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Asthma Canterbury turns 40

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asthma NEW ZEALAND THE LUNG ASSOCIATION

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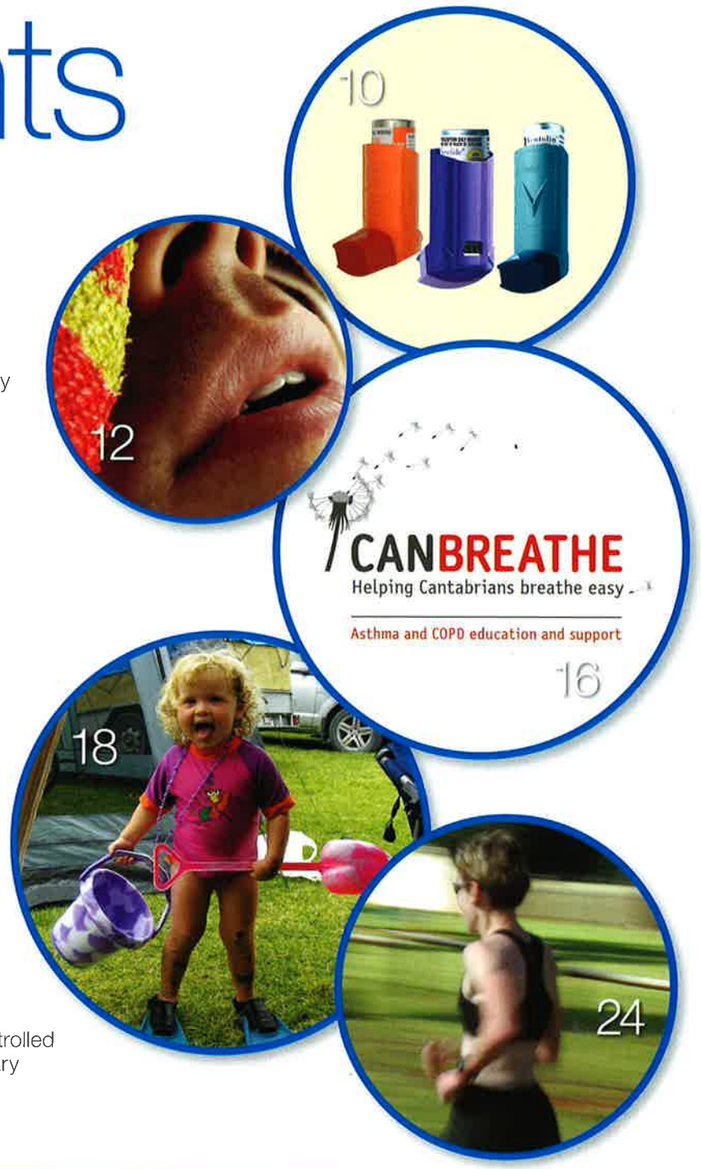

On the cover: Sandra Yates (Secretary for 25 years), Alison Wilkie (founding Secretary) and Anna Bullen (founding committee member). Photo supplied by George O'Brien.

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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 2013 and COPD Nursing Course for April 2013. 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, 931 nurses have enrolled over 38 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 is available for registered nurses.**

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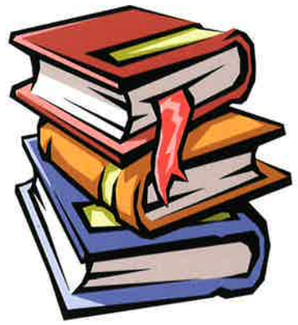

The closing date for enrolment is
11 February 2013 for Asthma Nursing Course
15 April 2013 for COPD Nursing Course

Upcoming events and courses

ASTHMA NEAT COURSE
20 March 2013
19 June 2013
18 September 2013

HALF DAY COPD COURSE
17 April 2013
17 July 2013
16 October 2013

WORLD ASTHMA DAY
7 MAY 2013

GLUTEN FREE FOOD &ALLERGY SHOW
Auckland
25-26 May 2013
ASB Showgrounds
Auckland
www.glutenallergy.co.nz

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

message to readers

One would have to admit that the challenges are increasing and more must be accomplished using lesser resources. In general terms there has been no funding increases from the District Health Boards and that does make life extremely difficult and places a great deal of pressure on the PR/Marketing Manager to develop funding so that Asthma New Zealand – The Lung Association (Inc) can continue to work productively across New Zealand.

I would like to take this opportunity to thank all the Societies, who worked with Asthma New Zealand throughout the past year and can promise you that we will endeavour to improve throughout the next financial year. A number of approaches are being made to the Ministry and to the local District Health Boards regarding the increase in funding so that more can be achieved.

I would also like to take this opportunity to thank Asthma Auckland in particular for its support for Asthma New Zealand – The Lung Association (Inc). I would also like to thank the PR/Marketing Manager for the tremendous achievements happening in this area and the nursing staff for their commitment and support of children and adults with asthma throughout New Zealand.

Yours sincerely

G.A. Hanna
Secretary/Treasurer
Asthma New Zealand
– The Lung Association (Inc)

Breathe Easy

asthma and your immune system

by Elaine Murray RN
Asthma Nurse Educator

The same system that helps protect you from infections, your immune system, can also be responsible for your worsening asthma. You may notice that at the same time you have a runny nose, watery eyes and sinus congestion, your peak flows are lower, you may be wheezing more, and you may experience shortness of breath. So what is the connection?

Firstly, let's look more closely at the immune system.

The immune system is a very complex and highly developed system, yet it has a very simple mission, seek and destroy invaders. The immune system consists of a network of lymphatic organs, tissues and cells which serve a dual purpose. The lymph system not only drains interstitial fluid back into the circulatory system but also helps to fight disease.

The lymphatic is full of white cells which use various methods to fight foreign bodies, viruses, bacteria and cancer cells. The white cells are essential to the immune system, and include the eosinophils, basophils, monocytes, macrophages, neutrophils and lymphocytes.

Lymphocytes are white cells or leukocytes that play a key role in both immunity and allergy. They are divided into two types, the T and B lymphocytes.

B lymphocytes produce antibodies to help identify and eliminate invading antigens (carried by bacteria or viruses). They are helped by circulating T lymphocytes and macrophages which engulf the virus and therefore protect the body from infection.

An allergic response is a reaction by the body to foreign irritants called allergens. When an allergen enters the body it causes the B cells to produce antibodies, which then attach themselves to the mast cells. The next time the allergen enters the body it is captured by the antibodies on the mast cell. The mast cells respond by releasing histamine, which produce the symptoms of the allergy. They also release a variety of chemical mediators (such as leukotrines, cytokines and prostaglandins) that cause allergic inflammation.

The body does not know which antigens it will encounter, but makes receptor sites for a large number of possible antigens. It is estimated that for the million or so antigens we encounter in our lifetime we have an equal number of specific lymphocytes for each possible antigen.

An allergy is an inflammatory immune response to a non-pathogenic antigen. If you are sensitive to the allergen, the body produces an inflammatory response to get rid of it. Allergic responses can range from mild tissue damage to fatal reactions. The immune response in allergies is called sensitivity or hypersensitivity to the allergen. Exposure to an allergen may be by ingestion, inhalation, injection, or direct contact.

Many asthmatics are atopic (an inherited predisposition towards allergy) where the immune system develops an exaggerated response to certain foreign substances or allergens. The immune system senses these allergens, recognises them as foreign and prepares to fight them off.

This process is often referred to as the allergic cascade, and is divided into three stages;

1. Sensitisation
The first time you are exposed to an allergen, you will not usually develop symptoms, but your body senses it as foreign.

2. Early Phase Response
With re-exposure to the allergen, the inflammatory cell mediators are released and cause allergy symptoms such as wheezing, coughing, feeling tight in the chest and short of breath as the airways become swollen and narrowed. It may cause a runny nose or watery itchy eyes. This response can occur within seconds or minutes of exposure to the allergen and may last for 3-4 hours.

3. Late phase Response
This usually begins at the same time as the early phase response but does not cause any symptoms for several hours (at least 4 hours) but may last as long as 24 hours. It is during this late phase that the tissues become red and swollen due to the arrival of eosinophils, neutrophils, lymphocytes and cytokines. Eosinophils are often present in great numbers in the blood of people with allergies. When they arrive at the site of the allergic reaction, they release chemicals that cause damage to the tissues and continue to promote the inflammation. Repeated episodes of this "late phase" reaction contribute to chronic allergic symptoms and make the tissues even more sensitive to subsequent exposure.

Common allergic asthma triggers are:
- Pollen from trees, grasses and weeds
- Animal dander and proteins in the saliva and urine of cats, dogs, horses and rabbits
- Dust mite and cockroach faeces
- Mould spores
- Food such as peanuts, dairy, wheat or egg

The most convenient way to treat allergies is with medication:
- Antihistamines are used for sneezing, itching and a runny nose (hay fever).
- Anti-inflammatory medications (preventer medication) such as inhaled corticosteroids, are used for asthma. They work on the inside of the airways reducing the hypersensitivity, swelling, mucous and also helps to prevent long term damage.
- Short acting reliever medications (bronchodilators or short acting beta antagonist[5]) relax the muscles around the narrowed airways, therefore relieving the symptoms of asthma e.g. cough, wheeze, shortness of breath or chest tightness.
- Symptom controllers (long acting beta antagonist) help to relax the muscles around the airways for up to 12 hours.
- A leukotriene modifier (Singulair) has recently been found to be helpful in treating asthma and is now subsidised for certain criteria (see article on Singulair)

Another consideration in controlling allergic asthma is to reduce the exposure to the allergen, but you need to know what you are allergic to. If you suspect you are allergic to any of the common allergens mentioned above, discuss with your GP about having a skin prick test or RAST test done, so you know for certain what allergies you may have, and you can therefore work towards avoiding them or reducing your exposure to them.

