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Example **DISCUSSION** of **History Results** for client Jane Starbright

Discussion

This client presents with chronic obstructive lung disease, diagnosed as bronchiectasis, and demonstrates crackling and gurgling sounds upon auscultation with episodes of heart palpitations (check also for tachycardia), with chest expansion still at 4cm. There appears to have been some lean weight loss as client demonstrates a lean body mass deficit of about 3kg, which in chronic respiratory disease is “thought to be due to a shift from aerobic to glycolytic metabolism in systemic muscle tissue, secondary to impaired oxygen and carbon dioxide exchange and high level free radical production.” Progression of chronic pulmonary inflammation can cause a rise in pulmonary vascular pressure and fibrosis causing changes in the hearts right atrium and right ventricle with consequent disturbances in the myocardial conducting system in turn triggering episodic supraventricular cardiac arrhythmias. **Monitor through MD.** As this client presents with both upper and lower respiratory disease that is both chronic and acute, the likelihood of uncontrolled inflammation and immune dysfunction is prominent and as such the use of diet, nutrients and phytonutrients to modulate these disease processes will be discussed later.

This client has a history (and family history) of **allergy**. Also atopy, a form of allergy in which a hypersensitivity reaction such as dermatitis or asthma may occur in a part of the body not in contact with the allergen, may also be active in this client and has a genetic component. Allergy is one of four forms of hypersensitivity and is called type I (or immediate) hypersensitivity. It is characterized by excessive activation of mast cells and basophils by the IgE antibody, resulting in an inflammatory response that can range from mild to extreme and life threatening (anaphylaxis). Allergen containing substances that trigger a reaction in this client include mould, dust mites, prawns, smoke and penicillin and probable other as yet unidentified foods and particles. As a child the client has had eczema and as an adult has what appears to be rhinitis and has been diagnosed lately with adult onset asthma although a second opinion from a different MD disputes this or at least suggests that night time symptomatic episodes are not asthma.

Eczema is characterised by inflammation of the epidermis and comes in many forms with as

many causes and manifests as redness, skin edema (swelling), itching and dryness, crusting, flaking, blistering, cracking, oozing, or bleeding and is often found on the flexor aspect of joints. Eczema can be initiated by an allergen (Atopic eczema also called atopic dermatitis) or an irritant (as in Contact dermatitis). Other less common forms of eczema exist. Atopic eczema can have an hereditary component and although this client's Mother does not seem to have any allergies or asthma, her sisters do while her father has a strong reaction (apparently allergy based) to morphine. Some substances are known to act as both an allergen and irritant (wet cement, for example). Other substances cause a problem after sunlight exposure, bringing on phototoxic dermatitis. Eczema has now stabilised in this client and this may in part be due to the corticosteroids that have been and are being used presently for the respiratory conditions.

Note however that "an underlying chronic allergic process commonly increases susceptibility to intercurrent microbial infection, particularly viral agents." Thus, as will be seen again and again it is important, especially in the presence of chronic bronchiectasis, to find, reduce and if possible eliminate the offending allergens, be they airborne, on surfaces or in food. MSQ scores, high in eyes, nose and throat, further indicate either an allergy reaction or an immune reaction to low level persistent infection (from lungs) with inability to clear due to overall low Ig levels (Lab results) with possible genetic immune deficiency a possible cause. (discussed later) In addition to the lower respiratory tract condition (Bronchiectasis), this client has concurrent acute and/or chronic upper respiratory conditions – possibly rhinitis (chronic and could be either allergic and/or non-allergic rhinitis) and sinusitis (periodically acute). Rhinitis is an inflammation of the nasal mucosa and as seen in this client is accompanied by runny nose and post nasal drip and some congestion. Colds and allergies are the most common causes of rhinitis.

Symptoms of rhinitis also include sneezing and stuffiness. Acute rhinitis commonly results from viral infections but may also be a result of allergies or other causes like emotion. Chronic rhinitis may be accompanied by acute bouts of sinusitis – as seen in this client - and/or chronic sinusitis (chronic rhinosinusitis).

REF: <http://www.merck.com/mmhe/sec19/ch221/ch221g.html>

Allergic rhinitis is caused by a reaction of the body's immune system to an environmental trigger. The most common environmental triggers include dust, molds, pollens, grasses, trees, and animals. Symptoms include itching, sneezing, runny nose, stuffiness, and itchy, watery eyes. It is roughly estimated that one in three people have an active allergy at any given time and at least three in four people develop an allergic reaction at least once in their lives. Allergic rhinitis can be 1/ Seasonal (commonly called hay fever) and occurs particularly during pollen seasons and does not usually develop until after 6 years of age and 2/ Perennial, which occurs throughout the year. This type of allergic rhinitis is commonly seen in younger children."

REF: <http://en.wikipedia.org/wiki/Rhinitis>

In Australia allergic rhinitis prevalence is lower in coastal regions (21%) than in dry inland areas (31%) while about 40% - 50% of rhinitis patients have NON allergic rhinitis. [REF: IMVS Newsletter Number 39 Spring 2000](#). Thus management of precipitating factors in BOTH allergic and nonallergic rhinitis can lead to marked improvement of symptoms and sometimes eliminate the need for long term medication (as seen in this client). Also important to check for deviated nasal septum and/or polyps (recheck in this client) and use Skin testing and RAST IgE blood tests. (more to order in this client)

The following important abstract from the The Medical Journal of Australia highlights the link between upper and lower airway disease and rhinitis and asthma [REF: Janet Rimmer and John W Ruhno, MJA 2006; 185 \(10\): 565-571](#).

