

Role of Diet in Dermatological Conditions

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Abstract

In recent years nutritional deficiency diseases have been reported in developed countries. Nutritional deficiencies can be due to inadequate intake, abnormal absorption or improper utilisation. Many nutrients are essential for life, an adequate amount of nutrients in the diet is necessary for providing energy, building and maintaining body organs and for various metabolic process. Skin disorders have long been associated with nutritional deficiencies. Nutrition is one of the most important parameters that are involved in modulating skin health and condition. Nutritional status plays an important role in maintenance of healthy skin. A variety of nutritive factors such as small peptides, minerals, vitamins, macronutrients and micronutrients work together to maintain the barrier functions of skin in the face of everyday challenges. Changes in nutritional status that alters skin structure and function can also directly affect skin appearance. In general dermatological problems where diet plays a role are atopic eczema, urticaria, dermatitis herpetiformis, psoriasis, pityriasis, rubra pilaris, purpura, scurvy, Refsum's disease, ichthyosis, vitiligo, hypo pigmentary conditions, acne, rosacea, kwashiorkor, marasmus, stomatitis, phrynoderma, pellagra, acrodermatitis enteropathica, homocysteinuria, Hartnup disease, gout, porphyrias, xanthomas, hyper carotenemia and lycopopenemia. Dietary changes or modification might help to prevent recurrences of many skin diseases.

Introduction

Many nutrients are essential for life, and an adequate amount of nutrients in the diet is necessary for providing energy, building and maintaining body organs, and for various metabolic processes [1].

Skin disorders have long been associated with nutritional deficiencies. Nutrition is one of the most important parameters that are involved in modulating skin health and condition [2].

In general dermatological problems where diet plays a role are atopic eczema, urticaria, dermatitis herpetiformis, psoriasis, pityriasis rubra pilaris, purpura, scurvy, Refsum's disease, ichthyosis (nutritional), vitiligo, hypopigmentary conditions, acne, rosacea, kwashiorkor, marasmus, stomatitis, phrynoderma, pellagra, acrodermatitis enteropathica, homocystinuria, Hartnup disease, gout, porphyrias, xanthomas, hypercarotenemia and lycopopenemia [3].

Earlier nutritional deficiency diseases were assumed to be limited to the underdeveloped and developing countries. In recent years nutritional deficiency diseases have been reported in developed countries [4,5]. Nutritional deficiencies can be due to inadequate intake, abnormal absorption or improper utilization [1]. Nutritional status plays an important role in the maintenance of healthy skin [6-9]. Deficiencies of several vitamins, minerals, and fatty acids have clear cutaneous manifestations [10-13].

Many attempts have been made to improve skin health and beauty by changing or by supplementing the diet [14]. In 2001, Boelsma et al. [15] reviewed the effects of vitamins, carotenoids and fatty acids supplementation in optimizing skin condition and preventing skin diseases and concluded that nutritional factors show potential beneficial actions on the skin.

Macronutrients (carbohydrates, proteins, and lipids) and micronutrients (vitamins and nutritionally essential minerals) work together to maintain the barrier functions of skin in the face of everyday challenges. Changes in nutritional status that alter skin structure and function can also directly affect skin appearance [16].

Excessive inflammation of the skin is known to increase the requirements of specific nutrients like folic acid and protein [10,17].

Primary requirement for skin cells is glucose also provides carbohydrate backbones for modification of proteins (glycoproteins) and lipids (glycolipids) that comprise the extracellular environment of the epidermis. Aberrant glucose handling drastically affects skin structure and appearance [18].

Much of the role of nutrition in skin health emphasizes on the effects of deficiency, since the structural components of the skin are supported by a variety of nutritive factors, such as small peptides, minerals, and vitamins, which serve as enzyme cofactors, activators, or inhibitors [9].

Many micronutrients were initially recognized for their impact on skin health. The effect of micronutrient supplementation on skin health is a relatively new field of study, as associations between diet and skin conditions are only now starting to emerge [19] (Table 1).

Vitamin A

Deficiency of vitamin A results in hyperkeratinization with reduced number of sebaceous glands and blockage of sweat glands [10].

Hypovitaminosis also affects the skin by causing xerosis, generalized hyperpigmentation, and sparse and fragile hair. Plugging of the follicular openings with spiny horns is one of the classic signs of vitamin A deficiency as in phrynoderma [20].

Retinol (Vitamin A), carotenoids (provitamin A) and retinoids (Vitamin A metabolites) are absorbed better with parallel intake

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Vitamins	Skin conditions
Vitamin A	Hyperkeratinization, sparse and fragile hair, phymoderma, herpes, wound healing, acne, photoaging
Vitamin C	Scurvy
Vitamin B12	Hyperpigmentation, vitiligo, angular stomatitis, hair changes
Vitamin B3	Pellagra
Vitamin D	Photo damage
Vitamin E	Anti-inflammatory effects-erythema, edema. Wound healing, photodamage
Macronutrients	Skin conditions
Carbohydrates, proteins	Galactosemia, Kwashiorkor (Protein malnutrition)
	Marasmic-Kwashiorkor
Fatty acids	Photoprotection, photoaging, wound healing, skin sensitivity
Minerals	Skin conditions
Zinc	Weeping dermatitis, secondary infections, excessive fragile hair and spars, alopecia, nail defects, acrodermatitis, poor wound healing.
Iron	Spoon shaped nails, hair loss, glossitis with loss of papillae, angular cheilitis and pruritis.

