Advanced Nutrition and Food Therapy

Clinical Research Assignment 50%

Psoriasis in middle aged adults (50 years +)

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Advanced Nutrition and Food as Therapy
Assignment, weighing 50%
3,000 words

An Evidence Based Nutritional Approach to a Population Group
A written literature review of the naturopathic nutritional approach to a health disorder or a specified life stage in a population group. The emphasis is on presenting and critically evaluating current information on the health disorder from primary resources.

OBJECTIVES

1. Understand and analyse current nutritional research on the intake of specific conditions or life stages of key population groups & identify specialist nutritional treatment to either optimise health and/or correct specific health complaints.

2. Compare and contrast nutritional agents and dietary intakes and use available research to recommend effective treatment strategies for key population groups.

3. Develop a coherent argument for the utilisation of nutritional agents and dietary intake within key population groups.

4. Critically evaluate available research to inform the choice of nutritional agents and dietary therapy used in the treatment of the key population groups.

5. Conceptualise the biochemical mode of action within the nutritional and dietary treatment generated for a key population group and validate the treatment hypothesis with available peer-reviewed research.

Introduction – 300 words (5 marks) General overview of the population group.

Nutritional causes or deficiencies (20 marks)
Clearly differentiate between nutritional cause and deficiency affecting a life state or disorder and nutrients that have been disrupted as being therapeutic agents. Eg smoking – nut cause that depletes zn & vit C which predisposes to dx.

Treatment and evidence based research (70 marks) Most research focussed here.

Compare Australian RDI's (10 marks) Identify any that might be inadequate.

Conclusion (20 marks) briefly review evidence for your condition and summarise quality of the evidence and future direction for research. Give conclusion for best treatment option.

Structure, style, formatting (10 marks)

References (15marks)

Total 150

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1. INTRODUCTION (5 marks)

Psoriasis

Is a chronic inflammatory, non-infectious skin disease characterised by well-defined erythematous plaques with silvery scales that prefer extensor surfaces and scalp, by a chronic fluctuating course. There are 2 epidemiological patterns: 1. Onset in teenage and adult years, often associated with a family history. 2. Onset occurs at 50-60 years of age. Exact mechanisms are poorly understood. (Davidson's, 2002) It is this later (50-60yrs old) group, that this nutritional analysis is based on.

Aietiology: keratinocytes hyperproliferate with increased mitotic index and abnormal pattern of differentiation with retention of nuclei in the stratum corneum (normal dead corneum cells don't have nuclei). Large inflammatory cell infiltrate of inflammatory mediators is seen. Hyper-proliferation may be secondary to inflammatory infiltrate, increasing keratinocyte proliferation. (Davidson's, 2002) The rate of cell division in psoriatic lesions is 1000x greater than normal skin cells. (Pizzorno, 2006) Cell division is regulated by cAMP and cGMP balance. cGMP is associated with proliferation and higher amounts are seen in patients with psoriasis. (Voorhes, 1975; Robbins, 1979)

Disordered cell proliferation is reflected by the increase in number of mitoses visible in psoriatic plaque. The transit time for keratinocytes in the basal layer, to leave the epidermis, is shortened in psoriasis from 28 to 5 days. (Davidson's, 2002)

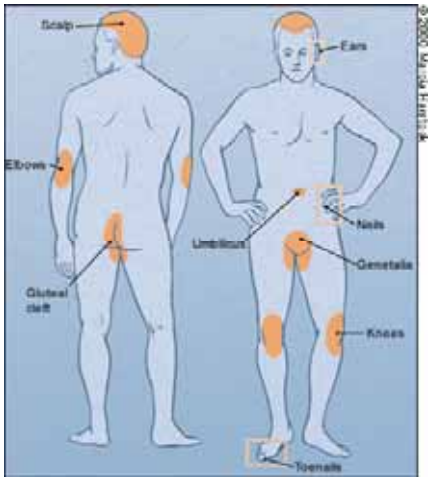

FIGURE 1. Common areas of distribution of psoriasis. Lesions are usually symmetrically distributed and characteristically located on the ears, elbows, knees, umbilicus, gluteal cleft and genitalia. The joints (psoriatic arthritis), nails and scalp may also be affected. (AFP, 2000)

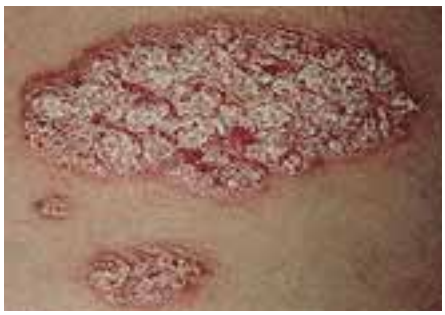

FIGURE 2. A primary lesion of plaque-type psoriasis. Typical lesions are well-demarcated, thick, erythematous plaque with a silvery scale.

Large familial association: if one parent has psoriasis, chances of inheritance are 15-20%. If both, probability of inheritance is 0.5. (Davidson's, 2002)

Precipitating factors: chronic disease characterised by variation in temporal and special extent. Factors thought to exacerbate the disease are: trauma (scratching, surgical wounds),

infection (ß-haemolytic streptococcal throat infections can precipitate guttate psoriasis), sunlight (UV can worsen psoriasis – rarely), drugs (anti-malerials, ß-blockers, lithium), emotion (anxiety). (Davidson's, 2002)

There are 5 classifications of psoriasis (See Appendix 1)

Management: patient's state of mind is imperative to treatments, particularly due to unsightliness of the condition. (Davidson's 2002)

Treatment involves topical agents (corticosteroids, vitamin D agonists, 'weak' tar preparations), UV therapies, systemic agents (retinoids, immunosuppressives) eg. methotrexate. Major concerns with methotrexate: decrease in white blood cells due to immunosuppressive action, hepatic fibrosis and cirrhosis. May cause serious side effects. (Davidson's 2002)

Methotrexate may cause liver damage (therefore alcohol must be avoided), lung damage, kidney damage (high fluid intake is recommended especially with exercise), bone marrow suppression, intestine damage, severe rash and decreased immune system activity possibly leading to serious infections and increased risk of lymphoma. (NLM, 2007)

2. NUTRITIONAL CAUSES OR DEFICIENCIES (20 marks)

PREDISPOSING FACTORS
Hereditary link, stress, possible autoimmune presentation of psoriasis.