“The united airway disease (UAD) hypothesis proposes that upper and lower airway disease are both manifestations of a single inflammatory process within the respiratory tract. United airway disease is characterised by inflammation of the respiratory tract, in which asthma and rhinitis are the upper and lower respiratory tract manifestations, respectively, of the same disease process. Irrespective of cause, the upper and lower respiratory tract manifestations are characterised by a **systemic inflammatory response**.

Patients with rhinitis or asthma should always be assessed for coexistent disease in the reciprocal area.

Treatment of upper airway disease can modify the severity of lower airway disease and vice versa.

The potential for early treatment of allergic rhinitis to prevent progression to asthma merits further study.”

The table below that summarises the different inflammatory changes in upper and lower respiratory tissue as a result of an allergic stimulus. [REF: Janet Rimmer and John W Ruhno, MJA 2006; 185 \(10\): 565-571](#).

2 Inflammatory changes in the upper and lower airways in response to an allergic stimulus

	Nose	Bronchi
Eosinophils	Increased	Increased
Mast cells	Increased	Increased
Basophils	Present	Absent
T _H 2 lymphocytes	Increased	Increased
Inflammation intensity	Less	More
Vasculature	Large increase	Smaller increase
Basement membrane thickening	No	Yes
Epithelial disruption	No	Yes
Smooth muscle increase	No	Yes
Early and late allergic responses	Yes	Yes
Increase in AHR after exposure to allergen	Yes	Yes

AHR = airway hyper-responsiveness.

“Allergic disease is the result of an immune response to external antigens, which leads to production of antibodies that are typically, **but not exclusively, IgE antibodies**. Allergic airway disease results from hypersensitivity or IgE-mediated reactions when inhaled allergen reacts with cells bearing IgE antibodies (typically mast cells and basophils). Cross-linking of allergen-specific IgE molecules bound to cells by allergen particles results in the release of granule-associated mediators (eg, histamine, tryptase), membrane lipid-derived mediators (eg, leukotrienes) and cytokines. The initial reaction, the early allergic response (maximal at 10–20 minutes), is usually followed by a late allergic response (within 2–6 hours) in both the upper and lower airways. The later response is associated with an eosinophilic and T cell (CD4+) tissue infiltrate.”

“Although the nose is usually the first site of exposure to allergens or other noxious substances, the presence of nasal epithelial damage is minimal, **whereas, in the bronchi, marked epithelial disruption may be present.** ([important in this client](#)) Thus, it is postulated that the nasal mucosa has developed protective mechanisms that minimise remodelling and

enhance epithelial regeneration. In support of this hypothesis, animal models of airway inflammation show that, although the majority of allergen is deposited in the nose, more inflammation occurs in the lower airway. Basement membrane thickening, a consistent hallmark of lower airway remodelling in asthma, is present even in children with asthma, and also in atopic patients without asthma and patients with allergic rhinitis; however, it has not been reproducibly demonstrated to occur in the nose.

Inflammatory changes can be detected in both the upper and lower airways without accompanying clinical symptoms. In patients with rhinitis, lower airway inflammation can be demonstrated by increased levels of eNO and by eosinophils found in induced sputum, bronchoalveolar lavage fluid and bronchial biopsies. Similarly, in patients with asthma, nasal biopsies show eosinophilic inflammation, even in those who do not have symptoms of rhinitis. After direct allergen challenge in one part of the airway, an inflammatory response can be shown in both the target area and the reciprocal part of the airway within 24–48 hours of the challenge.”

Thus the **important** take home message from this paper is - find ALL possible allergens and irritants that may affect the upper airways and eliminate them as much as possible. Not only will rhinitis improve but more importantly symptoms of lower airway disease like **bronchiectasis** may ASLO improve.

Food antigens can also trigger both allergic rhinitis and sinusitis. Some cross-reactivity may occur. For example, someone allergic to birch pollen may also find that they have an allergic reaction to the skin of apples or potatoes and this occurs because of similarities in the proteins of the pollen and the food. There are many cross-reacting substances. Other comorbidities with allergies include eczema, asthma, depression and migraine, some of which are seen in this client.

Some of the investigations that have been published recently show that not previously considered foods can also cause allergy REF: <http://www.allergenbureau.net> The following abstract highlights recent findings that lentils (along with rice, eggplant and maize) can indeed cause an allergy reaction.

“Legume allergy, mainly to lentils and chickpeas, is the fifth most common cause of food allergy in Spanish children. Martínez San Ireneo and his colleagues conducted a study on 54 children with clinical allergy to legumes to further investigate these allergies. They found that allergy to lentil was the most frequently diagnosed legume allergy (43 children [80%]), followed by allergy to chickpea (32 children [59%]). The majority of children (69%) had allergy to more than one legume. The most frequent symptoms of allergy were **respiratory (rhinitis and/or asthma)** and skin reactions.

The researchers further investigated cross-reactivity among lentils, chickpeas, peas, white beans and peanuts. This was evaluated by ELISA inhibition experiments and oral food challenges. The oral legume challenges demonstrated that the most frequent associations were allergy to lentils and chickpeas (57%), allergy to lentils and peas (54%) and allergy to

lentils, chickpeas and peas (43%). The authors therefore concluded that there is a high degree of cross-reactivity among lentils, chickpeas and peas in Spanish children.” Even though these trials are small in sample number, they do highlight the theme of cross reactivity and previously unsuspected food allergies. *References: Martínez San Ireneo et al 2008. Clinical features of legume allergy in children from a Mediterranean area. Annals of Allergy, Asthma and Immunology Vol 101(2) pp. 179-84. AND Martínez San Ireneo et al 2008. In vitro and in vivo Cross-Reactivity Studies of Legume Allergy in a Mediterranean Population. International Archives of Allergy and Immunology. Vol147 pp. 222-230.*

The abstract below from the following article begins to draw attention to cross-reactivity between birch pollen and some foods.

