Learning Outcomes

On successful completion you will be able to:

• Explain the pathophysiology of a range of common skin and respiratory disorders such as asthma and eczema.

• Explain the varied aetiologies which drive various skin and respiratory diseases.

• Illustrate how skin and respiratory disorders connect to pathologies of other body systems.

• Evaluate both orthodox medical and naturopathic nutritional diagnosis and therapy for skin and respiratory disorders.

• Show awareness of the importance of referral with ‘Red Flag’ symptoms.
Asthma

General Considerations:

• Bronchial asthma is a hypersensitivity disorder characterized by:
  – Spasm of the bronchi.
  – Swelling of the bronchial mucosa.
  – Excessive excretion of a viscous mucus.

Leading to breathing insufficiency.

• Prevalence is ca. 8% in the UK, and 10% in Ireland (in Ireland the prevalence is 4th highest in the world).

• Most common in children younger than 10.

• 2:1 male-to-female ratio in children, which equalizes by the age of 30.

(Murray and Pizzorno 2008; AU 2009; ASI 2009; )
Asthma

Diagnosis:

• Chest tightness and shortness of breath.
• Wheezing when breathing out (Expiratory wheeze).
• Rapid, shallow breathing that is easier when sitting up.
• Difficulty breathing.
• Neck muscles tighten.
• Coughing, especially at night, occasionally with thick, clear or yellow sputum.

(Murray and Pizzorno 2008)
Asthma

Diagnosis:

- The diagnosis of asthma is a clinical one; there is no standardised definition of the type, severity or frequency of symptoms, nor of the findings on investigation.

- The absence of a gold standard definition means that it is not possible to make clear evidence based recommendations on how to make a diagnosis of asthma.

- Central to all definitions is the presence of symptoms (more than one of wheeze, breathlessness, chest tightness, cough) and of variable airflow obstruction.
Asthma

Diagnosis Cont.:

• More recent descriptions of asthma in children and in adults have included airway hyper-responsiveness and airway inflammation as components of the disease.

• How these features relate to each other, how they are best measured and how they contribute to the clinical manifestations of asthma, remains unclear.

• Although there are many shared features in the diagnosis of asthma in children and in adults there are also important differences. The differential diagnosis, the natural history of wheezing illnesses, the ability to perform certain investigations and their diagnostic value, are all influenced by age.

(British Guideline on the Management of Asthma, revised 2012)
Asthma

• Some of the classifications used include age of onset and intrinsic v.s. extrinsic.
  – **Extrinsic or atopic asthma**: Is generally considered an immunologically mediated condition with a characteristic increase in serum antibodies- IgE.
  – **Intrinsic**: A bronchial reaction that is due, not to antigen-antibody stimulation, but rather to such factors as chemicals, cold air, exercise, infection and emotional upset – however recent evidence suggests IgE may also be involved in these – particularly in exercise induced asthma.

• Phenotyping by atopic status is not considered particularly useful as lung tissue of atopic adult and children presents with very similar histology to non-atopics, and does not predict response to corticosteroid treatment.

• Further development and definition of asthma types is desirable to help to identify responses to treatment and prognosis.

(Bel, E, 2004; Murray and Pizzorno, 2008; E, Lommatzsch et al 2013)
Asthma - Pathophysiology

- Environmental trigger
- Increased T-helper (2) cell response
- Mast cells, IgE, Eosinophils
- Inflammatory mediators
  - Examples: leukotrienes, prostaglandins, histamine, thromboxanes, platelet activating factor, etc.
- Bronchial constriction
- Breathing obstruction
- Increased mucus production
- Wheezing
- Coughing
Asthma - Pathophysiology

- The most potent chemical mediators in asthma are the **lipoxygenase products - leukotrienes** that are 1000 times more potent as stimulators of bronchial constriction than histamine.

- Asthmatics have an **imbalance in Arachidonic acid metabolism**, leading to a relative increase in lipoxygenase products.

(Murray and Pizzorno 2008; Yen and Morris 1981)
Asthma

Conventional Treatment:

- **Mild intermittent asthma** is treated on an as-needed basis with inhaled beta-agonists (bronchodilators).

- **Mild persistent asthma** treatment involves daily inhalation of a corticosteroid, along with a short-acting beta-agonist for breakthrough symptoms.

- **Moderate persistent asthma** is treated with inhaled corticosteroid and/or the addition of a long-acting beta agonist or leukotriene antagonist (drugs that inhibit leukotrienes).

- **Severe asthma** requires high-dose inhaled corticosteroids or oral corticosteroids, along with other controller medicines.

(Murray and Pizzorno 2008; Yen and Morris 1981)
Asthma

Aetiology:

• The incidence of asthma is rising rapidly in the West, especially in children; possible reasons:
  – Earlier weaning.
  – Earlier introduction of solid foods to infants.
  – Replacing breastfeeding with infant formulas.

• Ideally an infant should be breastfed for 6-9 months; weaning earlier than 6 months and feeding formula increases the likelihood of food allergy including asthma.

(Murray and Pizzorno 2008; Oddy et al 1999; Friedman and Zeiger 2005)
Asthma

Aetiology:

- **Food additives:**
  - **Food colourings:** Azo dyes-tartrazine (orange)
  - **Preservatives:** Benzoates, sulfur dioxide, and sulphites

- The largest sources of sulphites are:
  - Prepared salads.
  - Salad dips servered in restaurants.
  - Wine and beer.

- Average sulphite consumption in the West is 2-3g; Single restaurant meal can contain 25 -100mg!

_Freedman 1977; Stevenson and Simon 1981; Sikorski 2007; Papaioannou and Pfeiffer 1984_
Asthma

Aetiology:

• Food allergy
  – The presence of food allergies is thought to be responsible for asthmatics having "leaky guts".
  – As a result of increased gut permeability, there is increased antigen load on the immune system.
  – This increases the likelihood of developing additional allergies, as well as increasing bronchoconstrictive compounds into the circulation

(Benard et al 1996)
Asthma

Aetiology:

• **Candida Albicans:**
  – An overgrowth of the yeast Candida albicans in the gastrointestinal tract has been implicated as a causative factor in allergic conditions including asthma.
  – Apparently the **acid protease** produced by C. albicans is the responsible allergen.

(Akiyama et al 1994)
Asthma

Aetiology:

• Salt:
  – Studies show that an increased intake of salt increases bronchial reactivity and mortality from asthma.
  – The degree of bronchial reactivity to histamine is positively correlated with the 24-hour urinary sodium excretion and rises with increased dietary sodium.

(Carrey et al 1993; Burney et al 1987)
Asthma

Aetiology:

• **Higher incidence of obesity** - There is a clear association between obesity and asthma, although the nature of this relationship is still not clear; possible factors include:
  
  – Increased leptin levels in the obese may alter functioning of their immune system and cause proinflammatory changes.
  
  – Obese clients have different breathing patterns (more shallow and frequent) that may predispose them to asthma.
  
  – Higher oestrogen levels due to excess adipose tissue have been linked to asthma; although the mechanism is unknown.