The last approach to the management of allergies attempts to interfere with the allergic antibody immune response. Allergy shots, (immunotherapy) involves desensitising a patient by injecting increasing amounts of the allergens to which the person is allergic. Over time, the immune system becomes less reactive to these allergens, generates less IgE in response to them, and becomes more tolerant upon re-exposure to them.

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Allergic asthma Symptoms, Treatments, Allergy Triggers, and more
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Human Physiology/ The Immune System
http://en.org/wiki/Human_Physiology/The_ Immune_ System

asthma nz hits #1 in the medical category in the apple app store in nz!

The Asthma New Zealand mobile asthma management app hit #1 in the Medical category in the New Zealand App Store last week.

The Asthma NZ App allows users to track, control and manage their Asthma on a daily basis. Thanks to its sponsors it is free to download on the Apple App Store.

With five Star App Store reviews, users in rural and urban areas the app is meeting the promise of its developer (VADR) and mHealth. Gerry Hanna, CEO of Asthma New Zealand says "The app is proving to cut across ethnic and geographical barriers and deliver health services to users in need of assistance."

The developer and Asthma New Zealand have further enhancements planned. The most recent enhancement has been the addition of a quick add menu.

More information about the app is available at www.breatheeasy.co.nz

The Asthma NZ App is free to download on the Apple App Store.

★★★★★
"Awesome App. Reminds me when I am out and forget to take me medication to take it. Such a good App and so easy!!!"

For more information contact:
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Asthma New Zealand
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dear nurse

Dear nurse I am sure that my child does not have asthma as we have never heard her wheeze, the Doctor has given her all these puffers and I don't think that she needs them.

Dear mum, you cannot always hear a wheeze with asthma, ask your Doctor if he has heard a wheeze when he listens to your daughter's chest with his stethoscope. Do you hear a rattling when she breathes or does she say that her chest feels tight? Is there a history of allergy in your family or does your daughter cough at night? A simple way to check if there is narrowing of the airways is to find out her best peak flow (so long as your child is over 5 years of age) then give her four puffs of her blue puffer through the spacer. Re-measure her peak flow after 20 minutes. If it increases more than 15% it is indicative of asthma. Please do not stop your child's medication until you have discussed it with your doctor or asthma nurse.

Dear nurse, my friend said that the orange puffer will make my child's teeth rot and fall out, is this true?

Dear worried this is not true. Oral cortisone such as Redipred is 1000 times stronger than the orange puffer your child is having. High frequent doses of oral steroids can lead to loss of bone that supports teeth so it makes it even more important that your child's asthma is controlled by Flixotide (the orange puffer). There is a very slight chance of sore throat and oral thrush with Flixotide that is why it is important to gargle and spit or rinse your mouth out after Flixotide.

Dear nurse, my one and a half year old son has had five episodes of Bronchiolitis this year and has needed Redipred each time. Why doesn't the Doctor prescribe a preventer?

This is a common question. Bronchiolitis is a virus, not asthma and usually does not respond to asthma medication. Doctors are reluctant to diagnose asthma before the age of 2yrs. However it is important to consider family history of asthma and allergies such as hay fever and eczema. Does your child have any symptoms of asthma such as coughing at night when he is well or have you heard him wheeze? If his cough is improved by a blue puffer it could well mean the diagnosis of asthma should be considered.

Dear Nurse, I am a 25year old woman and I moved to New Zealand three years ago from India. I have just been diagnosed with asthma. How can this be possible when none of my family in India has asthma and I have never had any signs of asthma until I came here?

Welcome to New Zealand. This can happen with children and adults who move to New Zealand. Triggers that can cause asthma vary from person to person and different countries may have different triggers. Asthma can be diagnosed at any age and does not require a family history for a person to have this diagnosis. Does your family have a history of hay fever, eczema or recurrent bronchitis as these may be an indication that there is a family history of allergy.

Dear Nurse, my aunty said I should not start the orange puffer (Flixotide) as my son will be become addicted to it and need it all his life. Is this true?

No your child will not become addicted. All the orange puffer will do is to reduce the swelling inside his small breathing tubes so that the signs of asthma such as coughing and wheezing will lessen and hopefully disappear. This is not addiction but you do need to take this medication twice daily every day even when well to maintain this improvement. The medication is low dose compared to oral steroids.

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

Heartiest Congratulations on successfully completing Unitec /Asthma New Zealand Asthma and COPD Nursing Courses 2012 – 1st semester

Therese Clarke	Christchurch	Megan Asplin	Palmerston North
Grace Maromo Ndaiziveyi	Wellington	Fiona Clarke	Timaru
Judith Leppard	Levin	Sharron Erbacher	Auckland
Maria Catharina Van Rijzingen	Nelson	Adrienne Riddell	Wellington
Cecille Guimbangan	Manukau	Judith Leppard	Levin
Chris Colbourne	Wanaka	Janine Hayes	Oamaru
Jennifer Helen Broad	Hamilton		

Unitec NEW ZEALAND Te Whare Wānanga o Wairaka

metered dose inhalers

by Adie Riddell RN
Asthma Nurse Educator

As health professionals we are dealing with patients who have respiratory illnesses every day and we are seen as the experts in aerosol therapy. With the cost of medication forever rising and the choices available in drug delivery constantly increasing, it is very important that we have a high level of expertise in delivery devices. For medication to be effective it is important to match the patient's ability to use the device correctly. The five 'rights' that apply to all medication delivery apply also to aerosol therapy: – the right patient, the right medication, the right time, the right route, and the right dose.

There are three common devices used for inhaled drug delivery – the small volume nebulizer, the pressurized metered dose inhaler (pMDI), and the dry-powder inhaler (DPI). Lung deposition may range from 1-50% with clinical aerosol delivery systems. This is largely dependent on the patient, the drug, the disease being treated, and technique. An example of this is 2 actuations (doses) of Ventolin (from a pMDI) dispensing 200 micrograms (ug), only 20-40 ug will reach the lungs with good technique. The remaining drug is lost on the oropharynx, in the device, or in the exhaled breath.[1]

How much do you know about Metered Dose Inhalers and the propellant used in them?

Pressurized Metered Dose Inhalers (pMDI) are one of the most commonly used medication delivery systems for treating asthma, chronic pulmonary obstructive disease (COPD) and other respiratory diseases. They are presently used to administer beta-2 agonist, anticholinergics, anticholinergic/beta-2 agonist combinations and corticosteroids. The MDI became very popular in the 1950s as a popular medication delivery system and was not only portable but perceived as being easy to use becoming the most popular form of inhalation delivery. It has been proven to be a convenient and effective way to deliver medication to the lower airways. The medication covers a large surface area and stays in the airways for a long period of time.[1]

The pMDI consists of a canister, a formulation made up of the drug, a liquefied gas propellant and in many cases, stabilising excipients, a metering valve, the mouthpiece and actuator. The medication represents only 1-2% of the mixture that is emitted from the pMDI and is suspended in the propellant mixture. The propellant of the pMDI makes up 80% of the mixture. The propellant liquid is both the MDI's power source and the suspending, or dissolution medium for the drug. Originally propellants used in MDI's were a chlorofluorocarbon (CFC), but this changed in 2008 due to international agreement to phase out the use of CFC propellants due to their potential impact on the ozone layer.[2] MDI's are now available using hydrofluoroalkane (HFA) as HFA134a and HFA227ea, alcohol and oleic acid propellant which have enhanced the therapeutic index of the drugs, and substantially diminished the number and degree of side effects[4] and improved the distribution of the drug in both large and small airways. These propellants have been shown to have no toxic effects.

The job of the propellant is to effectively provide a force to generate an aerosol cloud and act as a medium to suspend or dissolve the active drug. The volatile propellant very quickly breaks up into droplets which rapidly evaporate resulting in the generation of an aerosol consisting of micrometre-sized particles that are then inhaled. Over 99% of the actuated dose will be propellant. The pMDI is designed as a 'press and breathe' mechanism. By depressing the canister into the actuator the drug/propellant mixture is released and then expands and vaporizes to convert the liquid medication into an aerosol.

Suitable propellants for MDI's must pass a stringent set of criteria. They must be nontoxic, non-flammable and compatible with the elected drug formulation and have appropriate boiling points and densities. They must also be able to dissolve common additives.

There are some important differences between CFC and HFA propellants. CFC's use surfactant for dispersion whereas HFA's contain no surfactant or use alcohol. The feel and taste will be different with HFA, pMDI's having a softer spray and a much warmer spray temperature which is particularly beneficial for those clients sensitive to cold mist inhalation. Whereas with CFC's 'priming' was important following short periods of non-use, this is no longer an issue with HFA's with a longer time of non-use allowed without priming.

A single metred dose of the formulation which includes the medication either dissolved or suspended in the propellant is released when the patient presses down on the top of the canister.

All MDI inhalers available in New Zealand for asthma control are now CFC free. HFA134, in the propellant has been shown to have no toxic effects at very high vapour concentrations in animal studies. Alcohol and oleic acid, the excipients commonly used, will have evaporated prior to reaching the small airways.