Current understanding of cross-reactivity of food allergens and pollen. Vieths S, Scheurer S, Ballmer-Weber B, Paul-Ehrlich-Institut, Department of Allergology, Paul-Ehrlich-Str. 51-59, D-63225 Langen, Germany. Viest@pei.de, Ann N Y Acad Sci. 2002 May;964:47-68.

“Pollen-allergic patients frequently present allergic symptoms after ingestion of several kinds of plant-derived foods. The majority of these reactions is caused by four distinct cross-reactive structures that are present in birch pollen. Proteins that share common epitopes with Bet v 1, the major birch pollen allergen, occur in pollens of several tree species: **apples, stone fruits, celery, carrot, nuts, and soybeans**. Approximately 70% of our patients who are allergic to birch pollen may experience symptoms after consumption of foods from these groups. In contrast, two minor allergenic structures—profilins and cross-reactive carbohydrate determinants (CCD)—that sensitize approximately 10-20% of all pollen-allergic patients are also present in grass pollen and weed pollen.

Moreover, IgE-binding proteins related to the birch pollen minor allergen Bet v 6 have been found in many vegetable foods such as **apple, peach, orange, lychee fruit, strawberry, persimmon, zucchini, and carrot**. Frequently, the occurrence of cross-reactive IgE antibodies is not correlated with the development of clinical food allergy. In particular, the clinical relevance of sensitization to CCD is doubtful. Generally, pollen-related allergens tend to be more labile during heating procedures and in the digestive tract compared to allergens from classical allergenic foods such as peanut. However, recent DBPCFC studies have shown that **both cooked celery and roasted hazelnuts still pose an allergenic risk for pollen-sensitized subjects.**”

The following snippet highlights that immune activation (via food in birch pollen sensitive people) can even happen via non IgE mediated means – further emphasising the need to identify ALL possible allergens in an atopic or allergic patient (as in this client)

The impact of pollen-related food allergens on pollen allergy, Bohle B., Allergy. 2007 Jan;62(1):3-10

Patients with birch pollen allergy frequently develop hypersensitivity reactions to certain foods, e.g. apples, celery, carrots and hazelnuts. These reactions are mainly caused by IgE-antibodies specific for the major birch pollen allergen, Bet v 1, which cross-react with

Interestingly all states are different and Northern Territory doesn't have any ryegrass pollen release at all.

Therefore important to further research all possible cross reactive allergens in this clients geographical area and test accordingly and further confirm by elimination diet and challenge if needed. This may have significant impact on acute episodes and progression of lower respiratory tract manifestations like bronchiectasis and asthma. Also important to note is that cooking a food that is known to contain cross reactive allergens can denature the protein in those allergens and thus (but not always) reduce immunological activation and subsequent allergic symptoms. Raw foods are more problematic in cross reactive allergy sufferers.

The link here - www.nlabs.com.au/products/brochures/foodstats_booklet.pdf - contains **great** information about food families and cross reactivity and an example from this document follows -

Aster/Daisy Family (Asteraceae)

Artichoke, burdock root, chamomile, chicory, dandelion, endive, escarole, globe artichoke, jerusalem artichoke, (artichoke flour), **lettuce** (iceberg lettuce, romaine lettuce, bib and red leaf lettuce), safflower oil, salsify, (oyster plant), stevia, sunflower seeds, tansy, tarragon, wormwood, (Absinthe) and yarrow, coneflower (echinacea) and milk thistle

Inhalants: Dog fennel, rough marshelder, common mugwort, poverty weed, rabbit bush, short ragweed, Western ragweed, common sagebrush

Related plants and chemicals: Aster, bachelor's button, mum, chrysanthemum, cosmos, saffron, zinnia, blessed thistle, tansy, feverfew, and lavender cotton. Pyrethrum is used in insect sprays and in mothballs. If you are very sensitive to the members of this family and related plants, avoid insect sprays containing pyrethrum.

Thus in this document, all foods are derived from either a plant or animal source, and are grouped into families according to their origin. With some food groups, in certain individuals, an adverse reaction to one member of a family may result in a similar reaction to other members of the same group - known as cross-reactivity. **This article is useful especially when testing for IgG based food immunological activation.** For example if IgG results come back as positive for the above foods in blue (common IgG tested foods) then it can be useful to know the other members in these foods family as cross reactivity is possible within this family and/or within the listed inhalents. **However this is NOT a list of foods to avoid.** It is simply a guide to help identify possible cross reactive allergens.

Below is a simple table of common inhalant and/or food cross-reactions. (list is not necessarily complete)

Ragweed Pollen

Honeydew, banana, apple, watermelon, gourds, chamomile tea, honey, nuts,

Birch Pollen

Apple, hazelnut, almond, peach, apricot, nectarine, carrot, potato, parsnips, pear, plum, prune, cherry, fennel, parsley, coriander, buckwheat, celery, kiwi, honey, peanut, peppers, spinach, walnut, wheat

Alder Pollen

Almond, apple, celery, cherry, hazelnut, parsley, peach, pear

Grass Pollen

Melon, oranges, Swiss chard, tomato, watermelon, wheat, fennel, celery, kiwi, peanut

Mugwort Pollen

Celery, carrot, coriander, fennel, melon, parley, pappers, spices, sunflower seeds, watermelon

Hazel Tree

Hazelnut

Latex

Banana, avocado, chestnut, almond, potato, tomato, kiwi, pear, hazelnut, papaya, melon, apple, celery, carrot, cherry, peach

Cow's milk ; Sheep, lamb, goat, buffalo

Pork Meat ; Cat epithelia

And of interest for this client, from a previous discussion we have the following -

With respect to **shellfish allergy**, muscle **tropomyosin** is the major allergen. The following articles highlight the allergen involved - "Tropomyosins give rise to important cross-sensitization between shellfish, but also with gastropods, mites, cockroaches and other arthropods. The myosin light chain has been shown to be an allergen in 60% of white shrimp-allergic patients. A muscle arginine kinase has been recognized as an allergen in a third of patients sensitized to black tiger shrimp. A sarcoplasmic calcium-binding protein of the same tiger shrimp is recognized by IgE antibodies of 60% of shrimp-allergic patients.