(Murray and Pizzorno 2008; Weiss and Shore 2004)
Asthma

Aetiology:

• ‘Hygiene Hypothesis’:
  – The immune response of T helper cells is the main mechanism of mediated airway inflammation.
  – There are two types of T helper cells: Th1 and Th2 helper cells.
  – Asthma and atopic conditions like allergies reflect increased response of Th2 helper cells.
  – The "hygiene hypothesis": minimizing exposure to infectious agents due to more hygienic lifestyle choices has favored the dominance of Th2 immune responses and the encouragement of asthma and atopic diseases.

(Joos et al 2004; McGeady et al 2004; von Mutius et al 2001)
Asthma

Aetiology:

• ‘Hygiene Hypothesis’:
  – Today’s children are exposed to a much lesser variety of natural antigens than children even 20-30 years ago due to:
    • Excessive use of detergents/cleaning agents at home.
    • Spending less time playing in natural surroundings like earth, sand, grass; and more time inside.

• Result: The immune system is deprived of its ‘learning tools’ - various antigens in early life which then leads to the inability of the immune system to deal with them later in life.

• Natural antigens stimulate Th1 response - which is thought to be protective against asthma.

(Joos et al 2004; McGeady et al 2004; von Mutius et al 2001; Ball et al 2000)
Asthma

Aetiology:

• Greater chemical pollution in the air:
  – Various mechanisms.

• Increased antibiotic use in children:
  – A meta-analysis of seven studies involving more than 12,000 youngsters, found that those prescribed antibiotics before their first birthday **were more than twice as likely** as untreated kids **to develop asthma**.
    • Antibiotics may contribute to the state of ‘excessive hygiene’.
    • Antibiotics disrupt normal gut and respiratory bacterial flora.

(Diaz-Sanchez et al 2003; Miller 2001; Marra et al 2006)
Asthma

Aetiology:

• **Pertussis (Whooping Cough Vaccine):**

  • A British study of 448 children found the relative risk of developing asthma was:
    – 1% in children who receive no immunizations.
    – 3% in those who receive vaccinations other than pertussis.
    – 11% in those who receive pertussis vaccine.

• However, in the group not immunized to pertussis - 16 developed whooping cough compared with only 1 in the immunized group

(Murray and Pizzorno 2008; Odent et al 1994)
Asthma

Nutritional Considerations - Epidemiological Evidence:

• Numerous studies suggest that people who have a **diet rich in fruit and vegetables** have a lower risk of poor respiratory health.

• **Large epidemiologic review**: Higher consumption of fresh fruit among children has been related to a lower prevalence of asthma symptoms and higher lung function.
  – Fruit high in vitamin C was shown to be protective.
  – Fish consumption was also related to lower airway hyper-reactivity in this study.

(Denny et al 2003; McKeever et al 2002; Romieu et al 2001)
Nutritional Considerations - Epidemiological Evidence:

- **A study of Scottish adults examining** a dose/response relationship between fruit consumption and pulmonary function found: increased fruit consumption led to decreases in phlegm and better pulmonary function.

- **In a study of 607 asthma patients and 864 controls:** Higher consumption of apples and moderate red wine consumption decreased asthma severity.

- The hypothesis is that fruit and wine are sources of **antioxidants** which may exert beneficial effect on asthma (see below).

(Kelly et al 2003; Shaheen et al 2001)
Asthma

Nutritional Considerations - Epidemiological Evidence:

• Diet in pregnancy and the risk of future asthma in children:
  – Closely following a Mediterranean diet during pregnancy (i.e. high in fruits and vegetables) was protective for asthma and atopy at age 6.5 yrs.
  – In a separate study, maternal fish consumption during pregnancy was also found to be protective for atopy-related illnesses among non-breastfed children (the same effect was not noted for breastfed infants).

(Chatzi et al 2008; Romieu et al 2007)
Asthma

Nutritional Considerations - Foods and Nutrients:

- **Onions and garlic:** Inhibit lipoxygenase and cyclooxygenase enzymes, which generate inflammatory mediators.

- **Omega 3 EFA/Cold Water Fish:** Children who eat fish more than once a week have one third the risk for asthma of children who do not eat fish regularly.

- Supplementation with omega-3 fatty acids offers significant benefits in asthma.

- These benefits are related to increasing the ratio of omega-3 to omega-6 fatty acids in cell membranes, thereby reducing the availability of arachidonic acid which leads to a significant shift in leukotriene synthesis from the extremely inflammatory 4-series to the less inflammatory 5-series.

Nutritional Considerations - Foods and Nutrients:

• Some children with asthma have a defect in tryptophan metabolism and reduced platelet transport of serotonin - a known broncho-constricting agent in asthmatics.

• Therefore foods high in tryptophan and supplemental tryptophan should be removed from the diet.

• Or supplemental vitamin B6 could be used to aid in the metabolism of tryptophan.

• When used in a study that included 76 asthmatic children, it produced significant reductions in symptoms and in the dosages of drugs (bronchodilators and corticosteroids) required.

(Unge et al 1983; Reynolds et al 1985; Collipp et al 1975)
Asthma

Nutritional Considerations - Foods and Nutrients:

- **Vitamin B6**: In adult asthmatics, blood levels of B6 have been found to be significantly lower than those in healthy controls.

- B6 supplementation has produced decreases in frequency and severity of wheezing and asthmatic attacks, except in clients dependent upon steroids.

(Unge et al 1983; Reynolds et al 1985; Collipp et al 1975; Sur et al 1993)
Asthma

Nutritional Considerations - Foods and Nutrients:

• **Antioxidants:**
  – Inhibit leukotriene formation and histamine release from mast cells.
  – Increase the integrity of the epithelial lining of the respiratory tract.
  – Protect the lung against free radicals and other oxidizing agents that may stimulate bronchial constriction and increase reactivity to other agents.

• **Antioxidants work in synergy;** it is best to include a variety of antioxidant nutrients in the client’s diet such as the **carotenes, vitamins A, C, and E**, the mineral cofactors essential for antioxidant defense actions such as **zinc, selenium, and copper**, and **flavonoids**, particularly **quercetin**.

(Murray and Pizzorno 2008; Johnston et al 1993; Foreman 1984)
Asthma

Nutritional Considerations - Foods and Nutrients:

• **Magnesium:** Is a natural bronchodilator, magnesium stabilizes mast cells and relaxes muscles, dilating bronchioles and quickly opening up airways.

• Dietary magnesium intake is independently related to lung function and asthma severity.

• Oral magnesium supplementation has been shown to be of clinical benefit in asthma studies with children and adolescents (200-300mg/day).

(Murray and Pizzorno 2008; Britton et al 1994; Gontijo-Amaral et al 2006; Bede et al 2003)
Asthma

Therapeutic Protocol:

• The development of an appropriate treatment plan includes the following steps:
  – Determining and rectifying the underlying defect that allows the development of sensitization.
  – Determining and balancing the underlying metabolic defect that causes an excessive inflammatory response.
  – Finding the allergens and developing a lifestyle, diet, and environment that allow the allergens to be avoided.
  – Modulating the inflammatory process to limit the severity of the response.