EXCITATORY
Allergies, Infections, lowered immunity, inflammation, reduced bowel mucosa nutritional deficiencies, low EFA's, incomplete protein digestion and other nutrients poor liver function and bile production, stress and anxiety.

SUSTAINING
Inflammation, hyperproliferation of keratinocytes, low-grade infection and therefore immune system, low bile production. (Many of these factors fall in both Excitatory and Sustaining causes). Diet, stress, high IgE.

Immune system. Activation of T lymphocytes and release of cytokines results in proliferation of keratinocytes. This response is due to 'unidentified antigens' causing maturation of epidermal antigen-representing cells that migrate to regional lymph nodes. There, the antigen-presenting cells interact with naïve T cells, resulting in T cell activation. (Lebwohl, 2003) Active immune cells enter circulation, extravasating through blood vessels to inflammatory sites in the skin (Pizzorno, 2006). With compromised immune response, nutrients to support immune function are required.

Interestingly, methotrexate was initially given to suppress cell growth and multiplication, however it is postulated that it's action is primarily through its influence on the immune system. (Davidson's, 2002)

Bowel mucosa is compromised in psoriatic patents (even without bowel symptoms present) and microscopic lesions and increased intestinal permeability are seen. (Scarpa et al, 2000; Humbert et al, 1991) This provides greater understanding for the presence of food allergies, inflammation and reduced liver function often seen in psoriasis patients. (Pizzorno, 2006)

Many gut-derived toxins are implicated in psoriasis, notably bacteria streptococcus, raised IgE and IgA immune complexes. (Rosenberg, 1982) A low fibre diet is related to increased gut-derived toxins (Pizzorno, 2006). Gliadin, a glycoprotein found in gluten, induces zonulin release in intestinal epithelial cells. Zonulin regulates intestinal permeability, an increase

allows undigested food particles, toxins, bacterial and viral particles access to the immune system. Activation of the zonulin pathway by PKC mediated cytoskeleton re-organisation and tight junction opening leads to a rapid increase in intestinal permeability. (Clemente, 2003, Celiac centre) Gluten sensitivity has been observed in psoriasis patients and a gluten free diet may see reduction in symptoms. (Further discussion below) (Michaëlsson et al, 2000)

Nutritional deficiencies & incomplete protein digestion or poor absorption can result in elevations of polypeptides and amino acids in the bowel, where they are metabolized into toxic compounds. Toxic metabolites of arginine and ornithine, known as polyamines, are higher in psoriatic patients. These polyamines inhibit cAMP, increasing proliferation rate. (Pizzorno, 2006)

Ineffective protein digestion and assimilation can place stress on the liver to break it down, and excretion via the kidneys. A low protein diet may benefit psoriasis when there are kidney problems. (Morrision, 2001) Unrestricted intake of protein-rich foods is accompanied by sustained increases in glomerular capillary pressures and flows. Progressive loss of renal function may be retarded by restriction of protein intake. Protein restriction appears to preserve renal function by limiting intra-renal capillary hypertension and hyperperfusion. (Myer et al, 1983) However, soy protein may have beneficial effects and provide an alternative to animal protein. (Anderson, 1993)

Malabsorption. Prevalent among psoriatic patients and coeliac disease, bacterial overgrowth, parasitic infestations and eosinophilic gastroenteritis have been cited as possible causes. (Ojetti et al, 2006) This is of additional importance in this over 50 age group, particularly as they move into their 70's where their ability to produce hydrochloric acid is reduced and therefore digestive function and absorption is compromised.

Liver function is integrally linked to psoriasis. Its major function is detoxification and filtration of blood returning through the portal circulation from the bowels. (Pizzorno, 2006) Hepatic structure is altered in psoriasis. (Pietrzak, 1998) Psoriasis is linked to microbial byproducts in the blood, thus if the liver is overwhelmed by toxins it's detoxification function is compromised and systemic toxin levels rise along with psoriatic symptoms. Another reason why alcohol should be avoided. (Pizzorno, 2006)

Bile production is important to detoxify endotoxins in the intestine and prevent them translocating into the bloodstream causing pathological conditions and inflammation. A study by Gyurcsovics et al (2003) showed 78.8% of the 551 subjects receiving bile acid became asymptomatic and a staggering 95.1% in the acute psoriasis group. Alongside these findings, colon epithelial cell proliferation index was significantly lower. Long-term use of bile acid supplementation is linked to cancer. However, it is important to support and increase bile production and function in safer ways eg. bitter foods to stimulate bile secretion. (Haas, 2006)

Infection. There is a strong link between parasitic infection and psoriasis, malabsorption and pancreatic insufficiency. Anthelmintic therapy has shown positive effects via decreasing the degree of acanthosis, increasing intracellular regeneration of epidermocytes, and suppressing inflammatory reaction of the derma and hyperplasia of immunocompetent cells. (Nepomnyashchikh et al, 2004)

Enhancement of general immunity along with cell-mediated immunity is vital in reducing the severity and frequency of outbreaks. (Pizzorno, 2006)

Stress and Anxiety. The impact of psoriasis on patients' quality of life may be quite destructive, looking at disease status alone does little to describe the true burden of illness. (Sampogna, 2004) A common feature of the disease, stress and anxiety exacerbate symptoms.

Fortune et al (2003) discusses the existence of a brain-skin axis, linking psychological distress and excessive worrying with significant detrimental affects on treatment outcome in patients with psoriasis. Concluding that adjunctive psychological intervention before and during treatment may be of benefit. (Fortune et al, 2003)

In conclusion, the mechanisms and factors contributing to psoriasis can lead to either nutritional deficiencies (eg free radical damage from stress increasing the need for antioxidants), reduced tolerance to foods such as gluten or a high protein diet or susceptibility to infection and compromised immune function. The following research looks at specific nutrients commonly used in the treatment of psoriasis.