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singulair – the latest funded medication for asthma

by Ann Wheat BN
Asthma Nurse Educator

Asthma affects 1 in 4 children and 1 in 6 adults in New Zealand and costs the country over $825 million dollars a year.[1] The main medication for all people with asthma are inhaled corticosteroids, short and long acting beta agonists; the majority are well maintained on these so long as they use their medications as prescribed. There is, however, a group of patients that may still have problems with asthma control and require further add on treatment. One such treatment is Singulair.

As from 1st August this year, Singulair (montelukast sodium) has been funded for the treatment of asthma in people that meet certain criteria. Singulair is now funded for people with exercise-induced asthma and for pre-school children from 2 years of age whose asthma is not well controlled on maximum therapy. It also has specific funding criteria for aspirin desensitization. Singulair has been available in New Zealand for many years (from 1998) but was not funded so the cost was a barrier for most patients in using this medication even if they had been told about it.

What is Singulair and why it is used?

Singulair is a cysteinyl leukotriene antagonist which works to inhibit the leukotriene receptors in the airways. Leukotrienes are inflammatory molecules/chemicals that are mainly produced in the mast cells in the airways.[2] Leukotrienes can have multiple effects in the airways which are airway inflammation, bronchoconstriction and increased airway hyperresponsiveness, decrease in respiratory cilia activity, increase in mucous secretion, vasoconstriction, increased eosinophilic migration into the airway mucosa and airway smooth muscle proliferation.[3] The main effect though is bronchoconstriction in acute episodes of asthma (leukotrienes are 100 to 1000 times more effective than histamine in inducing bronchoconstriction[5]) but they can also be involved in chronic asthma when eosinophils, which also produce leukotrienes, cause airway hyper-reactivity or muscle twitchiness. Singulair therefore inhibits the receptors in the smooth muscles which results in reduced airway eosinophilic inflammation and can lessen the symptoms associated with airway obstruction.[4]

Exercise Induced Asthma

Exercise-induced asthma (EIA) or as it can sometimes be called exercise-induced bronchoconstriction (EIB) can occur in up to 90% of people not treated with anti-inflammatory medication as well as 40% who have allergic rhinitis and up to 12 – 15% of the general population.[5] It is the most common trigger of bronchoconstriction in children with asthma[5] plus children and young adults with asthma are more affected by EIB than adults with asthma.[6] Running is more likely to cause EIA than swimming and more intense activity is more likely to cause symptoms especially if warm up exercises are not completed before the activity.[6] For people with asthma it is essential that they maintain an active lifestyle yet many do not for fear of having an asthma episode. There are several theories as to how EIA occurs including the water loss theory where water is lost from the airways when we breathe with an open mouth and the second that when we breathe in cooler air it dries and cools the airway thereby triggering the asthma reaction.[6] Singulair is used for those people that are on maximum therapy (combination medications and still using short acting reliever therapy more than twice weekly) and still have significant episodes of EIA. It works quickly, is easy to take and people do not become tolerant to it.[6]

For Children from 2 – 5 years

Asthma is often difficult to diagnose in the under 5-year-olds but the condition often starts in this age group and has its greatest prevalence.[7] They have intermittent symptoms often triggered by the common cold.[7] Children often seem to have a greater susceptibility to respiratory infections and in fact viral infections cause up to 85% of childhood asthma exacerbations plus daily symptoms and exacerbations in children and adults with asthma.[7] Leukotrienes are increased for up to 28 days after the onset of a viral-induced respiratory infection and can be found in the nasopharyngeal secretions in children with viral-induced wheeze.[7]

Singulair is given to children from 2-5 years with severe intermittent wheezing, are on maximum therapy of ICS up to 400mcg of beclomethasone or budesonide or 200mcg of fluticasone daily for at least a month and who have had three acute exacerbations a year requiring hospital admission or extended emergency department visits. It significantly reduces the rate of acute asthma exacerbations[4] and increases the time to the exacerbation in those with intermittent symptoms.[7]

Singulair is generally well tolerated. The side effects include headache, behaviour and mood related changes, stomach pain and occasionally hypersensitivity including a skin rash, anaphylaxis or angioedema (swelling of the face and throat).[8]

In conclusion therefore, Singulair is now fully funded for those you meet the criteria. If children from 2-5 who are not well controlled (using preventer medications and still requiring frequent use of a short acting beta agonist – reliever medication) or if children and adults over 5 have EIA despite being on maximum therapy (preventer medications and long acting beta agonist) then they could qualify for this medication and it would be important to see a general practitioner.

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silent night
– or how i learned to love my CPAP

by John Titchener
National Secretary Sleep Apnoea Association of New Zealand

I always knew that I snored. (Well, actually, I didn't know that I snored. I guess I was asleep at the time). But other people certainly told me I snored. On tramping trips, I was often told to leave my chainsaw at home! On one occasion, I was even banished from a hut. And no-one wanted to share a tent with me!

At home, my wife was very tolerant, but eventually, she convinced me to talk to a doctor about it. Reluctantly, I did, and the doctor gave me a referral to a clinic for a sleep study. Which showed that, yes, I snored; that was no surprise. What WAS a surprise, was to be told that I was stopping breathing – effectively, I was suffocating – over 200 times, every night!

Welcome to the world of the sleep apnoeac! How could something as mundane as getting a good night's sleep, be so difficult? What was wrong? At that time, I wasn't particularly overweight; I had a fairly stressful job, with lots of call-outs at night, which left me always tired, but otherwise, I was reasonably healthy. What was this thing they said I had, called Obstructive Sleep Apnoea (OSA)?

Well, "apnoea" simply means "not breathing". "Sleep" means it only happens when I'm asleep. And "obstructive" means that my "not breathing" is caused by my airway closing, or "being obstructed", rather than the much worse disease, "central sleep apnoea", which is a neurological condition where, for some unknown reason, the brain doesn't tell the body that it needs to breathe. With OSA, the airway collapses; and no air can flow until I have woken enough to breathe again.

Anyone with any form of breathing disorder knows all too well, breathing is a fundamental part of "being alive". We need to breathe, to get oxygen into our lungs, and our blood, and from there into every cell in our bodies. And we need to breathe, to get rid of the carbon dioxide that those cells have produced. The level of oxygen in our blood (the oxygen saturation, or SO2), is one of the most critical measures of how well we are. (That is what is being measured with that little clothes-peg thingey they put on your finger when you are in hospital).

"Normal" oxygen saturation is about 95 percent, +/- say 2%. When I stop breathing for about 10 seconds, my SO2 drops to 90%, perhaps to 85%, When I stop breathing for 30 seconds, my SO2 can fall as low as 70%, or even lower. This is not healthy. But when I have OSA, I stop breathing for 20 to 30 seconds, or even longer, many times an hour; in my case, over 200 times a night.

So – I have OSA. What can I do about it? Here came the second surprise: there is no cure for it.

Losing some bodyweight might help a bit, but at that time, I didn't have a lot of excess weight to lose. Surgery helps in some cases, where there is found to be a problem with the airway, but it is expensive; it is quite risky; and for me, there was no indication that there was anything that surgery could fix anyway.

Wearing a kind of "mouthguard" at night (which rejoices in the name of a mandibular advancement device), to hold my lower jaw forward while I am asleep, works in some cases, but not all; again, it is expensive; in most cases, I have to pay the full cost, even if I then find that it does not work; and it requires that the patient has sound teeth, which unfortunately, I do not have.

Breathing therapies, such as Buteyko, or even yoga, have been reported to help some people, and their enthusiasts certainly claim great success. I have not tried them myself, so I cannot make any personal recommendation. My reservations about them are that, by definition, sleep apnoea occurs while I am asleep, so any therapy has to work while I am asleep, and not just when I am awake. And there does seem to be very little research evidence to support the claims made for these therapies. But if they work for you – great; go for it.

The next question is, what would happen, if I did nothing at all? How many times have you heard of people actually dying from OSA? The fact is that very few death certificates state that the direct "cause of death" was sleep apnoea. We are far more likely to die from one of the big five killers – heart disease; lung disease; diabetes; cancer; or in a motor vehicle crash. BUT – and it is a very big "BUT" – every one of those big five killers are made worse, and are more likely to kill me, if I also have untreated OSA. So for me, doing nothing was not an option.

Having eliminated the alternatives, I was left with one treatment – a Continuous Positive Airway Pressure (CPAP) machine. CPAP (pronounced See-pap) is not a "cure", but it does, very effectively, treat the symptoms of OSA. I will have OSA till the day I die; but, provided I use my CPAP, my death, when it comes, will not have been hastened in any way by my having OSA. My life expectancy will be exactly the same as if I did not have sleep apnoea.

Originally invented by an Australian doctor, Colin Sullivan, CPAP works by blowing a constant stream of air, through a mask, and into my airway. It doesn't "fill my lungs with a torrent of air", as some people say. It simply maintains just enough positive pressure to keep my airway open, and so allows me to breathe in the normal way.

CPAP does take a bit of getting used to. There is very little noise; in fact, the new models available now are virtually silent. Having the mask strapped onto my face is a bit daunting at first, but you soon get used to it Some people find the air supplied by the CPAP is a bit cold, or dry, but this can be treated by heating and/or humidifying the air first. This is where it is especially valuable to have access to a "support group", to help you through the first few weeks.

There is quite a range of CPAP machines, and masks, on the market. So if one model does not feel "right", do try one of the alternatives, rather than just throwing the whole thing away. And in fact, some of the world's best quality CPAP machines and masks are designed and made right here in NZ.

For many people, the greatest resistance to using CPAP is simply "vanity". Let's face it – a CPAP mask is NOT the sexiest of nightwear. But, which am I going to prefer – to sleep well; to wake in the morning feeling rested and energetic; and to live longer – or to go on suffering from sleep apnoea; and to die sooner than I need to?