(Murray and Pizzorno 2008)
Asthma

Dietary Protocol:

• Increase the amount of fruits and vegetables in the diet:
  – Unless the client is allergic to onions and garlic, encourage them to consume those vegetables liberally.
  – Encourage the client to liberally consume sources of antioxidants like vit C, carotenes, vit E, Se, flavonoids.

• Decrease the amount of food sources of arachidonic acid (meat and dairy).

• Increase the amount of cold-water fish in the diet.

• Eliminate food additives, decrease salt.

(Murray and Pizzorno 2008; Demir et al 2004)
Sources of Arachidonic Acid

Refined carbohydrates and excess oils stimulate inflammatory response, see section on Acne for more details.

(Simopolous 2006; Ghosh and Myers 1997)
Asthma

Dietary Protocol:

• **Eliminate Food Allergens:** Adverse reactions may be immediate or delayed.

  – **Immediate onset sensitivities** are usually due to (in order of frequency): eggs, fish, shellfish, nuts, and peanuts.

  – Foods most commonly associated with **delayed-onset sensitivities** include (in order of frequency): milk, chocolate, wheat, citrus, and food colorings.

(Murray and Pizzorno 2008)
Asthma

Dietary Protocol:

- **Elimination diet:** Elimination diets have been successful in identifying allergens and treating asthma.

- Elimination of common allergens during infancy (the first 2 years) has been shown to reduce allergenic tendencies in children with a strong familial history of asthma.

(Murray and Pizzorno 2008; Hodge et al 1996)
Asthma

Dietary Protocol:

• **Allergen identification diet:** Another approach is to put a client for 1 week, on a hypoallergenic diet – consisting of only turkey, rice, carrots, pears, and sweet potatoes (least allergenic foods).

• Reintroduce one new food every day for 3 days and monitor for adverse reactions.

• This approach can only be applied in **otherwise healthy clients** with no major nutrient deficiencies!
Asthma

Dietary Protocol:

• **Vegan Diet:** Yet another approach is to put a client on a whole-food vegan diet.

• A long-term trial of a vegan diet (elimination of all animal products) provided significant improvement in **92%** of the 25 treated patients who completed the study.

• The diet excluded all meat, fish, eggs, and dairy products.

• Drinking water was limited to spring water (chlorinated tap water was specifically prohibited), and coffee, ordinary tea, chocolate, sugar, and salt were excluded.

• Most fruits, vegetables, pulses and were allowed freely (exception soya and green peas); **most grains were eliminated.**

(Murray and Pizzorno 2008; Lindahl et al 1985)
Asthma

Dietary Protocol:

• **Vegan Diet - Mechanisms:**
  
  – Elimination of major food allergens.
  
  – Altered prostaglandin metabolism.
  
  – Decreasing the availability of arachidonic acid (derived from animal products) as a substrate of inflammatory prostaglandins and leukotrienes appears to explain some aspects of the efficacy of the vegan diet.
  
  – Increased intake of antioxidant nutrients and magnesium: A well-planned vegan diet is characterised by high intake of Mg and antioxidant nutrients.

(Murray and Pizzorno 2008; Lindahl et al 1985)
Asthma

Suggested Supplemental Protocol:

• **EPA** - 1.8g; **DHA** - 1.2 /day or according to client’s individual needs.
• **Vitamin B<sub>6</sub>**: 25 to 30mg/day.
• **Vitamin C**: 10 mg for every kg body weight in divided dosages.
• **Vitamin E**: 400IU/(238mg)/day.
• **Magnesium**: 200 to 350 mg/day.
• **Quercetin**: 400 mg 20 minutes before meals.
• **Selenium**: 100-200 μg/day.

(Murray and Pizzorno 2008; Bender 2002)
Asthma: Protocol - Red Flags

- Make sure you adjust EFA supplementation to client’s dietary EFA intake; against Omega 3 Dominance Syndrome!

- Vit B6 dosage of more than 50mg/day may cause adverse reactions.

- Vitamin E, Selenium can interact with numerous medications (anticoagulants, statins, antibiotics, barbiturates, etc.) check with the client’s GP before prescribing
Asthma

Other Naturopathic Considerations:

• Advise the client to avoid allergens in their environment.

• **Herbal Medicines to Consider:**
  – Ginkgo biloba: Inhibits PAF (platelet activating factor), a key mediator in asthma, inflammation and allergies - improves respiratory function & reduces bronchial reactivity: 120mg q.d.
  – Glycyrrhiza glabra (licorice root): Inhibits phospholipase A$_2$ which cleaves AA from membrane phospholipid, initiating eicosanoid synthesis – also acts as an expectorant.
  – Aloe Vera: Only if client is not dependent on corticosteroids – exact mechanism unknown believed to restore protective mechanisms & augment the immune system: 5ml of 20% solution b.i.d.
  – Ephedra sinica (Ma Huang): Similar to adrenaline acts as a powerful stimulant – banned in the USA it may only be lawfully supplied in the UK within a registered pharmacy and while a pharmacist is present
Asthma

Other Naturopathic Considerations:

- ‘Buteyko‘ is an effective drug-free approach for the management of asthma and other breathing related problems such as COPD, it is a technique designed to help people regain control of their breathing.

- Usually taught at 4-6 day workshop in groups of 5-10 people.

- Clinical trials (1994) Mater Hospital, Brisbane: Asthmatics reduced their symptomatic medication intake by 90% and their steroid medication by 30%.

- [http://www.buteyko.co.uk/](http://www.buteyko.co.uk/)
Acne

General Considerations:

• Acne is the most common of all skin disorders.

• The lesions occur predominantly on the face and, to a lesser extent, on the back, chest, and shoulders.

• It is more common in males, and onset is typically at puberty (somewhat later for the conglobata form).

(Murray and Pizzorno 2008)
Diagnosis - Acne Vulgaris:

- **Open comedones** - Dilated follicles with central dark, horny plugs (blackheads).

- **Closed comedones** - Small follicular papules with (red papules) or without (whiteheads) inflammatory changes.

- **Superficial pustules** - Collections of pus at follicular opening.

- **Nodules** - Tender collections of pus deep in dermis.
Acne

Diagnosis - Acne Conglobata:

- **Cysts** - From nodules that fail to discharge contents to the surface.

- Large deep pustules - From nodules that break down adjacent tissue, leading to **scars**.
Pathophysiology:

- This is what a healthy follicle/pore looks like:
Pathophysiology:

- In acne, sebum (oil) which normally drains to the surface, gets blocked by excess skin cells (hyperkeratinization) lining the inside of the follicle and bacteria begin to grow.

