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Emphysema Takes Your Breath Away — September 2008

BIOLOGIC LUNG VOLUME REDUCTION (BLVR) SYSTEM TRIAL ACHIEVES PRIMARY EFFICACY *Endpoint; Preparations for Phase 3 Trials are Underway*

Aeris Therapeutics, Inc. today announced that the primary efficacy endpoint in Phase 2 clinical trials for its Biologic Lung Volume Reduction (BLVR) System has been achieved. BLVR is designed to provide a safe non-surgical alternative to lung volume reduction surgery, one of the few available treatments for patients with advanced emphysema. The BLVR system is the first biologic therapy that has been successfully used to remodel the lung and improve breathing function.

"The Phase 2 clinical trial data show that BLVR is well tolerated and is associated with significant improvements in breathing function, symptoms and quality of life," commented Dr. David Dove, CEO of Aeris. "Based on these positive results, we are currently preparing for the initiation of Phase 3 trials."

Aeris conducted two U.S. Phase 2 studies enrolling 44 patients with upper lobe predominant emphysema to define the optimal dosing regimen for BLVR treatment. Additional trials focusing on the use of BLVR for homogeneous emphysema are in progress.

The primary endpoint for the Phase 2 trials was a statistically significant reduction in lung volume at three months following treatment, and was achieved in both studies. In addition, for patients in the high dose group, more than half had clinically significant improvements in breathing function, and more than three quarters had clinically significant improvements in quality of life measures, including activity level, mood and ability to work. BLVR was well tolerated and the median hospital length of stay was two days. As a comparison, patients undergoing open-chest lung volume reduction surgery have a median hospital length of stay of 10 days.

The primary endpoint was measured using the Residual Volume to Total Lung Capacity Ratio (RV/TLC). Improvement in breathing function was assessed using FEV1, a standard pulmonary function test considered to be a primary indication of lung function. This test measures the amount of air a patient can forcibly expel from his or her lungs in one second. Mean FEV1 improvements in the high dose group exceeded 17 percent at three months.

The Biologic Lung Volume Reduction (BLVR) System is a non-surgical treatment for emphysema that is currently under investigation. A bronchoscope is used to deliver a proprietary mixture of drugs and biologics that form a bioabsorbable gel at diseased areas of the lungs. The gel is a mixture of clotting agents that help deliver the treatment, an antibiotic to prevent infection, and drugs designed to cause a reaction in targeted areas of the lungs--actually using the body's natural scar formation response to permanently collapse diseased areas of the lungs. This reduction in lung volume creates more space for the non-diseased parts of the lungs to function more effectively. http://tinyurl.com/5njnvp/

CHRONIC OBSTRUCTIVE PULMONARY DISEASE: *Beyond Respiratory System*

Abstract

Chronic obstructive pulmonary disease (COPD) is defined as set of breathing related problems i.e. chronic bronchitis, expectoration and exertional dyspnea characterized by presence of airflow obstruction that is not fully reversible. However, COPD is a preventable and treatable disease with some extra pulmonary effects including skeletal muscle dysfunction, nutritional abnormalities and systemic inflammation that may contribute to the severity of the individual patients. The current clinical focus on respiratory symptom should be expanded to include assessment and prevention of diverse negative effects of the disease.

Introduction

Chronic obstructive pulmonary disease (COPD) which includes chronic bronchitis and emphysema is a progressive disease characterized by airflow limitation/obstruction that is either not reversible at all or only partially reversible. It is generally difficult to separate out the two conditions (chronic bronchitis and emphysema), hence these are grouped together as COPD. The airflow obstruction in COPD is associated with abnormal inflammatory response of the lungs to chronic inhalational exposure from smokes, dusts and gases. This definition, as well as those published by many other societies and organizations focuses exclusively on the lungs. Recently, besides the typical pulmonary pathology of COPD (i.e. chronic bronchitis and emphysema), several effects occurring outside the lungs often associated with extra pulmonary abnormalities and have been described as the so called systemic effects of

COPD.There is increasing realization that these systemic effects are clinically relevant and may contribute to better understanding and management of the disease. In the present review, extra pulmonary effects of COPD along with their potential mechanism and clinical implications have been discussed.

Systemic Inflammation

It is currently accepted that an excessive/inadequate inflammatory response of the lungs to a variety of noxious inhaled gases or particles (mostly cigarette smoke) is a key pathogenic mechanism in COPD. Various studies have shown that the lung inflammatory response is characterized by:

1) increased numbers of neutrophils, macrophages and T-lymphocytes with a CD8+ predominance;

2) augmented concentrations of proinflammatory cytokines, such as leukotriene B4, interleukin (IL)-8 and tumor necrosis factor (TNF)-alpha, among others; and

3) evidence of oxidative stress caused by the inhalation of oxidants (tobacco smoke) and/or the activated inflammatory cells mentioned above. It is less often realized that similar inflammatory changes can also be detected in the systemic circulation of these patients, including evidence of oxidative stress, the presence of activated inflammatory cells and increased plasma levels of proinflammatory cytokines.

Nutritional Abnormalities

Malnutrition contributes to respiratory muscle weakness resulting in increased frequency of hospitalization, cor pulmonale and increased mortality. Several etiologies have been proposed for the nutritional deficiency observed in patients with COPD Imbalance between energy intake and energy expenditure, due to decreased intake or increased expenditure, seems to be the factor involved in most cases. Mechanisms or causes of Progressive Weight Loss:

Hypotheses

1. Decreased food intake: Several studies have demonstrated that COPD patients consume more calories than estimated for normal people or by measured energy requirements. Lewis and co-workers found that caloric intake was 150% of the calculated basal energy expenditure (BEE) in COPD patients with weight loss.

2. Elevated resting energy requirements: Malnourished COPD patients show incomplete metabolic adaption to weight loss and have greater than predicted resting energy requirements.

Diet-induced thermogenesis: An obligatory energy expenditure occurs during assimilation of food into the body.

This is abnormally elevated in malnourished COPD patients and contributes to their increased basal metabolic rate (BMR). Carbohydrate based diets (53% of calories from carbohydrates) cause a 20% rise in resting energy expenditure (REE) while fat based diets (55% of calories from fat) cause a 14% rise in REE.

Increased daily energy expenditure – energy cost of daily activities: The increased metabolic requirements in malnourished COPD patients are the result of the increased oxygen costs of augmented ventilation relative to 2.61 in normally nourished COPD patients .Hyperinflation with the associated mechanical disadvantage of the respiratory muscles, reduced ventilatory muscle strength and increased mechanical load, but also reduced ventilatory muscle efficiency.

The terms “malnourishment” and “cachexia” are often used indiscriminately in discussion of the nutritional abnormalities in COPD; however, important differences exist between these terms. Both terms share several biochemical characteristics, but their origin and, importantly, response to dietary supplementation are very different. Several observations suggest that patients with COPD may suffer from cachexia rather than malnourishment. For instance, the caloric intake of patients with COPD is normal or even greater than normal, not lower, as in malnourishment; their metabolic rate is usually increased whereas it is decreased in malnourished patients; and their response to nutritional support is often poor.