More important is the response of my bed-partner. My wife tells me my CPAP is better than my snoring! And nothing could be as bad as listening to me, stopping breathing for 20, 30, 40 seconds; and then gasping and choking as I start breathing again; and then stopping again … and again …

Are there any long-term problems with using CPAP? I have been sharing my bed with my little friend for sixteen years now, and I am not aware of any down-side. The worst thing for me is that I now depend so much on my CPAP, in order to sleep at all. Before CPAP, I did not sleep "well" – in fact, I slept very badly. But I did get some sleep. Now, if I try to sleep without my mask on, my airway closes almost immediately, and I am roused fully awake by that awful feeling of suffocation.

And, of course, I am totally dependent on having electricity for my CPAP. At home, this is not a problem, so long as we don't have a major earthquake, or a storm like "Sandy" in the US recently. Most modern CPAP machines are designed to run on either mains supply, or on batteries, so if I am a serious CPAP user, I need to have my own batteries. Travelling with a CPAP is another story, for another day. It can be interesting, but so far for me, has not been impossible.

Life with CPAP certainly has some challenges. Sometimes I wish I could do without it. But I can't. Whatever the challenges, it beats the alternative!

nordic walking

by Sharron Erbacher RN BN
Asthma Nurse Educator

Some of you may have seen groups of people walking along the beach with poles in both hands. If you are anything like me your first thought was something along the lines of 'looks a bit daft- where's the snow?' After a little reflecting and some 'googling' on my lap top, I found out that these groups were doing something called Nordic walking and it did indeed start out as a summer training exercise for cross country skiers. Always one to try out something new, I thought I would give Nordic walking a go.

My first stop was a Nordic walking session with Auckland based coach June Stevenson. June explained to me that Nordic walking aims to encourage a more anatomically correct walking position by promoting a fuller range of motion in the torso and arms. She says that often people tend to walk with their heads down to fend off wind, or else walk with hands in pockets, or walk holding a chain attached to the family pet. The end result of such poor posture when walking is that you end up feeling tired faster. Unsurprisingly, recent research agrees with June. Testing of physiological responses related to Nordic walking suggests that Nordic walking tends to increase calorie burn with a lower perceived exertion when compared to normal walking.

Other studies are showing that Nordic walking can be a great option for COPD patients looking for a simple and convenient way of adding to their exercise regime. June's view is that, for COPD patients, the poles provide a postural point of reference. Nordic walking allows the diaphragm and chest to expand. "It's a great way for people to participate in a low impact, total body workout that can use up to 40 per cent more calories than regular walking," she said.

June states, "Nordic walking is all about technique but limb movements should be controlled." The aim is to have coordinated limb movements with the upper body becoming more involved and powerful.

Trying to establish a rhythm between arms and legs, which move opposite to one another, is fundamental to the sport. June explains that participants obtain the best results when they exhibit trunk rotation, which in turn, engages the abdominal muscles encouraging a longer stride and the usually tight hip flexors and hamstrings begin to release. Muscles in the abdominal core, shoulder girdle, chest and arm are all targeted during exercise.

Because it is low impact compared to many other forms of exercise, Nordic walking can be ideal for the elderly, many of whom are discarding their walking sticks in favour of poles. June shared an interesting observation with me based on a recent visit she had made to a rest home. She had noticed the number of people using walking frames to get around and that these people were tilting forward to push the frame along. Should the frame be removed and the person is still leaning forward, the obvious could happen.

Asthma invited June to host a talk on Nordic walking at one of our (COPD) chronic obstructive airway disease groups. Reactions ranged from very interested through to very doubtful and, I am sure, at least one person feeling that snow should be involved somehow. A few walking sessions later, however, and they looked quite inspired with one person mentioning her confidence would be much better as she would be less likely to fall.

To summarise the acute physiological effects of Nordic walking, it increases the energy consumption of the body compared to regular walking with the same speed without poles both in women and men and in fit and less fit individuals. The increase is due to larger working muscle mass in the upper body. The increase varies individually according to walking speed and technique. If the speed is very fast, there is less time for efficient pushing off with poles and thus decreased upper body muscular involvement. Similarly to energy consumption the increase in heart rate is variable. Because perceived exertion in pole walking is often less than true physiological strain, controlling heart rate may be beneficial for those tending to overreach. The resulting increases in energy consumption and heart rate in Nordic walking mean that the cardiovascular strain induced by Nordic walking is greater compared to walking without poles at the same speed.

This is desirable for those people who have difficulty reaching their training heart rate by walking – instead of having to start running they can start using walking poles and continue walking. Walking involves less harmful impacts to the lower extremities compared to running, and therefore may prevent injuries.

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Gerry Hanna and Linda Thompson of Asthma New Zealand with Prof. Ian Town and Angela Francis of the Asthma Foundation

asthma canterbury rebrands as "CANBREATHE"

"Winning is the most important thing in our life – after breathing. Breathing firstly, winning next."

George Steinbrenner

It was an honour to be invited to Asthma Canterbury's 40th birthday and launch of their new brand CANBREATHE.

Patron, Dick Tayler

Linda Thompson, Pamela O'Brien (President – Asthma NZ and CanBreathe) with Gerry Hanna

Andrew and Lynley Tomlin of Torlesse Wines (Sponsor)

Dick Tayler with partner Elspeth Simpson

It was a particular delight to be among a special group of people who have served Asthma Canterbury in its early years and who continue to offer support alongside current committee members and staff.

Alison Willkie, who was the very first Secretary, Anna Bullen who was also on the founding committee and Sandra Yates who was secretary for years and years (around 25 in fact) gathered to celebrate and later were the official cake cutters.

It is through their efforts, and those who followed, that the society has grown to have such a significant presence in the Canterbury community as it has today.

Many fundraisers and 40 years on things have changed and today the face of CanBreathe is a very professional team of nurses and management supported by a dedicated team of volunteer Board Members.

Manager, Teresa Chalecki, spoke about the history of the Society, what it had achieved and where it is heading in the next 40 plus years. From humble beginnings to the professional society it is today. She spoke of the changes since 1972 including improved understanding of asthma triggers and management, advances in medications and the establishment of asthma nurse educators. "Asthma remains a significant health concern; affecting one in five people and especially children". Supporting and educating people with asthma, COPD and other respiratory conditions in self-management techniques remains a key focus of the society. She thanked past and present board members for their support and of course everyone who helped to make the evening a success.

President of Asthma New Zealand and CanBreathe, Pamela O'Brien, was radiant in purple and couldn't contain her pride as she spoke of her amazing team including Teresa and other staff – Louise, Greer and Mary along with the very important Board who are all hands on

north & south
NEWS FROM AROUND THE REGIONS ...

CANBREATHE
Helping Cantabrians breathe easy

Asthma and COPD education and support

Sandra Yates, Alison Wilkie and Anna Bullen

starting at the top with Patron, Dick Tayler, who gave a great speech "lap by lap" of his amazing achievement at the 1974 Commonwealth Games where he won the 10,000 metre event; a testimony that having asthma does not have to limit success in sport or anything for that matter. He waxed lyrical about Arthur Lydiard, his admiration of him obvious. Dick became a national identity following this win and among other titles he was named "Sportsman of the Year 1974. Dick is held in high esteem throughout Canterbury and is enthusiastic about supporting CanBreathe to increase its profile and support the community any way he can.

Canterbury, like all societies in New Zealand, is fortunate that there are two national organiations spearheading research and education of asthma, supporting local societies by providing their resources and expertise. To that end both Gerry Hanna of Asthma New Zealand and Prof. Ian Town of the Asthma Foundation spoke briefly about the issues we face and congratulated Asthma Canterbury for 40 years of service.

The future of societies is and always will be reliant upon the support and presence maintained locally!

Linda Thompson
PR / Marketing Manager
Asthma New Zealand

Teresa Chaleki, Sandra and John Yates

Dianna Green (HRV), Teresa Chaleki and Niall Crawford (HRV Canterbury South)

Linda Thompson and Gerry Hanna, Asthma NZ

Mary (past President) and David Wells

the camp

Written by Karen Little
Asthma Nurse Educator

Joe was so excited that he was allowed to go on camp again after being sent home early last year.

He was sent home not because he got covered in mud, ate 10 sausages or put a weta in Sam's bed but because he could not stop wheezing and coughing, especially at night.

This year was going to be awesome because the asthma nurse had visited and given him a plan to get ready.

Mum was helping him get ready as she was upset that he had to come home early last year especially as she was having a relaxing time with Aunty Robyn and the cousins in New Plymouth.

Joe had now been taking his orange puffer through his spacer morning and night for three months.

His teacher, Miss Scott, said he could sleep in the top bunk. Joe had a skin prick test last year and found out that he was allergic to dust mites. When Joe saw the picture of what a dust mite looked like he really freaked out. He told his friends they looked even worse than the creature in the movie Aliens. The nurse kindly told him that everyone had dust mites and even the Queen in England had them in her castle. The dust mites were so small, that even when he tried looking for them with his mum's glasses he could not see them. You had to have a microscope. By sleeping in the top bunk he would not have them or their poo fall down on top of him when the person in the top bunk rolled over. Mum had bought him a new sleeping bag, pillow and blanket so he knew that they would not make him wheeze. Joe was glad to know that dust mites do not bite you instead they eat the dead skin that falls off your body. It was not the mites that were making him wheeze but their poo!

The teachers now knew how much of the blue puffer to give through the spacer if he needed it and what signs to look for if he started to get asthma.

