THE NZ JOURNAL OF RESPIRATORY HEALTH

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THE NZ JOURNAL OF RESPIRATORY HEALTH

ISSN 1176-7847

PUBLISHER
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Auckland 1024
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ON THE COVER
World Asthma Day
(Photo: Asthma New Zealand)

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Upcoming events and courses

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ASTHMA NEAT COURSE
17 June 2015
8 July 2015 (School Nurses)
16 September 2015

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15 April 2015
15 July 2015
21 October 2015

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WORLD ASTHMA DAY

5th May 2015

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

MESSAGE TO READERS

Welcome to the first issue of O_2 for 2015. This year, we're hoping for some major changes in the health sector, and we've had some big developments already.

This year, I'm aiming to meet with the Ministry of Health, with a goal of making asthma a government health priority. Ideally, we'd like to meet with Minister of Health, Jonathan Coleman, although previous Ministers and Ministry staff have proven somewhat elusive. We tried in vain to organise meetings in 2014, and their unresponsiveness indicates that the government doesn't take asthma and similar respiratory conditions for the serious issues that they are. We think this needs to change.

New Zealand's asthma rates are alarming, due to our damp climate, high pollens and poor housing stock. With one in six adults and one in four children affected, it's our families who are shouldering the costs of the government's inaction. We support thousands of people nationwide, yet our current allocation of funding is nowhere near proportional to the number of people affected by asthma.

This year we'll be pushing to get in front of government decision-makers and lobby for the prevention and management of asthma to be dedicated the resources and funding it needs. This only makes sense. Being the fourth leading cause of death in New Zealand, COPD has already been recognised by the government as a health priority, and it's widely known that unmanaged asthma is a contributing factor to the development of COPD. Currently there is no sustainable long-term solution in place, which drastically needs to be addressed. The management and prevention of asthma needs substantial and sustainable government funding. This will allow us to extend our services, visit more families affected by asthma, provide more information to educational facilities and healthcare practices, and continue to lobby for better living conditions for New Zealanders.

We've got exciting plans ahead for World Asthma Day also. Established by GINA (Global Initiative for Asthma), World Asthma Day is celebrated on the first Tuesday of May, with the aim of raising awareness worldwide around the precautions and prevention of asthma. This year, we'll be focusing on primary school children, a group of New Zealanders who are particularly vulnerable to developing asthma. On May 5th, we've organised a fun activation in primary schools to empower and encourage children to take care of their respiratory health.

The 'Best Bubble Competition' will get kids active, raise funds for Asthma New Zealand and make schools and families more aware of our services and how we can help, while giving schools the chance to win great prizes. There's more information available online, so mark it in your calendar and head to our website to find out more!

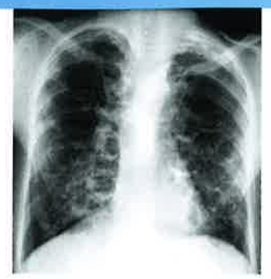
Another of our focuses for the year is making products to help with the management of asthma more readily available for people throughout New Zealand, so we've established an online store, providing allergy-friendly cleaning supplies, Miteguard bedding, anti static spacers and a variety of other products including the AsthmaMinder™, a useful tool that houses an MDI preventer and toothbrush together. It was invented to make people more compliant with medication and to remind people to take their preventer twice a day before brushing their teeth. With reasonable prices and nationwide shipping, this is your go-to shop for making life with asthma easier, and it's perfect for people living rurally. You can see what's available by clicking on 'Shop' on our website.

2015 will be an important year for the asthma community. We're continuing to support and educate New Zealanders with asthma and other respiratory conditions, and we're appreciative of your support. Together, we can make a difference to the lives of thousands of New Zealanders, and build a healthier future.

Linda Thompson
Executive Director
Asthma New Zealand

BRONCHIECTASIS...

By Cathy Gasparini, RN, BHSc, PG Cert.
Asthma Nurse Educator



As an asthma nurse educator, I was working with a 2 year old child who did not respond to his asthma medications as was expected, and required frequent hospitalisations for his asthma, despite taking his preventer inhaler every morning and every night. It was then discovered that he also had bronchiectasis. This article will attempt to explain what this condition is, how to recognise it, and how it is treated.

Bronchiectasis was first described in 1819 by the inventor of the stethoscope, Rene Laennec, while he was observing patients with tuberculosis, post pneumonia.[1] Bronchiectasis was a common condition with a poor prognosis, until the advent of immunization campaigns and the introduction of effective antibiotics, which occurred in the mid-1950s. From then, there was a sharp reduction in the incidence of childhood infections – the prevalence of bronchiectasis declined and the prognosis improved.[2]

However, bronchiectasis has now re-emerged as a serious health risk, and currently, there is a worldwide increase in the incidence of bronchiectasis. Statistics show that between 2001-2003, 3.7 out of 100,000 children aged under 14 years old were diagnosed with bronchiectasis in New Zealand. Of this group, 80% were Maori or Pacific peoples.[3]

The word bronchiectasis originates from Greek which literally means "stretching of the windpipe". It is defined by permanent and abnormal widening of the bronchi, which usually occurs as a result of obstruction and/or inflammation of the airway.[4] This obstruction or inflammation can be due to underlying disorders, such as cystic fibrosis, or primary ciliary dyskinesia, or may be due to childhood pneumonia and infections. Infection is the most common cause of bronchiectasis, and some of the infections that cause bronchiectasis are TB, whooping cough, pneumonia, or measles. Other risk factors for bronchiectasis include primary immune deficiency (such as AIDS), recurrent aspiration (gastroesophageal reflux and foreign body inhalation), and other diseases that cause inflammation in other parts of the body, for example, rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease.[3]

The respiratory tract is lined with cells that contain cilia, which are hair-like structures that are in the mucous layer which lines the airways. These cilia continually waft to propel the mucus out of the lungs. Mucus traps bacteria and particles, and mucociliary clearance is an important defence mechanism for the bronchial tubes.[8]

The pathogenic sequence that is seen in bronchiectasis is bronchial dilation, inflammation, and weakening, which causes airway distortion and scarring, thereby altering both the structure and function of the mucociliary apparatus, impairing the ability of the patient to clear secretions.[2] Cole (1997) describes a vicious cycle theory for the pathogenesis of bronchiectasis, where an initial triggering insult compromises the first line of defence in the bronchial system, being mucociliary clearance. Without being able to clear this mucus adequately, bacteria remains longer than usual in the bronchial tree, allowing those microorganisms to colonize the mucus, then stimulate an inflammatory response by the host. When the inflammatory response fails to eliminate the colonised microorganisims, the inflammation becomes chronic. Inflammatory cells continue to be recruited to the site of infection, which include neutrophils and macrophages, which release increasing amounts of cytotoxic agents. In an effective inflammatory response, these cytotoxic agents, called proteolytic enzymes, would be neutralized by corresponding antiproteolytic agents, which would then prevent damage to adjacent tissues. However, when inflammation persists, an ongoing chemical reaction occurs which results in progressive, irreversible damage to both the bronchial wall, and airway cilia. The inflammation and damage widens the airways, causing extra mucus to form, which is then less easily cleared. The mucus tends to pool, which makes those airways with pooled mucus prone to infection.[2,4,5]

Coughing up a lot of sputum is the main symptom of bronchiectasis. According to the Thoracic Society of Australia and New Zealand, recurrent episodes of wet or productive cough (three or more episodes) lasting more than four weeks, is the most obvious sign of bronchiectasis. There may be an alteration in sputum colour – from clear to yellow, to green, and in rare cases haemoptysis (blood in sputum) is seen in adults. Sometimes, but not always, a fever is present. People with bronchiectasis will also have shortness of breath, and in some people there is also a wheeze. Sometimes there is a coexistent history of asthma and according to Starship, approximately one third of children with bronchiectasis also have asthma.[2,6]

Clinical signs include low or borderline oxygen saturations during the day when having an acute exacerbation, increased respiratory rate, increased work of breathing, crackles, wheeze, chest wall abnormalities, and digital clubbing.[2]

The gold standard for diagnosing bronchiectasis is through high resolution CT chest scanning. Other investigations undertaken to diagnose bronchiectasis are sputum tests, blood tests to check inflammatory indicators, such as a Full Blood Count (FBC), C-Reactive Protein (CRP), and Erythrocyte Sedimentation Rate (ESR), which are likely to be elevated. A chest x-ray is also taken.[2]

Antibiotic therapy and chest physiotherapy are central treatments for children with bronchiectasis. When the child is having an acute exacerbation, chest physiotherapy is given twice a day while they are on antibiotics, which is usually over two weeks. Otherwise chest physiotherapy is given once daily. This is to help expectorate the sputum. Many children also use active cycle of breathing and postural drainage techniques, and òthers use positive expiratory pressure devices or sometimes acapella devices to help expectorate the sputum.[2,6]

Bronchodilator inhalers can be used in bronchiectasis for wheezing and breathlessness, if it helps, but is not prescribed routinely, but used only on an individual basis. Steroid inhalers are only used in bronchiectasis if there is also asthma present.[6]

Lifestyle factors that are recommended for children and adults with bronchiectasis are regular physical activity; good nutrition; no smoking, and avoidance of second hand smoke exposure. Keeping immunizations up to date is important in children as well as having annual flu vaccinations.[6]

In a study by Monash University, Melbourne, it was found that bronchiectasis is most commonly found in children in their first five years of life, or in people aged 50 years and over. In two studies, it has been shown that people with child-onset bronchiectasis may show an improvement with adulthood and then show clinical deterioration again beyond the age of 50 years old.[1] This could be due to the growth of new pulmonary tissue in children which occurs rapidly until about the age of 6 years old, and then tapers off. Therefore injury to the lungs at an early age may be compensated for by growth of normal healthy lung tissue.[4]

Studies have also shown that patients with asthma and bronchiectasis have more severe asthma than patients who have asthma alone, but more studies are needed in this area.[7]

In conclusion, as effective management influences prognosis and quality of life, early diagnosis of bronchiectasis is important, and therefore a heightened awareness of this condition is needed by health professionals.