Skeletal Muscle Dysfunction

Skeletal muscle dysfunction is common in patients with COPD . It is characterized by specific anatomic changes (e.g., fiber-type composition and atrophy) and functional changes (e.g., strength, endurance, and enzyme activities) and contributes significantly to limited exercise capacity and reduced quality of life. The respiratory muscles, in particular the diaphragm, appear to behave quite differently from skeletal muscles in patients with COPD, from both the structural and functional points of view. The skeletal muscles are generally underused, whereas the diaphragm is constantly working against an increased load. Sedentarism, tissue hypoxia, and systemic inflammation are likely to be relevant pathogenic factors in skeletal muscle dysfunction

Musculoskeletal Effects

The musculoskeletal system is among the extra pulmonary organ systems most frequently affected by chronic obstructive pulmonary disease (COPD). Initially described as weight loss and cachexia, the involvement of the musculoskeletal apparatus in COPD is now better understood as a loss of fat-free mass (FFM) and bone mineral density (BMD). Previous studies have described these processes separately and established the body mass index (BMI) as a predictor of loss of FFM and BMD. Increasing severity of COPD is found to be associated with decreasing FFM and BMD that includes progressive deconditioning and inactivity, greater number of exacerbations, increased use of corticosteroids, and increasing systemic inflammation.

Cardiovascular Effects

COPD increases the risk of cardiovascular disease by two- to threefold (19) For every 10% decrease in FEV1, there is cardiovascular mortality increases by approximately 28% and risk of non-fatal coronary events increase by approximately 20% in mild to moderate COPD.Besides reduced lung function is an independent and significant predictor of cardiovascular morbidity and mortality along with first-time stroke and fatal stroke. Several studies have shown that the endothelial function in COPD is abnormal in both pulmonary and systemic (renal) circulations . Tobacco smoking is a shared risk factor for both COPD and cardiovascular disease. The inflammatory process seen in patients with COPD might be the mechanism responsible for this association.

Renal And Hormonal Abnormalities

Renal and hormonal abnormalities, usually manifested as oedema or hyponatraemia, are encountered frequently in patients with chronic obstructive pulmonary disease. Edema in patients with COPD has been attributed to "cor pulmonale with backward heart failure"—that is, pulmonary hypertension induced by hypoxia and by structural changes in pulmonary arteries, increased systemic venous pressure, and reduced cardiac output. Experimental evidence has accumulated in support of the hypothesis that, in the advanced stages of COPD, imbalances in hormones that regulate body Na+ and water homeostasis—namely, the renin-angiotensin aldosterone axis and the arginine vasopressin system—are potential contributors to edema and hyponatraemia. (The most consistent alteration in renal function in hypoxemic hypercapnic patients with COPD is the reduction in Effective Renal Plasma Flow ERPF.

Osteoskeletal Effects

The prevalence of osteoporosis is increased in patients with COPD. Osteoporosis can have multiple causes, singly or in combination, including malnutrition, sedentarism, smoking, steroid treatment and systemic inflammation. Since most of them are already considered potential pathogenic factors of Skeletal muscle dysfunction in COPD, they could theoretically also contribute to osteoporosis, and, in this context, excessive osteoporosis in relation to age could also be considered a systemic effect of COPD.

Endocrinal Disturbances

Disturbances in the anabolic hormone system may also impair the anabolic responses needed for skeletal muscle performance.

Growth hormone/insulin-like growth factor-I

Growth hormone provides stimulation for muscle growth and development. In addition to increasing age, systemic corticosteroids (commonly used to treat COPD exacerbations) are known to down-regulate the growth hormone system IGF-I levels in stable COPD patients tend to be low consistent with the impression that the growth hormone axis is suppressed by chronic disease.In COPD, physiological stress like chronic hypoxia and broncho constriction could possibly induce an increase in growth hormone.

Thyroid hormone

An important function of thyroid hormone is regulation of metabolism and thermo genesis. Abnormalities in thyroid function potentially influence energy balance and body composition. Hyper metabolism is commonly observed in patients with COPD; this has been attributed to increased energy expenditure both at rest and during physical activities. A hyper metabolic state in combination with insufficient dietary intake will result in a negative energy balance and may conceivably contribute to weight loss in COPD Low FEV1 was associated with low basal and stimulated TSH levels.

Testosterone

Accumulating data indicate that testosterone levels are low in COPD. The mechanism of these alterations is unclear, but it has been speculated that chronic hypoxia, disease severity, smoking, corticosteroid therapy and chronic (inflammatory) illness contribute to low testosterone levels. One of the suggested underlying factors for hypogonadism is hypoxaemia, which is present in a portion of the COPD population. SEMPLE et al. found low testosterone levels in acutely ill, hospitalised COPD patients with hypoxaemia (Pa,O2 ranging from 5–10 kPa). The degree of testosterone depression was correlated to the severity of arterial hypoxaemia and hypercapnia.

Nervous System Defects

The energy metabolism of the brain is altered in these patients. Patients with hypoxic COPD have evidence of a sub clinical parasympathetic autonomic neuropathy with apparent preservation of sympathetic function. The abnormality may occur in stimulus reception, afferent nerve conduction, central processing, efferent nerve conduction, motor end-plate or end-organ (e.g. heart) response. Kinsman et al. showed a high frequency of sensory disturbances in COPD patients with few clinical signs. Cigarette smoking is a major etiological factor in the development of COPD and COPD-related peripheral neuropathy. It has several potential neurotoxic actions; carbon monoxide exacerbates tissue hypoxaemia, nicotine has stimulant actions and cyanogens may interfere with nerve function

Psychological Dysfunction

Many patients with COPD develop psychological symptoms in addition to physical complaints. For example, depression (?42%) and anxiety (?50%) are two to three times more prevalent in COPD patients than in the general population. In addition, panic disorder may occur in as many as 32% of depressed patients with COPD. Indeed, studies have shown that anxiety and depression play a larger role in determining a patients' quality of life than COPD severity. Overall, psychological distress compounds the negative symptoms of COPD and other respiratory illnesses, resulting in longer hospital stays, adverse medical outcomes, and higher mortality rates. Patients with breathing disorders are predisposed to anxiety and depression, since symptoms of these three overlap. COPD patients who develop anxiety or depression face greater levels of cognitive decline, more functional limitations, lower self-efficacy, and more serious life events than those with only breathing difficulties.

Clinical Relevance

The systemic effects reviewed above are likely to have a profound clinical impact on the management of COPD. First, weight loss and skeletal muscle dysfunction clearly limit the exercise capacity of these patients and, therefore, have a direct negative effect on their quality of life. Second, weight loss is a prognostic factor in patients with COPD that, importantly, is independent of other prognostic indicators, such as FEV1 or PaO2, that assess the degree of pulmonary dysfunction. Thus, weight loss identifies a new systemic domain of COPD not considered by the traditional measures of lung function. These observations indicate, therefore, that in addition to the severity of lung disease, the clinical assessment of patients with COPD should take into consideration the extra pulmonary consequences of COPD, with weight loss being a critical indicator.

Conclusion

COPD must be considered a systemic disease, and the extra pulmonary manifestations must be considered in the evaluation of its severity. In addition, the treatment of these manifestations could modify the prognosis of these patients. Further studies elucidating the systemic manifestations, especially those affecting nutritional status and peripheral skeletal muscle function, are needed for the development of new treatment strategies, which might improve the exercise tolerance and the overall health status of these patients. http://tinyurl.com/6deolcmedical records (EMR) of 47 general practices in Flanders. GPs had not received special instructions for testing specific patients. For each patient the mean cholesterol level per year was calculated. A patient belonged to the group with lipid-lowering drugs if there was at least one prescription of the drug in a year in his EMR. Mixed model linear regression models were used to quantify the effect of covariates on total cholesterol values.