3. TREATMENT & EVIDENCE BASED RESEARCH (70marks)

TREATMENT AIMS & NUTRIENTS

Decrease bowel permeability / toxicity
Improve intestinal flora and mucus membranes, probiotics
Rebalance fatty acid levels
EFA's, n-3's
Reduce inflammatory processes in the skin
Anti-inflammatory's, n-3's, Vitamin E
Balance/reduce abnormal cell proliferation
Vitamin D, A, C, Zinc
Increase immunity & address immune dysregulation & Th1 dominance
Vitamin D, probiotics Antivirals, Antibacterials eg garlic
Increase lymphatic circulation
Improve channels of elimination: bowels, skin, liver, lymph
Eradicate underlying infection
Antimicrobial, antiviral, antibacterial, anthelmintic eg garlic
Enhance stress response (brain / skin axis dysregulation)
B vitamins, whole grains.
Address dietary and nutritional deficiencies
Increase complex carbohydrates, good quality protein, good fats, reduce trans and saturated fats, increase raw foods.

Nutrients/phytochemicals x 4 (4 articles for each)
Vitamin C, Vitamin D, Zinc, Gliadin
Foods x4 (2 articles for each)
Fish, Yoghurt, Tumeric, Aloe Vera

NUTRIENTS / PHYTOCHEMICALS

VITAMIN C

Vitamin C is involved in corticosteroid, collagen and neurotransmitter biosynthesis, protection of folic acid reductase (which converts folic acid to folinic acid). Maintenance of connective tissue: vitamin C is essential for collagen formation. If collagen is produced in the absence of vitamin C, it is unstable and cannot produce the triple helix necessary for tissue structure formation. It is important for biosynthesis of elastin, proteoglycans, and bone matrix (Hall et al, 1998). Ascorbate is a cofactor for biosynthesis of noradrenaline to production of serotonin. (Bornstein et al, 2003) It is an immune stimulant, favourably modulating lymphocytes and phagocytes and regulating NK cells, influencing antibody and cytokine synthesis. Its can

reduce histamine and is involved wound healing and promotes formation of the epidermal barrier. (Russell, 2001)

Deficiency is associated with muscle weakness, drying of the skin and mucus membranes and susceptibility to infection. (Braun, 2007)

Along with its direct action on the skin, the actions listed above are related to underlying pathology in psoriasis. Vitamin C supplementation has shown results with high levels of activity in recovery, restoring immunity and inflammatory responses and counteracting oxidative stress and changes to adrenal hormones. (Thompson, et al, 2001)

Although listed in many complementary text for use in the treatment of psoriasis, little research has been conducted relating vitamin C treatment directly to the condition.

VITAMIN D

Vitamin D helps to control keratinocyte proliferation and increase type 2 helper Tcell cytokine expression and increase interleukin-10 (IL-10) and lower IL-8. Treatment should not be used in conjunction with UV light therapy. (Pizzorno, 2006)

An article by van de Kerkhof et al, 1998 titled claims that Vitamin D3 analogues have revolutionized the topical treatment of psoriasis. Mode of action is via modulation of the transcription of genes with vitamin D3 response elements in their promoter region. They state that vitamin D3 analogues cause inhibition of various aspects of cutaneous inflammation and epidermal proliferation with enhancement of normal keratinization. In vivo, active vitamin D3 analogues have substantial antipsoriatic effect and are contained in ointments such as: calcipotriol, tacalcitol and calcitriol shown to have an antipsoriatic effect in placebo-controlled studies.

In 1992 Kragballe et al discuss that a high-affinity receptor for the bioactive form of vitamin D3, 1,25-dihydroxyvitamin D3 (1,25[OH]D3), existing in most skin cells which led to the finding of previously unknown effects of vitamin D on epidermal growth and on the skin immune system. They showed 1,25(OH)2D3 inhibits epidermal proliferation and promotes epidermal differentiation, providing the rationale for introducing 1,25(OH)2D3 in the treatment of psoriasis vulgaris. They conclude that compared with 1,25(OH)2D3, calcipotriol is about 200 times *less* potent in its effects on calcium metabolism, although similar in receptor affinity. In double-blind, placebo-controlled, randomised studies, topical calcipotriol (50 micrograms/gm, up to 100 gm weekly) showed to be efficacious and safe for the treatment of psoriasis.

Hakamataa et al, 2008 agree that Vitamin D receptor (VDR) ligands are therapeutic agents for the treatment of psoriasis, along with showing therapeutic potential for autoimmune diseases and cancers of the skin amongst others. They further specify that LG190178 is a novel non-secosteroidal ligand for VDR. After synthesizing and evaluating stereoisomers of LG190178 they found that only an (2S,2′R)-analogue of LG190178 (YR301) had strong activity.

Lehmann et al, 2005 highlight that the skin is the only body tissue that completes ultraviolet-B (UV-B)-induced pathway from 7-dehydrocholesterol to hormonally active calcitriol (1 alpha,25-dihydroxyvitamin D-3). Epidermal synthesis of calcitriol regulates cellular functions in keratinocytes and immunocompetent cells. Due to their antiproliferative and pro-differentiating effects, calcitriol and other vitamin D analogs are highly efficient in the treatment of psoriasis vulgaris. The known antipsoriatic effect of UV-B light could, at least in part, be mediated via UV-B-induced synthesis of calcitriol. In addition, mounting evidence indicates that cutaneous vitamin D-3 synthesis may be indicated in the prevention of diseases including various malignancies. Lehmann discusses recent sun-protection

guidelines and their aim to prevent internal cancers. He claims a clearer understanding of vitamin D metabolism via the skin may offer a new perspective for therapeutic applications of vitamin D analogs.