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Yes it is essential to put ordinary sheets, pillow covers and duvet covers on top as this will protect the dust mite cover. If you do not use these, then the cover will wear out much quicker than it should and would need to be replaced at your own cost. Your sheets need to be washed weekly and the dust mite covers then will only need to be washed every 2 – 3 months minimum.

Dear Nurse, my daughter has put on weight and she does not want to take her Flixotide for her asthma as someone told her it would make her gain even more weight, is that correct?

No Flixotide will not make your daughter put on weight. Flixotide is a very weak corticosteroid. Oral steroids such as prednisone and Redipred are 1000 times stronger than Flixotide. Oral steroids taken frequently or in high doses may cause fluid retention and increase appetite. Your daughter may have put on weight because she finds it difficult to exercise if her asthma is not controlled. If she wheezes or coughs when exercising she will need to take a preventer morning and night even when she is well.

Dear Nurse, I went to stay with my cousins and we were playing cricket. I started to cough and wheeze and had to stop playing because I could not breathe properly.
My auntie gave me an inhaler to use. It was different from my inhaler. My cousin told me that I shouldn't use other people's inhalers, is that correct?

Your cousin was right.

Anyone who has asthma must take their blue reliever inhaler with them at all times as you never know when you might need it. The inhaler that your auntie gave you to use was prescribed for her to use for her medical condition, which may not have been asthma.

Remember, inhalers are prescribed by doctors for each individual person depending on their diagnosis. Inhalers work in different ways. It may be dangerous to use an inhaler that has not been prescribed for you.

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IF YOU HAVE A QUESTION PLEASE EMAIL OR POST TO:
editor@asthma.org.nz or Dear Nurse, Asthma New Zealand, PO Box 67066, Mt Eden, Auckland 1349.

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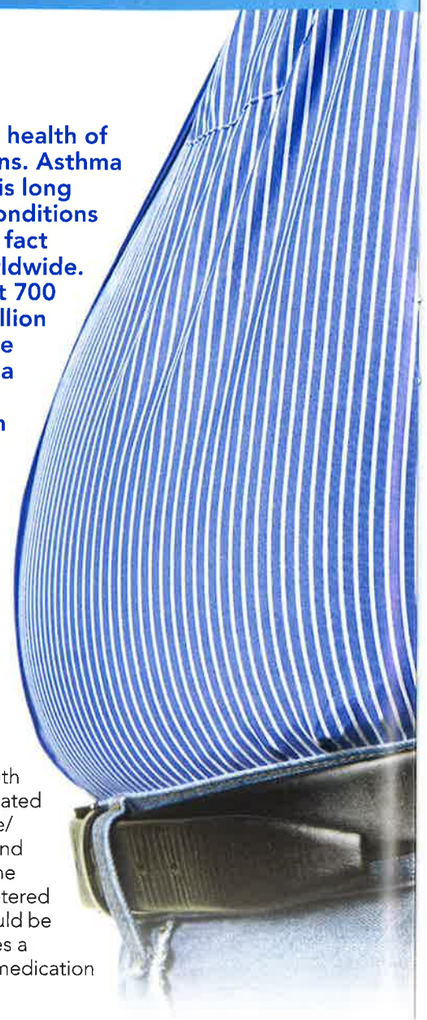
IS OBESITY A PROBLEM IN ASTHMA?

By Ann Wheat RN BN
Asthma Nurse Educator



In themselves both obesity and asthma are important problems for the health of individuals who have them and can lead to other more serious conditions. Asthma can lead on to Chronic Obstructive Pulmonary Disease if the condition is long term and not well managed. Obesity is associated with other chronic conditions such as diabetes, hypertension and obstructive sleep apnoea (OSA).[1] In fact both asthma and obesity are considered major public health issues worldwide. In fact by this year (2015), the World Health Organisation considers that 700 million people over the age of 14 years of age will be obese[1] and 2.3 billion will be overweight.[3] It is now apparent that people with asthma who are obese or overweight have poorer asthma control including more asthma exacerbations and have less response to corticosteroid therapy.[2,4] In fact they are beginning to think that asthma and obesity share common genetic determinants and that obesity may result in a distinct asthma phenotype.[4] This may be particularly true in adults. It has been demonstrated that in adults, being overweight increases the chance of asthma by 38%, and if a person is obese the risk is 92% compared to someone of normal weight.[3] In children and adolescents it is becoming clearer that body mass index is strongly related to wheeze and asthma, especially in affluent countries.[4]

Over the last 20 years, asthma has increased dramatically worldwide with rates over 2.5 times higher in that time.[3] The latest figures we have indicate that there are approximately 235 million people with asthma in the world and that there are an estimated 250,000 deaths in the world annually.[5] It is also believed that there is a strong relationship with very low birth weight children and the development of overweight children who also have asthma by the age of 12 years.[3]

So why is obesity so important for people with asthma?

Asthma is a chronic inflammatory condition of the airways. It is often associated with allergy and the main cells occurring in allergy are eosinophils. The airways become red and swollen and over produce mucous. In obesity there is a marked increase in inflammation throughout the body including the lungs.[3] According to one research study the cells involved in the inflammation in obesity are neutrophils which are found both in sputum and blood.[6] So it is not surprising that if a person has asthma, and are female and obese, their asthma will be less well controlled and they will not respond to the normal amounts of corticosteroid medication which is the mainstay of asthma control, especially in allergic asthma.[6] It is also thought that there is a smaller improvement in lung function such as FEV1 with preventer medication.[6] Accordingly people who are obese complain of more daytime and night-time symptoms, less social activity and poorer exercise tolerance.[1] There also appears to be an increased risk of hospital admissions in obese asthmatic people.[1]

So what can be done for people who are obese and have asthma?

The most important factor that must be taken into consideration is using the prescribed preventer medication twice daily even when well. Preventer medications work on a 12 hour cycle so must be taken twice daily to be effective. They take up to three weeks to come up to full efficiency so it is important to continue using them even when well. The use of the medication device must also be correct and a person with asthma should be educated in the correct technique/ use of both preventer and reliever medications. The use of a spacer with Metered Dose Inhaler (MDI) should be encouraged as this gives a greater percentage of medication to the lungs.

The second most important factor is to lose weight. Even a small weight loss will improve asthma control and even reduce the need for asthma medication such as rescue medication.[1] Losing weight by 10% also improves lung function both FEV1 and FVC by a 92ml and 73 ml respectively.[1] It is important that this weight loss is achieved by both exercise and eating a healthy diet.

Exercise is really essential not only for a person who is obese but also for non-obese people with asthma. It is very important to discuss any exercise plan with a doctor before commencing to exercise. Exercise should be undertaken slowly at the beginning. This could just be a short walk down the street and then gradually increasing this as a person feels able. Another good exercise for people with asthma is swimming. This will not only help with lung function but is a good aerobic workout.[7] Exercise can also be a trigger for exercise-induced asthma so a person with asthma must be educated in the correct techniques to help control this.

Diet is also very important when trying to control weight. Eating a healthy diet with lots of fruit and vegetables provides the body with plenty of antioxidants. These foods have Vitamin A, C and E which can help reduce inflammation in the lungs, reduce exacerbations and improve lung function.[7,8] It is important to eat a diet that is low in dietary fats. Dietary fats are known to worsen asthma symptoms and can lead to an increased concentration of inflammatory markers.[8] Dietary fats can also reduce the efficacy of bronchodilator medications such as salbutamol.[8] So if you have asthma and are obese then it is important to avoid diets that are high in fat, processed foods and take-aways especially in children.[8] High soft drink consumption can be associated with asthma so it is vital to not drink large quantities.[8]

Conclusion

In answer to my opening question, then yes obesity is very important when trying to maintain asthma control. If someone is obese then it is crucial that they lose weight to not only be physically healthy but also to have better asthma control. Keep Active and Eat Healthy.

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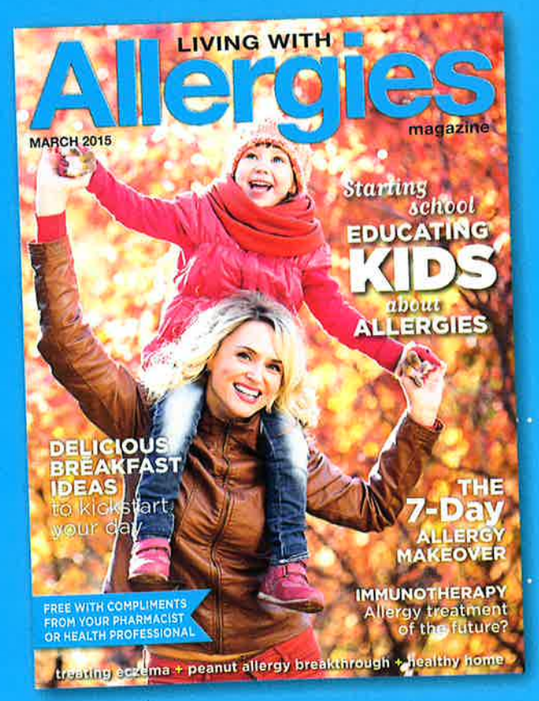

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ASTHMA TREATMENT:
DO COMPLEMENTARY AND ALTERNATIVE APPROACHES WORK?