- Both whiteheads and blackheads start out as a **microcomedone** (the picture below is a microcomedone)
Acne

Pathophysiology - Non-Inflammatory Acne:

• **Whiteheads:** When the trapped sebum and bacteria stay below the skin surface, a whitehead is formed; whiteheads may show up as tiny white spots, or they may be so small that they are invisible to the naked eye.
Acne

Pathophysiology - Non-Inflammatory Acne:

- **Blackheads:** A blackhead occurs when the pore opens to the surface, and the sebum, which contains the skin pigment melanin, oxidizes and turns a brown/black color; it is not dirt and cannot be washed away.
Acne

Pathophysiology - Inflammatory Acne:

• A blackhead or whitehead can release its contents to the surface and heal.

• Or, the follicle wall can rupture and inflammatory acne can ensue.

• This rupture can be caused by random occurrence or by picking or touching the skin.

• This is why it is important to leave acne prone skin relatively untouched.
Pathophysiology - Inflammatory Acne:

- **Papule**: A papule occurs when there is a break in the follicular wall. White blood cells rush in and the pore becomes inflamed.
Pathophysiology - Inflammatory Acne:

- **Pustule**: Forms several days later when white blood cells make their way to the surface of the skin; this is what people usually refer to as a "zit" or a "pimple".
Acne

Pathophysiology - Inflammatory Acne:

• An inflamed lesion can sometimes completely collapse or explode, severely inflaming the surrounding skin, and sometimes engulfing neighbouring follicles.

• These lesions are called nodules or cysts.
Acne

Conventional Treatment:

• A number of *topical therapies* are used to treat non-inflammatory acne:
  – *Retinoids* decrease follicular hyperkeratinization.
  – *Acid preparations* (e.g., salicylic acid, azelaic acid, glycolic acid) also decrease follicular hyperkeratinization.
  – *Benzoyl peroxide* is a topical treatment that has antibacterial and comedolytic properties.

• **Severe acne:** Can be treated with intensive topical treatment and/or oral antibiotics that are usually prescribed for 6 months.

• *Isotretinoin* is usually reserved for the most severe cases; it is effective, but has many adverse effects, including teratogenicity.
Acne

Aetiology:

- The severity and progression of acne are determined by a complex interaction among:
  - **Hormones:** The predominant aetiologic factor.
  - Bacteria
  - Inflammation

(Murray and Pizzorno 2008)
Aetiology – Hormones:

- Acne is considered to be linked to an **androgen and other hormone excess**.
  - Free testosterone
  - Dehydroepiandrosterone
    - (DHEA – The most abundant steroid in the body).
  - Dehydroepiandrosterone sulfate
    - (DHEAS – the sulfated version of DHEA via the adrenal cortex).
  
These all stimulate hyperkeratinization of follicles, increase sebum production & stimulate acne lesions.

- High levels of insulin, IGF-1 (insulin-like growth factor mediates the effects of growth hormone).
- Low sex-hormone-binding globulin levels.
- The skin of acne clients shows greater activity of **5-alpha-reductase**, the enzyme that converts testosterone to a more potent androgen, **dihydrotestosterone**.

(Melnik 2009; Cordain 2005; Pochi 1982; Takayasu et al 1980)
Acne

Nutrition and Acne:

• In western industrialized societies, acne is a ubiquitous skin disease.

• Some degree of facial acne has been found in 54% of women and 40% of men older than 25 years of age.

• In contrast, acne has been reported to be absent in non-westernised populations such as the Okinawa islanders (Japan), Ache hunter-gatherers (Paraguay) and Kitavan islanders (Papua New Guinea).

• The complete absence of acne in non-westernised populations points strongly to underlying environmental factors, including diet.

(Goulden et al 1999; Steiner 1946; Cordain et al 2002)
Nutrition and Acne - 1st Mechanism:

Foods causing high glucose, IGF-1 and insulin response.

Chronic high insulin and IGF-1 levels.

Low levels IGFBP-3 (protein binding IGF-1).

Increased sebum production.

Stimulation of hyperkeratinization of follicles.

IGFBP-3 blocks excessive hyperkeratinization, its low levels lead to excessive skin cells lining the follicle.

(Cordain et al 2002, Melnik 2009; Deplewski 1999)
Nutrition and Acne - 2nd Mechanism:

Foods causing high glucose, IGF-1 and insulin response.

- Stimulation of **androgen production** in ovarian and testicular tissue.
- Decreasing levels of SHBP; protein binding excess androgens.

More free circulating androgens.

- Stimulation of sebum production; acne lesions.

(Cordain 2005; Cara 1994; Bebakar 1990; Crave et al 1995;)

Acne
Nutrition and Acne - 3rd Mechanism:

- Foods causing pro-inflammatory changes.
- Stimulation of pro-inflammatory cytokine production.
- Stimulation of sebum production; Acne lesions.

(Gollnick et al 2003; Guy et al 1998)
Nutrition and Acne - 4th Mechanism:

- Excess fat and processed carbohydrates in the diet.
- Increased sebum and lipid secretion in human skin.
- Stimulation of acne lesions production.

(Wolf et al 2004)
Nutrition and Acne – Hyperinsulinaemia:

- No better diet that promotes high insulin levels as a typical Western diet.

  - Low in fibre: Impairs insulin action
  - High in fats: Impairs insulin action
  - High in refined carbs: Increases insulin levels
  - High in animal protein: Increases insulin levels

Nutrition and Acne – Hyperinsulinaemia:

• Unlike processed carbohydrates, animal protein increases postprandial (after the meal) insulin levels without increasing blood glucose to a great extent.

• The food that has the most potent effect on postprandial insulinaemia is milk! (Insulinaemia = abnormally high levels of insulin in the blood).

• The addition of 200 ml milk to a meal with a low GI, increased the insulin response by 300% to a level typically seen from a meal with a very high GI like white bread.

• Higher intake of milk increased insulin levels among 8-year old boys by 103% within 7 days!

(Hoppe et al 2005, Melnik 2009; Liljeberg and Bjorck 2001)
Nutrition and Acne - High IGF-1 Levels:

• Dietary factors promoting high IGF-1 levels:
  – High energy intake.
  – High animal protein intake.
  – Dairy products!!

(Heber 2006)
Nutrition and Acne - High IGF-1 Levels:

- An increase in milk intake from 200 to 600 mL/d corresponded to a 30% increase in circulating IGF-I in children.

- 3 servings of skimmed milk = 10% IGF-1 increase in adult women

(Heaney et al 1999; Allen et al 2002; Chan et al 2002)
Milk: Powerful Insulin and IGF-1 Promoter:

(Melnik et al 2009)
Acne

Dietary Factors **Reducing** Insulin / IGF-1 Levels:

- Plant protein/avoidance of excess protein.
- Lots of fibre.
- Low/moderate dietary fat.
- Eating less energy dense foods.

(Fliesen et al 1989)
Nutrition and Acne – Factors that Increase Inflammation:

- Excess oils (even the ‘good’ oils!).
- Processed carbohydrates.
- Products high in saturated /trans-fats.
- These all initiate a cascade of free radicals and inflammatory change in the bodies almost immediately after the meal!

Omega 6: Omega 3 ratio >5 (ideal <5) (see Lecture 8, Year 1 for more details).

