introduction of inhalers and mechanical ventilators in the 1960's heralded a new era of therapy. Pulmonary Rehabilitation and Homecare were introduced as a concept at the 8th Aspen Emphysema Conference in 1965.

Oxygen therapy was trialled in the mid 1960's by researchers from the University of Colorado Medical Centre in Denver. But it was not until the 1980's that it was further developed and today long term oxygen therapy is common place in COPD.[5]

In 1982, The Lung Division of the National Heart Lung and Blood Institute (NHLBI), organised the Lung Health Study. There were almost 600 volunteers between the ages of 35-60, all heavy smokers with constricted breathing. The results of this study became the basis of identifying COPD and advocating smoking cessation as a way of improving survival from this disease.[3]

The 1990's saw a growth in the use of medication management of the symptoms of COPD to regulate pulmonary function.[4]

We have come a long way in the diagnosis and treatment of COPD and we hope with smoking cessation and better education that the rapidly rising numbers of those with COPD will show a decline in the future.

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Pediatr Allergy Immunol
The association of preterm birth with severe asthma and atopic dermatitis: a national cohort study;
Trønnes H, Wilcox A, Lie R, Markestad T, Moster D; Pediatric Allergy and Immunology (Dec 2013)

BACKGROUND: Asthma and atopic dermatitis are both regarded as atopic diseases. Being born too early is associated with increased risk of asthma, but some studies have indicated that the opposite might be true for atopic dermatitis. We explored in more detail the associations between preterm birth, asthma, and atopic dermatitis.
METHODS: We analyzed data from Norwegian registries with prospectively collected data. All live births in Norway from 1967 through 2001 were followed through 2005 by linking the Medical Birth Registry of Norway to the National Insurance Scheme and to Statistics Norway. Only severe asthma and atopic dermatitis were registered in the National Insurance Scheme.
RESULTS: Of a total of 1,760,821 children, we identified 9,349 cases (0.5%) with severe asthma and 6,930 cases (0.4%) with severe atopic dermatitis. Compared with children born at term (37-41 wk gestation), preterm birth was associated with increased odds for severe asthma (odds ratio (OR) 1.7 (95% confidence interval (CI): 1.6-1.8) for 32-36 wk gestation and OR 3.6 (95% CI: 3.1-4.2) for 23-31 wk) and decreased odds for severe atopic dermatitis (OR 0.9 (95% CI: 0.8-1.0) for 32-36 wk gestation and OR 0.7 (95% CI: 0.5-1.0) for 23-31 wk). Adjustment for perinatal and socio-demographic factors weakened the association between gestational age and severe asthma, while slightly strengthening the association between gestational age and severe atopic dermatitis.
CONCLUSIONS: Preterm birth was associated with increased risk of severe asthma and decreased risk of severe atopic dermatitis.

Pediatr Allergy Immunol
Antibiotic exposure in the first two years of life and development of asthma and other allergic diseases by 7.5 yr: A dose-dependent relationship;
Hoskin-Parr L, Teyhan A, Blocker A, Henderson A; Pediatric Allergy and Immunology (Dec 2013)

BACKGROUND: Antibiotic use in infancy disrupts gut microflora during a critical period for immune system development. It is hypothesized that this could predispose to the development of allergic diseases. We investigated the associations of antibiotic use in the first 2 yr of life with the development of asthma, eczema or hay fever by age 7.5 yr in a longitudinal birth cohort.
METHODS: Subjects were 4952 children from the Avon Longitudinal Study of Parents and Children (ALSPAC). Child antibiotic use and asthma, eczema and hay fever symptoms were maternally reported. Atopy was assessed by skin prick tests at age 7.5 yr. The total number of antibiotic courses was considered as the main exposure. Data were analysed using multivariate logistic regression.
RESULTS: Children reported to have taken antibiotics during infancy (0-2 yr) were more likely to have asthma at 7.5 yr (OR 1.75, 95% CI 1.40-2.17), and the odds (OR, [95% CI]) increased with greater numbers of courses: once 1.11 [0.84-1.48]; twice 1.50 [1.14-1.98]; three times 1.79 [1.34-2.40]; four times or more 2.82 [2.19-3.63]. Increased antibiotic use was also associated with higher odds of eczema and hay fever but not atopy. The effect appeared to be associated with cumulative rather than a critical period of exposure during the first 2 yr.
CONCLUSIONS: A robust and dose-dependent association was found between antibiotic use in the first 2 yr of life and asthma at age 7.5 yr but did not appear to be mediated through an association with atopy.