By Adie Riddell RN
Asthma Nurse Educator

It is not uncommon that many people with chronic diseases often look to 'alternative' therapies to either replace their current medication regime or to complement it. It can be said that the goal of alternative treatments for asthma is to bolster the body's ability to protect itself from asthma triggers and to strengthen the lungs and immune and hormonal system. However, as with many alternative treatments for any condition, opinions are often mixed as to the efficacy of some of these treatment methods, and there are few or no research studies on most of them. Therefore the effectiveness and safety of most of them are unknown. In this article we are talking primarily about asthma.

A recent information paper from the Australian National Asthma Council[1] provides a review of complementary therapies based on their efficacy on clinically relevant outcomes of asthma, looking specifically at symptoms and quality of life, lung function and need for medication. They have based their evaluations on literature searches from a variety of databases and reviewed the effectiveness of each therapy. While this review did not include all alternative therapies used in asthma, it provided information on those whose treatments were evaluated in controlled published clinical studies.

These treatments include herbs, dietary supplements, yoga, acupuncture, chiropractic and massage therapy, and biofeedback. While some of these treatments are often used alone, others are used to compliment the traditional medications prescribed by your doctor (complimentary therapy).

Of the nutritional and dietary supplements, there was evidence that magnesium, vitamin D, and fish oil supplements provided possible benefits. There was strong evidence of the effectiveness of caffeine on resting lung capacity and exercise induced asthma.

There was only one controlled study found on diet restriction – dairy elimination which may have the potential to impact on growth and bone density.

Herbal medicines are vast and complex ranging from Japanese herbs, Indian, Chinese herbal medicines, and western herbal medicines. It would appear that there are limited conclusive reviews and conflicting results. Many of the clinical trials have a very small number of participants. According to a review of herbal remedies in data produced by the Mayo Clinic[2]; treatment often involves a blend of herbs, and that by taking certain herbs in combination, may be more effective than using only one herb. Clients should be warned that some herbs are known to have the potential to cause severe allergic reactions in predisposed individuals with asthma such as echinacea, royal jelly, willow tree bark extracts, and·camomile.

There was some evidence that acupuncture which involves the insertion of very thin needles into your skin at specific points on your body, may improve symptoms.[3] But more definitive studies are needed to fully assess the usefulness of acupuncture for treatment of asthma.

The results of six randomised trials failed to show any benefit of homeopathy with regard to lung function, with only one study showing benefit. Hence there was little evidence of effectiveness for routine asthma control.

In studies relating to mind-body medicine such as relaxation therapy, meditation and hypnosis the results were inconclusive. However, there may be some positive impact on anxiety and attitude.

Breathing techniques used for controlling asthma symptoms including the Buteyko breathing technique, Tai Chi, Qigong, and yoga breathing (pranayama), are aimed at reducing hyperventilation and regulating breathing. Review of studies indicates that they don't seem to improve the underlying allergic reaction that causes asthma symptoms. But these exercise regimes may be useful in relieving stress and anxiety – a common asthma trigger, and in reducing the need for reliever medication. Aerobic exercise training showed benefits in three studies for improving lung function and symptoms.

Other therapies reviewed were osteopathy yoga, swimming, reflexology, Chiropractic and breathing exercises of which there was either limited data or variable results which made it difficult to rate in efficacy. Current evidence does not support the use of manual therapies (chiropractic, osteopathy and related modalities or acupuncture) for treating patients with asthma.

Based on the available evidence, reviews of the literature and expert medical guidelines appear to have come to conflicting conclusions about the efficacy of complimentary therapies for asthma treatment, although some of these alternative therapies may contribute to easing symptoms such as anxiety, stress or over-breathing. Before trying any alternative therapies you should check with your doctor to ensure that the methods are safe and right for you and that they do not interfere with other medication you may be taking.

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SPACERS

By Vicki Lyford, RN
Asthma Nurse Educator

As an Asthma Nurse Educator I advocate the use of a spacer when using an asthma Metered Dose Inhaler (MDI) AT ALL TIMES, no matter what your age.

Asthma and COPD Medications come in three different styles, MDI's, turbuhalers and accuhalers. Spacers are used with **MDI's**, both relievers and preventers.

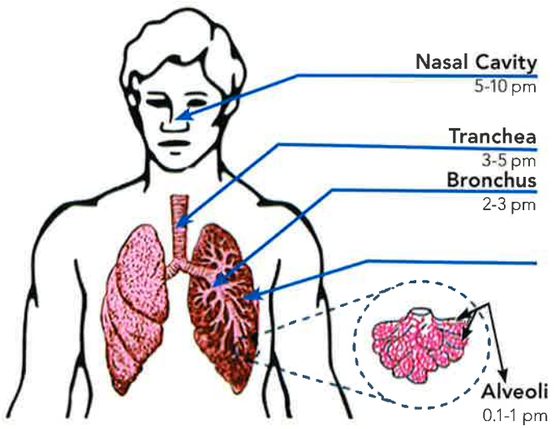
So what exactly is a spacer?

It is a valved cylindrical device that is used solely with a MDI. The aim of this device is to make it easier to inhale the aerolized medication prescribed for asthma or Chronic Obstructive Pulmonary Disease (COPD). It does this by enabling the medicine to break into smaller particles by mixing with the air in the cylinder. The particle size goes from >5µm to particles of 0.5 – 4.7µm, which are more easily inhaled into the smaller bronchioles of the lungs.[1] It reduces the amount of medication deposited in the upper respiratory tract thus ensuring more medication makes it to the lungs where it is most effective.[2]

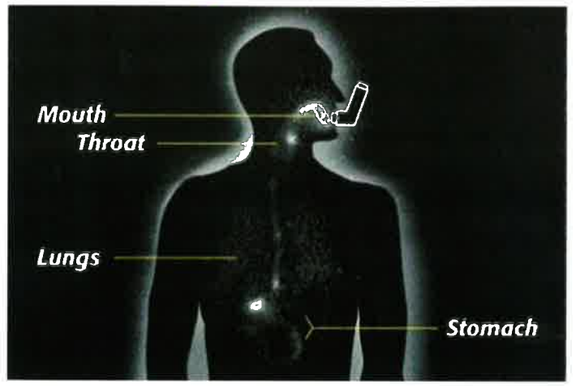
In an MDI the medication is mixed with propellant in a pressurized canister. When activated, it is expelled from the casing at 100 km/hr.[1] If used straight into the mouth, because of the force, the medicine sticks to all aspects of the oral cavity: the tongue, upper hard palate, uvula, tonsils (if you have them), cheeks and the back of your throat. Only a small proportion of the medication actually makes it into the lungs.

So how do I use a spacer?

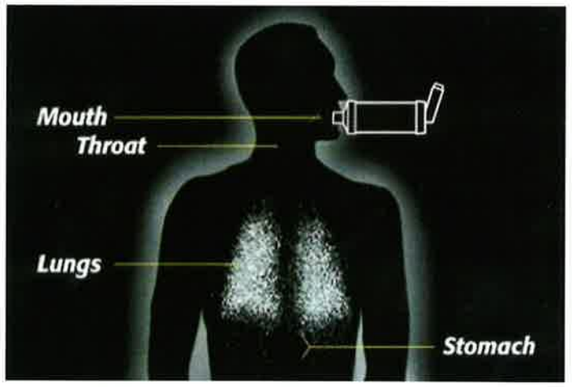
- Sit upright in a comfortable chair with your spacer and inhaler.
- Once the inhaler has been shaken it is placed into the fitted end of the spacer.
- The mouthpiece is then inserted into the mouth; you form a tight seal with your teeth and lips.
- Hold it at a right angle to the face; push the canister, expelling aerolized medication into the chamber where it mixes with the air.
- This is then inhaled into the lungs by breathing normally for 6 breaths.
- This is repeated again if required.[4]



Why use a spacer with an inhaler?



Inhaler alone



Inhaler used with spacer drive

- You can use a face mask for smaller children until they are old enough to use the mouthpiece.
- When you breathe in, the valve near the mouthpiece opens to allow only the mixture of air and medication in the space chamber to be inhaled into the lungs. On exhalation, the valve in the spacer closes to the chamber and the air and carbon dioxide are expelled via the openings near the mouthpiece into the open air.
- Once you have finished taking your prescribed amount of medication it is advisable to rinse your mouth out with water, or gargle with water to remove any medicine residue in the oral cavity that may lead to side effects like oral thrush, voice hoarseness, or sore throat.
- Wash the face of a child who has used a face mask.
- A Haleraid can be used to assist a person who has difficulty depressing the canister to expel the medication from the inhaler casing.[2]

The advantages of using a spacer are:

1 You get more of the medicine into the lungs where it is needed (at lease 3 times more medication). Even when you are short of breath you can still get your reliever medication by breathing in through the spacer.
2 You do not have to hold your breath for a count of 10, which, in an asthma attack, is extremely difficult!
3 It does not require good coordination of medicine actuation and inhalation to obtain maximum efficiency[1]
4 It is easier for parents to get children to take their medication. A face mask may need to be used if the child is very young.
5 It reduces the side effect of thrush in the mouth and throat by ensuring most of the medication goes to the lungs and is not deposited in the mouth or throat.[3]

So which spacer should I use?

There are several different types of spacers available to you.
1 **Volumetric Spacer**. This spacer has a volume capacity of 800 mls and is used by older children and adults alike.
2 **Space Chamber Plus** has a volume capacity of 230 ml and is used by children. A face mask can be attached for very young children.