(Esposito and Giugliano 2005, Cordain 2005)
Nutrition and Acne - Factors that Increase Inflammation

- **INSULIN RESISTANCE**
- **High GL foods**
- **High Fat Foods (irrespective of the fat type content!)

**INFLAMMATION**
- ↑ IL-6, ↑ TNF α;
- ↑ sICAM-1;
- ↑ sVCAM-1;

Hyperglycemia → Reactive Oxygen Species (ROS)

Hypertriglyceridemia → oxidizable Lipids

ox-LDL

Protection by Antioxidant Micronutrients e.g. Dietary Flavonols

Endothelial Dysfunction

Diabetic complications | Atherosclerosis

(Esposito i Giugliano 2006; Reaven et al 1993; Reaven et al 1996; Steinberg et al 1991)
Acne

Inflammation - It is Easy to Overload on Omega 6!

• **International Society for the Study of Fatty Acids and Lipids:**
  – Linoleic Acid (n-6) LA-2% (of total calories)
  – Alpha Linolenic Acid (n-3) ALA-0.7%

• A healthy upper limit of the intake of LA -3%!

• **European guidelines on cardiovascular disease prevention**
  – LA –between 5-8%

(ISSFAL 2008; Graham et al 2007)
Acne

Inflammation - It is Easy to Overload on Omega 6!

- **1 tbs of sunflower oil**: 9g of LA; 81 kcal from LA, 4% if 2000 kcal
- **1 tbs of grapeseed oil**: 9.5 g of LA; 85 kcal from LA, 4.5% if 2000 kcal
- **1 tbs of olive oil**: 1.3 g of LA; 85 kcal from LA, 0.5% if 2000 kcal
- **8 Brazil nuts**: 8.2 g of LA; 75 kcal from LA, 3.75% if 2000 kcal
- **1.3 oz pumpkin seeds**: 8.2 g of LA; 75 kcal from LA, 3.75% if 2000 kcal

(USDA Nutrient Database)
Acne

Inflammation:
Watch Out for Omega 6:Omega 3 Ratio!

- Spinach: 0.2:1
- Strawberries: 1:1
- Mangoes: 0.25:1
- Lettuce: 0.4:1
- Kale: 0.7:1
- Salmon: 0.04:1
- Kidney Beans: 0.5:1
- Beef: 2.71:1
- Cheese: 2:1
- Walnuts: 4:1
- Pumpkin Seeds: 100:1
- Sunflower Seeds: 320:1
- Brazil nuts: 1025:1
- Olive Oil: 13:1

(USDA Nutrient Database)
Acne

Nutritional Protocol:

- Low fat; high fibre
- Adequate (not excessive!), mostly plant protein
- Adequate (not excessive) calories
- Lots of whole plant foods
- Minimize oils, choose nuts/seeds with beneficial n-6: n-3 ratio

Minimize dairy products!

↓ Insulin
↓ Growth factors (IGF-1)
↓ Inflammation
↓ Glucose
↓ Excessive n-6
Lifestyle Protocol:

- **Exercise lowers IGF-1 and insulin levels**
  - After 11 days of moderate exercise and low fat plant based diet IGF-1 was reduced by 20% and IGF-1 clearance increased by 53%.

(Ngo et al 2002 ; Hawley and Lessard 2008)
Supplemental Considerations:

- **Chromium** (or chromium-rich yeast) is known to improve glucose tolerance and enhance insulin sensitivity; and has been reported in an uncontrolled study to induce rapid improvement in clients with acne.

- **Zinc** (sulphate and gluconate) has been shown to be effective in acne treatment in some studies.

- **Vit A (retinol)** has been very effective in treating acne, reducing sebum production and the hyperkeratosis of the follicles; however, retinol has been shown to be effective in treating acne when used at high, potentially toxic, dosages (i.e. 300,000 to 400,000 IU/day for 5 to 6 months) - only under medical supervision!

- **Vit E and Selenium** - Preliminary studies suggest that vit E and Selenium can be effective, possibly by reducing levels of free radicals and thus inflammation.

Acne

- Nicotinamide inhibits *Propionibacterium acnes*-induced IL-8 production in keratinocytes through the NF-κB and MAPK pathways.

- Nicotinamide gel (2-4%) is available over the counter and has been found to be an effective therapeutic for moderate acne and useful in reducing sebum production.

Acne

Suggested Supplement Protocol:

• **Vitamin E**: 400IU/(238mg)/day

• **Zinc**: 20 mg/day

• **Selenium**: 100-200 µg/day

• **Yeast (brewer's)** - 1 tablespoon twice a day (if client is susceptible to gout, use chromium supplement instead).

(Murray and Pizzorno 2008; Bender 2002)
Acne: Protocol - Red Flags:

- A low-fat, plant-based, high fibre diet can significantly lower insulin and glucose levels; clients taking insulin or oral hypoglycaemic drugs might need to decrease their medications; work with the client’s doctor!

- Long-term Zn supplementation can impair absorption of other minerals, advise the client as to the duration of supplementation.

- Vitamin E, Selenium and Zinc can interact with numerous medications (anticoagulants, statins, antibiotics, barbiturates, etc.) - check with the client’s GP before prescribing.
Acne

Other Naturopathic Considerations:

• Tea tree oil (5% to 15%) preparations.

• Azelaic acid (20%) preparations (found in wheat, rye & barley).

• Thorough daily cleansing with calendula soap.

• Adequate sun exposure.

(Murray and Pizzorno 2008)
Atopic Dermatitis

Diagnosis:

• Chronic, pruritic, inflammatory skin condition.

• Skin is dry and hyperkeratotic.

• Lesions include papules, eczema (patches of erythema, exudation, and scaling with small vesicles formed within the epidermis), and lichenification (hyperpigmented plaques of thickened skin).

• Scratching and rubbing leads to lichenification.

• Personal or family history of atopy.

(Murray and Pizzorno 2008)
Atopic Dermatitis

Aetiology:

- Atopic dermatitis (AD) (eczema) is a common condition with a prevalence of 2.4 - 7% of the population.

- It is largely an immediate hypersensitivity disease:
  - Serum immunoglobulin (Ig) E is elevated in 80% of clients.
  - Most clients have positive skin, and other allergy tests.
  - There is a positive family history in two thirds of eczema clients.
  - Many eventually develop allergic rhinitis or asthma, or both.
  - Most improve with an elimination diet.

(Murray and Pizzorno 2008)
Atopic Dermatitis

Pathophysiology:

• AD can be divided into 2 forms:
  – **Extrinsic**: IgE-mediated (70 to 80% of cases).
  – **Intrinsic**: Non IgE-mediated (20 to 30% of cases).
Atopic Dermatitis

Pathophysiology:

• **Extrinsic AD**: This form occurs when environmental exposures trigger immunologic, usually allergic (i.e. IgE-mediated), reactions in genetically susceptible people.