Results

In the period 1994-2003 total cholesterol was tested in 47,254 out of 139,148 different patients. Twelve percent of those tested took lipid-lowering medication. The proportion of patients with at least one cholesterol test a year, increased over a period of ten years in all age groups, but primarily for those over the age of 65. The mean cholesterol level decreased in the treated as well as in the non-treated group. Of the patients with a cardiovascular antecedent who were on lipid-lowering drugs in 2003, 56% had a cholesterol level [less than or equal to]199 mg/dl, 31% between 200-239 and 13% over 240 mg/dl.

Conclusions

The indications for testing and treating cholesterol levels broadened considerably in the period examined. In 2003 cholesterol was tested in many more patients and patients were already treated at lower cholesterol values than in previous years. Comparisons of cholesterol levels over different years should therefore be interpreted with caution as they are a reflection of changes in medical care, and not necessarily of efficacy of treatment.

Source: BioMed Central Ltd; BMC Family Practice 2008

target for cell-based therapy. This disease is associated with:

1) loss of resident reparative cells;
2) loss of the extracellular matrix that transmits pre-stress signaling;
3) and loss of pre-stress itself, the very signal required to trigger regenerative responses.

Although these factors represent obstacles to the development of regenerative therapeutic strategies for emphysema, preliminary studies in our lab show that modulation of mesenchymal and epithelial cell proliferation using members of the fibroblast growth factor family complexed to carrier molecules in a biocompatible polymer can promote expansion of parenchymal tissues. The polymer scaffold, an air containing foam with mechanical properties similar to healthy lung tissue, effectively transmits stress to reparative/progenitor cells to promote proliferation and remodeling. Studies proposed here will test the hypothesis that therapeutic post-natal lung tissue growth in emphysema can be achieved

- by augmenting the lung's innate healing response using growth factors to direct endogenous reparative lung cells following a localized mild injury; and that
- the magnitude of this response can be modulated by altering pre-stress using concomitant bronchoscopic lung volume reduction therapy to increase transpulmonary pressures. We intend to advance this approach, known as pneumografting, into human trials under a physician-sponsored Investigation New Drug Application. http://tinyurl.com/6k3mve

MUSCLE STRENGTH BOOSTING SUPPLEMENT 'DOESN'T HELP' COPD PATIENTS

A nutritional supplement popularly known for boosting athletic performance and muscle strength does not improve exercise outcomes in patients with chronic obstructive pulmonary disease (COPD), says a new study.

The randomized, placebo-controlled, double blind study provided the most powerful evidence to date that the effect of Creatine (Cr) supplementation was negligible at best among these patients.

We have evidence to suggest Cr uptake into muscles [in COPD patients] but are unable to explain why an increase in muscle Cr did not enhance training," wrote the study"s lead author, Sarah Deacon, M. D., specialist registrar at the Institute for Lung Health at Glenfield Hospital in Leicester, England.

The results were published in the first issue for August of the American Journal of Respiratory and Critical Care Medicine by the American Thoracic Society (ATS).

Cr supplementation has been shown to improve short-burst, high-intensity exercise function in athletes, as well as enhancing isometric muscle strength, lower body endurance and lean body mass in the elderly.

To determine whether Cr supplementation could similarly enhance the physical condition of COPD patients, Dr. Deacon and co-researchers recruited 100 patients with COPD to either receive Cr or a placebo over the course of a seven week pulmonary rehabilitation program.

Those who were randomized to the placebo group were give lactose supplements that appeared identical to the Cr-containing supplements. Following a five-day loading period each subject followed maintenance dosing of 3.76 or 4 g of Cr or lactose respectively.

Of the original 100 subjects, 80 successfully completed the study. In both control and Cr groups, there were statistically significant improvements in functional and muscular performance during the loading phase, but no differences were seen between the groups.

The Cr group also showed a greater, but non-significant percentage of improvement in the incremental shuttle walking test with loading and after pulmonary rehabilitation, but additional analysis still showed no overall effect between it and the placebo group.

"The most likely explanation is that any benefits of creatine have been submerged by the large training effect of

physical training alone," wrote Dr. Deacon.

This study, therefore, further validates that there is no substitute for the old-fashioned hard work that is an essential element of pulmonary rehabilitation. (ANI)

The results of the study are published in the Journal of Exercise Physiology. http://tinyurl.com/66ckxe

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THE RESPIRATION CONNECTION

How dysfunctional breathing can cause upper body pain and injuries

This article will trace the steps between respiratory dysfunction and a constellation of painful upper body conditions: from garden variety stiffness to seemingly unlikely consequences such as rotator cuff injuries and whiplash.

The connection between dysfunctional breathing is straightforward in principle: if the diaphragm doesn't do its job, muscles in the upper chest (pectoralis minor) and throat (sternocleidomastoid and scalenes) try to take over.1 Unfortunately, these muscles aren't built for routine respiration, and they exhaust and eventually injure themselves. A cascade of potential consequences results.

If the diaphragm doesn't do its job, muscles in the upper chest (pectoralis minor) and throat (scalenes) try to take over. This scenario is common. About 75% of my clients consistently try to inhale by lifting their rib cages with muscles in the upper chest and throat. Most are able to stop doing so when they put their mind to it, but easily slip back into the habit. A few are unable to make the change at all without diligent practice.

That's the connection. But what is dysfunctional breathing, and why do we ask such unsuitable muscles to work so hard in the first place?

Hydraulics

If you understand the principle of hydraulics, you can quickly grasp how breathing should work. When your diaphragm moves, your belly has to move — if it doesn't, you aren't using your diaphragm. The diaphragm is your primary breathing muscle. It is a thin, wide sheet of muscle that separates the rib cage from the abdomen. It has a high domed shape which flattens out significantly when it contracts.

The hydraulics of respiration

The diaphragm flattens as it contracts, pushing the abdominal walls outwards. When the diaphragm flattens, it pushes downwards on the viscera like a hydraulic plunger. Since the watery viscera cannot be compressed,4 they have to get out of the way. So where do they go?

They go outwards! The abdominal contents are forced down and out. When you inhale with your diaphragm, your belly expands. Hence, good breathing is usually described as "abdominal breathing" or "diaphragmatic breathing."

The Buddha belly

The way in which the belly sticks out during healthy inhalation can often be seen in statuary of the Buddha. Good breathing and a flexible pot belly are associated with enlightenment, apparently!

Okay, maybe this Buddha is just overweight... but they almost all have pot bellies!

Enlightenment aside, good breathing is generally associated with vitality (as well as some other unusual phenomena). Only the liveliest people are breathing well: athletes and martial artists, dancers and actors and people who belly laugh a lot. The rest of us struggle to maintain both our breath and our vitality.

Eight reasons people don't breathe diaphragmatically

If breathing diaphragmatically was good enough for Buddha and is a key to vitality, why doesn't everyone do it? Monkey see, monkey do — no else does, so we don't. Shallow breathing is a pattern that we start imitating when we are infants.

Breathing is emotionally stimulating and expressive, and most people avoid emotions and expressiveness as carefully as they steer clear of pot bellies.

Men and women alike are afraid to have little pot bellies like Buddha, even for a moment. Belly sucking-in may be the most popular postural habit.