Because these two spacers are made of plastic they are non-conducting and they can therefore build up an electrostatic charge on their surface. This results in the medication particles being attracted to the plastic instead of being inhaled into the lungs. They have a cross valve technology giving low resistance on inhalation.[4] These spacers are free from your GP practice rooms and Asthma Nurse Educator.

3 **E-Chamber Spacer** – Turbo and La-Petite 3 – volume capacity of 220 mls and La Grande – volume capacity of 510 mls.
These spacers are made from a durable and soft thermo-grade silicone, are anti-static, and no priming is needed. They are latex and BPA free. They have a patented butterfly one way valve which means that minimal force is required for opening and closing the valve during inhalation and exhalation. You can also hear when you give adequate breaths. They are small and easy to fit in a purse, pocket, and glove box. The La Petite spacer comes apart and the MDI can be placed inside so you can carry both together, very convenient! However these spacers will cost you a minimal fee.[3] These spacers are available from your Asthma Nurse Educator or offices of Asthma New Zealand – The Lung Association.

Care of your spacer

Because they are plastic you do need to wash the spacer weekly in warm soapy water then leave to air dry. The detergent coats the inside of the spacer helping to negate the static charge. Washing also helps to dispel the residue from the medication. The spacer should be housed either in the box it came in or a cloth bag, NEVER in a plastic bag as this just reinforces the static charge.

Therefore it pays to have two spacers so you have one available when the other is being cleaned.[2]

The spacer should be washed before first use, however if it has not been used in a while and you need to use it, you can squeeze 10 puffs of your reliever inhaler into the spacer to prime it (holding your hand over the mouthpiece), then use as normal.

They should be replaced every 6-12 months as small scratches and abrasions decrease its effectiveness.[5]

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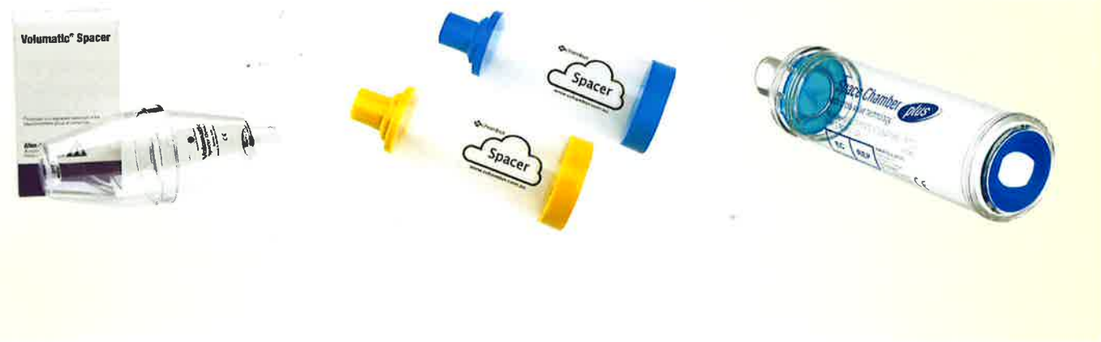
3 3. http://www.asthma-nz-shop.org.nz/index.php?route=product/product&product_id=175
4 4. http://www.medicaldev.com/shop/space-chamber-plus-with-cross-valve-technology/
5 5. http://www.medicaldev.co.uk/use-asthma-spacer/ *



WORLD ASTHMA DAY

World Asthma Day is approaching! A project developed by the Global Initiative for Asthma (GINA), World Asthma Day is celebrated on the first Tuesday of May, encouraging people around the world to take control of their asthma and set goals for better health.

Alarmingly, New Zealand has the second-highest rate of asthma in the world. One in four Kiwi kids struggle with asthma, and it is the leading cause for children having time off school. This leads to a huge loss of education, with young children in low social-economic areas being particularly affected. The foundation of children's education is set during primary school, where kids develop learning styles and attitudes towards development. When primary school education is disrupted, ongoing consequences that affect further learning are common.

This year, Asthma New Zealand's focus for World Asthma Day is on primary school-aged children. World Asthma Day is May 5th, and we're empowering kids to take care of their respiratory health and have fun through the Best Bubble Competition fundraiser!

We're demonstrating to kids around New Zealand that having a healthy respiratory system is important and allows them to do fun activities, while showing that there are services available to help keep them healthy.

We're asking primary schools all around the country to have their students donate a gold coin to enter the Best Bubble Competition, and submit videos of their student's best bubbles to Asthma New Zealand. They'll then go into the draw to win awesome prizes for the whole school!

There are five categories for schools to enter:

- **Biggest bubble**
- **Most creative bubble blowing instrument (ie: wand)**
- **Biggest amount of people blowing bubbles at once!**
- **Best bubble photo**
- **Anything goes!**

We'll select a winner from each category, who will win great prizes for their school, like books, sports equipment and other school essentials. We'll also share the winning videos on our website and YouTube channel, so make sure you watch!

The Best Bubble Competition is a great opportunity for kids to take control of their health in a way that is fun and creative, while learning more about our services and the challenges that their friends with asthma face. It'll raise funds to help develop our services, and will make children and schools aware that we are available to help, support and educate them about asthma and other respiratory conditions.

All funds raised through the Best Bubble Competition will be used to continue and improve our services, to allow us to better support children and families with asthma. It's about working together to keep Kiwi kids happy and healthy. Plus, all donations over $5.00 are tax-deductible!

Now's the time to help support a great cause in Term Two! If your school would like to join the Best Bubble Competition, please contact Judith at anz@asthma.org.nz We'll send you an info pack with everything you need to get involved… Easy!

Olivia Premium Food's "Bear Smile" campaign supports Asthma New Zealand

Recognising that healthy breakfast cereals often don't appeal to children, Olivia Premium Foods have created the delicious Lil' Bites cereal, especially for kids.

Made with 100% New Zealand ingredients, Lil' Bites is packed full of dried fruit, seeds and nuts, and comes in cute, child-friendly packaging, to give kids a delicious, healthy start to the day.

Olivia Premium Foods have been donating funds to Asthma New Zealand, through their "Bear Smile" campaign. Lil' Bites cereal comes packaged in an adorable box featuring a bear, and buyers are encouraged to upload photos of themselves doing a "bear smile", with the hashtag… For each post to social media, Olivia Premium Foods will donate 10 cents to Asthma New Zealand!

We're extremely grateful for their support, and we love the cereal too! It's a great way to ensure kids get a good breakfast, so head to your local supermarket to get a box! Don't forget to upload your bear smile!

NORTH & SOUTH

Asthma Wellington will now be much more visible out in the community thanks to Johnston Ebbett in Wellington. Managing Director, David Johnston is supporting the Society by very generously sponsoring us with a brand new Holden Barina Spark. We even got to choose the colour – blue of course! – in keeping with our branding! They had it sign written for us so you will see us coming – it is bold and bright! It will help us raise awareness of the service, putting us in touch with more people in need of our support, and will make it much easier for staff to get out and about. Look out in the next O_2 to see our new edition in action!
Kim, Adie and Alice

JOHNSTON EBBETT

EBBETT
MARQUE OF EXCELLENCE

HOLDEN

Managing Director, David Johnston with Alice Paul, asthma nurse educator.

Adie and Alice our two asthma nurse educators.

Lost and Found

Shameer Mohamed's a keen cricket player. He goes to practice after a day at Dominion Road School.

Shameer has asthma, so he carries his medication and equipment to school and cricket practice, in a cool bag that Asthma New Zealand gave him.

It's really handy for Shameer - he has all he needs to manage his asthma in one place, and he never forgets anything.

But on the way to cricket practice one day, he dropped the bag... How annoying!

For a whole month Shameer couldn't keep his asthma gear all together. Sometimes he forgot things - what a pain!

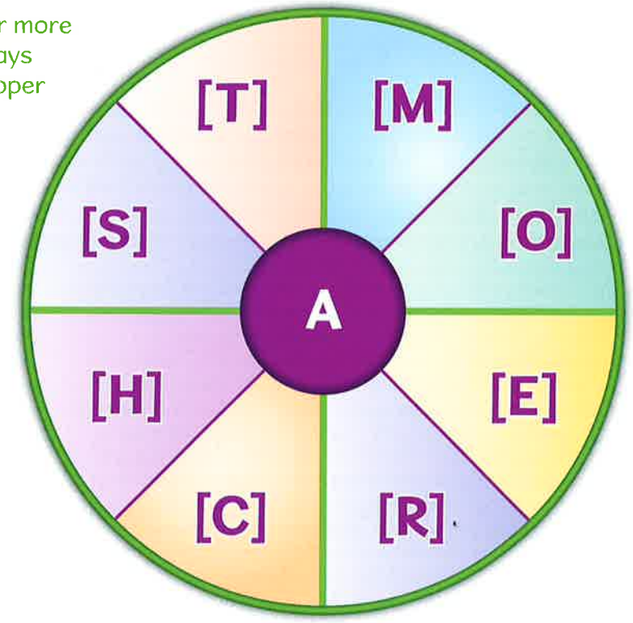
Then one day, his mother Sithy had a knock at the door.... It was a policeman!

He had Shameer's asthma bag. Someone had found the bag and kindly handed it in. Shameer had written his address on the bag, so the police could return it to him easily.... What a relief!