• Common environmental triggers include:
  – **Foods** (e.g. milk, eggs, soy, wheat, peanuts, fish).
  – **Airborne allergens** (e.g. dust mites, molds, dander).
  – **Staphylococcus aureus** colonisation on skin due to deficiencies in endogenous bactericidal peptides.
  – **Topical products** (e.g. cosmetics).
Atopic Dermatitis

Pathophysiology:

- **Intrinsic AD**: this form is not mediated by IgE.

- Intrinsic AD is non-familial and its pathophysiology is generally not well understood.
Atopic Dermatitis

Pathophysiology:

- Leukocytes from people with atopic dermatitis have:
  - Decreased cyclic adenosine monophosphate (cAMP) levels due to increased cAMP-phosphodiesterase activity.
  - Decreased level of prostaglandin precursors which suggests a deficit in the function of the delta-6-desaturase enzyme.
  - The lack of intracellular cAMP results in increased histamine release and decreased bactericidal activity.

(Saarinen et al 1995)
Atopic Dermatitis

Conventional Treatment:

• **Antihistamines** to relieve itching symptoms.

• **Antibiotics** when clients develop a bacterial infection in the affected area or have pustular disease.

• **Topical corticosteroids** are used in the lowest possible therapeutic strength to treat active atopic dermatitis; occasional use of topical steroids between episodes are used to reduce the likelihood of recurrence.

• **Systemic corticosteroids**, such as prednisone, may be used for a short duration when exacerbations occur.

(Murray and Pizzorno 2008)
Atopic Dermatitis

Nutrition Considerations - Food Allergy:

• Studies have documented the major role of food allergy in AD.

• The most common allergens are: milk, eggs, peanuts, fish, soy, wheat, citrus, and chocolate.

• Research shows that breastfeeding offers significant prophylaxis against atopic dermatitis, as well as allergies in general.

• In breastfed infants who develop atopic dermatitis, it is usually the result of transfer of the above mentioned allergic antigens in the breast milk.

• Maternal avoidance of these common allergens is associated with complete resolution in the majority of cases

(Saarinen et al 1995; Murray and Pizzorno 2008; Cant et al 1986)
Atopic Dermatitis

Nutrition Considerations: Food Allergy - Leaky Gut:

• The presence of food allergies is thought to contribute to the development of “leaky gut” (increased gut permeability).

• In increased gut permeability, there is increased antigen load on the immune system which subsequently overwhelms the immune system and increases the likelihood of developing additional allergies.

• Studies confirm that hypoallergenic diets decrease gut permeabilities and are associated with improvements in atopic eczema.

• Elimination of allergenic foods appears to stop the development of new allergies.

(Majamaa and Isolauri 1996; Agata et al 1993)
Atopic Dermatitis

Nutrition Considerations - Candida Albicans:

• An overgrowth of the common yeast Candida albicans in the gastrointestinal tract has been implicated as a causative factor in allergic conditions including atopic dermatitis.

• Elevated levels of anti-Candida antibodies are common in atopic individuals.

• The severity of lesions tends to correlate with the level of antibodies to Candida antigens.

• Anti-Candida therapy may result in significant clinical improvement of atopic dermatitis.

(Savolainen et al 1993)
Atopic Dermatitis

Nutrition Considerations - EFA and Prostaglandin Metabolism:

• Clients with atopic dermatitis appear to have altered EFA and prostaglandin metabolism.

• Analyses of fatty acids in plasma, red blood cells, and monocytes in clients with atopic dermatitis demonstrated:
  – A tendency for linoleic acid levels to be increased.
  – Gamma-linolenic acid and the long-chain omega-3 oils-eicosapentaenoic acid (EPA) and docosahexanoic acid (DHA)-tend to be relatively low.

(Manku et al 1982; Lindskov and Holmer 1992)
Nutrition Considerations - EFA and Prostaglandin Metabolism:

• Supplementation with Omega 3 EFA (especially the long chain ones) and/or eating fatty fish twice a week may bring some benefits for people with atopic dermatitis.

• A meta-analysis (2004) of placebo-controlled trials has not shown clinically relevant effects of EFAs on atopic dermatitis. Supplementation with evening-primrose oil did not show any significant benefit. Response to specific EFA therapy is likely to depend upon the individuals current EFA ratio and their individual needs.

• Consumption of fish oil by the mother during pregnancy is associated with positive modulation of immune response to common allergens and reduction in atopic dermatitis in the first years of life extending to adolescence in some participants.

(Calder et al 2011; Dunstan et al 2003; Van Gool et al 2004)
Nutrition Considerations - Inhibitors of Excess Histamine Release:

• Agents that stimulate cAMP production or inhibit cAMP phosphodiesterase - reduce the inflammatory process in atopic dermatitis by reducing the shunting to histamine.

• The following botanicals, phytochemicals have been shown to inhibit diesterase:
  – Liquorice
  – Quercetin
  – Rutin
  – Naringen
  – Grape seed
  – Green tea
  – Ginkgo biloba

Atopic Dermatitis

Dietary Protocol:

• The client should begin a 4-day rotation diet and eliminate all major allergens (milk, eggs and peanuts account for the offending food in approximately 81% of cases).

• As the client improves, allergens can be slowly reintroduced and a stringent rotation diet can be reduced.

• Limit animal products and oils rich in Omega 6 (sources of proinflammatory mediators).

• Include plenty of sources of antioxidants and flavonoids.

• Add fatty fish such as salmon, mackerel, herring and halibut or plant sources of Omega 3.

• Follow anti-candida protocol.

(Murray and Pizzorno 2008)
Atopic Dermatitis

Suggested Supplement Protocol:

• Zinc supplementation may be helpful given that low zinc levels are common in atopic dermatitis and given the importance of zinc to proper EFA metabolism.

• **Zinc:** 20 mg/day (decrease as condition clears).

• **Quercetin:** 200 to 400 mg three times/day (5 to 10 minutes before meals).

• **EPA and DHA:** 540 and 360 mg daily or flaxseed oil 10 g daily.

• **Probiotics: dosage:** 1 to 10 billion viable Lactobacillus acidophilus and Bifidobacterium bifidum cells daily.

(David et al 1990, Murray and Pizzorno 2008)
Atopic Dermatitis

Other Naturopathic Considerations:

• Advise the client to avoid allergens in their environment.

• Bathing in Dead Sea salt solutions – Magnesium is likely to be responsible for improved barrier function, cell differentiation and hydration.

• Herbal Medicines to consider:
  – Glycyrrhiza glabra: Anti-inflammatory & anti-allergenic effects.
  – Ginkgo biloba: Anti-PAF activity.

(David et al 1990, Murray and Pizzorno 2008; Proksch et al 2005)
Other Naturopathic Considerations Cont.:

- **Topical preparations:**
  - Chamomile: Anti-inflammatory.
  - Witch Hazel: Anti-inflammatory.
  - MSM cream may help dry (not weeping) eczema.

- Client’s with gene mutations to *filaggrin* – a type of protein found in the skin – have been found to struggle to stay free from symptoms for longer than 5mths at a time.