In fish allergy, the major allergens are the β isoforms of muscle parvalbumins. Fish parvalbumins share among themselves important homologies in amino acid composition, which gives rise to extensive cross-sensitizations. Allergic cross-reactions with frog parvalbumin have been described. Due to the frequent sensitization to parvalbumins, the role of other allergens is masked. Allergy to fish gelatin has been reported. Additional muscle allergens have been incompletely described, but their exact role remains to be defined."

[*Fish and shellfish allergens, F. Hentges, Revue Française d'Allergologie, Volume 49, Issue 3, April 2009, Pages 156-159*](#)

"Most food allergens are major proteins, polyvalent molecules with at least two or more IgE-binding sites, and are recognized as foreign molecules (hence immunogenic). A number of major food allergens have been recently characterized, and amino acid sequences determined. Tropomyosin is the only major allergen of shrimp. A number of IgE-binding epitopes have been identified in this molecule, though they may vary from one shrimp-allergic individual to another. Single amino acid substitutions within epitopes based on that of

homologous, nonreactive tropomyosins can substantially enhance or abolish IgE antibody binding.”

Current Understanding of Food Allergens, SAMUEL B. LEHRER et al., Annals of the New York Academy of Sciences, Volume 964 Issue 2006 GENETICALLY ENGINEERED FOODS: ASSESSING POTENTIAL ALLERGENICITY, Pages 69 – 85

Article below brings to attention the link between **house dust mites, cockroaches and crustaceans allergy**. – **as seen in this client**

“Among food allergens, crustaceans, such as shrimp, crab, crawfish and lobster, are a frequent cause of adverse food reactions in allergic individuals. The major allergen has been identified as the muscle protein tropomyosin. This molecule belongs to a family of highly conserved proteins with multiple isoforms found in both muscle and nonmuscle cells of all species of vertebrates and invertebrates. Its native structure consists of two parallel alpha-helical tropomyosin molecules that are wound around each other forming a coiled-coil dimer. Allergenic tropomyosins are found in invertebrates such as **crustaceans** (shrimp, lobster, crab, crawfish), **arachnids (house dust mites)**, **insects (cockroaches)**, and molluscs (e.g. squid), whereas vertebrate tropomyosins are nonallergenic. Studies of cross-reactivities among crustaceans and the high degree of sequence identity among them suggest that tropomyosin is probably the common major allergen in crustaceans. **Furthermore, immunological relationships between crustaceans, cockroaches and housedust mites have been established and may suggest tropomyosin as an important cross-sensitizing pan allergen.**”

Tropomyosin: An Invertebrate Pan-Allergen, G. Reese, R. Ayuso, Samuel B. Lehrer Section of Allergy and Clinical Immunology, Tulane University School of Medicine, New Orleans, La., USA, Int Arch Allergy Immunol 1999;119:247-258

An action plan for this client could be to further investigate (with my help if needed) seasonal and perennial pollen/grass exposure in her area – through CSIRO, land care, etc – test for reaction via SPT and/or Rast IgE to these identified allergens – minimise exposure at known peak times - identify any cross reactive foods within the identified windborne allergen family and perform elimination/challenge tests to these foods. Thus the **allergenic load** (the combined quantitative exposure to allergens at any one time) would be reduced thus introducing the possibility of decreasing immune cell activation and degranulation, minimal rise in IgE levels and subsequent cytokines and a consequent decreased systemic inflammatory response. This is hard work and not without challenges and would require diligence and persistence but the payoff would be an improved quality of life and decreased progression of inflammatory based lower lung disease (bronchiectasis).

In addition it is **important to note** that what appears as an allergy type reaction to food can also be a **food based sensitivity reaction** to any or any combination of the following compounds-

Amines: histamine, tyramine, tryptamine, serotonin, dopamine, phenylethylamine Present naturally in foods and produced during fermentation, aging and storage in other foods.

Salicylates are aspirin- like compounds present in a wide variety of herbs, spices as well as fruit and vegetables. Reactions to these may be even more common than reactions to artificial colours and preservatives. Salicylates are concentrated in the surface areas of fruits and vegetables and levels decrease as the fruit ripens. Tea contains very high salicylate levels.

Glutamates: (E 620-623) MSG stimulates nerve endings, perhaps accounting for its function as a "flavour enhancer", amongst other properties. MSG is the sodium salt of glutamic acid, an amino acid found naturally in the human body and in all protein-containing foods such as cheese, meat and milk as well as some vegetables, without causing reactions.

Sulphites: (E 220 – 228) These chemicals are commonly found processed fruits, vegetables, meats/fish/poultry products, alcoholic and fruit drinks. They are sprayed onto foods to keep them fresh and prevent discoloration or browning. Their use is widespread and cannot be listed here.. Sodium metabisulphite (223) is commonly used as a flour treatment agent.

Benzoic acid and Parabens (E 210 – 219). Benzoates are both a preservative, and also occur naturally in many plant foods (e.g .berries, concentrated tomato products, spices)

Sorbates (E 200-203) – a preservative used in a wide variety of foods.

Food Colourings (E 100 – 180). This includes all the artificial colours, as well as the natural colour, annatto (code 160b).

Antioxidants: (E 319 - 321) Butylated hydroxyanisole(BHA) and Butylated hydroxytoluene (BHT) are found in high fat foods to prevent rancidity as well as some cereals.