Joe felt really good and did not think he would get asthma as he had been playing rugby three times a week without a wheeze. His doctor had told him that it would be a good idea to have two puffs of his blue puffer ten minutes before they started the big swimming race in the river at camp. He would also warm up his muscles before he jumped into the river. Joe was ready to win that race!

"Goodbye Mum and Dad, see you in a week".

Kid's Page

Unscramble words then name the pictures

1 epro
2 PPale
3 Rac
4 Tac
5 Reet
6 Sifh
7 Gdo
8 relahin
9 tah
10 ipg








Answers
1 rope
2 Apple
3 Car
4 Cat
5 Tree
6 Fish
7 Dog
8 Inhaler
9 Hat
10 Pig

Tree Farm

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

Acacia
Alder
Almond
Apple
Aspen
Balsa
Basswood
Beech
Birch
Boxwood
Butternut
Catalpa
Cedar
Cherry
Chestnut
Coconut
Dogwood
Ebony
Elder
Hemlock
Hickory
Holly
Larch
Laurel
Linden
Locust
Magnolia
Mango
Orange
Poplar
Redwood
Spruce
Tupelo
Umbrella
Walnut
Willow

9 November 2012 saw the event RISING STARS take place at the Waitakere Trusts Stadium in West Auckland.

This event was the inaugural Combined Combat Inc. fight night, and the Charity that we selected to align ourselves with was Asthma NZ.

Steve Oliver (son of Commonwealth Gold medallist Don Oliver) and Lolo Heimuli – the directors of Combined Combat Inc. – both feel strongly about the growing numbers of people with asthma in NZ and think it's a responsibility of everyone to get on board and help fight asthma together.

Steve, in particular, is a long time sufferer of asthma but has not let that stop him from achieving world titles. Having asthma since he was a child Steve has managed a healthy collection of titles and still holds the NZ Junior record for deadlift at 292 kg at 90 kg.

Lolo Heimuli is head coach of the famous Balmoral Lee Gar Muay Thai Kickboxing club – and has trained 100's of elite athletes, that have gone on to compete on the world stage, that have suffered from asthma.

We believe that Combined Combat Inc., along with Asthma NZ can work together to fight asthma head on in New Zealand. There is nothing more important than the health of both young and old that are suffering from this disease and believe more organisations need to align themselves with hard working charities like Asthma NZ.

As a part of the very first Combined Combat Inc. event RISING STARS – PR / Marketing Manager Linda Thompson from Asthma NZ was invited as a VIP guest to get a taste of what Combined Combat Inc. represented. Elite New Zealand athletes, competing on a first class stage many of whom have overcome the challenges of asthma to get where they are today.

Combined Combat Inc. have received items from NZ UFC greats such as Mark Hunt and Jamie Te Huna – these items will be auctioned off on TradeMe with 100% of those proceeds going to Asthma NZ.

The directors of Combined Combat Inc. have indicated that as they grow in New Zealand so will their contributions to Asthma New Zealand – the Lung Association.

Combined Combat Inc.

Keziah Matthews, Mark Hunt (NZ UFC legend), Tash Thompson, Linda Thompson (Asthma NZ), Nela Luau, Aaron Spellman and Vuna Tupou

Steve Oliver

Steve Oliver, Ali Campbell (UB40 legend), Linda Thompson and Mark Hunt

north & south
NEWS FROM AROUND THE REGIONS ...

GLUTEN FREE FOOD AND ALLERGY SHOW

The Wellington team attended the Gluten Free Food and Allergy show again this year and once again it was a very worthwhile weekend. With 30.7% of all attendees at the show being asthmatics we were kept busy with questions and requests for asthma education. It is great to have such an event in Wellington where it is possible to reach such a large target audience in such a short time.

Did you see us at the Johnsonville Mall on World COPD day? COPD (chronic obstructive pulmonary disease) is a major cause of illness, yet many people have it and don't know. Finding COPD early gives the best chance to prevent further lung damage, however treatments are available to help people at all stages of disease feel better and live a more active life. As with anything, awareness is the first step on the path to better health and that was our mission for the day. For further information about COPD go to www.goldcopd.org.

In the last three months we have introduced Asthma Clinics into pharmacies in the Wellington South and Kapiti areas. These have proved very popular with all appointments fully booked We plan

to extend this service in 2013. If your pharmacy would like to host a clinic please let us know.

Our 'Hailer' newsletter and the 'Hailer School Edition' have proved very popular. With each edition we are increasing our circulation. If you wish to receive a copy please let us know your email address.

Just a reminder our office will be closed from Thursday 20 December through to Monday 14 January. Feel free to leave a message during this time (04 237 4520) and we will contact you on our return.

We wish you all a safe and happy holiday season. Don't forget to take your medications with you when away from home and to continue with your preventers – not always an easy feat when out of your day to day routine!

Kim and Adie

Fully enclosed dust mite covers for mattresses, duvets and pillows.

If you have any covers that are no longer required please consider donating them to Asthma Auckland to distribute to those in need. If required we can collect from anywhere in the Auckland region.

north & south

NEWS FROM AROUND THE REGIONS ...

new zealand branch of the thoracic society of australia and new zealand (TSANZ) and new zealand society of respiratory science (ANZRS) annual scientific meeting

Ann Wheat and Karen Little

Ann Wheat and Karen Little were very grateful for the sponsorship from Tony Davison of Boehringer Ingelheim to enable them to attend the 2 day conference in Queenstown, 15th – 17th August 2012.

Invited speakers included

- Professor Richard Edwards, Head of department of Public Health, University of Otago, Wellington
- Dr Margot McLean, Medical Officer of Health, Wellington Public Health
- Dr Jeff Pretto, Scientific Director, Department of Respiratory and Sleep Medicine, John Hunter Hospital, Newcastle, Australia

Of particular interest was the lecture given by Debbie Hannah R.N. on Respiratory Nurse Clinics with Advance Care Planning. Debbie described the Dunedin respiratory Department experience on end stage Chronic Obstructive Pulmonary disease (COPD). The short film that the Department made called "A Good Death" was excellent viewing and thought provoking. The idea was to give permission for people with end stage COPD to talk about death and to look at different end of life scenarios. To plan in advance on how you would like to be treated at the end of life holds many advantages.

Dr Helen Snell spoke about the involved process enabling registered nurses practising in diabetes health to become designated prescribers. This was of particular interest as the process could be transferred to respiratory care. As asthma nurse educators it would be incredibly useful to be able to prescribe some asthma medications.

Other lectures included TB in the New Millennium, Achieving a smoke free New Zealand and Assessment of Hypoxia. There were sessions on case studies and young investigator presentations.

Unfortunately neither of us won the door prize of a brand new laptop.

It was also great to be able to network with other health professionals working in similar fields around New Zealand and to be able to listen and learn from their experience.

new asthma bags

Chelsea Makita (13)

Asthma Auckland is thrilled to announce that due to generous donations from Producers Trust and MiteGuard we have been able to produce cloth bags to store spacers and asthma medications in. We often see in the community that children misplace their asthma medications and spacers, because of this compliance is compromised. On the outside of the bag there are written instructions about spacer care, a reminder that preventers are to be taken twice daily even when well and what to do in an emergency. The children who have received this gift have been really pleased that we can print their name and phone number on it as this makes it especially theirs.

pulmonary rehabilitation for copd

by Janet Delooze RN
Asthma Nurse Educator

The most common symptom that people with COPD suffer from is breathlessness. This makes it increasingly more difficult to carry out everyday activities therefore they are less likely to want to exercise. This in turn causes muscle weakness and further breathlessness with activity and so starts a vicious cycle.

Regular exercise to strengthen muscles and improve fitness has been shown to slow down or reverse this cycle: "People who exercise regularly say that they are less breathless, have more energy and feel better in themselves".[1] It can also reduce the need for hospital admission.

Pulmonary rehabilitation has been defined as "evidence-based, multidisciplinary and comprehensive intervention for patients with chronic respiratory disease who are symptomatic and often have decreased daily activities".[2]

Pulmonary rehabilitation has been shown to reduce disability brought about by respiratory diseases.[3] Training and exercise can bring about a ten-fold improvement in normal lungs. In COPD, it can improve quality of life and exercise tolerance.[4] It can also help reduce breathlessness, anxiety and depression, and may reduce hospitalisation.[5]

Aims of the programme

- Increase exercise tolerance
- Improve adherence to recommended treatments
- Reduce frequency and severity of symptoms
- Improve mood and motivation
- Build self-management capacity
- Increase participation in everyday life
- Improve quality of life
- Reduce health care usage for patients, families, communities
- Improve survival.[6]

Who runs them?

Usually run by a physiotherapist with a team of other professionals which may include:

- Dietician or nutritionist
- Occupational therapist
- Social worker
- Mental health professional
- Relevant specialists or physicians
- Pharmacist
- Exercise physiologist
- Specialised nursing staff
- Speech therapist.[7]

What is involved?

Pulmonary rehabilitation programmes may vary from place to place but will have similar components and goals including:

Assessment – a baseline assessment of the current state of health and exercise tolerance

Exercise training – working out an individualised programme according to needs and abilities. Also, teaching people how to breathe correctly and techniques to help with breathlessness.

Education – this may include information on respiratory diseases such as COPD, benefits of exercise, medications and how to use them correctly, smoking cessation and support, nutritional requirements in respiratory disease, self-management using an action plan particularly seeking early medical attention with exacerbations.

Programme evaluation – assessing how the programme is working for the individual and adjusting the exercise programme as required.

Maintenance – help and support to maintain exercise levels once the programme has finished. Information is given on support services and exercise facilities out in the community.