Zinc

A study by Der Hautarzt et al 2000 claim the role of zinc in the pathogenesis of various dermatological conditions is controversial. Der Hautarzt et al used their own patient collective, aiming to determine variations in serum zinc levels in patients with atopic dermatitis and psoriasis comparative to levels in the normal population.

Serum zinc levels of 97 patients with atopic dermatitis and 88 patients with psoriasis were compared to those in 22 healthy subjects and subjected to statistical analysis. Their findings showed no statistically significant difference was found between the populations investigated, thus they conclude that zinc replacement therapy in patients with atopic dermatitis and psoriasis appears to be indicated only in those with a documented zinc deficiency. However, with such a small sample size, more evidence is needed to confirm these findings.

A study by Donadini et al 1980, showed that plasma zinc levels in psoriatic patients were significantly lower than in healthy controls.

Studies on the efficacy of zinc and dermatological conditions (other than in acne vulgaris) have been found to be conflicting. However, the role zinc plays in treatment of psoriasis is not just related to keratinocyte proliferation. Zinc is involved in many biochemical pathways relating to this condition such as growth and development, immune function and antioxidant effects. A study by Prasad et al in 1998 showed that even a mild zinc deficiency adversely affects T-cell function. Th1 over-expression is seen in both psoriasis and celiac disease. (Ojetti et al, 2006)

Zinc may prove to be effective in assisting mucus membrane and epithelial lining of the GIT in psoriatic patients. It is also involved in collagen synthesis. (Braun, 2007) As mentioned earlier stress is often associated with psoriasis, stress increases the utilization and loss of zinc. Lack of zinc can bring on a poor emotional response to stress. (Pizzorno, 2006)

GLIADIN ANTIBODIES

Gliadin Antibodies Linked to Psoriasis

Attention was drawn to the gliadin / psoriasis link when Michaëlsson et al hypothesized at least two abnormalities in the duodenal mucosa of psoriasis patients. 1. present in most patients and characterized by increased mast cells and eosinophils. 2. present in a subgroup of patients with antibodies to gliadin and increased number of duodenal intraepithelial lymphocytes. (Michaëlsson, 1997)

A further study indicated patients with raised antibodies to gliadin may improve on a gluten free diet even if they have no antibodies to endomysium or if the increase in duodenal intraepithelial lymphocytes is slight or seemingly absent. (Michaëlsson, 2000; Addolorato et al, 2003)

Ojetti et al found a high prevalence of malabsorption in psoriasis patients, however gliadin was only one contributing factor (Celiac disease 6%, bacterial overgrowth 21%, parasitic infections 3%, eosinophillic gastroenteritis 1%). They also question whether psoriasis may (hypothetically) be *responsible* for malabsorption due to psoriatic lesions developing in gut mucosa, leading to secondary malabsorption. They cite a draw-back in testing was difficulty accessing the small bowel without a biopsy. (Ojetti et al, 2006) A small sample size but showed significant results, further establishing a link between the gut and skin disorders.

A larger (but still relatively small) study of 130 subjects were tested (along with endoscopy and duodenal biopsy), for antigliadin (AG) antibodies. Along with showing a correlation between celiac-disease antibodies and greater psoriasis activity, results also showed a further rise in AG antibodies in those patients requiring immunosuppressant drugs such as methotrexate. (Woo et al, 2004)

More recently, Bozdek et al sought to bring clarity to the aetiopathogenesis of psoriasis and the link with asymptomatic celiac disease. Interestingly, Bozdek cites psoriasis as 'an auto-immunological disease induced by gluten consumption by genetically disposed people, which causes small intestine damage'. Again, a small subject size of 67 patients with intensified psoriatic lesions showed significantly higher mean concentrations of antibodies against tissue transglutaminase isolated and against gliadin for IgA. IgA antibodies for both tissue transglutaminase and gliadin positively correlate with psoriasis. They concluded a link between psoriasis and asymptomatic celiac disease/gluten intolerance. (Bozdek, 2008)

A correlation between gliadin antibodies and psoriasis is evident. However, as the sample size of most trials has been small, further testing would be worthwhile to establish clear pathology and mechanism of action. In light of clinical application, it is important to consider asymptomatic celiac disease when treating psoriasis. If simply the removal of dietary intake of gliadin will bring significant symptomatic effects it could even be considered mandatory.

FOODS

FISH

Psoriasis is accompanied by high amounts of arachidonic acid (AA) in plaques, and profound changes in metabolism of eicosanoids, further exacerbating proinflammatory agents. Fish oils may decrease inflammatory mediators, particularly leukotrienes. The EPA replaces AA in phospholipids resulting in weaker inflammatory responses. Not all psoriasis patients respond to fish oil supplementation and some can take up to 4mths to experience benefit. However, a study by Danno et al (1998) reported a 77% improvement in psoriasis vulgaris with fish oil supplementation, indicating that it is definitely a treatment worth considering.

EPA and DHA found in higher amounts in oily deep sea fish work on reducing inflammation by way of the Omega 3 pathway and stimulating prostaglandins (PGE3), Thromboxames (TXA), Prostacyclin (PGI 3), and inhibiting series 2 and 4 eicosanoids. These actions have anti-inflammatory effects. (Simopoulos, 2002) It is therefore relevant to hypothesise that the antiinflammatory effects of EPA/DHA will have a positive effect on the inflammatory condition of psoriasis.

Clinical trials and animal experiments involving fish oils show possible anti-inflammatory results, indicating potential usefulness in autoimmune and antiinflammatory conditions. Autoimmune conditions all show high levels of the pro-inflammatory cytokine interleukin 1 (IL-1) and the pro-inflammatory leukotriene LTB4 produced by omega 6 fatty acids. Placebo controlled human studies conducted in autoimmune conditions including psoriasis have shown significant benefits to these inflammatory conditions with the supplementation of fish oils (EPA/DHA) by decreasing elevated levels of pro-inflammatory cytokines. Benefits include a decrease in disease activity and a reduction in the use of anti-inflammatory drugs. (Simopoulos, 2002)

Based on profound changes in the metabolism of eicosanoids with increased concentrations of free arachidonic acid (AA) and pro-inflammatory metabolites observed in psoriatic lesions, Mayser et al, 1998 hypothesized that free EPA may compete with liberated AA and result in an anti-inflammatory effect. They conducted a double-blind, randomized, parallel group study across eight European centers.