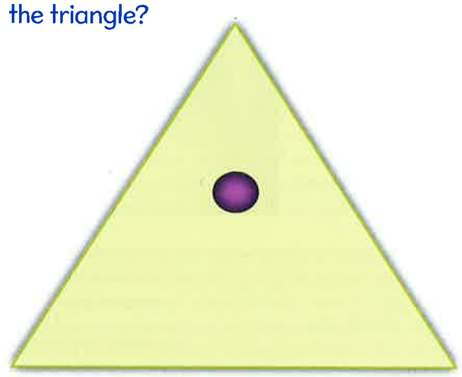
He had Shameer's asthma bag. Someone had found the bag and kindly handed it in. Shameer had written his address on the bag, so the police could return it to him easily.... What a relief!

Kid's Page

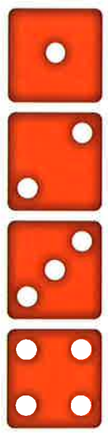
1 Create as many words of three letters or more using the given letters once only but always including the middle letter. Do not use proper names or plurals.



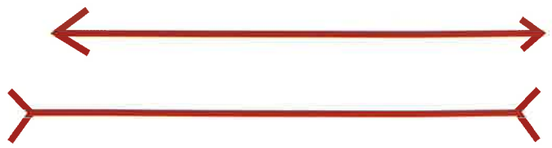
2 **Impossible triangle** – Look at the triangle below. How would you describe the position of the ball? Is it nearer the top or the bottom of the triangle?



3 **Dice Tower** - Four dice are stacked one on top of each other. What is the total value of the three pairs of hidden faces in the stack, given that the top face is 6 and the bottom face is a 3?



4 Which of the lines between the arrow heads is the longest?



5 Try the **Text Language**

10Q
1DAFUL
2QT
121
2B
XOXO
ZZZZ

Answers

1. Puzzle
Cat
Mat
Hat
Rat
Coat
Man
Mane
Tame
Came
Heat
Same
Ham
Tar
Ram

2. Puzzle
The Ball is in the middle

3. Puzzle
Total must be 19, since the opposite faces of a dice always add up to 7, the total must be (4x7) – 6-3+ 19

4. Puzzle
Both lines are the same length

5. Puzzle
Thank You
Wonderful
Too cute
One to one
To be
Hugs and Kisses
sleeping

THE CONNECTION BETWEEN PNEUMONIA, COPD AND ASTHMA

By Karen Little RN
Asthma Nurse Educator

Pneumonia is an inflammatory condition of the lungs primarily affecting the alveoli. It is usually caused by infection from a virus or bacteria and both may present with similar symptoms although viral pneumonia presents more commonly with wheezing than does bacterial pneumonia. A causative agent may not be isolated in approximately half of cases despite careful testing. Children with respiratory distress or oxygen saturations of less than 90% should be admitted to hospital.[1] Rates of pneumonia are greatest in children less than five-years-old and adults older than 75 years.[2]

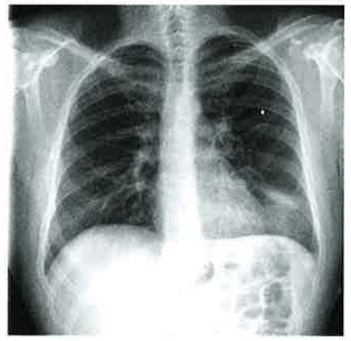
The Towards a Revolution in Chronic Obstructive Respiratory Disease (COPD) TORCH trial was designed to evaluate a possible survival benefit associated with the use of a combination of the long acting beta agonist (LABA) salmeterol and fluticasone propioate in comparison with placebo. This was a large randomised, double-blind trial over three years. The reduction in death from all causes among patients with COPD in the combination-therapy group was not statistically significant. However there were significant benefits in all other outcomes among these patients including reduced exacerbations and spirometric values. However, this trial reported for the first time an increased risk of pneumonia associated with the use of the inhaled corticosteroid (ICS) compared with the placebo group.[3] Since this trial, other studies and subsequent meta-analyses have reported a 50% to 70% increase in the risk of pneumonia associated with the use of ICS in COPD.

The Cochrane Collaboration (2014)[4] reviewed a total of 43 studies that met the inclusion criteria with more evidence for flucticasone (26 studies) than budesonide (17 studies). Evidence from the budesonide studies was more inconsistent and less precise, and the mean duration was shorter. No effect was observed for mortality, and increased rates of pneumonia seen with inhaled steroids were not significantly different between types of inhaled steroids. All reviews agree that evidence for harms associated with these medications needs to be assessed in conjunction with good evidence of the clinical benefit of inhaled steroids; notably, fewer exacerbations and improved quality of life.

One of the studies reviewed reported that current use of ICS is associated with a 69% increase in the rate of serious pneumonia. The increase in the rate of pneumonia rises with the dose, ranging from 24% for the lower doses to 86% with the highest doses. This study used a large population-based cohort of over 160 000 patients with COPD and followed up for 18 years. Fluticasone with a dose of 1000mg per day was associated with a 122% increase. The dose with budesonide was comparatively much lower with an increase of 17% and beclomethasone a 41% increase. These elevated risks disappeared within a few months of stopping the use of ICS. Cases of serious pneumonia were defined as a hospitalisation for, or death from, pneumonia.[5]

ICS do not appear to increase the risk of pneumonia in patients with asthma in clinical trials using budesonide.[6] Nor was an association seen in the smaller number of studies that used fluticasone as a comparator in asthma.

Systemic corticosteroids have been associated with increased risks of pneumonia in patients with rheumatoid arthritis. It is therefore not unexpected that high doses of ICS have similar effects on the incidence of pneumonia, as 1000mcg of inhaled fluticasone is estimated to be equivalent to 10mg per day of prednisone.[7]



While ICS are clearly effective for the treatment of asthma, their effectiveness in treating mild or moderate COPD is still controversial. The fact that ICS are now commonly combined in a single device with a long acting-acting bronchodilator has resulted in ICS now being used by over 70% of patients with COPD.[8] However, GOLD 2014[9] recommends that long-term treatment with inhaled corticosteroids are only recommended for patients with severe and very severe airflow limitation, and for patients with frequent exacerbations that are not adequately controlled by long-acting bronchodilators.

The role of respiratory tract infections in the pathogenesis of asthma is well established. Mycoplasma pneumonia (M. pneumoniae), primarily recognised as a causative agent of community acquired-pneumonia, has recently been linked to asthma in various ways: an infection with the organism may precede the onset of asthma, exacerbate asthma or make control difficult.[10] A large number of patients with asthma continue to harbour M. pneumonia in their airways. This leads to increased cytokine production, which may cause a continuing inflammatory response and affect asthma control. Laitinen *et al*, suggested that such mycoplasmal infection denuded the epithelial surface of the lung and exposed irritant receptors.[11] Macrolides such as erythromycin, are well known for their antibacterial properties, but they also possess anti-inflammatory properties that may contribute to clinical benefits observed in patients with airways inflammation, as in asthma. Kraft *et al* found that treatment with clarithromycin resulted in a marked improvement in forced expiratory volume only in M. pneumonia-positive patients.[12]

M. pneumoniae is commonly detectable by culture of the respiratory tract up to several months after recovery from acute pneumonia. Even after treatment with effective antibiotics, persistence of infection with this organism results in decreased expiratory flow rates and increased airways hyper-responsiveness in individuals without asthma.[12]

If pneumonia is suspected, it is important to seek medical attention promptly so that an accurate diagnosis can be made and appropriate treatment given.

The doctor will take a medical history and will conduct a physical examination. During the examination the doctor will listen to the chest with a stethoscope. Coarse breathing, crackling sounds, wheezing and reduced breath sounds in a particular part of the lungs can indicate pneumonia.

In order to confirm the diagnosis a chest x-ray is usually taken. The x-ray will show the area of the lung affected by the pneumonia. Blood tests may also be taken, and a sample of the sputum may be sent to the laboratory for testing. Typical symptoms include a cough, chest pain, fever, and difficulty breathing.

ICS if prescribed for COPD should follow established guidelines so they are not prescribed when they are not required or indicated. Your doctor will assess these criteria as well as taking into consideration quality of life, recent exacerbations, and individual circumstances.

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FUJITSU HEAT PUMPS
NEW ZEALAND'S FAVOURITE AIR™
AIRCON SERVICES LTD

A big thank you to Aircon Services Ltd who entered The Great Auckland Bed Race and won $1000 for Asthma NZ for the best supported team.
They raised a few extra thousands themselves for asthma and Fujitsu have donated a heat pump to be auctioned on Trade Me, free installation courtesy of Aircon Services Ltd and all proceeds goes to Asthma NZ. Keep an eye out on our Trade Me Charity Store.

THE GREAT AUCKLAND BED RACE
KING KOIL By A.H.BEARD

Asthma Auckland hosted a sausage sizzle fundraiser at Bunnings at Mt Roskill, raising our profile and a few dollars too. Thanks to those who supported us!
Pictured: Swarna Hemachandra, Anne Marie Roberts (visiting from UK), Asthma Nurse Educator – Ann Wheat.

A CASE STUDY

By Janet Delooze RN
Asthma Nurse Educator

This case study is about Aroha (pseudonym), a 44 year-old Maori lady, who contacted Asthma Auckland last year requesting some assistance with her poorly controlled asthma. An appointment was made to visit Aroha at home.

Aroha lived in a single-storey rented house that was reportedly cold and damp in the winter: cold and damp are well-known triggers for asthma. Her asthma started at about the age of 2 years and there was a family history in her paternal grandfather's family. Aroha had been a smoker for about 16 years having given up eight years ago.

Aroha had been prescribed Duolin (a short-acting combination) metered-dose inhaler (MDI) 100/20mcg 2 puffs 4 times daily and Symbicort turbuhaler (long-acting reliever and preventer combination) 200/6mcg 2 puffs twice daily. Aroha reported that she was using up to one canister of Duolin per week when her asthma was at its worse: she had no salbutamol on its own as a (short-acting) reliever. Duolin is usually prescribed up to 8 puffs daily so this far exceeded the recommended dose. Also, her Symbicort had been empty for a while.