J Allergy Clin Immunol
Anti-inflammatory and corticosteroid-enhancing actions of vitamin D in monocytes of patients with steroid-resistant and those with steroid-sensitive asthma;
Zhang Y, Leung D, Goleva E; Journal of Allergy and Clinical Immunology (JACI) (Jan 2014)

OBJECTIVE: Vitamin D regulation of responses in patients with steroid-resistant (SR) versus steroid-sensitive (SS) asthma has not been studied.
METHODS: Peripheral blood cells from 11 patients with SR asthma and 8 patients with SS asthma were preincubated with 1,25-dihydroxyvitamin D (1,25[OH]2D [VitD]), followed by dexamethasone (DEX) treatment and LPS stimulation. LPS-induced phosphorylated p38 mitogen-activated protein kinase (p-p38) in monocytes was examined by means of flow cytometry. Mitogen-activated protein kinase phosphatase-1 (MKP-1) mRNA expression, which inhibits p-p38, was analyzed by means of real-time PCR. Glucocorticoid receptor (GR) binding and histone H4 acetylation in the glucocorticoid response element of the MKP-1 promoter in monocytes were analyzed by means of chromatin immunoprecipitation.
RESULTS: DEX significantly inhibited LPS-induced p-p38 in monocytes from patients with SS asthma but not those from patients with SR asthma ($P < .01$). VitD inhibited LPS-induced p-p38 in monocytes from both patient groups ($P < .01$) but enhanced DEX suppression of LPS-induced p-p38 only in monocytes from patients with SS asthma ($P < .01$). VitD induced MKP-1 expression and enhanced DEX induction of MKP-1 in both patients with SS asthma and patients with SR asthma. VitD/DEX-induced MKP-1 mRNA levels remained significantly lower in monocytes from patients with SR asthma ($P < .05$). DEX-stimulated recruitment of GR and histone H4 acetylation at the glucocorticoid response element 4.6 kbp upstream of the MKP-1 gene were significantly lower in monocytes from patients with SR asthma compared with those from patients with SS asthma. VitD pretreatment enhanced DEX-induced GR binding and histone acetylation in monocytes from both patient groups. However, GR binding and histone H4 acetylation remained significantly lower in monocytes from patients with SR asthma.
CONCLUSION: VitD demonstrated anti-inflammatory and corticosteroid-enhancing effects in monocytes of patients with SR asthma and patients with SS asthma. However, the responses to corticosteroids in patients with SR asthma remained significantly lower than those in patients with SS asthma.

Eur Respir J
Hospital admissions and exercise capacity decline in patients with COPD;
Ramon M, Gimeno-Santos E, Ferrer J, Balcells E, Rodríguez E, de Batlle J, Gómez F, Sauleda J, Ferrer A, Barberà J, Agustí A, Gea J, Rodriguez-Roisin R, Antó J, Garcia-Aymerich J, the PAC-COPD Study Group ; European Respiratory Journal (Jan 2014)

Exercise capacity declines with time and is an important determinant of health status and prognosis in patients with chronic obstructive pulmonary disease (COPD). We hypothesised that hospital admissions are associated with exercise capacity decline in these patients.Clinical and functional variables were collected for 342 clinically stable COPD patients. The 6-min walk distance (6MWD) was determined at baseline and after a mean±sd of 1.7±0.3 years. Information on hospitalisations during follow-up was obtained from centralised administrative databases. Linear regression was used to model changes in exercise capacity.Patients were mostly male (92%), with mean±sd age 67.9±8.6 years, post-bronchodilator forced expiratory volume in 1 s 54±17% predicted and baseline 6MWD 433±93 m. During follow-up, 6MWD decreased by 21.9±51.0 m·year(-1) and 153 (45%) patients were hospitalised at least once. Among patients admitted only for COPD-related causes (50% of those ever admitted), the proportion presenting a clinically significant loss of 6MWD was higher than in patients admitted for only nonrespiratory conditions (53% versus 29%, $p = 0.040$). After adjusting for confounders, annual 6MWD decline was greater (26 m·year(-1), 95% CI 13-38 m·year(-1); $p<0.001$) in patients with more than one all-cause hospitalisation per year, as compared with those with no hospitalisations.Hospitalisations are related to a greater decline in exercise capacity in COPD.
Int J Clin Pract
Chronic bronchitis in COPD patients is associated with increased risk of exacerbations: a cross-sectional multicentre study; Corhay J, Vincken W, Schlesser M, Bossuyt P, Imschoot J; International Journal of Clinical