The sample size included 83 (aged 18-80) patients hospitalized for chronic plaque-type psoriasis with a severity score of least 15 according to the PASI who participated in a 14-day trial. They were randomly allocated to receive daily infusions with either a w-3 fatty acid–based lipid emulsion (Omegavenous; 200 ml/day with 4.2 gm of both EPA and DHA; 43 patients) or a conventional w-6-lipid emulsion (Lipovenous; EPA+DHA < 0.1 gm/100 ml; 40 patients). Efficacy of therapy was evaluated by changes in PASI, in an overall assessment of psoriasis by the investigator, and a self-assessment by the patient. In one center neutrophil 4- versus 5-series leukotriene (LT) generation and platelet 2- versus 3- thromboxane generation were investigated and plasma-free fatty acids were determined.

Results showed a total PASI score decrease by 11.2 ± 9.8 in the w-3 group and by 7.5 ± 8.8 in the w-6 group (p = 0.048). In addition, the w-3 group was superior to the w-6 group with respect to change in severity of psoriasis per body area, change in overall erythema, overall scaling and overall infiltration, as well as change in overall assessment by the investigator and self-assessment by the patient. Response was seen in 16 of 43 patients (37%) receiving the w-3 emulsion and 9 of 40 patients (23%) receiving w-6 fatty acid–based lipid emulsion. Within the first few days of w-3 lipid administration, (not in the w-6 supplemented patients), a manifold increase in plasma-free EPA concentration, neutrophil leukotriene B5 and platelet thromboxane B3 generation occurred. They concluded that Intravenous w-3-fatty acid administration is effective in the treatment of chronic plaque-type psoriasis.

Although a statistically significant result, this study was conducted on a relatively small sample size. Self assessment by participants may also have skewed results. No mention was made in regards to diet and possible pro-inflammatory foods consumed during the trial period.

In psoriasis, inflammatory products (LTB_4 and 12-hydroxyeicosatetraenoic acid) are elevated along with AA metabolism being altered. EPA/DHA supplementation showed an improvement in symptoms and a decrease in LTB_4 yet most research suggests fish oils used in combination with corticosteroids is more effective. Supplementation as an adjunct to drug therapy may be beneficial, however more stringent clinical trials with larger sample sizes on the efficacy of EPA/DHA in the treatment of psoriasis may clarify treatment efficacy, dosage and associated inflammatory pathways.

Additionally, most trials were conducted with dosage ranging from 3-4g EPA/DHA. It would be difficult to obtain this therapeutic amount from diet alone. Although fish is listed here as a food, a fish oil supplement may have a greater therapeutic effect.

YOGHURT – PROBIOTIC

The word probiotic is Greek for “life” describing the action of one organism stimulating the growth of another. Naidu et al, 1999 describes probiotics as ‘a microbial dietary adjuvant that beneficially affects the host physiology by modulating mucosal and systemic immunity, as well as improving nutritional and microbial balance in the intestinal tract’. Clinical application in psoriasis is in prevention of atopy, allergic reactions, intestinal hyperpermeability and lowered immunity. (Pizzorno, 2006)

Because probiotics are actually live bacteria, claims of effectiveness can vary greatly depending on quality, strains and colonisation of certain micro-organisms, refrigeration and shelf life. A survey by *Choice* (1999) showed few of the tested yoghurts showed adequate amounts of bacteria to be therapeutically viable. Brands showing desired effective strains in sufficient quantities were: Vaalia (Parmalat Australia Ltd), Jalna drinking yoghurt (Jalna dairy foods), Yoplait Yoplus (National foods) and Yakult milk drink (Yakult Australia Ltd). The natural, unsweetened varieties are preferred. (Hawrelak, 2005)

A study by Naidu et al (1999) on Lactic acid bacteria (LAB) states it's beneficial effects on the GIT include prevention of establishment, adherence and replication of enteric mucosal pathogens along with synergistic effects on digestion and alleviation of intestinal malabsorption. Specific components of LAB strains have strong adjuvant effects including modulation of cell-mediated immune responses and augmentation of cytokine pathways and regulation of interleukins and TNF. (Naidu et al, 1999)

Main characteristics of probiotics relevant to psoriasis:

1. Gastric acid and bile salt stability – allowing survival through the stomach and small intestine.
2. Ability to adhere to intestinal mucosa – essential for immune cell modulation and inhibition of pathogens.
3. Ability to colonise the intestinal tract – daily ingestion may not be needed due to multiplication in the intestines.
4. Production of antimicrobial compounds – via normalisation of GIT flora and suppressed growth of pathogens.
5. Assist in synthesising and enhancing nutrient bioavailability – via metabolism of the microbial population in the bowel, affecting faecal pH. (Hawrelak, 2005, Pizzorno, 2006).

There are many different micro-organism species used as probiotics. Actions of each can be specific to a certain *strain* of bacteria rather than the species of bacteria. If a particular species is proven to show therapeutic benefits, this does not mean the same applies for that species of bacteria (Hawrelak, 2005). This also relates to appropriateness and relevance to human conditions and imbalances.