At the visit, Aroha was quite breathless but able to hold a conversation. She reported that it had become increasingly difficult to carry out her daily activities and that her exercise tolerance had decreased considerably over time. She had been able to walk several kilometres the previous year but now she became breathless walking to the end of her drive. With less activity her weight had increased, giving her a BMI of 58 (healthy range: 18.5 to 24.9). Her peak flow reading was 280 l/min with an expected peak flow of 430 l/min. Twenty minutes after using her Duolin inhaler, her peak flow reading had increased to 300 l/min, an increase of 7% and she reportedly felt better.

At our educational visits, inhaler technique is always assessed. Aroha was using her Duolin inhaler directly into her mouth. Most of the medication would have been hitting the back of her throat or been swallowed. Using a spacer can increase the lung deposition from about 10% to around 30-40% especially with the new anti-static spacers.[1] A Volumatic spacer was given to Aroha with full instructions on its use and care.

Prescribers often assume that there can be no user errors when using a turbuhaler, however, in my experience this is often not the case. Indeed, a client I met recently thought that two inhalations from one dose were equal to two doses. Turbuhalers need to be correctly loaded, in the upright position, turned in the correct manner and inhaled with a good inspiratory flow for each dose. Clients often get confused about the amount of medication left in the device. Even when the counter is completely red, people assume that the powder they can hear moving around is medication; this is actually the desiccant that keeps the device dry.

At the end of the assessment, Aroha was advised to visit her doctor again as soon as possible to assess her condition, and to discuss her medications and obtain new inhalers. Duolin is not an inhaler for maintenance therapy in asthma though it is used in chronic obstructive pulmonary disease (COPD).[2] Salbutamol is essential for symptoms, with a cold/flu and especially for emergencies. I advised her to ask her doctor to complete her asthma action plan so that she had clear guidelines to follow for all eventualities. A written action plan is useful in aiding clients to self-manage with more confidence.[3]

Aroha was not aware of any lung function tests being carried out previously so I arranged to carry out spirometry at home at my next visit, and left plenty of appropriate literature for her to read later.

At the one month follow-up visit, Aroha reported that she had been in hospital with an acute exacerbation triggered by a chest infection. She had been treated with antibiotics and oral steroids but was feeling exhausted and breathless. The Ventolin inhaler given by the hospital had run out so she was relying on her Duolin inhaler for symptoms. I contacted her GP practice to arrange a new prescription for Ventolin and advised Aroha to make an appointment with her doctor for review, or if she became any worse, to call an ambulance, and to use 6 puffs of salbutamol every 6 minutes as per the action plan.

After a second course of oral steroids, Aroha was well enough to complete the spirometry test. Her results after the bronchodilator showed FEV^1 was 42% of the predicted value, FVC was 58% and FEV^1/FVC ratio 70%, indicating severe obstruction.[4] The results were sent to her GP with a letter recommending comprehensive lung function testing at the local hospital with a referral to pulmonary rehabilitation if the results were as low as the home spirometry.

At my next visit, Aroha had completed lung function testing and had been prescribed a Spiriva Handihaler once daily – a long-acting reliever – in place of her Duolin inhaler. This has to be prescribed on special authority, one of the criteria being that the FEV^1 must be below 60% of the predicted value. Although she now had a definite diagnosis of COPD, she was feeling much better overall, had been less breathless especially at night, had needed far less reliever and was now much more active than before. There is often a co-existence of COPD with asthma particularly if the client has smoked and/or their asthma has been poorly controlled for some time.[5] Although smoking is a trigger for asthma symptoms, anyone over the age of 40 who has smoked during their lifetime should be assessed by a doctor for COPD. It is diagnosed by a history of progressively worsening breathlessness, chronic cough and sputum production, and confirmed by spirometry where the FEV^1 is usually low with very little or no increase post bronchodilators (relievers): asthma is usually a reversible condition where there are periods without any symptoms. COPD is more common where there has been exposure to occupational dusts and chemicals, and biofuels, and Maori and Pacific peoples are also more likely to develop COPD.[6]

At the six-month visit to Aroha, she had obtained some exercise equipment with a goal to lose some weight and to increase her exercise tolerance. Her medications were well-stocked and up to date, and she was using them correctly. Symptoms of breathlessness, tight chest, fatigue and excess sputum production had reduced, and she had also moved into warmer, drier accommodation.

Aroha was put on the waiting list for Pulmonary Rehabilitation sessions. Alongside the appropriate medications, pulmonary rehabilitation has been shown to be the most effective therapy for COPD.[2] I hope that Aroha is able to maintain optimum health, and lead a fulfilling and symptom-free life. She has now been discharged from our services but is always welcome to refer herself again should she require any further support.

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THE IMPORTANCE OF AN EARLY DIAGNOSIS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) PATIENTS

By Elaine Murray RN
Asthma Nurse Educator

Chronic Obstructive Pulmonary Disease (COPD) is a common preventable and treatable disease. It is 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]

The symptoms of COPD are shortness of breath, chronic cough and chronic sputum production.

Episodes of acute worsening of these symptoms (exacerbations) often occur.

Worldwide, the most common risk factor for COPD is tobacco smoking. Air pollutants that can be outdoor, indoor or occupational are other major COPD risk factors. BUT, non-smokers may develop COPD.

There is also a genetic risk factor which is a severe hereditary deficiency of alpha-1 antitrypsin.

COPD risk is related to the total burden of inhaled particles a person encounters over their life time.[1]

- Tobacco smoke, including cigarette, pipe, and cigar as well as exposure to second hand smoke
- Indoor air pollution from biomass fuel used for cooking and heating in poorly ventilated dwellings, a risk factor that particularly affects women in developing countries
- Occupational dusts and chemicals (irritants and fumes) especially if the exposure is very high and prolonged
- Outdoor air pollution also contributes to the lung's total burden of inhaled particles, although it appears to have a relatively small effect in causing COPD

In addition, any factor that affects lung growth during gestation and childhood (low birth weight, respiratory infections and chronic asthma) has the potential to increase an individual's risk of developing COPD

Key indicators for considering a diagnosis of COPD are[1];

- Patient over 40 years of age
- Shortness of breath that is progressive (worsens over time), is persistent and usually worse with exercise
- A chronic cough, may be intermittent and unproductive
- Chronic sputum production
- Exposure to risk factors as above
- Family history of COPD

These indicators are not diagnostic. A spirometry test is required to make a clinical diagnosis of COPD.

Unfortunately the disease begins many years before a diagnosis is made and many patients with COPD remain undiagnosed and potentially unknown to health care providers until the more advanced stages of the disease.[2] A UK-based study published in The Lancet Respiratory Medicine journal finds that opportunities are being missed to diagnose COPD in up to 85% of people.[3]

COPD is estimated to affect 15% of all New Zealanders aged over 45 years. Amongst New Zealanders aged 45-64 years, Maori are approximately five times more likely to die from COPD-related causes than non-Maori and are affected by COPD up to 20 years earlier.[4]

Both patients and doctors can miss the early signs of lung disease that should be investigated. Despite being two distinct conditions, COPD is often misdiagnosed as asthma due to the overlap of symptoms.

Patients often accept their symptoms as part of ageing or believe it is due to "not being as fit as I used to be", and continue to ignore the symptoms such as breathlessness. As a consequence they reduce their activities or do less and less.

Others may dismiss their symptoms as "just a smoker's cough".

In a Dutch study, 74% of patients with symptoms or signs of COPD or asthma never consulted their GP for respiratory complaints, regardless of the severity of symptoms or lung function impairment.[5]

Early detection of COPD is crucial. Whilst COPD management previously focused on the reduction and control of symptoms, it is now appreciated that improving the health status and quality of life, preventing disease progression, preventing and treating exacerbations and complications, and reducing mortality are equally important.[6]

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines have proposed four main components of COPD management: (1) Assessing and monitoring disease (2); Reducing the risk factors; (3) Managing stable COPD, and (4) Managing exacerbations.[2]

Raising awareness of COPD, including its symptoms and progression, is essential for the early diagnosis of the disease.

The primary care team should be monitoring all at risk patients, encouraging them to discuss their symptoms at each visit and provide education and support, especially with smoking cessation. Spirometry should be offered to all at risk patients aged over 40 years. Due to the earlier age of onset and increased burden of COPD in Maori and Pacific peoples, testing for COPD in selected people who are at an increased risk should begin from 30 years of age.[4]

"Support not blame" is an important approach for health professionals engaging with people who have COPD. A feeling of judgement or blame, because of the association between smoking and COPD, may cause people to present to their doctor much later than they should.[5]

As an asthma nurse educators working in the community we often get a referral to visit a patient who has "asthma" but following a full assessment it is clear that the patient now has COPD but they have "never heard the word" or know anything about it and may not be on the correct inhalers.

Mild COPD patients may only have mild symptoms but maintaining their current lung function must be a priority. Therefore, it is important to encourage physical activity, provide education, especially with medication devices, have regular reviews with their GP and have a written management plan to follow for when they are well and for when they are not well.

If they are still smoking, smoking cessation is the most important treatment in the care of people with COPD. Smoking cessation will stop any further damage to the lungs and improve oxygen levels in their blood which will in turn give them more energy and they will feel less short of breath. They may also sleep better and regain their appetite and sense of smell, and food will taste better.

Smoking cessation is not easy but there is a lot of help and support available. Health care professionals should be offering advice at every available opportunity.