A comprehensive programme may include exercise training, smoking cessation, nutrition counselling and education.[8]

Who can attend?

Anyone with a lung disease that makes them breathless. People with COPD make up a large proportion of the groups in the Auckland area. WHO GOLD advocate pulmonary rehab for patients with FEV1 below 80% of the predicted value.

How long?

The minimum length of an effective programme is 6 weeks; the longer the program continues, the more effective the results.[9] The North Shore Hospital Pulmonary Rehabilitation works on an 8 week rolling programme. People can start at any time, once they have been offered a place. The format at each session is usually exercise and a speaker or some sort of education.

There are pulmonary rehabilitation groups in Auckland at Greenlane Hospital, North Shore Hospital, Waitakere Hospital and AUT physiotherapy department. At the moment, referrals will only be taken from doctors so people may need to ask their GP or specialist if a referral would be appropriate for them.

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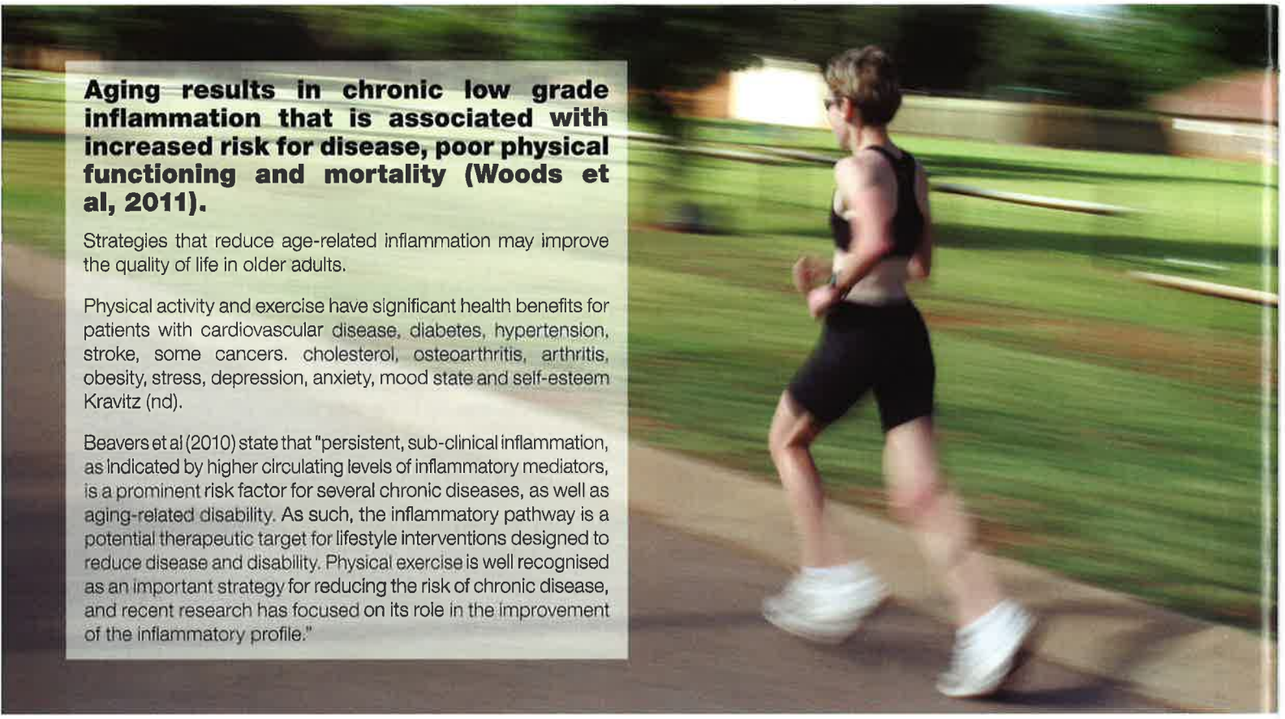
exercise

by Elaine Murray RN
Asthma Nurse Educator



Aging results in chronic low grade inflammation that is associated with increased risk for disease, poor physical functioning and mortality (Woods et al, 2011).

Strategies that reduce age-related inflammation may improve the quality of life in older adults.

Physical activity and exercise have significant health benefits for patients with cardiovascular disease, diabetes, hypertension, stroke, some cancers. cholesterol, osteoarthritis, arthritis, obesity, stress, depression, anxiety, mood state and self-esteem Kravitz (nd).

Beavers et al (2010) state that "persistent, sub-clinical inflammation, as indicated by higher circulating levels of inflammatory mediators, is a prominent risk factor for several chronic diseases, as well as aging-related disability. As such, the inflammatory pathway is a potential therapeutic target for lifestyle interventions designed to reduce disease and disability. Physical exercise is well recognised as an important strategy for reducing the risk of chronic disease, and recent research has focused on its role in the improvement of the inflammatory profile."

Exercise and asthma

Exercise is important for people with asthma as it helps to improve their fitness level. The fitter you are the better control you have of your asthma and you may have fewer episodes. You can participate in sport or exercise at the top level if you have good management of your asthma.

Asthma is not a reason to avoid exercise.

- With a proper diagnosis and the most effective treatment, you should be able to enjoy the benefits of an exercise program without experiencing asthma symptoms.
- Preventer medication must be used every day morning and night to control your asthma.
- Use a peak flow regularly to monitor your asthma.
- Know what your triggers are.
- Regularly review your asthma control with your GP.

Important

A person with asthma must always have their blue reliever with them at all times.

You can take 2 puffs of your blue reliever before doing a warm up. Do some gentle warm-up exercises slowly for 10-15 minutes before you start exercising/or playing sport. This will gradually increase your heart and breathing rate.

It is important to stop if you experience difficulty in breathing. Take some slow, deep breaths. Take another 2 puffs of the blue reliever. When symptoms improve you will be able to carry on.

If your symptoms continue or get worse, take 6 puffs of the blue reliever through a spacer. If still no improvement, seek medical help.

Remember

Do some cool-down exercise similar to your warm-up to get your breathing and heart rate to slowly return to normal.

Make sure your coach or sports instructor knows that you have asthma, and knows what to do if you have an emergency.

NB

- Don't exercise if you are unwell.
- Avoid the cold air if it is a trigger, indoor swimming pools if chemicals are your trigger, the outdoors if pollens are a problem.
- Swimming, cycling, running/jogging and yoga are activities recommended for asthmatics.
- These will help to expand the capacity of your lungs and therefore will help to improve your breathing and lung function, open up your airways and improve oxygenation.
- Yoga helps you to relax your entire body in general. With asthma, it is helpful for relaxing and improving breathing technique.

Exercise and COPD

When the structure of the lungs change, the amount of work it takes to breathe increases. Normally the diaphragm is the main breathing muscle. However, in severe COPD, the upper chest muscles become the main breathing muscles. And sometimes these muscles can be overused, causing muscle fatigue, general fatigue and further shortness of breath. Another common problem is hyperventilation – "over breathing" – when there is too much air volume with an increased breathing rate. Hyperinflation also increases muscle work and further shortness of breath.

The shortness of breath is often made worse by activity/exercise and stress.

Common ineffective breathing patterns are:

- Over using the upper chest; shoulders may be raised and neck muscles stand out
- Breath-holding
- Hyperventilation
- Gulping in air

The aim of effective breathing:

- Is to save energy
- Allows relaxation of the muscles
- Decreases shortness of breath
- Reduces stress
- Gives you breathing control
- Allows total relaxation of mind and body

Breathing control during exercise/activity

People with COPD often hold their breath during activity without being aware of it. This only makes things worse.

Don't hold your breath!!!

Use breathing control; find a rhythm that suits you or what you are doing.

For example

- When walking, focus on the rhythm of your breathing; use a long slow breath out
- When bending over, breathe out
- When raising your arms, breathe in
- When lowering your arms, breathe out.

Improvement through movement

Bodies like movement. Fear of breathlessness leads people with chronic breathing problems to do less and less.

Feeling short of breath with exercise is NOT harmful, but lack of exercise is.

It leads to muscle weakness, weak bones, weight increase, depression and less activity.

This is a vicious cycle. BUT, this cycle can be prevented or broken by exercise.

People with chronic breathing problems should exercise every day.

Walking is the easiest.

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the link between untreated or poorly controlled asthma and chronic obstructive pulmonary disease (COPD)

by Karen Little RN
Asthma Nurse Educator

Chronic obstructive pulmonary disease (COPD) is a slowly progressive disease mainly caused by cigarette smoking and is set to become the fifth highest cause of death in the world by 2020.[1] It is a chronic inflammatory condition, which progressively gets worse causing narrowing of the airways that is predominantly irreversible. COPD is an overlapping spectrum of disease processes which may manifest itself as a combination of three key pathological processes, all causing different symptoms including chronic bronchitis, emphysema and chronic uncontrolled asthma.[2] In this article I will be exploring the ramifications of untreated or poorly controlled asthma.

Remodelling of the small airways is a key factor in the development of the irreversible airflow limitation characteristic of COPD. Airway remodelling describes the persistent changes that occur within the structural components of the airways in response to inflammation and occurs in patients with either asthma or COPD.[3] Airways that are red, inflamed and swollen over a long period because of insufficiently controlled or untreated asthma can develop structural changes called fixed airways obstruction. If left unchecked remodelling (a form of thickening of the sub epithelial basement membrane) of the airways results, which contributes to irreversible airflow obstruction.