Strains indicated in psoriasis by way of skin and food allergies are:
Food allergies: *L.rhamnosus* GG, *B.lactis* Bb12*, L.paracasei* Shirota
Psoriasis/Eczema: *L. rhamnosus* GG*, Bifidobacterium lactis* Bb12, *Lactobacillus paracasei* Shirota
Immune function: *L. rhamnosus* GG, *B.lactis* HN019 (DR10), *Lactobacillus johnsonii* La1, *L.rhamnosus* HN001 (DR20), *Lactobacillus acidophilus* LA5
(Pizzorno, 2006)

Yoghurt is an easily accessible form of probiotic and can assist transport of probiotic bacteria into the GIT, enhancing it's survival. Other sources of probiotics are fermented vegetables such as sauerkraut and Kimchi. (Pizzorno, 2006)

Therapeutic uses relevant to Psoriasis	
Stimulation of the immune system	Enhanced function and motility of the GIT
Resistance to colonization of pathogens	Production of polyamines and SCFA's
Promote overall health	Intestinal hyperpermeability
Treatment and prevention of GIT infections	Stimulation of immunity – gastrointestinal and systemic
Food allergies	Atopic eczema
Gut flora stabilisation	

(Pizzorno, 2006)

Although studies exist on the therapeutic benefits of probiotics and skin conditions, there is not a lot on psoriasis alone, however there has been a larger focus on eczema, demonstrating positive results. With the knowledge of the gut-skin axis link in psoriasis, it could be hypothesised that probiotic use could also benefit psoriasis treatment. More clinical trials are needed to establish efficacy, relevant strains of bacteria and dosage.

TUMERIC

A recent study by Kohli et al, 2008 looked at curcumin as a possible safe, inexpensive and effective treatment of psoriasis, based on patient anecdotes of successful treatment with the

root. They conducted a phase II, open-label, Simon's two-stage trial of 4.5 g/d of oral curcuminoid C3 complex in patients with plaque psoriasis. End points included improvement in Physicians Global Assessment score, PASI score, and safety end points throughout the study. There were no study related adverse effects and results showed a low response rate, along with possible placebo effect or the natural history of psoriasis.

However sample size was small (18 participants) yet excellent results were seen in two patients results. It is therefore indicated that large placebo-controlled studies are necessary to determine therapeutic efficacy of oral curcumin in psoriasis treatment.

Kohli et al suggest a placebo controlled trial would need to enroll 254 participants to have statistical power of 80% to differentiate the response rate observed in their study from an expected PASI 75 response rate of 5% in the placebo group.

Interesting discussion on this topic Kohli et al, mention that in vitro curcumin has been shown to block pathways necessary to develop psoriasis, hypothesizing that it is possible that oral administration will not produce a desired clinical effect due to low bioavailability. They state orally administered curcumin has been shown to have low bioavailability in both animals and human beings due to curcumin being extensively reduced and conjugated in the intestinal tract (Ireson et al, 2002; Grant et al; 2000). Animal experiments suggest that independent of dose, 40% to 90% of orally administered curcumin is excreted in stool (Ammon HP, Wahl MA. Pharmacology of Curcuma longa. Planta Med 57:1-7, 1991). They administered a relatively high dose of oral curcumin (4.5 g) and hypothesize that administering curcumin at high doses or combining oral curcumin with agents that may enhance its absorption may result in better efficacy.

In another study concerning the antipsoriatic effects of turmeric Miquel et al 2002 added that not only did it have antiinflammatory effects, it found that curcumin's ability to inhibit interleukins may be an additional factor in it's therapeutic use. IL-6 and IL-8 are growth factors for keratinocytes, and therefore their inhibition may possibly directly reduce psoriasis-related keratinocyte hyper-proliferation. They are inhibited via anti-oxidant activity; an action of turmeric largely overlooked in previous studies.

Goel et al 2008 evaluated the indirect antipsoriatic effects of Turmeric by measuring its influence of phosphorylase kinase activity. This approach was taken with the knowledge that Turmeric is a potent selective inhibitor of phosphorylase kinase, and more importantly, that it is currently believed that increased levels may be a surrogate marker of psoriatic disease. They concluded "the decreased phosphorylase kinase activity in calcipotriol and curcumin-treated patients was associated with corresponding decreases in the expression of keratinocyte transferrin receptor (TRR), severity of parakeratosis, and density of epidermal CD8+ T cells."

It appears further research is needed, yet promising results have been demonstrated calling for further clinical trials of both oral and topical curcumin in the treatment of psoriasis. There is also research indicating tumeric has immunological, hepatoprotective and wound healing actions (Braun, 2007), all relevant in the management of psoriasis.

ALOE VERA

Although not technically a food, the use of aloe vera in the treatment of psoriasis is noteworthy. Some texts highly regard the use of aloe vera extract and yet research is conflicting on the topical use of aloe vera in the treatment of psoriasis.

Syed et al, 1996 conducted a double-blind, placebo-controlled study to evaluate the clinical efficacy and tolerability of topical Aloe vera extract 0.5% in a hydrophilic cream to cure patients with psoriasis vulgaris.

60 patients with slight to moderate chronic plaque-type psoriasis and PASI (Psoriasis Area and Severity Index) scores between 4.8 and 16.7 (mean 9.3) were involved in the trial. Impressive results showed progressive reduction of lesions, desquamation, erythema, infiltration and lowered PASI score. 83.3% of patients we considered 'cured'. (Syed et al, 1996) This was compared to the placebo cure rate of 6.6%. Also of note is that the study was followed up on a monthly basis for 8 months and there were no relapses. Although this was a small sample size, and no side effects were observed, the results indicate that topical application of aloe vera cream may be an efficacious treatment of psoriasis.

Active constituents of aloe vera have shown considerable analgesic, antipruritic, wound healing and anti-inflammatory properties. (Duke 1985)

Pugh et al, 2001 differs in their research in that they used aloe vera juice, characterising an immunostimulatory polysaccharide called Aloeride, whereas most studies use extract from the gel. They conclude that although Aloeride comprises only 0.015% of the aloe juice dry weight, its potency for macrophage activation accounts fully for the activity of the crude juice. Pharmaceutical development of Aloeride as an immunostimulant, either alone or in combination with other aloe components, may have significant potential for wound healing and immunotherapy. Aloeride polysaccharide could also be valuable for standardization of commercial aloe products, instead of the presently used, and most likely inactive, acemannan.

Although this study shows promising results the exact mechanisms of testing are difficult to ascertain. Testing was conducted in a laboratory rather than in vivo. Clinical trials on the ingestion of aloe vera are scarce and it would be of interest to assess further studies.