Life in chairs — with the hips flexed and the belly compressed from below — makes diaphragmatic breathing mechanically difficult.

Rat racey stress tends to accumulate high in the body: face, jaw, neck and shoulders. Many clients are too busy grinding their teeth to breathe from the gut.

A churning, tight belly is another common consequence of stress, and obstructs abdominal breathing even more effectively than facial tension distracts you from it.

Once lost, diaphragmatic strength is difficult to regain. It is one thing to be out of the habit of breathing abdominally (at age ten, say) and quite another to have lost the diaphragmatic strength and coordination for it (by age twenty-two, for instance). It's not like you can't breathe without strong diaphragmatic contraction — it's just more difficult. So perhaps the most insidious reason that people don't breathe with their diaphragms is because they can. Most people would rather stick with an understated respiratory style rather than work harder. They can get away with it, so they do.

But what exactly happens when you don't use your most important respiratory muscle? How do you get away with it?

The alternative: reverse breathing

When people don't breathe well, they tend to breathe in reverse: that is, the movement of their abdomen during respiration is the opposite of what is normal and healthy! Instead of letting the belly move outward during inhalation, they try to suck it in. And on exhalation, they relax the belly slightly. Of the next ten people you pass on the street, nine of them are probably reverse breathing.

Healthy versus "Reverse" Respiration

Inhalation - Healthy Breathing belly goes out belly sucks in
Exhalation - Reverse Breathing belly sucks in belly goes out

Even people who can breathe diaphragmatically when they go slowly will start to reverse breathe when they pick up the pace. It's purely a matter of coordination, a pat-your-head/rub-your-tummy challenge!

Reverse breathing is not inherently bad, but it's not a good idea to go it all the time. It's hard to ventilate thoroughly if you

are sucking in your belly while your diaphragm is trying to descend. It's so challenging to inhale while your belly is sucked in that it surprising that all those reverse breathers don't black out more often! In fact, it's so challenging to inhale while your belly is sucked in that it surprising that all those reverse breathers don't black out more often. Shouldn't the side walks be littered with dazed, gasping reverse breathers? How are they managing to get air at all, if not through contraction of the diaphragm?

Emergency breathing

The body is equipped with several emergency backup respiratory muscles. The diaphragm does not work alone. Any breath that uses extra muscle is considered to be forced respiration. Normal, healthy, relaxed breathing doesn't take much effort. Inhalation can be managed by the diaphragm alone, and exhalation takes no muscle contraction at all: the lungs collapse elastically, pushing air out effortlessly.

A sneeze or a cough, on the other hand, takes everything you've got: every fibre of muscle attached to your rib cage contracts violently. Yawning is not as heavy a recruiter, but is still much more intense than normal breathing. And, of course, you also use more breathing muscles when you exercise — depending on the intensity, this can range anywhere from just a little bit to quite a lot of extra breathing power.

Here are the muscles involved in respiration, and their roles in healthy breathing:

Diaphragm

This is your most important breathing muscle — it always has to work, even in relaxed breathing.

Intercostals

Tiny muscles between the ribs, recruited for slightly forced respiration.

Abdominals

The abdominals pull the rib cage down and push the diaphragm up, so they are vital for strong exhalation.

Quadratus Lumborum

"Quad" meaning "four-sided" and "lumb" as in "lumbar" — throw in some suffixes, and you've got some Latin.This is a low back muscles that pulls down hard on your bottom ribs. People sometimes tear it when they sneeze! It is recruited only for the strongest exhalations.

Pectoralis Minor

The pectoralis minor muscles are tiny chest muscles that pull up on the rib cage. The rib cage is heavy: this is definitely an emergency breathing muscle only.

Sternocleidomastoids

These prominent throat muscles form a distinctive V-shape. Like the pectoralis minors, they are rib cage lifters that should only be used when you absolutely, positively have to get something out of your trachea. I can breathe hard without ever feeling these muscles twitch!

Scalenes

Weirdest (and most important) of all the respiratory helper muscles are the scalenes. I have written entire articles about them (see Further Reading at the bottom of this article). They descend from the sides of the neck and attach to the uppermost ribs — and sometimes even attach to the top of the lungs. Their primary job is to move necks, but they also get involved in rib cage lifting when the need is great.

The muscles of respiration

Unfortunately, most people don't use their diaphragms to breathe, so they have to use their emergency breathing muscles. It's inefficient, so they have to work hard to take normal breaths, as if every moment was like a respiratory emergency! Does stress cause people to breathe poorly? Or does breathing poorly cause stress? It's both, obviously: each pattern aggravates the other. What a pickle.

So, other than always breathing like you're trying to run from lions, and giving your sternocleidowhatsis muscles a lot of exercise, what's the big deal? What's so bad about breathing with your chest and neck muscles?

The consequences

Chronic upper chest and neck breathing first exhausts and eventually damages the emergency breathing musculature, causing a list of bad news: exercise gets more difficult, and the risk of both acute and chronic injuries and painful conditions in the neck and shoulders increases dramatically — especially whiplash injuries.7

The importance of exercise should not be underestimated. Canadians suffer from a general plague of poor fitness — when "the burn" is much worse, because we can't breathe properly, how much more difficult is it to start and maintain an exercise program? The consequences of a sedentary lifestyle are far-reaching.

The risk of both acute and chronic injuries and painful conditions in the neck and shoulders increases dramatically. The increased risk of neck and shoulder injuries is easier to define, however, and I treat the symptoms of this disease in my practice every day. Exhausted muscles develop what I call "sick muscle syndrome" — they develop hard knots, technically known as myofascial trigger points, that radiate pain in characteristic patterns.8 The intensity of this phenomenon can range from mild to crippling, and it doesn't just hurt: anything that lies in the path of that radiating pain is vulnerable, interfering with normal function in a variety of ways.

Sick muscles don't do their own job very well, either: the more you've worn out your neck and chest muscles trying to breathe without your diaphragm, the more likely you are to have a problematic upper body posture and lousy mechanics of the shoulder and spinal joints, which leads to yet more injuries.

The combination of sick muscle syndrome and bad joint mechanics and posture cause and/or aggravate many problems:

A significant portion (probably more than half) of all upper back pain is caused by problems in the neck.

Shoulder tendonitises.

Whiplash. The more exhausted your neck muscles, the more a whiplash injury is going to hurt, and the longer it's going to take to heal — in many cases, people never really heal at all.

Frozen shoulder, a condition in which the shoulder joint mysteriously seizes up. While it is a strange condition whose ultimate causes are not known, it seems likely to me that it is at least aggravated by respiratory and upper body postural

dysfunction.

"Cricks" in the neck, most of which are probably caused by small mechanical problems in the spine (known as minor intervertebral derangements, or MIDs1011) that can cause days or weeks of painful protective spasm and months of stiffness.

Ringing in the ears and other hearing and balance problems — believe it or not. Bizarrely, referred pain from sick sternocleidomastoid muscles is well known to massage therapists to interfere with hearing, balance, and to cause tinnitus (ringing) either directly or indirectly via effects on the muscles of the jaw.

Numbness and pain in the entire arm can be caused by a condition called thoracic outlet syndrome (TOS), in which nerves and blood vessels that supply the arm are impinged by two specific muscles — two muscles that also happen to be the most abused respiratory muscles — pectoralis minor and scalenes!