Importantly it needs to be stressed that the inflammatory changes seen in COPD are not the same as those that occur in asthma and this probably explains the different responses to pharmacotherapy, for example, inhaled corticosteroid (ICS) responses, seen in the two diseases.[4] However, it has also been suggested that airway remodelling in asthmatic patients may be related to the development of COPD symptoms. Including non-reversible airway obstruction and accelerated decline in Forced Expiratory Volume (FEV1) suggesting functional and pathologic overlap between the two diseases.[5]

There is growing evidence regarding the slowing of remodelling with the use of anti-inflammatory drugs in the form of ICS and other treatments, which can be effective in reducing eosinophilic inflammation.[5] Eosinophilic inflammation of the airways is correlated with the severity of asthma. These cells are likely to play a part in the epithelial damage seen in this disease. Although eosinophilic airway inflammation is usually considered a feature of asthma, it has been demonstrated in large and small airway tissue samples and in 20%–40% of induced sputum samples from patients with stable COPD.

Disease exacerbation in both asthma and COPD can lead to an accelerated decline in lung function. Previous reports have shown an association between severe asthma exacerbation and an accelerated decline in forced expiratory volume in 1sec (FEV1), to a degree similar to that seen with smoking and COPD. Another important observation was that the decline in FEV1 seen in patients with infrequent exacerbation was similar to that in a population without asthma. These findings suggest that repetitive episodes of exacerbation may result in fixed airflow obstruction in asthma and contribute to the phenotypic overlap between asthma and COPD.[6]

It should be noted, however that the nature of this association is not yet fully understood, and the correlation between asthma airway remodeling and an increased risk of developing COPD requires further investigation.

COPD is a leading cause of preventable death and hospitalisation for Maori compared to non-Maori, Maori experience over twice the COPD prevalence, 3.6 times the COPD hospitalisations and 2.7 times the COPD deaths.[7] Asthma is the most common respiratory cause of hospitalisation for Maori. There is evidence to suggest that access to preventative health care and differential asthma treatment by ethnicity are factors contributing to asthma inequalities for Maori.[7] More needs to be done to ensure Maori children have the same opportunity as non-Maori to benefit from elements of best practice asthma care, including education, regular asthma reviews, appropriate medication and an asthma management plan.

There is some evidence that effective early introduction of anti-inflammatory treatment in children with asthma improves the prognosis in the sense that the earlier the treatment is started, the greater the improvement in lung function.[8] Many families are unaware that coughing at night may be a sign of asthma. It is important that asthma education and diagnosis is available to all families in New Zealand so that appropriate treatment may be commenced at the earliest opportunity.

As an asthma nurse educator we see poorly controlled and untreated asthma on a daily basis. The reluctance to introduce inhaled corticosteroids is a common feature usually due to misinformation on how these medications work and understanding the difference between preventers and relievers. Many days are lost from school and with some children these days lost may never be recovered in their education. Compounding this may be the fact that due to coughing at night the child will be extremely tired at school. We can measure the impact of untreated asthma by measuring peak flow. It is not uncommon for the peak flow to increase up to 40% after the administration of Ventolin via a spacer. A person living with uncontrolled asthma may become so accustomed to "running on half empty" that they are not aware of the quality of life that could be experienced if their asthma was well controlled. All of these signs and symptoms are quite apparent to a health professional and with explanation to the family involved. The hidden ongoing ramifications of untreated asthma may only become apparent in later life with decreased lung function and perhaps a diagnosis of COPD.

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Source: J Allergy Clin Immunol
Increased airway smooth muscle in preschool wheezers who have asthma at school age;
O'Reilly R, Ullmann N, Irving S, Bossley CJ, Sonnappa S, Zhu J, Oates T, Banya W, Jeffery PK, Bush A, Saglani S; Journal of Allergy and Clinical Immunology (JACI) (Oct 2012)

BACKGROUND: Increased airway smooth muscle (ASM) is a feature of established asthma in schoolchildren, but nothing is known about ASM in preschool wheezers. OBJECTIVE: We sought to determine endobronchial biopsy specimen ASM area fraction in preschool wheezers and its association with asthma at school age. METHODS: ASM area, reticular basement membrane thickness, and mucosal eosinophil and ASM mast cell values were quantified in endobronchial biopsy specimens previously obtained from preschool children undergoing clinically indicated bronchoscopy: severe recurrent wheezers (n = 47; median age, 26 months) and nonwheezing control subjects (n = 21; median age, 15 months). Children were followed up, and asthma status was established at age 6 to 11 years. Preschool airway pathology was examined in relation to asthma at school age. RESULTS: Forty-two (62%) of 68 children had 1 or more evaluable biopsy specimens for ASM. At school age, 51 of 68 children were followed up, and 15 (40%) of 37 preschool wheezers had asthma. Children who had asthma and an evaluable biopsy specimen had increased preschool ASM area fraction (n = 8; median age, 8.2 years [range, 6-10.4 years]; median ASM, 0.12 [range, 0.08-0.16]) compared with that seen in children without asthma (n = 24; median age, 7.3 years [range, 5.9-11 years]; median ASM, 0.07 [range, 0.02-0.23]; P = .007). However, preschool reticular basement membrane thickness and mucosal eosinophil or ASM mast cell values were not different between those who did or did not have asthma at school age. CONCLUSION: Increased preschool ASM is associated with those children who have asthma at school age. Thus a focus on early changes in ASM might be important in understanding the subsequent development of childhood asthma.

Source: J Allergy Clin Immunol
A population analysis of prescriptions for asthma medications during pregnancy;
Zetstra-van der Woude PA, Vroegop JS, Bos HJ, de Jong-van den Berg LT; Journal of Allergy and Clinical Immunology (JACI) (Oct 2012)

BACKGROUND: It is important to control asthma during pregnancy. However, some studies indicate that women stop or change their asthma medications when they become pregnant. OBJECTIVE: We used a population database to analyze changes in prescriptions for asthma medications to patients before, during, and after pregnancy. METHODS: We collected information from a pregnancy database that is part of the population-based pharmacy prescription InterAction Database from the northern Netherlands. Our study cohort comprised 25,709 pregnancies for which prescription data were available. We collected data over a study period of 1 year before pregnancy until 6 months after birth and analyzed data from pregnant women who received at least 1 prescription for asthma medication during the study period (n = 2072), identifying all prescriptions for asthma medication and oral corticosteroids. RESULTS: Prescriptions for asthma medications did not change during pregnancies from 1994-2003. However, during the 2004-2009 period, there was a significant decrease (P = .017) in prescriptions for asthma medications during the first months of pregnancy compared with the months before pregnancy, especially prescriptions of long-acting bronchodilators. Although most asthma prescriptions continued throughout pregnancy, prescriptions for controller therapies were reduced by 30% during the first months of pregnancy. CONCLUSIONS: Many women stop or reduce their use of asthma medications when they become pregnant. Strategies to safely control asthma during pregnancy are needed.

Source: Ann Allergy Asthma Immunol
Exhaled RANTES and interleukin 4 levels after exercise challenge in children with asthma;
Keskin O, Keskin M, Kucukosmanoglu E, Ozkars MY, Gogebakan B, Kul S, Bayram H, Coskun Y; Annals of Allergy, Asthma, & Immunology 109 (5), 303-8 (Nov 2012)

BACKGROUND: Despite the universality and clinical significance of exercise-induced bronchospasm (EIB), the mechanisms responsible for it are incompletely understood. OBJECTIVETo investigate the role of exhaled RANTES (regulated on activation, normal T-cell expressed and secreted) and interleukin (IL) 4 in EIB in children with asthma. METHODS: Fifty-six children with asthma were evaluated with exercise challenge and exhaled RANTES and IL-4 levels. Exhaled breath condensate was collected before and 30 minutes after exercise challenge. RANTES and IL-4 concentrations were determined using a specific immunoassay kit. RESULTS: A significant increase was found in RANTES levels after exercise challenge in the asthmatic children (P<.001). A statistically significant increase in RANTES levels was noted after exercise challenge in both the asthmatic children with EIB (n=25, P=.007) and in the non-EIB asthmatic group (n=31, P=.005). Our study revealed that exhaled RANTES level correlates significantly well with percentage of forced expiratory volume in 1 second (FEV(1)), exacerbation frequency, serum IgE, and body mass index. No statistically significant increase was found in IL-4 levels after exercise challenge. The maximal postexercise decrease in FEV(1) strongly correlated with total eosinophil count (P<.001, r=-0.61) and baseline ratio of FEV(1) to forced vital capacity (FVC) (P=.002, r=0.40). Results from multivariate regression analysis adjusted for age, sex, and atopy as covariates showed that eosinophil count and FEV(1)/FVC ratio were significantly associated with EIB. CONCLUSION: We found that exercise challenge, leading to hyperosmolar stimulus, may increase exhaled RANTES levels in children with asthma. In addition, exhaled RANTES levels correlate well with serum IgE, severity of asthma, FEV(1)/FVC ratio, and body mass index. RANTES and IL-4 may not be independent predictors for EIB. Furthermore, eosinophil count and FEV(1)/FVC ratio may predict the presence and severity of EIB in asthmatic children.