Nitrates and nitrites: (E 249 – 252). Used as a preservative in processed meats and some cheeses.

Other food ingredients capable of causing allergy or gut irritation:

Meat tenderiser, Papain can cause allergy and food intolerance (Processed meat products)

Irritant substances in foods such as paprika and chilli (capsaicin) or stimulant effects of naturally occurring substances in food such as caffeine in coffee and tea

Gums (E 400 – 418) (agar, guar gum, tragacanth, carragenan, xanthan gum) can cause abdominal distension and bloating when consumed in large quantities.

REF: Was it something you ate? By Sabine Spiesser BS, Grad Dip Dietetics. April, 2001, from - http://www.mecfscanberra.org.au/docs/food_allergy.htm

“It can be very difficult for people to identify food intolerances, because reactions can be inconsistent (depending on the dose eaten), can be delayed and build up over many days, individuals can react to several different food chemicals, and each food chemical can be found in many different foods, all contributing to the total dose.” Metabisulphite or sulphur dioxide induced **asthma** is an example of such a reaction. **Food intolerances are diagnosed by an elimination diet.**

The above author, although providing no references to back it up, makes the following observation –

“Certain non-histamine containing foods, and food chemicals, can trigger direct histamine

release from mast cells. IgE is not involved in the reaction and specific IgE antibodies to these foods are not elevated. Foods that have been implicated in this type of reaction include: **raw egg white, shellfish, strawberries, chocolate, citrus fruit, pineapple, tomatoes, alcohol, fish and pork meat, as well as salicylates and metabisulphites** (Sodium or potassium metabisulphite is used as a food additive, mainly as a preservative – found in wine and beer and other foods and is sometimes identified as E223). The histamine liberated in this reaction will cause symptoms that may mimic true food allergy.”

Chronic rhinitis is usually an extension of rhinitis caused by inflammation through allergy or an infection or irritants or a combination of all three. Chronic rhinitis can cause nasal obstruction, pus-filled discharge from the nose, and bleeding. Low humidity and airborne irritants along with viral infections and persistent inflammation contribute to chronic rhinitis. Any suspected underlying bacterial infection requires a culture or biopsy with appropriate treatment. Also **atrophic rhinitis** is a form of chronic rhinitis in which the mucous membrane thins (atrophies) and hardens, causing the nasal passages to widen (dilate) and dry out. The cells normally found in the mucous membrane of the nose—cells that secrete mucus and have hairlike projections to move dirt particles out—are replaced by cells like those normally found in the skin. A prolonged bacterial infection of the lining of the nose is also a factor.

And finally **Vasomotor rhinitis** (or non-allergic rhinitis) is a form of chronic rhinitis. While the pathology of vasomotor rhinitis is not very well-understood, it appears that hormones may play a role as vasomotor rhinitis is significantly more common in women than men and it appears that “oversensitive or excessive blood vessel dilation or contraction causes an overreaction to such stimuli as changes in weather, temperature, or barometric pressure, chemical irritants such as smoke, ozone, pollution, perfumes, and aerosol sprays, psychological stress and emotional shocks, certain types of medications, alcohol, and even spicy foods.” REF: <http://en.wikipedia.org/wiki/Rhinitis> In some people, the nose reacts strongly to irritants (such as dust and pollen), perfumes, and pollution. The disorder comes and goes but is worsened by dry air. Sometimes, people also have slight inflammation of the sinuses. When persistent, endoscopy of the nose or CT scan of the sinuses may be needed. Avoiding smoke and irritants and using a humidified central heating system or vaporizer to increase humidity may be beneficial.

Although some immunological/allergy tests have been done in this client such as gliadin and tTG (negative) and Rast IgE to pigeon feathers, pigeon droppings, bird mix, and aspergillus fumigatus (negative) and IgG also to aspergillus (also negative), a more extensive workup of possible food and airborne allergens is warranted and indicated in this client, particularly as any atopic reaction manifesting near or in the respiratory tract can further exacerbate this client primary condition and complaint – bronchiectasis and difficulty in breathing. Dust mite and mould allergy was diagnosed in 2005, presumably through IgE or skin prick tests. (results not seen). The client reports extensive mold around and in the house and dust mite allergen is pervasive in Australia. It is important to note that although these tests were positive for

these two allergens, a symptomatic or clinical response DUE specifically to these two allergens may not be present within the client. Furthermore and in contrast, just because a skin prick test comes back negative, does not rule out a symptomatic reaction to that allergen within the client. An interesting and important experiment for this client would be to temporarily, at least, relocate to an area free from known allergens and dustmites AND during which time optimisation of nutritional status, metabolic function and detoxification capacity is implimented and maintained, along with monitoring of any improvement in symptoms.

From a nutritional perspective it is critical to identify any possible allergy promoting foods and while some of these foods may only initiate a low level IgE response, the cumulative and cross reactive effects between foods and wind born allergens can produce clinical symptoms of respiratory disease. (prawns promote an asthma response in this client). Patients with inhalent allergy frequently exhibit symptoms of Irritable bowel Syndrome (**need to investigate this further in this client**) and migraine – indicative of food reactivity. Importantly **“sensitivity to gluten containg grains and milk products commonly underlies apparent apparent inhalent allergy reactions and avoidance of these foods often reduces or eliminates the response to inhalent allergens.”** Foods not coming up as positive in an IgE based screening may in fact come up as positive in an IgG4 test and although controversial, some symptom relief has been reported from eliminating foods from the diet identified by the IgG4 test. Thus recommend an [Allergix IgG4 screen](#) (from Metamatrix) to 90 common foods in addition to a systematic [screening for IgE reactions](#) to 4 panel foods. (can do 4 X 4 IgE food panels during a 1 year period under medicare and off course more albeit non – medicare). In addition the IgE tests would include SPECIFIC weeds, grasses, trees and pollens and fungi (including candida). This has not been done this thouroughly before in this client.