Smoking cessation is followed by a marked reduction in the irreversible accelerated decline in FEV_1 (forced expiratory volume in first second) which is associated with the development of the disease.[5]

In moderate COPD the breathlessness can be very limiting on daily activities due to the extra work of breathing and poor breathing patterns. Referral to pulmonary rehabilitation will help improve their fitness and understanding of their condition. The physiotherapist will help the patient to learn ways of controlling their breathlessness. They will learn to understand their limitations as to what they can do without causing extreme breathlessness. This can lead on to anxiety and panic which may cause them to hyperventilate and cause more breathlessness. The physiotherapist will also teach effective ways to cough and clear the phlegm which may become problematic especially during an exacerbation.

It is important to prevent deterioration by decreasing exacerbations and reducing a decline in the patient's quality of life. Regular assessments of patient's breathlessness and medication (along with inhaler technique) are imperative.

In conclusion, raising awareness of COPD, including its symptoms and progression, is essential for early identification of the disease. Health professionals need to encourage patients to discuss their symptoms. Primary care professionals are at the forefront of COPD diagnosis and management and are, therefore, ideally placed to provide education and support to their patient.[2]

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SURFACTANTS AND THEIR ROLE IN ASTHMA AND COPD

By Alice Paul RN
Asthma Nurse Educator

What is surfactant?

Surfactant: A fluid secreted by the cells of the alveoli (the tiny air sacs in the lungs) that serves to reduce the surface tension of pulmonary fluids; surfactant contributes to the elastic properties of pulmonary tissue, preventing the alveoli from collapsing. (Medicine Net.com)

Most of us associate surfactant with infant respiratory distress syndrome due to surfactant deficiency. However many studies have been conducted and are under way to isolate the role surfactant plays in asthma and other respiratory conditions.

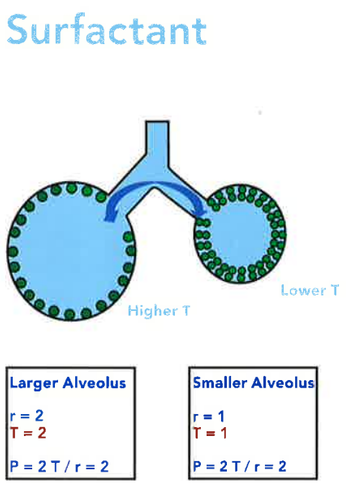
Relatively few studies have been carried out in airway surfactant because of major limitations in that there is no method for selective sampling of surfactant from the conducting airways.[1]

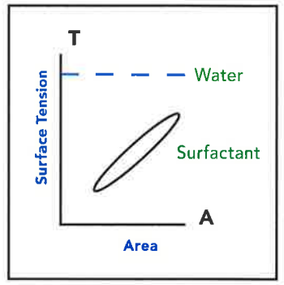
The main role of surfactant is to prevent collapse of the alveoli thereby reducing the effort needed to expand the lungs during inspiration (breathing in) and allow gas exchange to take place. Surfactant therefore helps breathing to be relatively effortless.[1]

Liu et al. found that surfactant – containing fluid allowed a free airway through the airways whereas saline led to spontaneous refilling of the capillary. The ability of surfactant to maintain free airflow was lost with the addition of albumin or fibrinogen (two potent surfactant inhibitors). The study also revealed that surfactant dysfunction by proteins was further disturbed by cooling which may explain the finding of increased airway resistance in patients with exercise induced asthma where airway surfactant with sufficient surface activity becomes seriously inactivated due to cooling during exercise with hyperventilation of cold air.[3]

Surfactant also contributes to the regulation of airway fluid balance, improves bronchial clearance and sets up a barrier to inhaled agents.It has also been shown to enhance mucocilary clearance partly by increasing cilary beat frequency. Other studies have also suggested that surfactant sets up a barrier to the diffusion of inhaled agents, including bacteria, allergens and drugs.[1]

In a study by Carla Winkler and Jens M. Hohlfeld they stated that the course of airway response upon allergen challenge in sensitised individuals can be divided into an early phase and late phase. The early phase develops very rapidly after allergen exposure and is characterised by mast cell degranulation accompanied by histamines and leukotriene release leading to bronchoconstriction and mucous hypersecretion. The late phase develops hours after allergen contact, with severe inflammation caused by activation of allergic specific effector cells and a massive influx of eosinophils into the airway. These effector cells in turn maintain an inflammatory environment in the lung leading to airway remodelling and airway obstruction. The pulmonary surfactant system is essential for reducing surface tension at the air – liquid interface and for regulating pulmonary immune responses. The soluble surfactant proteins in particular bind to regulate a variety of immune /effector cells present in the course of allergic airway inflammation.[2]





Lowers surface tension T

Surface tension increases with **area**

Tends to stabilize alveoli

(counteracts radius effect)

Hohlfeld points out in the study 'the role of surfactants in asthma' that there is no direct proof that surfactant dysfunction in human asthma causes airway obstruction, however some published data supports the concept that poor functioning surfactant contributes to the pathophysiological scenario in asthma.

There are two different ways to improve the surfactant balance in the airways. Firstly, the use of corticosteroids which are commonly used in asthma treatment and secondly treatment with exogenous surfactant has been shown to improve allergic airway obstruction in animal models of asthma. Human data are rare: a small randomised controlled trial demonstrated a significant improvement in pulmonary function data after inhalation of surfactant in patients with acute asthma attacks.[4]

Hohlfeld concludes that pulmonary surfactant with an optimal function in the airways is important because it stabilizes the conducting airways, prevents fluid accumulation within the airway lumen, improves bronchial clearance, acts as a barrier against the uptake of inhaled agents and has important immunomodulatory properties. In asthma it has been demonstrated that there is a surfactant dysfunction mainly due to inhibition by proteins that enter the airways during the inflammatory process. This provides an understanding of the pathophysiological scenario of airway obstruction in respiratory disease.[1]

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NEWSTREAM

Source: **Eur Respir J**
Tiotropium and olodaterol fixed-dose combination versus mono-components in COPD (GOLD 2-4); Buhl R, Maltais F, Abrahams R, Bjermer L, Derom E, Ferguson G, Fležar M, Hébert J, McGarvey L, Pizzichini E, Reid J, Veale A, Grönke L, Hamilton A, Korducki L, Tetzlaff K, Waitere-Wijker S, Watz H, Bateman E; European Respiratory Journal (Jan 2015)
Efficacy and safety of tiotropium+olodaterol fixed-dose combination (FDC) compared with the mono-components was evaluated in patients with moderate to very severe chronic obstructive pulmonary disease (COPD) in two replicate, randomised, double-blind, parallel-group, multicentre, phase III trials. Patients received tiotropium+olodaterol FDC 2.5/5 µg or 5/5 µg, tiotropium 2.5 µg or 5 µg, or olodaterol 5 µg delivered once-daily via Respimat inhaler over 52 weeks. Primary end points were forced expiratory volume in 1 s (FEV1) area under the curve from 0 to 3 h (AUC0-3) response, trough FEV1 response and St George's Respiratory Questionnaire (SGRQ) total score at 24 weeks. In total, 5162 patients (2624 in Study 1237.5 and 2538 in Study 1237.6) received treatment. Both FDCs significantly improved FEV1 AUC0-3 and trough FEV1 response versus the mono-components in both studies. Statistically significant improvements in SGRQ total score versus the mono-components were only seen for tiotropium+olodaterol FDC 5/5 µg. Incidence of adverse events was comparable between the FDCs and the mono-components. These studies demonstrated significant improvements in lung function and health-related quality of life with once-daily tiotropium+olodaterol FDC versus mono-components over 1 year in patients with moderate to very severe COPD.

Source: **Am J Respir Crit Care Med**
Asthma-COPD Overlap: Clinical Relevance of Genomic Signatures of Type 2 Inflammation in COPD; Christenson S, Steiling K, van den Berge M, Hijazi K, Hiemstra P, Postma D, Lenberg M, Spira A, Woodruff P; American Journal of Respiratory and Critical Care Medicine (Jan 2015)
RATIONALE: COPD is a heterogeneous disease, and likely includes a subgroup that is biologically comparable to asthma. Studying asthma-associated gene expression changes in COPD could add insight into COPD pathogenesis and reveal biomarkers that predict a favorable response to corticosteroids.
OBJECTIVE: To determine whether asthma-associated gene signatures are increased in COPD and associated with asthma-related features. **METHODS:** We compared disease-associated airway epithelial gene expression alterations in an asthma cohort (n=105) and two COPD cohorts (n=237, 171). The Th2 Signature ('T2S') score, a gene expression metric induced in Th2-high asthma, was evaluated in these COPD cohorts. The T2S score was correlated with asthma-related features and response to corticosteroids in COPD in a randomized placebo-controlled trial (GLUCOLD, n=89).
MEASUREMENTS AND MAIN RESULTS: The 200 genes most differentially expressed in asthma versus healthy controls were enriched among genes associated with more severe airflow obstruction in these COPD cohorts ($p<0.001$), suggesting significant gene expression overlap. A higher T2S score was associated with decreased lung function ($p<0.001$), but not asthma history, in both COPD cohorts. Higher T2S scores correlated with increased airway wall eosinophil counts ($p=0.003$), blood eosinophil percentage ($p=0.03$), bronchodilator reversibility ($p=0.01$), and improvement in hyperinflation following corticosteroid treatment ($p=0.019$) in GLUCOLD.
CONCLUSION: These data identify airway gene expression alterations that can co-occur in asthma and COPD. The association of the Th2 signature with increased severity and 'asthma-like' features (including a favorable corticosteroid response) in COPD suggests Th2 inflammation is important in a COPD subset that cannot be identified by clinical history of asthma.