Source: Respir Res
Analysis of longitudinal changes in dyspnea of patients with chronic obstructive pulmonary disease: an observational study;
Oga T, Tsukino M, Hajiro T, Ikeda A, Nishimura K; Respiratory Research 13 (1), 85 (Sep 2012)

ABSTRACT: BACKGROUND: Guidelines recommend that symptoms as well as lung function should be monitored for the management of patients with chronic obstructive pulmonary disease (COPD). However, limited data are available regarding the longitudinal change in dyspnea, and it remains unknown which of relevant measurements might be used for following dyspnea. METHODS: We previously consecutively recruited 137 male outpatients with moderate to very severe COPD, and followed them every 6 months for 5 years. We then reviewed and reanalyzed the data focusing on the relationships between the change in dyspnea and the changes in other clinical measurements of lung function, exercise tolerance tests and psychological status. Dyspnea with activities of daily living was assessed with the Oxygen

Cost Diagram (OCD) and modified Medical Research Council dyspnea scale (mMRC), and two dimensions of disease-specific health status questionnaires of the Chronic Respiratory Disease Questionnaire (CRQ) and the St. George's Respiratory Questionnaire (SGRQ) were also used. Dyspnea at the end of exercise tolerance tests was measured using the Borg scale. RESULTS: The mMRC, CRQ dyspnea and SGRQ activity significantly worsened over time ($p<0.001$), but the OCD did not ($p = 0.097$). Multiple regression analyses revealed that the changes in the OCD, mMRC, CRQ dyspnea and SGRQ activity were significantly correlated to changes in forced expiratory volume in one second (FEV1) (correlation of determination ($r2$) = 0.05-0.19), diffusing capacity for carbon monoxide ($r2$ = 0.04-0.08) and psychological status evaluated by Hospital Anxiety and Depression Scale ($r2$ = 0.14-0.17), although these correlations were weak. Peak Borg score decreased rather significantly, but was unrelated to changes in clinical measurements. CONCLUSION: Dyspnea worsened over time in patients with COPD. However, as different dyspnea measurements showed different evaluative characteristics, it is important to follow dyspnea using appropriate measurements. Progressive dyspnea was related not only to progressive airflow limitation, but also to various factors such as worsening of diffusing capacity or psychological status. Changes in peak dyspnea at the end of exercise may evaluate different aspects from other dyspnea measurements.

Source: Respir Med

Short term and long term effects of pulmonary rehabilitation on physical activity in COPD;

Egan C, Deering BM, Blake C, Fullen BM, McCormack NM, Spruit MA, Costello RW; Respiratory Medicine (Oct 2012)

The central purpose of pulmonary rehabilitation is to reduce morbidity by improving functional capacity through exercise. It is still unknown if improvements in functional capacity are maintained in the long-term and if this leads to increased physical activity levels as measured by a free-living activity monitor. The hypothesis of this study was that pulmonary rehabilitation would lead to a sustained increase in standard outcome measures and in daily physical activity. METHODS: A prospective study of 47 subjects with COPD was performed, registered at ClinicalTrials.gov (Clinical Trial Number NCT 0112943). The primary outcome was a maintained improvement in standard outcome measures with a secondary aim of an increase in daily physical activity. A convenient sample of the cohort ($n = 17$) was re-evaluated at a third time point at 1 year. RESULTS: A seven week hospital based outpatient pulmonary rehabilitation program led to a significant reduction in total energy expenditure ($p < 0.044$) and breathlessness (Borg, $p < 0.011$) and improved exercise capacity (ISWT, $p > 0.001$, 6MWT, $p > 0.002$) PiMax ($p > 0.007$) and quality of life scores (SGRQ, $p > 0.001$, EQ5D, 0.025). However, pulmonary rehabilitation did not significantly change the average number of daily steps taken, time spent sedentary activity, METs consumed or daily physical activity. Indeed, all of the standard and freeliving values had returned towards the baseline value at 1 year. DISCUSSION: These findings show that while pulmonary rehabilitation increased exercise capacity this was not transmitted into increased daily physical activity. Hence, alternative methods to alter/affect behavioural change need to be addressed.

Source: Int J Chron Obstruct Pulmon Dis

Exacerbation frequency and course of COPD;

Halpin DM, Decramer M, Celli B, Kesten S, Liu D, Tashkin DP; International Journal of Chronic Obstructive Pulmonary Disease 7 653-61 (2012)

BACKGROUND: Exacerbations affect morbidity in chronic obstructive pulmonary disease (COPD). We sought to evaluate the association between exacerbation frequency and spirometric and health status changes over time using data from a large, long-term trial. METHODS: This retrospective analysis of data from the 4-year UPLIFT(®) (Understanding Potential Long-term Impacts on Function with Tiotropium) trial compared tiotropium with placebo. Annualized rates of decline and estimated mean differences at each time point were analyzed using a mixed-effects model according to subgroups based on exacerbation frequency (events per patient-year: 0,>0-1,>1-2, and>2). Spirometry and the St George's Respiratory Questionnaire (SGRQ) were performed at baseline and every 6 months (also at one month for spirometry). RESULTS: In total, 5992 patients (mean age 65 years, 75% male) were randomized. Higher exacerbation frequency was associated with lower baseline postbronchodilator forced expiratory volume in one second (FEV(1)) (1.40, 1.36, 1.26, and 1.14 L) and worsening SGRQ scores (43.7, 44.1, 47.8, and 52.4 units). Corresponding rates of decline in postbronchodilator FEV(1) (mL/year) were 40, 41, 43, and 48 (control), and 34, 38, 48, and 49 (tiotropium). Values for postbronchodilator forced vital capacity decline (mL/year) were 45, 56, 74, and 83 (control), and 43, 57, 83, and 95 (tiotropium). The rates of worsening in total SGRQ score (units/year) were 0.72, 1.16, 1.44, and 1.99 (control), and 0.38, 1.29, 1.68, and 2.86 (tiotropium). The proportion of patients who died (intention-to-treat analysis until four years [1440 days]) for the entire cohort increased with increasing frequency of hospitalized exacerbations. CONCLUSION: Increasing frequency of exacerbations worsens the rate of decline in lung function and health-related quality of life in patients with COPD. Increasing rates of hospitalized exacerbations are associated with increasing risk of death.

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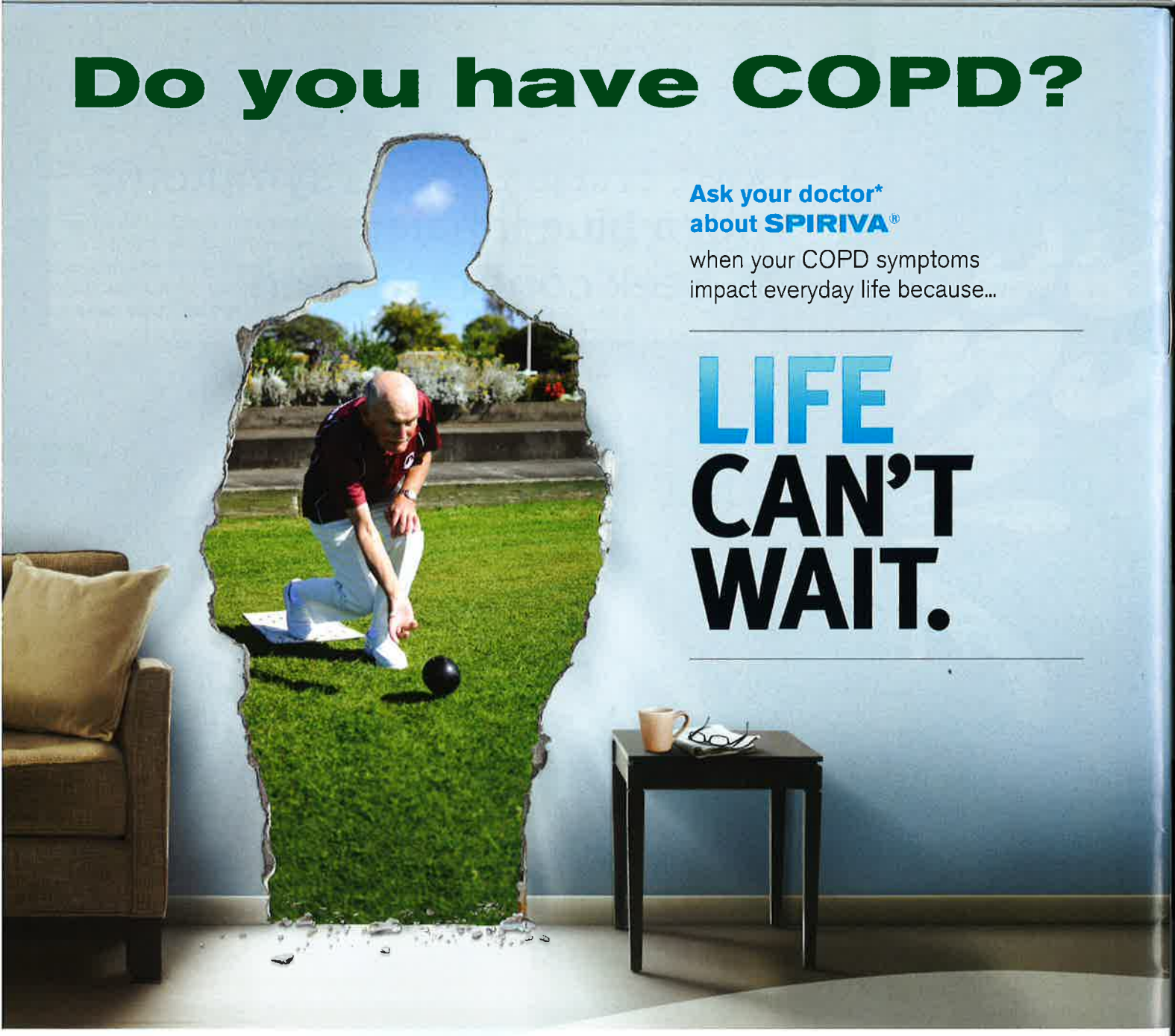

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