(David et al 1990, Murray and Pizzorno 2008; Proksch et al 2005)
Psoriasis

General Considerations:

• Psoriasis is a chronic, non-contagious, multisystem, inflammatory disorder.

• 2-5% of the Western population suffer from psoriasis.

• Incidence of psoriasis is dependent on the climate and genetic heritage of the population.

• It is less common in the tropics and in dark-skinned persons.

• It is slightly more common in women than in men.

(Gordon and Rosh 2009)
Psoriasis

**Diagnosis:**

- Sharply bordered reddened rash or plaques covered with overlapping silvery scales.

- Characteristically involves the scalp, the extensor surfaces (back of the wrists, elbows, knees, buttocks and ankles) and sites of repeated trauma.

- Family history in 50% of cases.

- Nail involvement results in characteristic "oil drop" stippling (yellowish brown spots under the nail plate).

- Possible arthritis.

(Murray and Pizzorno 2008)
Psoriasis

Aetiology and Pathophysiology:

• Aetiology and pathophysiology of this condition is **still not understood**.

• Multiple theories exist regarding triggers of the disease process including infectious episode, traumatic insult and/or stressful life event.

• In many people no obvious trigger exists at all.

• Once triggered, there is a substantial immune cell recruitment to the dermis and epidermis resulting in the characteristic psoriatic plaques.

• It has been suggested that activated immune cells release cytokines resulting in proliferation of keratinocytes.

(Murray and Pizzorno 2008; Gordon and Rosh 2009)
Aetiology and Pathophysiology:

- Psoriasis is a hyper-proliferative skin disorder; the rate of cellular division rate in psoriatic lesions is 1000 times greater than in normal skin.

- Excessive replication is the result of an imbalance in the ratio between two internal compounds that control the rate at which skin cells divide: cyclic adenosine monophosphate (cAMP) and cyclic guanidine monophosphate (cGMP).
  - Increased levels of cGMP - Increased levels of cell proliferation (in psoriasis).
  - Increased levels of cAMP - Enhanced cell maturation and decreased cell replication.

(Murray and Pizzorno 2008; Voorhees 1975; Robbins and Cotran 1979)
Psoriasis

Aetiology and Pathophysiology:

• There is a clear relationship of psoriasis with conditions like coeliac disease and Crohn's disease.

• Bowel mucosa of psoriatic clients without bowel symptoms have shown microscopic lesions and greater intestinal permeability.

• **Naturopathic hypothesis**: Factors leading to poor intestinal function encourage greater intestinal permeability and inflammation allowing antigens to travel through the blood stream and initiate activated immune cascades in susceptible tissues.

(Ogetti et al 2003; Najarian et al 2003; Scarpa et al 2000; )
Psoriasis

**Conventional Treatment:**

- **Topical corticosteroids** are used for widespread plaques and lesions that are resistant to other therapies; resistance to steroid creams can develop quickly, and withdrawal may cause exacerbation of disease; linked to numerous side effects.

- **Vitamin D analogues** (e.g. calcipotriene) slow keratinocyte growth, flatten lesions, and remove scale.

- **Anthralin** is a medicine that has been used for more than a century; it is believed to normalize DNA activity in skin cells; causes skin irritation.

- **Tazarotene**, a retinoid, normalizes DNA activity in skin cells, but may cause skin irritation and is contraindicated in pregnant women.

(Ojetti et al 2003; Najarian et al 2003; Scarpa et al 2000; )
Psoriasis

Nutritional Considerations - Incomplete Protein Digestion:

• Some improperly digested or poorly absorbed polypeptides and amino acids are metabolised by bowel bacteria into toxic polyamines (putrescine, cadaverine, spermidine).

• These polyamines:
  – Inhibit formation of cAMP, inducing excess cell proliferation.
Nutritional Considerations - Bowel Toxaemia:

• Gut-derived toxins can cause increases in cGMP levels-increasing rate of cell proliferation.

• These toxins include:
  – Endotoxins (cell wall components of bacteria).
  – Candida albicans.
  – Immune complexes.

• A diet low in fibre is associated with increased levels of gut-derived toxins since fibre components bind bowel toxins and promote their excretion.

(Rosenberg and Belew 1982; Rao and Field 1984; Juhlin and Vahlquist 1983)
Psoriasis

Nutritional Considerations - Impaired Liver Function:

• Psoriasis has been linked to several microbial byproducts in the blood.

• The liver filters and detoxifies the blood.

• If the liver is overwhelmed by excessive levels of microbial toxins in the blood or if the liver’s detoxification ability is impaired.
  – The level of toxins in the blood will increase.
  – With potential negative effects on psoriasis.

• Alcohol consumption worsens psoriasis since it increases toxin absorption from the gut and worsens liver function.

(Weber and Galle 1983; Pietrzak et al 1998; Monk and Neill 1986)
Psoriasis

Nutritional Considerations – EFA:

• In the skin of individuals with psoriasis, the production of inflammatory leukotrienes from arachidonic acid is many times greater than normal.

• Leukotrienes promote increased cGMP levels.

• Meat, animal products and dairy are rich sources of arachidonic acid.

• Several studies have demonstrated that supplementing the diet with 10 to 12 g of fish oils (providing 1.8 g EPA and 1.2 g DHA) results in significant improvement.
  – This is due to the competition of EPA for arachidonic acid binding sites, which results in the inhibition of the production of inflammatory compounds.

(Vorhees et al 1983; Murray and Pizzorno; Bittiner 1988; Grimmunger ; Maurice et al 1987)
Studies on Different Dietary Regimes:

- Several dietary regimes have been shown to be helpful:
  - Fasting
  - Vegetarian diets
  - Gluten-free diets
  - Elimination diets

- Probably due to decreased levels of gut-derived toxins and polyamines and proinflammatory mediators.

- Probably due to decrease in antigenic load.

(Lithell et al 1983; Douglas 1980)
Psoriasis

Dietary Protocol:

- **Consume a nutrient-dense diet:**
  - Rich in whole, unprocessed, preferably organic foods.
  - Emphasise plant foods (fruits, vegetables, beans, seeds, nuts, whole grains).
  - Cold water fish (ca. 150g of wild salmon, mackerel, herring) and ground flaxseed (1 tbsp) / or fish-oil supplements.
  - Eliminate wheat and other sources of gluten (rye, barley - check if client tolerates oats).
  - Minimise animal products.
  - Minimise processed foods and alcohol.

(Murray and Pizzorno 2008)
Psoriasis

Supplemental Considerations:

- **Omega-3 fatty acids**: If client is unwilling to consume fish and/or flaxseeds; choose only high quality products with no lipid peroxides.

- **Zinc and Vit A**: Decreased levels of vitamin A and zinc are common in clients with psoriasis.
  - Vitamin A inhibits bacterial decarboxylase, the enzyme that converts undigested amino acids into polyamines.
  - Vit A deficiency may promote gut permeability.
  - Both Zn and vit A play critical role in the health of skin.