That's the tip of the iceberg, but it gives you a good sense of the complex interconnections and the potential for totally exhausted chest and neck muscles to wreak havoc on your upper body. Most people are suffering from these and many other consequences of inefficient respiration by — no kidding — about age twenty. In other words, most of the people who walk into my office are suffering from problems which can be traced, at least in part, to respiratory dysfunction.

Some clarifications

To really understand this phenomenon, it's necessary to go just a bit deeper into respiratory mechanics. This section is for the devoted reader who wants to understand exactly what's going on when breathing goes wrong. It's easy enough to understand that breathing without the benefit of diaphragmatic contraction is probably not such a good thing. But why, exactly, is it such a problem?

Exhalation without the aid of the diaphragm is no big deal — even strong exhalation. The diaphragm simply isn't used for that in the first place. The muscles that pull the rib cage down are aided by gravity: those ribs are heavy, and it doesn't take much to pull them down a little more. And the muscles we use for the job are quite large and strong: the abdominals and the quadratus lumborum are large, each of them bigger than any of the muscles used to assist inhalation. And, furthermore, we don't really have any psychological inhibitions against contracting our abdominal muscles — it's the relaxed belly we shy away from.

It's inhaling without the diaphragm that is so difficult. Without the diaphragm, inhalation is extremely hard work: somehow or other, you're going to have to get that rib cage lifted up against the pull of gravity, and against the pull of abdominal and back muscles that don't like to relax.

Exhaling without the diaphragm is no big deal — it's inhaling without the diaphragm that's a challenge!

The only muscles that are really designed for serious rib-lifting are the intercostals, and they can only do so much. So people end up recruiting the pectoralis minors, sternocleidomastoids, and (worst of all) the scalenes. And even that is not, in itself, necessarily a bad thing: the trouble is when you do it all the time, for ordinary breathing. Imagine a handful of muscles the size of pencils trying to lift your rib cage several times per minute.

All day long, every day. For years. That is the ultimate and specific problem with not using your diaphragm.

So now what?

The solutions

Once you're a believer, the solution is (drum roll please) … diaphragm exercise!

To stop breathing with your chest and throat muscles, you must learn how to breathe with your diaphragm. Absolutely, positively the only way to do this is to practice using your diaphragm — but there are many ways to practice.

This is a challenge! The diaphragm is s a muscle you can't see and can't feel directly. It's like trying learn how to wiggle your ears. A lifetime of bad habits may stand in your way. Above all, you will doubt that it is really necessary — it's an awful lot of trouble for a muscle you barely knew you had. But it's the only way.

Take heart, though: it's no different than what any singer or martial artist has to learn. It's difficult, but hardly impossible. The following sections offer several useful perspectives and strategies that can serve you well.

Solution Idea No. 1: Strength is coordination

When people start a weight training program, it takes weeks for the first obvious increases in strength to manifest. These initial gains are not due to an increase in muscle mass — that comes later. The increase comes from coordination alone. Specifically, a beginner learns to "recruit" more muscle fibres. Every muscle consists of millions or billions of microscopic fibres. Individual nerves stimulate clusters of fibres — together, nerve and fibres are called a "motor unit." The more motor units you can stimulate at once, the stronger a contraction you can generate.

Recruiting a lot of motor units at once takes coordination, a physical skill that can only be learned with practice, practice, practice. It's true for your biceps, and it's true for your diaphragm. Learning to breathe with your diaphragm is mainly about learning how to recruit more of its motor units every time you inhale.

It's important to understand this, so that you realize that your goal is not exactly a big, beefy diaphragm, but simply a well-coordinated one — you just want to learn how to use what you've got. But how do you do that when you can't even feel it?

Solution Idea No. 2: Book lifting

Learning to breathe diaphragmatically really is like learning to raise one eyebrow or wiggle your ears — only worse, because it's difficult to even tell when you've succeeded. To learn to use your diaphragm, you have to make the results visible. Here's how:

Find yourself a good, heavy book. This book should pass the "thunk test" — it should make a good, satisfying thunk when you drop it on the ground.

Lie down on your back with your knees up.

Place the book square on your belly.

Take a deep breath.

If the whole book lifts up, you used your diaphragm. If it didn't lift up, or lifts unevenly, you didn't use your diaphragm. Laws of hydraulics. It is absolutely impossible to contract your diaphragm without your belly sticking out.

So there you go: visual feedback is how you're going to learn when you are actually contracting your diaphragm. Now, do that at least twenty times in a row, and your diaphragm isn't strong unless you can lift it at least two inches every time. Four inches would be better.

Solution Idea No. 3: Water breathing

Standing up to your chin in a swimming pool is an even more ingenious way of providing resistance to diaphragmatic contraction. Can you see why?

Strength training is usually called "resistance" training by professionals, because "weight" training is too narrow a term. Weights such as barbells and stacks of iron plates in a machine are only one way of providing resistance to muscle contraction. It is also possible to use big elastic bands, springs, body weight, other muscles, and even just stationary objects. And water …

The physics of water breathing

Water pressure resists expansion of the abdomen uniformly on all sides — and therefore it resists diaphragm contraction.

Water pressure is strong: even just a couple of feet under water, the pressure on one square foot of abdominal surface is an amazing 150 pounds! Every square inch of your torso has about a pound of pressure on it — less closer to your chest, and more closer to your waist. That's a lot of resistance to abdominal expansion! And it's perfectly uniform.

Anything that resists abdominal expansion is resisting diaphragmatic contraction, of course. Lifting the book, as described above, obviously resists abdominal expansion: but not much, and only in one direction, and somewhat awkwardly. That exercise is really intended for the visual feedback, not the resistance.

Breathing while standing in water, however, requires the diaphragm to overcome a strong, unrelenting water pressure evenly distributed over the abdomen. It's like wearing a broad, elastic garter belt. It's a phenomenal strength (resistance) training exercise for the diaphragm.

In fact, it's so difficult that most beginners will hardly be able to budge their diaphragm, and will — uh oh! — end up trying to lift their rib cage instead. It's actually a great way to demonstrate how the chest and neck muscles tend to get recruited when the diaphragm isn't being used, or can't be used — you can really feel all those secondary muscles kicking in and trying to take over!

So do experiment with this in the early stages, just so you can see what it feels like, but don't try to use it as a strength-building exercise until you've mastered book lifting. Happy water breathing!

Solution Idea No. 4: Bioenergetic or round breathing

Everyone's emotionally constipated, unless you're a sociopath or still in diapers — the price of maturity is that you repress much of your Genuine Self, and end up with a comfort zone that is often suffocating, respiratory dysfunction, and upper body pain. Ain't life grand?

So, shallow breath and emotional constipation usually go together, and they can only be fixed together. Oddly enough, the best cure for shallow breathing is … deeper breathing. Gee, this is rocket science, isn't it?

Solution Idea No. 5: The abdominal lift

This exercise is straightforward and is vital for mastering many breathing techniques. It has several benefits: in addition to strongly stimulating diaphragmatic breathing, this ancient yoga exercise will also …

- exercise your "other" abdominal muscles (the obliques and transversus abdominis, which are often neglected even by professional trainers, even though they are very much relevant to core stability and belly shape).
- wake you up in the morning by mobilizing the significant amount of blood that is "stored" in your viscera over night.
- strengthen your pelvic floor muscles, touted as an essential component of sexual prowess for women and men! Also helpful in the prevention of bowel and urinary dysfunction. And a host of other minor benefits. This exercise still offers more bang-for-buck than any other single exercise. It's what I call a "lifer," best done once a day for the rest of your life.