And as mentioned allergy type symptoms can also be produced by food sensitivities (not an immune based reaction) to substance such as salicylates, histamines and mono amines (Common in many foods and drinks that this client consumes). Failure to identify any food allergies would stimulate a search for these food sensitivities (through illimination diets).

Thus **immune system dysfuntion**, as appears to be present in this client, is often seen in chronic and recurrent Upper Respiratory Track Inflammation (URTI) and in addition to the allergic and sensitivity reactions outlined above, **persistant sub-clinical infection** and **Nutrient insufficiency** are causal mechanisms of URTI.

Persistant sub-clinical or relapsing infection can be due to viruses such as Epstein Barr Virus, Herpes-6 virus (HLV-6), Ross river Virus (RRV) and Cytomegalovirus. Would be useful to test client for these as viral load may be maintained in the presence of chronic corticosteriod use (which dampens the immune system)

Note that client reported *“In 2003 went to Singapore and contracted severe chest infection - SARS was prevalent at time although SARS was not Dx. and antibiotics given for 2-3 weeks.*

Expressed feeling "different after this infection" and Asthma symptoms started to develop after that."

Severe Acute Respiratory Syndrome (SARS) is a respiratory disease in humans which is caused by the SARS coronavirus (SARS-CoV). It originated in China apparently in 2003 and by 2003 has spread to other countries around the world including Singapore. Although client was not Dx. With SARS at the time it is interesting to note -

"With the identification and sequencing of the RNA of the coronavirus responsible for SARS on 12 April 2003, several diagnostic test kits have been produced and are now being tested for their suitability for use. Three possible diagnostic tests have emerged, each with drawbacks. The first, an ELISA (enzyme-linked immunosorbent assay) test detects antibodies to SARS reliably but only 21 days after the onset of symptoms. The second, an immunofluorescence assay, can detect antibodies 10 days after the onset of the disease but is a labour and time intensive test, requiring an immunofluorescence microscope and an experienced operator. The last test is a polymerase chain reaction (PCR) test that can detect genetic material of the SARS virus in specimens ranging from blood, sputum, tissue samples and stools. **The PCR tests so far have proven to be very specific but not very sensitive.** (not sure how this client was diagnosed at the time) This means that while a positive PCR test result is strongly indicative that the patient is infected with SARS, **a negative test result does not mean that the patient does not have SARS.** There is currently no rapid screening test for SARS and research is ongoing." REF: <http://en.wikipedia.org/wiki/SARS>

Any persistent viral infections can lead to Post Viral Syndrome, Chronic fatigue syndrome and Fibromyalgia.

Other non virus organisms although intracellular and obligate (Obligate intracellular parasite, is a parasitic microorganism that cannot reproduce outside their host cell) are more prevalent than previously thought and may be causally linked with Lower Respiratory Tract Inflammation (LRTI) along with URTI

These organisms can include – 1/ **Mycoplasma** a genus of bacteria which lack a cell wall. Without a cell wall, they are **unaffected by many common antibiotics such as penicillin or other beta-lactam antibiotics** that target cell wall synthesis. They are usually parasitic. Several species are pathogenic in humans, including *M. pneumoniae* (***Mycoplasma pneumoniae***), which is an important cause of atypical pneumonia and other respiratory disorders. There are over 100 recognized species of the genus *Mycoplasma*, and are unusual among bacteria in that most require **sterols** for the stability of their cytoplasmic membrane. Sterols are acquired from the environment, usually as **cholesterol** from the animal host. 2/ **Chlamydia** – a genus of bacteria that are obligate intracellular parasites, many of which are pathogenic. Chlamydia infections are the most common bacterial sexually transmitted infections in humans and although not specific to the Respiratory tract may contribute to systemic inflammation. However ***Chlamydia pneumoniae***, like *M. pneumoniae* is a small bacterium (0.2 to 1 micrometer) that undergoes several

transformations during its life cycle and is a major cause of pneumonia and in addition is associated with atherosclerosis, Alzheimer's disease and **asthma**. The first known case of infection with *C. pneumoniae* was a case of sinusitis in Taiwan and this atypical bacterium commonly causes **pharyngitis, bronchitis** and atypical pneumonia mainly in elderly and debilitated patients but in healthy adults also. REF:

http://en.wikipedia.org/wiki/Chlamydomphila_pneumoniae and 3/ **Rickettsiae** “a genus of motile, Gram-negative, non-sporeforming, highly pleomorphic bacteria that can present as cocci (0.1 µm in diameter), rods (1–4 µm long) or thread-like (10 µm long). Obligate intracellular parasites, the Rickettsia survival depends on entry, growth, and replication within the cytoplasm of eukaryotic host cells - typically endothelial cells. The majority of Rickettsia bacteria are susceptible to antibiotics of the tetracycline group.” REF:

<http://en.wikipedia.org/wiki/Rickettsiae>

In addition to these viruses and virus like pathogens, bacterial infection and/or colonisation from ***Pseudomonas aeruginosa*** and an unidentified fungus were detected in the sputum of this patient and may exhibit persistence and chronicity albeit sub clinical or asymptomatic at times. (Note that colonisation by *Pseudomonas aeruginosa* – NOT infection- is seen in many normal healthy people in both the gut and the respiratory tract. *Pseudomonas aeruginosa* is found in soil, in plants, water, skin flora. The symptoms of infections are generalised inflammation and sepsis. It is a Gram-negative, aerobic, rod-shaped bacterium with unipolar motility. An opportunistic pathogen of immunocompromised individuals, *P. aeruginosa* typically infects the pulmonary tract, urinary tract, burns, wounds, and also causes other blood infections. It is the most common cause of infections of burn injuries and of the external ear (otitis externa), and is the most frequent colonizer of medical devices (e.g., catheters). *P. aeruginosa* uses virulence factors so that eukaryotic cells cannot synthesize proteins and using an exoenzyme, phospholipase S, which degrades the plasma membrane of eukaryotic cells, leading to necrosis and lysis. The release of intracellular contents further induces an immunologic response recruiting immune cells that further release cytokines thus promoting further inflammation in an already chronic inflammatory state.