(Murray and Pizzorno 2008; Majewski 1989)
Psoriasis

Supplemental Considerations:

• Selenium and Vit E:
  – Glutathione peroxidase (GP) and selenium levels are low in psoriatic clients.
    • Possibly due to alcohol abuse, malnutrition and the excessive skin loss.
    • The depressed levels of GP normalize with oral selenium and vitamin E therapy.

(Juhlin et al 1982; Serwin et al 2003; Michaelsson et al 1989)
Psoriasis

Supplemental Considerations:

- **Vit D**: Has anti-proliferative properties and controls the rate of cell replication.
  - Clients with severe psoriasis have been found to have significantly low serum levels of active vit D.
  - Oral supplementation with vit D resulted in significant improvement in clients with psoriasis.

(Morimoto et al 1986, Takamoto et al 1986; Staberg et al 1987)
Psoriasis

Supplemental Protocol:

• **EPA**: 1.8g; DHA- 1.2g /day or according to client’s individual needs

• **Zinc**: 20mg/day

• **Vit A**: 5000IU

• **Selenium**: 100-200 μg/day

• **Vitamin E**: 400IU/(238mg)/day

• **Vitamin D**: 5-10μg/day

(Murray and Pizzorno 2008; Bender 2002)
Psoriasis

Psoriasis: Protocol - Red Flags:

• Never recommend vit A supplements to pregnant women due to potential teratogenic effects!

• Make sure you adjust EFA supplementation to client’s dietary EFA intake.
  – Guard against Omega 3 Dominance Syndrome.

• Long-term Zn supplementation can impair absorption of other minerals, advise the client as to the duration of supplementation.

• Vitamin E, Selenium and Zinc can interact with numerous medications (anticoagulants, statins, antibiotics, barbiturates, etc) - check with the client’s GP before prescribing.
Psoriasis

Other Naturopathic Considerations:

• Herbal medicines to consider:
  – H. canadensis (goldenseal)
  – S. marianum (milk thistle)

• Adequate sun exposure.
Seborrheic Dermatitis

General Considerations:

• Seborrheic dermatitis is a common condition similar in appearance to eczema.

• In adolescents and adults, seborrheic dermatitis is commonly called "dandruff"; in babies it is known as "cradle cap"; although it may occur on different body parts.

• The condition occurs either in infancy (usually between 2 and 12 weeks of age) or in the middle-aged or elderly and has a prognosis of lifelong recurrence.
Seborrheic Dermatitis

Diagnosis:

• Superficial reddened small bumps and flaking, white scaly patches on the scalp, eyebrows, forehead, cheeks, behind the ears, over the breastbone (sternum), and/or skin folds (around the nose, the armpit, groin, and neck).

• Scales anchor to hair shafts.

• Usually does not itch.

• The scale may be yellowish and either dry or greasy.

• Seasonal; worse in winter.

(Murray and Pizzorno 2008)
Seborrheic Dermatitis

Aetiology and Pathophysiology:

• The cause of seborrheic dermatitis is unknown.

• Implicated factors:
  – Genetic predisposition.
  – Emotional stress.
  – Diet
  – Hormones
  – Infection with yeast-like organisms.

(Murray and Pizzorno 2008)
Seborrheic Dermatitis

Aetiology and Pathophysiology:

- Seborrheic dermatitis is now recognized as one of the most common manifestations of acquired immunodeficiency syndrome (AIDS), affecting as many as 83% of persons with the syndrome.

- This recent observation suggests that infection might be the cause of seborrheic dermatitis.
Seborrheic Dermatitis

Conventional Treatment:

- **Anti-dandruff shampoos** containing **salicylic acid** (some brand names: Scalpicin, X-Seb), **Selenium sulfide** (brand names: Exsel, Selsun Blue) **zinc pyrithione** (some brand names: DHS Zinc, Head & Shoulders); or **coal tar** (some brand names: DHS Tar, Neutrogena T/Gel, Polytar).

- **Seborrheic dermatitis of the skin creases**: steroid lotions are used in adolescents and adults.

- **Cradle cap** is treated with products that are not as strong as those used in adults (e.g. a mild, non-medicated baby shampoo); brushing baby's scalp with a soft brush, like a toothbrush, can help loosen scales or flakes.
Seborrheic Dermatitis

Nutritional Considerations - Food Allergy:

• Seborrheic dermatitis usually begins as "cradle cap" and, although not primarily an allergic disease, has been associated with food allergy.

• 67% of clients exhibit some form of allergy by 10 years of age.
Seborrheic Dermatitis

Nutritional Considerations - Biotin Deficiency:

• The underlying factor in infants appears to be a biotin deficiency.

• Because a large portion of the human biotin supply is provided by intestinal bacteria, the absence of normal intestinal flora may be responsible for biotin deficiency in infants.

• A number of articles have demonstrated successful treatment of seborrheic dermatitis with biotin in both the nursing mother and the infant.

(Niesenson 1957; Niesenson and Barness 1972)
Seborrheic Dermatitis

Nutritional Considerations - B-vitamin Deficiency:

• In adults, treatment with biotin alone is usually of no value.

• It has been postulated that long-chain fatty acid synthesis is impaired in seborrheic lesions.

• B vitamins are vital for fatty acid metabolism and may prove beneficial in treatment, although there are not many studies supporting the efficacy of this approach.

(Murray and Pizzorno 2008)
Nutritional Considerations - Candida Albicans:

- It has been suggested that the infection with Candida Albicans may exacerbate the symptoms.

(Murray and Pizzorno 2008)
Seborrheic Dermatitis

Dietary Protocol:

• **Prescribe a nutrient-dense diet:**
  – Identify and eliminate all possible allergens.
  – In infants and their nursing mothers: Alleviate biotin deficiency.
  – In adults: Correct possible vitamin B deficiencies.
  – When infection with Candida suspected apply anti-Candida dietary protocol.

(Murray and Pizzorno 2008)
Seborrheic Dermatitis

Suggested Supplemental Protocol:

• **Biotin**: 3 mg twice/day

• **B complex**: 50 mg /day

• **Additionally Murray and Pizzorno (2008) recommend:**

  • **Zinc**: 20 mg/day

  • **Flaxseed oil**: 1 tbsp/day

  As direct (flaxseed oil) and indirect (Zn as cofactor in EFA metabolism) anti-inflammatory measures.

(Murray and Pizzorno 2008; Majewski 1989)
Seborrheic Dermatitis

Seborrheic Dermatitis: Protocol - Red Flags:

- Vit B6 dosage of more than 50mg/day may cause adverse reactions.

- Long-term Zn supplementation can impair absorption of other minerals, advise the client as to the duration of supplementation.
Seborrheic Dermatitis

Other Naturopathic Considerations:

• **Topical Treatments:**
  – Pyridoxine (B6) ointment
  – Aloe vera gel

• **Homeopathy** has been shown to be effective in a double-blind, placebo-controlled study.

(Murray and Pizzorno 2008; Vardy et al 1999; Smith et al 2002)
Additional Reading

• Remember to do your additional readings, see your Learning Guide.

Thank you.