Here are the instructions:

Stand with your upper body supported on your knees.

Take at least three deep breaths to prepare yourself.

When you feel you have oxygenated sufficiently, blow all of your air out, hold your breath, and then suck your belly in hard against your spine. Particularly focus on your low belly, below the navel.

Hold the position and your breath for several seconds (go as long as you can).

Relax the belly before breathing again (if you try to breath first and then relax, it can hurt a bit).

Repeat at least three times, or until you are exhausted.

Solution Idea No. 6: Stay out of chairs

My father always told me to stay out of bars, advice which has served me well, I think. I wish he had also told me to stay out of chairs — they are just as corrupting and dangerous!

This is not easy advice to follow, of course, but it is good advice nevertheless. Chronic sitting is an obvious mechanical barrier to diaphragmatic breathing: the belly is compressed, and cannot expand as easily or as far.

And, of course, if you're in a chair … you're not getting any exercise, are you? Says the massage therapist who has spent thousands of hours in a chair creating this website. Do as I say, not as I do! Seriously, if you can't avoid working in chairs, do everything you can to mitigate the harmful effects. Above all, take micro-breaks!

Solution Idea No. 7: Stress reduction

"Reduce your stress level" is the most vague and unhelpful

advice I can imagine! It's a huge topic, and I can't properly address it here. For the purposes of this article, I just want to instill in you some respect for the consequences of stress: if you are so stressed out that you can't breathe properly, what else is stress doing to you? It's time to take a serious look solving some problems in your life, and/or changing the way you react to challenges — for the sake of your health.

We are a society of shallow breather

Quick conclusion

We are a society of shallow breathers: afraid of moving our bellies, afraid of expressing ourselves, living our lives in chairs, and stressed out by our busy minds. Instead of breathing with the diaphragm, people tend to breathe with upper body musculature that is inadequate to the task, with a cascade of musculoskeletal consequences and vulnerabilities. These are the solutions to dysfunctional breathing:

- the book lifting exercise, to learn how to recruit the diaphragm
- water breathing, to increase your diaphragmatic strength and coordination
- the abdominal lift exercise, to strengthen and stimulate the abdominal musculature and increase your body awareness of abdominal movement during breathing
- stay out of chairs as much as possible, so that your diaphragm actually has room to do its job
- embark on a program of stress reduction, whatever that means to you

http://saveyourself.ca/articles/respiration-connection.php

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GARLIC HELPS LOWER HIGH BLOOD PRESSURE

Garlic supplements may lower blood pressure just as effectively as some drugs used to treat hypertension can, according to a new research review.

"Supplementation with garlic preparations may provide an acceptable alternative or complementary treatment option for hypertension," Dr Karin Ried and colleagues from The University of Adelaide in South Australia write.

Research to date on garlic and blood pressure has had "inconclusive" results, they note, while the last meta-analysis - in which the results of several studies are analyzed collectively - only included studies done up until 1994.

To provide an updated perspective, Ried and her team included more recently published studies in their analysis, identifying 11 studies in which the patients were randomly assigned to garlic or placebo. In most studies, participants given garlic took it in powdered form, as a standardized supplement. Doses ranged from 600 mg to 900 mg daily, which study participants took for 12 to 23 weeks.

When the researchers pooled the data from the trials, they found that garlic reduced systolic blood pressure (the top number in a blood pressure reading) by 4.6 mm Hg, on average. An analysis limited to people with high blood pressure showed garlic reduced systolic blood pressure by 8.4 mm Hg, on average, and diastolic blood pressure (the bottom number) by 7.3 mm Hg. The higher a person's blood pressure -was at the beginning of the study, the more it was reduced by taking garlic.

The effects were similar to those of widely used drugs for treating hypertension, for example beta blockers, which reduce systolic blood pressure by 5 mm Hg, and ACE inhibitors, which produce an 8 mm Hg average drop in systolic blood pressure, the researchers note.

The 600 mg to 900 mg dosage used in the studies is equivalent to 3.6 mg to 5.4 mg of garlic's active ingredient, allicin, Ried and her team point out. A fresh clove of garlic contains 5 mg to 9 mg of allicin.

In the population as a whole, they note, reducing systolic blood pressure by an average of 4 to 5 points and diastolic blood pressure by 2 to 3 points could cut the risk of heart disease and heart disease-related death by up to 20%.

More research is needed to determine whether garlic supplementation might have a long-term effect on heart disease risk, the researchers conclude.

http://tinyurl.com/5o7688

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FRUIT JUICE 'COULD AFFECT DRUGS'

Drinking fruit juices may not be as healthy an option as thought - they could reduce the effectiveness of some medicines, it is being claimed. Research presented at a US conference suggested a chemical in grapefruit juice could stop anti-allergy drugs being absorbed properly.

A University of Western Ontario team said oranges, and possibly apples, had similar ingredients. Grapefruit juice is already known to interfere with blood pressure drugs. "This is just the tip of the iceberg - I'm sure we'll find more and more drugs that are affected this way," said Dr David Bailey, of the University of Western Ontario.

Some medications carry a warning that taking them alongside grapefruit juice could cause an overdose. However, the latest finding, presented at the American Chemical Society conference in Philadelphia, points to a different problem with researchers saying it was "the tip of the iceberg".

In this case, he found that the grapefruit juice had the reverse effect on fexofenadine, an antihistamine drug, making it less potent rather than more potent. Volunteers took the drug with either a single glass of grapefruit juice, or just water. When it was taken with juice, only half the drug was absorbed, potentially reducing its effectiveness.

Orange warning

Researchers believe that an active ingredient of the juice, naringin, appears to block a mechanism which moves drug molecules out of the small intestine into the bloodstream. Study author Dr David Bailey said that orange and apple juices appeared to contain naringin-like substances which might have a similar effect.

"Recently, we discovered that grapefruit and these other fruit juices substantially decrease the oral absorption of certain drugs undergoing intestinal uptake transport. The concern is loss of benefit of medications essential for the treatment of

serious medical conditions."

So far, the three types of juice have been found to affect etoposide, a chemotherapy drug, some beta-blocker drugs used to treat high blood pressure, and cyclosporine, taken by transplant patients to prevent rejection of their new organs.

However, Dr Bailey said: "This is just the tip of the iceberg - I'm sure we'll find more and more drugs that are affected this way."

Colette McCready, from the National Pharmacy Association, said: "The effect of grapefruit juice on some medicines is well established and where this applies it is clearly detailed in Patient Information Leaflets. "Pharmacists will usually draw this matter to patients' attention when dispensing their medicines. This new research showing that apple and orange juice may enhance or reduce the effects of some medicines is interesting but it is only one study.

"Usually further research is needed to establish that these interactions are significant."

Professor James Ritter, a clinical pharmacologist at King's College London, said: "The observation is very interesting. It will need more work to establish how important such interactions are in clinical practice and for what drugs and juices." news.bbc.co.uk/go/pr/fr/-/1/hi/health/7572500.stm

EXPERIMENTAL DRUG SHOWS EARLY PROMISE AGAINST CYSTIC FIBROSIS

The molecule causes flawed gene to work properly, researchers say

An experimental drug that blocks the genetic flaw responsible for one form of cystic fibrosis has worked well in an early trial, Israeli researchers report.