Practice 67 (12), 1294-301 (Dec 2013)
BACKGROUND AND AIMS: Chronic bronchitis (CB) in chronic obstructive pulmonary disease (COPD) patients is associated with increased mortality, frequent exacerbations and faster disease progression. This study investigates the prevalence of CB in a large population of COPD patients to identify features associated with CB.
METHODS: Cross-sectional multicentre study in patients with Global Initiative for Chronic Obstructive Lung Disease (GOLD) stages 2-4 from Belgium and Luxembourg.
RESULTS: The 974 patients included were on average 67.8 ± 9.6 years old; 72% were male, FEV1 was 52.5 ± 15.8% of predicted. The prevalence of CB was 64% (622/974). In patients with CB, the number of pack-years smoked and the prevalence of chronic respiratory failure, cachexia and skeletal muscle wasting were significantly higher, whereas FEV1 and FEV1 /VC were lower. The prevalence of CB increased with GOLD stage and was higher in patients with emphysema and those exposed to occupational risk factors. The CB group had more exacerbations, a higher percentage of patients with frequent exacerbations (37.3% vs. 14.2% of patients; $p < 0.0001$), increased COPD-related, non-intensive care unit hospitalisations and all-cause hospitalisation rates. In multiple logistic regression analysis, frequent exacerbation was the most important independent variable associated with CB, followed by current smoking, chronic respiratory failure, COPD duration and age.
CONCLUSIONS: CB prevalence in GOLD stage 2-4 COPD patients is high. CB is related to current tobacco smoking, and prevalence increases with COPD severity and duration, emphysema and age. CB could be the hallmark of a subtype of COPD easy to identify in clinical practice, associated with increased disease severity and increased risk of exacerbation.

Chest

Passive smoking exposure is associated with increased risk of COPD in never-smokers;

Hagstad S, Bjerg A, Ekerljung L, Backman H, Lindberg A, Rönmark E, Lundbäck B; Chest (Dec 2013)

ABSTRACT BACKGROUND: Passive smoking, or environmental tobacco smoke (ETS), is a risk factor for lung cancer, cardiovascular disease and childhood asthma, but a relationship with COPD has not been fully established. AIM To study ETS as a risk factor for COPD in never-smokers. METHODS: Data from three cross-sectional studies within the Obstructive Lung Disesease in Northern Sweden (OLIN) database were pooled. Of the 2182 lifelong never-smokers 2118 completed structured interviews and spirometry of acceptable quality. COPD was defined according to the GOLD criteria using post-bronchodilator spirometry. The association of COPD with ETS in single and multiple settings was calculated by multivariate logistic regression adjusting for known risk factors for COPD. RESULTS: COPD prevalence was associated with increased ETS exposure: 4.2% (no ETS), 8.0% (ETS ever at home), 8.3% (ETS at previous work) and 14.7% (ETS ever at home and at both previous and current work), test for trend p=0.003. Exclusion of subjects aged ≥65 years and subjects reporting asthma yielded similar results. ETS in multiple settings, such as ever at home and at both previous and current work was strongly associated to COPD, OR 3.80 (95% CI 1.29-11.2). CONCLUSIONS: In this population-based sample of never-smokers, ETS was independently associated with COPD. The association was stronger for ETS in multiple settings. ETS in multiple settings was, after age, the strongest risk factor for COPD and comparable to personal smoking of up to 14 cigarettes/day in comparable materials. The findings strongly advocate measures against smoking in public places.

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