Choi et al, 2001 also looked to isolate the active constituent effective in wound healing. Their methods included Chromatography, electrophoresis and spectroscopic methods. The cell-proliferation activity of each component isolated was measured by a [3H]thymidine uptake assay. The cell-proliferation activity of the effective component was tested on a three-dimensional raft culture (cell culture technique by which artificial epidermis is made from keratinocytes).

A glycoprotein fraction was isolated and named G1G1M1DI2. It showed a single band on sodium dodecyl sulphate–polyacrylamide gel electrophoresis. It exhibited significant [3H]thymidine uptake in squamous cell carcinoma cells. The effect of G1G1M1DI2 on cell migration was confirmed by accelerated wound healing on a monolayer of human keratinocytes. When this fraction was tested on a raft culture, it stimulated the formation of epidermal tissue. Furthermore, proliferation markers (epidermal growth factor receptor, fibronectin receptor, fibronectin, keratin 5/14 and keratin 1/10) were markedly expressed at the immunohistochemical level.

The glycoprotein fraction enhanced wound healing in hairless mice by day 8 after injury, with significant cell proliferation. They concluded that this glycoprotein fraction is involved in the wound-healing effect of aloe vera via cell proliferation and migration.

Although beneficial in isolating the active constituent and possible mechanism of action, it would be interesting to convert this information into human trials.

Results were very different in a trial by Paulsen et al, 2005 where they tested the effect of a commercial, preserved, but otherwise untreated *Aloe vera* gel in psoriasis. A small sample size of 41 with stable plaque psoriasis were included in a randomized, double-blind, placebo-controlled right/left comparison. The study comprised a 2-week wash-out period followed by a 4-week treatment period with two daily applications and follow-up visits after 1 and 2 months. Results showed score sum of erythema, infiltration and desquamation decreased in 72.5% of the *Aloe vera*-treated sites compared with 82.5% of the placebo-treated areas

from week 0 to week 4, which was statistically significant in favour of the placebo treatment (P = 0.0197).

Fifty-five per cent of the patients reported local side-effects, mainly drying up of the skin on test areas. They concluded *Aloe vera* gel on stable plaque psoriasis was modest and not better than placebo. However, the high response rate of placebo indicated a possible effect of this in its own right, which would make the *Aloe vera* gel treatment appear less effective.

This latter study shows that aloe vera extract should not be used as a mandatory treatment of psoriasis but with consideration and an eye on current research. Further studies with larger sample sizes, more detailed explanation of the chemical composition of gel used and clarification of the species of aloe vera plant used.

Compare Australian RDI's (10 marks)

NUTRIENTS	RDI	THERAPEUTIC DOSE
Vitamin C	45mg p/d (Braun, 2007)	100-400mg p/d
Vitamin D	Adults 50-70yrs 10mcg Adults 70+yrs 15mcg	Sunlight Cod liver oil (See Appendix2) 1000 IU (equiv 25mcg)
Zinc	Adult men > 19yrs 14mg/d Adult women > 19yrs 8mg/d (Braun, 2007) Doses greater than100-150mg for extended periods can interfere with copper metabolism. (Braun, 2007)	Dependant on severity of symptoms and deficiency state. Approx range from RDI – 100mg.
Gliadin	Elimination of gluten containing foods	
FOODS	**RDI**	**THERAPEUTIC DOSE**
Fish	Dietary intake of fish will not suffice to obtain a therapeutic dose of 3-4g/d EPA/DPA for psoriasis patients. The most concentrated sources of EPA and DHA are found in cold water deep sea fish such as: mackerel, halibut, salmon and hering. (Braun, 2003). Results from a study by Meyer et al indicate that the majority of Australians are failing to meet recommended intake n−3 PUFA and emphasize the need for strategies, to increase consumption of n−3-containing foods. (Meyer et al, 2003) The csiro recommends 3-5 serves of fish per week (www.csiro.com.au, 2007), whereas Food Standards Australia New Zealand recommend a proclaimed 'conservative' 2-3 serves per week (FSANZ newsletter, 2004). Methyl mercury concerns: Methyl mercury generally accumulates in larger, predatory fish. According to FSANZ, methyl mercury found in Australian fish is low (FSANZ, 2004). Rule of thumb for fish	3-4g p/d EPA/DHA

	consumption: Limit intake of larger species at the top of the food chain as they are more likely to accumulate higher levels of mercury. To maximize the nutritional benefit of eating fish, eating a wide variety of species is recommended (FSANZ, Media release, 2004).	
Aloe vera	Aloe vera gel from fresh living plant or stabilised juice: 25ml up to 4xp/d Standardised extracts to acemannan: preparation containing up to 800mg p/d Topical application: gel, cream, ointment: as needed 1.5-4.5ml daily of 1:10 tincture of resin (Braun, 2007)	Topical use 3x p/d on psoriasis. (Syed et al, 1996)
Tumeric	Powdered: 1.5-3g/d in water or cooking Liquid extract (1:1) in 45% ethanol: 5-15ml p/d Powdered extract standardised to 95% curcumin: 100-300mg/d. Higher doses for arthritis and cancer. External use: ½ cup standardised extract combined with 1 tspn carbonate of soda, mixed with hot water to make a paste. Spread on gauze and apply as a poultice or paste to affected area. (Braun, 2007)	4.5 g/d of oral curcuminoid C3 complex (Kohli et al, 2008)
Yoghurt	Minimum dose for therapeutic yoghurts (containing > 10^8 viable bacteria per mL) = 100g serving (½ cup) per day. Note: this size serving of yoghurt contains 3.1-3.5g of lactose, and should be suitable for those with lactose sensitivity as it is substantially below the threshold for lactose-intolerance individuals. (Pizzorno, 2006)	½ - 1 cup per day.

CONCLUSION

Nutrient, phytochemical and food as medicine treatment options for psoriasis analyzed in this paper shows both solid and conflicting results.

Vitamin C shows benefit for its corticosteroid, collagen and neurotransmitter biosynthesis and maintenance of connective tissue. However, research is lacking in the direct application of vitamin C on psoriasis.