"The results have been promising," said Dr. Eitan Kerem, head of pediatrics at Hadassah Hebrew University Hospital in Jerusalem, and lead author of a report published online Thursday in the journal The Lancet.

Cystic fibrosis is an inherited chronic disease caused by a flaw in genes for the channels that allow salt to enter cells, causing the body to produce unusually thick, sticky mucus. It affects the lungs and digestive system of about 30,000 children and adults in the United States and 70,000 people worldwide. Intensive treatment can prolong the lives of patients into their 30s, 40s and beyond, according to the Cystic Fibrosis Foundation.

The people treated in the Israeli trial have cystic fibrosis because of a mutation in the salt channel gene that produces a false "stop" signal, so that the proteins forming the channel aren't produced. The new drug was developed by a New Jersey biotechnology company, PTC Therapeutics, through a screening program that singled out molecules that block such genetic "stop" signals.

The form of cystic fibrosis in the Israeli trial is responsible for about half of all cystic fibrosis cases in that country and for about 10 percent of cases in the United States, where other genetic flaws in channel production predominate.

In the study, 23 Israelis with cystic fibrosis received the drug, designated PCT124, in two cycles -- three doses a day for 14 days, then three higher daily doses after a 14-day pause.

Salt flow reached the normal range in 13 of the 23 cases during the first treatment cycle and in nine of 21 cases in the second cycle. Kerem said the results were "encouraging," but added, "This was a short-term study without placebo, so it shows that a longer-term trial should be done." A longer trial probably will be started "early next year," Kerem said.

"We are planning a larger long-term study that will be international, in the United States and Canada and also in Europe," said Dr. Langdon Miller, chief medical officer of PTC Therapeutics, which sponsored the study.

PTC124 was developed through a screening program in which hundreds of thousands of compounds, with a light-emitting molecule called luciferase, were aimed at the genetically altered "stop" signal, Miller said. Several compounds hit the target and lit up. "We selected the best ones, did chemistry to modify them and came up with PTC124," he said.

Several initial studies, including the one in Israel, have shown that PCT124 can help produce salt-conducting channels that are "full length and fully functioning," Miller said.

The drug may be useful for a number of other genetic disorders caused by the same sort of mutation, he added. It already is being tried for another genetic condition, Duchenne muscular dystrophy.

http://www.nlm.nih.gov/medlineplus/news/fullstory_68376.html (*this news item will not be available after 11/19/2008)

BP DRUGS MIGHT ACTUALLY CAUSE DIABETES

In what could be termed as a significant discovery, a recent research in UK has revealed that some of the most popular drugs prescribed for hypertension may actually increase the risk of diabetes.

The class of medicines in question here are called beta blockers. These include drugs called atenolol, inderal and metaprolol. It has been found that these drugs can increase the level of blood sugar in patients suffering from diabetes. In other cases it can become a reason for the onset of the disease among patients of high BP. The latest research carried out at National Heart and Lung Institute, Imperial College, London, actually puts forward the baseline predictors of new-onset diabetes in hypertensive patients.

The study took into account 19,257 hypertensive patients. These people were randomly assigned to receive one of the two anti hypertensive regimens using beta blockers. It was found that 14,120 were at the risk of developing diabetes at baseline. On the other hand, 1,366 people, i.e. 9.7% subsequently developed NOD during median follow-up of 5.5 years.

Beta blockers are basically used for managing cardiacdefine arrhythmias and cardio protection after a heart attack. They were used extensively for the treatment of hypertension. But somehow their role was downgraded in June 2006 in the United Kingdom because they showed an

unacceptable risk of provoking type 2 diabetesdefine. But later on December 18, 2007 the beta blocker named Bystolic was approved by the FDA for the treatment of hypertension.

Dr. Anoop Mishra, director and head (diabetes and metabolic diseases) Fortis Hospitals said, “In patients with hypertension, beta blocker drugs are no longer frontline therapy. These drugs may not only increase blood sugar levels in those who don't have diabetes, but may worsen sugar control in those with diabetes and also blunt warning symptoms when low sugar occurs."

But Dr. Mishra didn’t deny the fact that these drugs are still useful for patients suffering from diabetes and hypertension with associated heart disease. He added that the latest beta blockers may have some advantages over the previous generation drugs.

As of now, the doctors have started reducing the use of beta blockers among patients affected by diabetes and high blood pressure.

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INNATE IMMUNE SYSTEM TARGETS ASTHMA-LINKED FUNGUS FOR DESTRUCTION

A new study shows that the innate immune system of humans is capable of killing a fungus linked to airway inflammation, chronic rhinosinusitis and bronchial asthma. Researchers at Mayo Clinic and the Virginia Bioinformatics Institute (VBI) have revealed that eosinophils, a particular type of white blood cell, exert a strong immune response against the environmental fungus Alternaria alternata. The groundbreaking findings, which shed light on some of the early events involved in the recognition of A. alternata by the human immune system, were published recently in the Journal of Immunology.

Eosinophils typically combat parasitic invaders of the human body larger than bacteria or viruses, such as flukes or parasitic worms (collectively known as helminths). Evidence from different experimental approaches suggests that asthma and chronic sinusitis can arise when the body perceives that it has encountered a disease-causing organism. Environmental fungi such as Alternaria do not typically cause invasive infections like parasites but for some reason, in certain people, the body responds as if it is being attacked and Principal Investigator Hirohito Kita, M.D., from Mayo Clinic, remarked: "Our results strongly demonstrate that eosinophils have the capacity to recognize and exert immunological responses to certain fungi such as Alternaria. We have shown that CD11b receptors on the surface of eosinophils recognize and adhere to beta-glucan, a major cell wall component of the fungus. This in turn sets in motion the release of toxic granule proteins by the white blood cells, leading to extensive damage and ultimate destruction of the fungus. To the best of our knowledge, this is the first time that live eosinophils and not just the intracellular components have been shown to target and destroy a fungus."

The researchers used fluorescence microscopy to determine the outcome of the interaction between eosinophils and A. alternata. The contact of fungus with eosinophils resulted in bright red fluorescence due to the damaged fungal cell wall and subsequent death of Alternaria. Immunohistochemistry confirmed the release of toxic granular proteins by eosinophils due to contact with the fungus.

Dr. Chris Lawrence, Associate Professor at VBI and the Department of Biological Sciences at Virginia Tech, remarked: "T helper 2 (Th2) cells in the immune system typically produce cytokine signaling molecules or interleukins that lead to the recruitment of eosinophils for the dysregulated immune response commonly associated with airway inflammatory disorders. Continual exposure of sensitized individuals to common environmental fungi like Alternaria may result in Th2 cells being constantly activated to recruit eosinophils and this sustained defense mechanism results in chronic inflammation. It has been shown previously that degranulation of eosinophils causes damage of airway mucosa and enhances inflammation. The next step in our transdisciplinary research collaboration will be to use recombinant fungal proteins and fungal knockout mutants for specific genes to dissect the different molecular steps involved in the development and progression of this acute immune response."