An interesting recent article shows that *Pseudomonas aeruginosa*, at least in the gut, has 2 rather distinct phenotypes – one that is fairly benign for the host while the other phenotype produces toxins and demonstrates more tissue destruction. In the gut of patients who had surgery or were immunocompromised they found that **low phosphate** levels correlated with increased virulence of this pathogen.

[Red death in *Caenorhabditis elegans* caused by *Pseudomonas aeruginosa* PAO1, Alexander Zaborina et al, PNAS, April 14, 2009, vol. 106, no. 15, 6327– 6332](#)

“Based on results from the present study, phosphate depletion may represent a previously unappreciated environmental cue in the intestinal tract of severely injured and physiologically stressed patients that has a major influence on *P. aeruginosa* lethality. A better understanding of how *P. aeruginosa* senses and responds to phosphate depletion within the intestinal micro-environment is critical for the development of strategies to contain this pathogen which

continues to be among the most antibiotic resistant organisms infecting hospitalized patients. Data from the present study provide compelling evidence that **phosphate depletion** induces virulence systems in *P. aeruginosa* associated with phosphate, quorum sensing, and **iron** signalling.

We have identified several of these compounds as immune elements (**IFN- gamma**), **opioids** (morphine, dynorphin) and end-products of hypoxia (**adenosine**), all of which are released into the intestinal tissues and lumen during surgical injury, ischemia, and inflammation. The local concentration of extracellular phosphate is one of the multiple local environmental cues within the intestinal tract of a surgically injured host that might converge to activate a lethal phenotype in *P. aeruginosa*. Phosphate depletion is known to rapidly develop following major surgery and organ injury and independently predicts the development of lethal sepsis. We have recently documented that following surgical injury, **phosphate becomes rapidly depleted within intestinal mucus** (does this happen in Lung also? – my notes) to levels that are associated with the expression of important virulence determinants in *P. aeruginosa*.”

Red death in Caenorhabditis elegans caused by Pseudomonas aeruginosa PAO1, Alexander Zaborina et al, PNAS, April 14, 2009, vol. 106, no. 15, 6327– 6332

Interesting to follow up on this research, particularly for someone like this client who most likely has chronic drug resistant colonization of *P. aeruginosa* in the lung, with acute pathogenic outbreaks possibly linked to progressive tissue destruction via bronchiectasis.

Fungal infection, particularly *Candida albicans*, needs to be further investigated in this client as indications discussed previously (medication induced fungal colonisation) and lab results point to possibilities of colonisation at least. Also consider Allergic bronchopulmonary aspergillosis (ABPA), which describes a syndrome in which patients with asthma harbor the saprophytic growth of aspergillus species, most often *aspergillus fumigatus*, within their airways. An intense allergic inflammation results, as ABPA represents an immunoglobulin IgE based hypersensitivity to either *A. fumigatus* antigens or other non - aspergillus antigens. Recurring clinical exacerbations can lead to bronchiectasis (both onset and progression).

“ABPA, like rhinosinusitis, gastroesophageal disease (GERD) and chronic obstructive pulmonary disease (COPD), should be included whenever considering and asthma diagnosis” refer to MD

REF: BOOK; Bronchial Asthma: A guide for practical understanding and treatment, by M Eric Gershwin

This client has indications of unidentified fungus in sputum. IgE load appears however to be low, although total IgE (along with all other Ig's) is also low which may be secondary to an underlying protein deficiency (also indicated in this client), thus introducing the possibility of a false negative regards specific IgE results.

Nutrient insufficiency and immune dysregulation, particularly nutrients that are important for optimal immune function, is present to some extent in this client (seen from physical, lab results and diet) and as such leads to recurrent and/or chronic low grade infection. Nutrients of particular importance for optimal immune support include Vitamins A, B, C, D and E and include the minerals Calcium, magnesium, zinc, iron and copper. Protein depletion, as seen

in this client, along with Vit B6, zinc and iron deficiency are the commonest causes of nutrient related immuno deficiency. *ref: Course 6 notes.*

This client also is exposed to **environmental chemicals**. Apart from some atmospheric pollutant exposure that is regarded as minimal (by the client) , this client is also exposed to weed spray - glyphosate360, once per month. Other household cleaning products should also be investigated for any contribution to precipitating URTI. The Materials Safety Data sheet for **Glyphosate 360** indicate - Inhalation - Low irritant. Over exposure may result in mucous membrane irritation of the nose and throat with coughing. And “The active ingredient has been in use for many years in Australia and in other countries. There are no reports of inhalation health effects in humans. No animal inhalation studies have been carried out. However, it may be mildly irritant to mucous membranes.” Searching the Australia Government service called **Australian Inventory of Chemical Substances** (AICS) revealed CAS No:38641-94-0

Chemical Name: Glycine, N-(phosphonomethyl)-, compound with 2-propanamine (1:1)

Molecular Formula: C₃H₉N.C₃H₈NO₅P

Assessed by NICNAS: No

Note that NICNAS, Provides a national notification and assessment scheme to protect the health of the public, workers and the environment from the harmful effect of industrial chemicals; and Assesses all chemicals new to Australia and assesses those chemicals already used (existing chemicals) on a priority basis, in response to concerns about their safety on health and environmental grounds.