Studies on vitamin D has shown to reduce or control keratinocyte proliferation and increase type 2 helper Tcell cytokine expression, its use is commonly accepted in medical literature. However, there are many studies claiming efficacy of different vitamin D analogues. Greater clarity in this area would be beneficial.

Zinc has also demonstrated efficacy in wound healing, skin conditions and enhancing immune function. Yet, actual research on its role in psoriasis is often lacking in stringent methodology and larger sample sizes.

The gliadin antibody link to psoriasis is interesting. The link is related to abnormalities in the duodenal mucosa of psoriasis patients. The safety of this treatment option is that it is simply removal of a potential allergen, rather than a prescribed supplement. There is varying research on malabsorption, varied GIT dysfunction a strong link between the gut and skin disorders. Sample sizes have been small yet the safety compared to possible positive effects is certainly worth strong consideration in treatment.

The use of fish oils showed positive results in decreasing inflammatory mediators. No research was found on the dietary intake of fish alone. Most clinical trials used dosages

ranging 3-4g, an amount unsustainable from a dietary source. Depending on severity of the psoriasis, a supplement such as cod liver oil may be an appropriate source of omega 3, as it also contains vitamin D, E and A. Vitamin A inhibits polyamine formation, thus reducing the rate of cell proliferation. (Pizzorno, 2006)

Research on probiotics has focused more on skin conditions in general, particularly eczema. Many other conditions associated with the gut/skin axis indicate that probiotics may play a valuable role in treatment. However, clinical trials are lacking and further investigation into mechanism of action and specific involvement would be beneficial.

Tumeric is probably the most promising area of research for this condition. A variety of studies show efficacy in both oral and topical application. Most potent actions include antioxidant and anti-inflammatory. Further clinical trials may result in curcumin extract becoming a mainstay in the treatment of psoriasis.

Trials on aloe vera extract have shown vastly conflicting results, one study concluded that the placebo group produced statistically significant results. (Paulsen et al, 2005) It is certainly worth following up with further trials, however this last study may indicate that the application of a topical solution, may give patients a feeling of control in their illness. Therefore topical relief should be valued as part of the treatment strategy as a whole.

Other considerations in treatment: See Appendix 3

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Appendix 1

5 CLASSIFICATIONS OF PSORIASIS

Stable plaque: red dry silvery-white scaling, usually on elbows, knees, lower back. Other sites include the scalp (possibly due to infection), nails (pitting and onycholysis (separation from the nail bed) and subungual hyperkeratosis (excessive thickening), flexures (natal cleft, submammary & axillary folds – shiny red and symmetrical), palms (often difficult to recognise). (Davidson's, 2002)

Guttate: most common in children and adolescents, usually following streptococcal sore throat. This is often the first indication of the disease. Patients usually respond to treatment, however, most develop plaque psoriasis later in life. (Davidson's 2002)

Erythrodermic: universally red or scaly skin. Temperature regulation can become problematic with danger of hypothermia or hyperthermia. (Davidson's 2002)

Pustular varieties: 1.Serious. Onset sudden with large numbers or sterile pustules erupting on a red base. Patient may become ill and need hospital admission. 2. Localised pustular psoriasis, affecting palms and soles. (Davidson's 2002)

Arthropathy: occurs in 5-10% (Davidson's 2002)

Appendix 2

Arctic cod liver oil – Nordic Naturals

Ingredients: purified arctic cod liver oil, vitamin D3 (cholecalciferol in sesame oil), d-alpha tocopherol, natural lemon flavor, rosemary extract

One Teaspoon Contains:
EPA: 410 mg
DHA: 625 mg
Other Omega-3s: 225 mg
Vitamin A: 650–1500 IU
Vitamin D: 1000 IU
Vitamin E: 30 IU

Appendix 3

OTHER CONDIDERATIONS IN TREATMENT

Dietary Inclusions

- High fibre – reduce gut-derived toxins, maintain healthy colon, promote excretion of toxins. (Pizzorno, 2006)
- 150g cold water fish (mackerel, herring, salmon) 3-4x p/wk (Pizzorno, 2006)
- Predominantly vegetarian diet to assist reducing gut-derived toxins and polyamines. (Pizzorno, 2008)
- Eat slowly, chew well to increase assimilation and absorption of nutrients, especially protein. (Haas, 2006)
- Consume proteolytic enzymes to assist digestion: paw paw, kiwi fruit (actinidin), fig (ficin), pineapple (bromelain). (Morrison, 2001)
- 3 brazil nuts p/d. Rich in selenium. Low levels seen in patients with moderate-severe psoriasis and use of methotrexate. (Michaelsson et al, 1989)
- Bitter foods to stimulate bile secretion; bitter greens, rocket, endive, lemon.
- Vitamin C rich foods: particularly blackcurrants, red and yellow capsicum, oranges. Also cabbage, mango, papaya, cantaloupe. (Braun, 2007)
- Fresh vegetable juices to promote elimination (Haas, 2006)

Avoid

- Sugar & alcohol. (Pizzorno, 2006)
- Eliminate gluten
- Identify/remove food allergens
- Limit intake of animal products as they increase AA. (Pizzorno, 2006)
- Reduce arginine rich foods, increase lysine rich foods

Psychological

Stress is implicated in many psoriasis cases but not all. Often onset is directly linked to a traumatic event. (Pizzorno, 2006) Stress management and relaxation techniques can be beneficial. (Fortune, 2000)

Physical

Sunlight is beneficial and a good source of vitamin D. UV light has had some success however it requires diligent monitoring. (Pizzorno, 2006)

Topical

Tumeric, aloe vera, zinc (see above)
Licorice: glycyrrhetinic acid exerts similar if not superior properties to hydrocortisone. It potentiates hydrocortisone and may be used in conjunction with it. (Pizzorno, 2006)
Chamomile and Chickweed are vulnerary, anti-inflammatory and anti-allergic. (Bone, 2003)