Hirohito Kita added: "We have taken an important step in showing that the innate immune system of eosinophils is capable of targeting an asthma-associated fungus for destruction. The biological significance of these results will need to be verified further in animal models and in humans and our collaborative efforts with Dr. Lawrence's research group for proteomics and functional genomics will be invaluable in this respect. We suspect that the dysregulated immune responses to Alternaria, other filamentous fungi, and perhaps chitin-encased insects, such as mites and cockroaches, may play a pivotal role in chronic inflammation and the subsequent development of bronchial airway disease."

http://tinyurl.com/64d8kb

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A FUNGUS THAT'S ACTUALLY GOOD FOR YOU?

White button mushrooms are boring? Au contraire, mon frere. This champion of the champignon is the most widely cultivated (and least expensive) mushroom at the market. The button mushrooms you bought at the store aren’t just a pretty kabob filler. With a dash of this, a smidgen of that, voila! Bon appetit!

Those beauties may have talent, too. Like the ability to rev up the body’s self-defenses against things like cancer and viral infections.

Tumor Terminators

Western medicine has only recently begun to study the concept, but early animal research suggests fungi may have some pretty serious health-promoting powers. For example, powdered white button mushrooms recently boosted production of natural killer cells in mice. If the same thing happens in humans, that’s great news, because killer cells help defend against tumors and virus-infected cells.

Source: Real Age

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WOMEN TYPICALLY GET HEART DISEASE MUCH LATER THAN MEN

But not if they smoke, researchers say

In fact, women who smoke have heart attacks more than a dozen years earlier than women who don't smoke, Norwegian doctors reported in a study presented to the European Society of Cardiology. For men, the gap is not so dramatic; male smokers have heart attacks about six years earlier than men who don't smoke.

"This is not a minor difference," said Dr. Silvia Priori, a cardiologist at the Scientific Institute in Pavia, Italy. "Women need to realize they are losing much more than men when they smoke," she said. Priori was not connected to the research.

Dr. Morten Grundtvig and colleagues from the Innlandet Hospital Trust in Lillehammer, Norway, based their study on data from 1,784 patients admitted for a first heart attack at a hospital in Lillehammer.

Their study found that the men on average had their first heart attack at age 72 if they didn't smoke, and at 64 if they did. Women in the study had their first heart attack at age 81 if they didn't smoke, and at age 66 if they did.

That works out to eight and 15 years, respectively, for men and women. After adjusting for other heart risk factors like blood pressure, cholesterol and diabetes, researchers found that the difference for men was about six years for women about 14 years.

Previous studies looking at a possible gender difference have been inconclusive.

Doctors have long suspected that female hormones protect women against heart disease. Estrogen is thought to raise the levels of good cholesterol as well as enabling blood vessel walls to relax more easily, thus lowering the chances of a blockage.

Grundtvig said that smoking might make women go through menopause earlier, leaving them less protected against a heart attack. With rising rates of smoking in women _ compared with falling rates in men _ Grundtvig said that doctors expect to see increased heart disease in women.

"Smoking might erase the natural advantage that women have," said Dr. Robert Harrington, a professor of medicine at Duke University and spokesman for the American College of Cardiology.

Doctors aren't yet sure if other cardiac risk factors like cholesterol and obesity also affect women differently.

"The difference in how smoking affects women and men is profound," Harrington said. "Unless women don't smoke or quit, they risk ending up with the same terrible diseases as men, only at a much earlier age."

http://tinyurl.com/6js2qd

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TIRED OF THE SAME OLD, SAME OLD CHICKEN? NOT TONIGHT!

GRILLED CHICKEN BRUCHETTA

1/4 cup KRAFT Sun-Dried Tomato Dressing, divided
4 small, boneless and skinless chicken breast halves (1 lb.)
1 medium tomato, finely chopped
1/2 cup KRAFT Shredded Low-Moisture Part-Skim Mozzarella Cheese
1/4 cup chopped fresh basil or 1 tsp. dried basil leaves

PLACE large sheet of heavy-duty foil over half of grill grate; preheat grill to medium heat. Pour 2 Tbsp of the dressing over chicken in resealable plastic bag; seal bag. Turn bag over several times to evenly coat chicken with the dressing. Refrigerate 10 min. to marinate.

REMOVE chicken from marinade; discard bag and marinade. Grill chicken on uncovered side of grill 6 min. Meanwhile, combine tomatoes, cheese, basil and remaining 2 Tbsp dressing.

TURN chicken over; place, cooked side up, on foil side of grill. Top evenly with tomato mixture. Close lid. Grill an additional 8 min. or until chicken is cooked through (165˚F).

SUGGESTION: Serve with steamed or grilled vegetables and noodles.

Great recipe and easy to make. I cooked a box of frozen chopped spinach and added it to the tomato mix. You can also used mild salsa instead of tomatoes. You do need the listed salad dressing. Really yummy. JJ

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YOU SAY TOMATO, I SAY LET'S EAT!

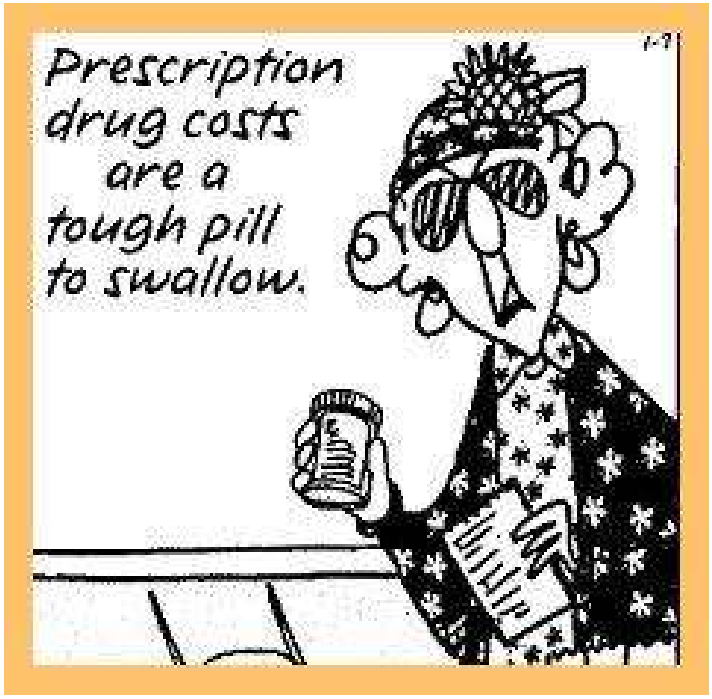
Nothing says summer like red vine-ripened tomatoes. But did you know tomatoes have significant health benefits? They are low in calories, have no sugar or cholesterol, and are very high in vitamins A and C. And possibly most important, tomatoes contain lycopene, a powerful antioxidant that may aid in the prevention of cancer and heart disease. So go ahead and eat those tomatoes.

CREAM OF FRESH TOMATO SOUP

2 cups ripe tomatoes, chopped
1 medium onion, chopped
1/2 bay leaf 1/2 tsp. salt
1/8 tsp. pepper
2 tbsp. butter
2 tbsp. flour
2 cups milk
Fresh chopped basil

Combine first 5 ingredients in a saucepan; simmer for 10 to 20 minutes. Strain mixture. In a saucepan, melt butter. Blend in flour, stirring constantly. Cook 1 minute. Stir in milk and cook until thickened. Slowly stir in hot tomato mixture. Heat thoroughly. Sprinkle with basil.

FarmersAlmanac.com



Information in this newsletter is for educational purposes only. Always consult with your doctor first about your specific condition, treatment options and other health concerns you may have.

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