REF: <http://www.nicnas.gov.au/Industry/AICS/Search.asp>

In a paper entitled **Asthma, Children and Pesticides - What you should know to protect your family. 2005** From <http://www.beyondpesticides.org/> comes some of the following research and information -

“A 2003 study of over 3,000 Lebanese children similarly found correlations between exposures to pesticides and respiratory diseases. Pesticide exposure includes home and garden pesticide use, occupational use by a household member, and living in proximity to a treated field. All of these exposures are associated with chronic respiratory disease and symptoms, and particularly with asthma.”

And “people with asthma are especially sensitive to pesticides and at risk of attacks when exposed to even small amounts. Pesticides can trigger asthma attacks by **increasing airway hyper-reactivity**, which makes the airway very sensitive to **any allergen** or other stimulus. Subsequent exposure to a stimulus can cause an extreme reaction in a hyper-reactive airway. In these situations, researchers at Johns Hopkins Bloomberg School of Public Health have shown that pesticides somehow alter the nerve function controlling the smooth muscle lining of the airway, causing the airway to contract and restrain airflow, which is exactly what occurs during an asthma attack. Pesticides can also trigger asthma attacks by directly damaging cells that line the lungs. “

And **“Glyphosate (Roundup®)**: Glyphosate is one of the most commonly used pesticides on

lawns and landscapes. Exposure to glyphosate can cause asthma-like symptoms and breathing difficulty. Undisclosed, or proprietary, ingredients (called “inert ingredients”) in Round-up®, a common formulation of glyphosate, have been linked to pneumonia and damage to the mucous membrane tissue and the upper respiratory tract.” Even though his article is presented more as a brochure it is scientifically referenced.

Finally in this article entitled - *Pesticides and Atopic and Nonatopic Asthma among Farm Women in the Agricultural Health Study*, Jane A. Hoppin et al, *AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE VOL 177 2008*, the authors, in a very detailed study involving over 25,000 women found and concluded that a total of 7 of 16 insecticides, 2 of 11 herbicides, and 1 of 4 fungicides were significantly associated with atopic asthma; only permethrin use on crops was associated with nonatopic asthma and in conclusions these findings suggest that pesticides may contribute to atopic asthma, but not nonatopic asthma, among farm women.

In summary to effectively deal with **URTI or LRTI** it is paramount to remove exposure to chemicals and if possible inhalent allergens and particularly food allergens and food ingredients causing sensitivities, as outlined in the above discussion. Go through the **Diet** with a fine toothed comb. Minimal exposure time should be organised to inhalent allergens and other irritants so that concurrent nutrition therapy can **optimise nutrition status**, metabolic function and detoxification capacity. A diligent search for food sensitivities often reveals **frequently eaten foods**.

Next enhancing **digestive efficacy** is crucial as undigested food in combination with any intestinal permeability would significantly expose an already hyper-inflammatory overwhelmed individual to greater allergen load. Also in many chronic disease conditions as in this case of bronchiectasis, the individual may be in a protein depletion state (as in this client) and as such get caught in a viscous cycle of impaired gastric acid production and diminished pancreatic digestive enzymes that then further leads to poor protein assimilation. The excessive mucus production seen in this client must also contribute to protein loss. Low protein status also reduces activity of microsomal oxidase enzymes that are responsible for neutralising many environmental chemicals. The corticosteroids are known to cause gastro intestinal disturbances and as such may negatively impact on digestive efficacy.

Next Essential **Fatty acid balance and metabolism** needs to be addressed as increased consumption of saturated fats and trans fats and increased intake of $\Omega 6$ foods (vegetable oils, nuts and seeds) at the expense of $\Omega 3$ fatty acids containing foods (fish oil, flaxseed oil, chia seeds or hemp seeds) will increase propensity of inflammation as eicosenoid production shifts to pro-inflammatory mediators and up regulation of pro-inflammatory genes.

Next **antioxidant status** must be optimised as “ high dose therapy with Vit C, tocopherols and flavanoids reportedly reduces hyperactive inflammatory processes by down regulating polymorphonuclear free radical release, mast cell histamine release and immune cell cytokine production”

Next enhance **hepatic detoxification** as environmentally sensitive individuals may become more hypersensitive to chemicals and food particles and immune complexes and in this case concurrent drug molecules (corticosteroids) due to these individuals having possible impaired and overwhelmed hepatic detoxification mechanisms leading to excess free radical production with subsequent glutathione depletion. On the other hand inhalation of glutathione can reduce respiratory inflammation.

Next correcting any **bowel dysbiosis** is essential and in this client even though the physical and MSQ relieved minimal abdominal discomfort and symptoms, research into the effects of the drugs being taken by this client show that gastro intestinal disturbance is a side effect with overgrowth of candida also indicated. Also Bronchiectasis has been linked to irritable/inflammatory bowel disease. In addition antibiotic therapy, particularly on the scale seen in this client, can SIGNIFICANTLY cause the overgrowth of toxigenic bowel flora, particularly (again) candida. Any resulting dysbiosis can contribute to immune hyperactivity via undue antigen uptake by enteric macrophages, thus activating T cell activity and increasing cytokine production. To correct dysbiosis will need to investigate bacteria growth in GIT. Use the Metamatrix GI effects DNA stool analysis and treat accordingly.

And finally **Specific disease therapeutics** should look at further enhancing immune system function and equilibrium, as continual escalation of infection is detrimental to the bronchiectasis and in a viscous loop the prevalent hyper inflammation seen in bronchiectasis promotes pathogen build up and stasis.

Diet and key nutrient therapeutics will be discussed later.

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It is a sample of the work carried out on a real client during a full Nutrition Medicine Health Assessment and treatment. It is designed to highlight the attention to detail and individualised care given to each client during an assessment. It is not intended to help in any form of self diagnoses or self treatment.

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