Source: **Am J Respir Crit Care Medl Posted 3 weeks ago**
Occupational Exposures are Associated with Worse Morbidity in Patients with COPD; Paulin L, Diette G, Blanc P, Putcha N, Eisner M, Kanner R, Belli A, Christenson S, Tashkin D, Han M, Barr R, Hansel N, for the SPIROMICS Research Group ; American Journal of Respiratory and Critical Care Medicine (Jan 2015)
RATIONALE: Links between occupational exposures and morbidity in individuals with established chronic obstructive pulmonary disease (COPD) remain unclear.
OBJECTIVES: To determine the impact of occupational exposures on COPD morbidity.
METHODS: A job exposure matrix (JEM) determined occupational exposure likelihood based on longest job in current/former smokers (n=1075) recruited as part of the SPIROMICS cohort study, of whom 721 had established COPD. Bivariate and multivariate linear regression models estimated the association of occupational exposure with COPD, and among those with established disease, the occupational exposure associations with 6-minute walk test-distance [6MWT], Modified Medical Research Council Dyspnea Scale [mMRC], COPD Assessment Test [CAT], St. George's Respiratory Questionnaire [SGRQ], 12-item Short-Form Physical Component [SF-12], and COPD exacerbations requiring health care utilization, adjusting for demographics, current smoking status, and cumulative pack-years.
MEASUREMENTS AND MAIN RESULTS: Intermediate/high risk of occupational exposure by JEM was found in 38% of participants. In multivariate analysis, those with job exposures had higher odds of COPD (OR=1.44, 95% CI (1.04-1.97)). Among those with COPD, job exposures were associated with shorter 6MWT distances (-26.0m, $p=0.006$), worse scores for mMRC (0.23, $p=0.004$), CAT (1.8, $p=0.003$), SGRQ (4.5, $p=0.003$), and SF-12 Physical (-3.3, $p<0.0001$); and greater odds of exacerbation requiring health care utilization (OR=1.55, $p=0.03$).
DISCUSSION: Accounting for smoking, occupational exposure was associated with COPD risk and, for those with established disease, shorter walk distance, greater breathlessness, worse quality of life, and increased exacerbation risk. Clinicians should obtain occupational histories from patients with COPD, as work-related exposures may influence disease burden.

Source: **Lancet Respir Med**
The effect of an electronic monitoring device with audiovisual reminder function on adherence to inhaled corticosteroids and school attendance in children with asthma: a randomised controlled trial; Chan A, Stewart A, Harrison J, Camargo C, Black P, Mitchell E; Lancet Respiratory Medicine (Jan 2015)
BACKGROUND: Suboptimum adherence to preventive asthma treatment is associated with substantial morbidity and mortality, yet adherence often remains poor. We aimed to investigate whether use of an inhaler with audiovisual reminders leads to improved adherence and asthma outcomes in school-aged children who presented to the emergency department with an asthma exacerbation.
METHODS: We did a randomised controlled trial in patients aged 6-15 years who attended the regional emergency department in Auckland, New Zealand with an asthma exacerbation and were on regular inhaled corticosteroids. Using a simple, unrestricted block randomisation with block sizes of

200, we randomly assigned patients to receive an electronic monitoring device for use with their preventer inhaler with the audiovisual reminder functions either enabled to support adherence to inhaled corticosteroids (intervention group) or disabled (control group). Participants were followed up every 2 months for 6 months. The primary outcomes were adherence to preventive inhaled corticosteroids and number of days absent from school for any reason. Asthma control was assessed as a secondary outcome. All analyses were done in the intention-to-treat population. This trial is registered with the Australian New Zealand Clinical Trials Registry, number ACTRN12613001353785.
FINDINGS: The study took place between May 10, 2010, and Feb 26, 2012. We randomly assigned 220 patients, 110 to the intervention group and 110 to the control group. Median percentage adherence was 84% (10th percentile 54%, 90th percentile 96%) in the intervention group, compared with 30% (8%, 68%) in the control group (p<0·0001). The proportion of days absent from school for any reason was 1·9% (10th percentile 0·0%, 90th percentile 7·9%) in the intervention group and 1·7% (0·0%, 8·6%) in the control group. The change in asthma morbidity score from baseline to 6 months was significantly greater in the intervention group than in the control group (p=0·008), with a reduction of 2·0 points from a mean baseline score of 9·3 (SD 2·2) to 7·3 (2·1) in the intervention group, compared with a reduction of 1·2 points from a baseline of 9·2 (2·5) to 8·0 (2·2) in the control group.
INTERPRETATION: Use of an electronic monitoring device with an audiovisual reminder led to significant improvements in adherence to inhaled corticosteroids in school-aged children with asthma. This intervention could be beneficial for the improvement of asthma control in patients for whom poor asthma control is related to poor adherence.

Source: **J Asthma**
Mobile-based asthma action plans for adolescents; Burbank A, Lewis S, Hewes M, Schellhase D, Rettiganti M, Hall-Barrow J, Bylander L, Brown R, Perry T; Journal of Asthma 1-4 (Jan 2015)
ABSTRACT PURPOSE: To examine feasibility and utilization of a mobile asthma action plan (AAP) among adolescents.
METHODS: Adolescents (aged 12-17 years) with persistent asthma had their personalized AAP downloaded to a smartphone application. Teens were prompted by the mobile application to record either daily symptoms or peak flow measurements and to record medications. Once data were entered, the application provided immediate feedback based on the teen's AAP instructions. Asthma Control Test (ACT®) and child asthma self-efficacy scores were examined pre- and post-intervention.
RESULTS: Adolescents utilized the mobile AAP a median 4.3 days/week. Participant satisfaction was high with 93% stating that they were better able to control asthma by utilizing the mobile AAP. For participants with uncontrolled asthma at baseline, median (interquartile range) ACT scores improved significantly from 16 (5) to 18 (8) [p = 0.03]. Median asthma attack prevention self-efficacy scores improved from 34 (3.5) to 36 (5.3) [p = 0.04].
CONCLUSIONS: Results suggest that personalized mobile-based AAPs are a feasible method to communicate AAP instructions to teens.

Source: **J Asthma**
Increased body mass index predicts severity of asthma symptoms but not objective asthma traits in a large sample of asthmatics; Bildstrup L, Backer V, Thomsen S; Journal of Asthma 1-22 (Jan 2015)
ABSTRACT AIM: To examine the relationship between body mass index (BMI) and different indicators of asthma severity in a large community-based sample of Danish adolescents and adults.
METHODS: A total of 1,186 subjects, 14-44 years of age, who in a screening questionnaire had reported a history of airway symptoms suggestive of asthma and/or allergy, or who were taking any medication for these conditions were clinically examined. All participants were interviewed about respiratory symptoms and furthermore height and weight, skin test reactivity, lung function, and airway responsiveness were measured.
RESULTS: A total of 516 individuals had asthma. The mean BMI was 24.9 kg/m(2) (SD=5.1). Asthma severity measured by GINA score increased with increasing BMI (p=0.009). The result remained significant after adjusting for age, sex, medication use for asthma and smoking (p=0.010). Severity of individual asthma symptoms; cough (p=0.002) and chest tightness (p=0.023) was also significantly related to BMI, whereas severity of wheezing and shortness of breath was not. Airway obstruction was more pronounced in subjects with increased BMI (p<0.001) but the effect disappeared after adjustment for covariates (p=0.233). Lung function, airway responsiveness, and atopy were not significantly related to BMI as were use of medication for asthma and adherence to treatment.
CONCLUSIONS: In adults, increased body mass index predicts severity of asthma symptoms but not objective asthma traits.

Source: **J Asthma**
Association between vitamin D and respiratory outcomes in Canadian adolescents and adults; Niruban S, Alagiakrishnan K, Beach J, Senthilselvan A; Journal of Asthma 1-33 (Jan 2015)
ABSTRACT BACKGROUND: Asthma is one of the most prevalent chronic diseases worldwide, affecting more than 200 million people. Vitamin D deficiency has been reported among individuals with asthma and might play a role in asthma exacerbations. In this cross-sectional study, we investigated the association of serum 25-hydroxy vitamin D [25(OH)D] levels and current asthma, ever asthma, and lung function.
METHODS: Data from 3,937 subjects aged 13-69 years who participated in the Canadian Health Measures Survey-Cycle 1 were considered in this study. Serum 25(OH)D levels were categorized into ≤49 nmol/L (low), 50-74 nmol/L (moderate) and ≥75 nmol/L (high).
RESULTS: The proportion of subjects with current and ever asthma was greater in the lower 25(OH)D category than in moderate and high categories. After adjusting for potential confounders, subjects in the low 25(OH)D levels were more likely to have current asthma than those in the moderate levels (OR:1.54, 95% CI:1.01-2.36). Low 25(OH)D levels were also associated with ever asthma (OR:2.12, 95% CI:1.40-3.21) among those with a family history of asthma and this association was stronger in those with asthma onset before 20 years of age. High 25(OH)D levels were associated with lower mean value of FEV1/FVC ratio. No significant association was observed between 25(OH)D levels and other lung function measurements.
CONCLUSION: In this study, 25(OH)D levels below 50 nmol/L were associated with an increased risk of current and ever asthma. Further exploration of this relationship is needed to determine the optimal level of vitamin D in the management of asthma in adolescents and adults.

Asthma New Zealand wishes to thank the generous support of the following funders and business –

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4. Pharmaceutical Schedule April 2014, PHARMAC.

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