Table 1: List of Macronutrients and Micronutrients Related to their Skin Conditions.

of vegetable oils [21-26]. Vitamin A rich food items are useful in controlling the Pityriasis rubra pilaris [27]. Excess intake of vitamin A itself can lead to side effects including shedding of skin and hair loss [28]. Source of Vitamin A: Liver, heart, kidney, milk, cod liver oils, fish liver oils, butter, eggs, carrots, cabbage, vegetables, green leaves, mangoes, potatoes, tomatoes, spinach, papaya etc. [29].

Vitamin B12

Cutaneous appearances associated with vitamin B12 deficiency are skin hyperpigmentation, vitiligo, angular stomatitis, and hair changes. Malabsorption is the most common reason of vitamin B12 deficiency

Sources: Milk, liver, kidney, muscle, butter, chicken, fish, yeast, cheese, raw egg, white grains, green vegetable such as spinach, peanuts, fruits such as apple, orange etc. [29].

Vitamin B3

Classic pellagra is a nutritional disease considered deficiency of vitamin niacin. Other factors, such as, mycotoxins, excessive dietary leucine intake, estrogens and progesterone, chronic alcoholism, and various medications, might also lead to the development of pellagra [11,30,31]

Vitamin C

Scurvy is a deficiency disease of ascorbic acid manifested in the decreased production and increased fragility of collagen [1].

Sources: Guava, amla, green chilli, amaranth leaves, citrus fruits, green vegetables, potatoes, tomatoes, cheese, milk etc. [29]

Vitamin D

It can cause photodamage.

Sources: Fish liver oils e.g., cod liver oil, halibut liver oil etc. butter, milk, eggs, liver. In subcutaneous tissue, 7 dihydrocholesterol is converted to vitamin D by UV light [29].

Antioxidants

Antioxidants are effective in reducing free radical damage of collagen and elastin, the fibers that support the skin structure, and in preventing wrinkles and other signs of premature aging [32].

Supplementation with β carotene and other carotenoids [33-35] such as dietary tomato paste containing lycopene [36] protects against UV light induced erythema in humans. However, they are reported to exacerbate UV carcinogenesis under certain dietary conditions, by acting as prooxidants, at high oxygen pressure and under oxidative stress [37].

Selenium is an antioxidant mineral accountable for tissue elasticity. It also acts to avoid cell damage by free radicals. Associated skin signs contain hypopigmentation of the skin and hair and whitening of the nails [13]. It may play an important role in preventing skin cancer, as it can protect the skin from damage from excessive ultraviolet light.

Sources of selenium include wheat germ, seafood such as tuna and salmon, garlic, Brazil nuts, eggs, brown rice, and whole wheat bread [1].

Fatty Acids

Numerous studies have revealed that clinical imbalances of specific essential fatty acids are associated with a variety of skin problems. Hence dry, itchy, scaly skin is a hallmark sign of fatty acid deficiency [38].

Omega-6 fatty acids are believed to persuade more pro-inflammatory mediators and have been related with the development of inflammatory acne [39,40]. On the other hand, intake of high levels of omega-3 fatty acids is linked with declines in inflammatory factors [41]. In addition there are epidemiological studies that demonstrate that increasing the intake of omega-3 fatty acids through a diet rich in fish and seafood results in lower rates of inflammatory disease [42]. There are also studies claiming that sebum production is increased by the consumption of dietary fat or carbohydrate [43] and that variations in carbohydrates could also affect sebum composition [44,45].

Minerals Deficiency

Cutaneous manifestations of zinc deficiency are weeping dermatitis, secondary infection, poor wound healing, excessively fragile hair and sparse or no scalp and pubic hair [1]. Dermatitis, alopecia, and nail defects are also associated with zinc deficiency [11].

Zinc deficiency is accompanying with delayed wound healing. Lim et al, have hypothesized the association of dietary zinc in activating the nuclear factor kappa B (NF κ B), expression of proinflammatory cytokines (interleukin1b and tumor necrosis factor), and in neutrophil infiltration during the early stage of cutaneous wound healing [46]. Turmeric, red pepper, cloves, ginger, cumin, anise, fennel, basil, rosemary, garlic, and pomegranate, can block NF κ B activation of inflammatory cytokines [47].

Acrodermatitis enteropathica develops in a zinc deficient patient and a combined nutritional deficiency of zinc, EFAs, albumin and amino acids may result in acrodermatitis enteropathica [48].

The activities of lysyl oxidases that initiate the crosslinking of collagen and elastin decline with copper deficiency [49].

Chronic iron deficiency has resulted in spoon shaped nails (koilonychia), hair loss, glossitis with loss of papillae, angular cheilitis, and pruritus. Iron found in Liver, eggs, meat, dark and green vegetables, lentils, potatoes, soybeans, chick peas, black beans, spinach, etc. [29,50].

Carotenemia

Carotenemia is caused by too much intake of carotene rich food

such as oranges and carrots. β -Carotene is present in carrots. Hence excess eating of carrots has to be avoided [3].

Kwashiorkor

Kwashiorkor is the edematous form of protein energy malnutrition. Evidence has been linking aflatoxins, free oxygen radicals, leukotrienes; zinc deficiency, and essential fatty acid deficiency [4]. In the 1970s, Vasantha et al. [6] demonstrated that kwashiorkor, a severe protein/calorie deficiency, was related with skin biochemical changes in children [51].

Sources of protein Peas, beans, poultry, cereals, lentils, milk, cheese, eggs, meat, wet and dry fishes, pulses, and nuts [29].

Pemphigus

The role of thiol and tannin rich foods in exacerbating pemphigus have been reviewed by Tur E and Brenner S [52]. Substances such as thiols, thiocyanates, phenols and tannins can precipitate the pemphigus in a genetically predisposed individual [53].

Foodstuffs covering tannins include a wide variety of vegetables (cassava, eggplant), fruits (mango, cashew apple, guarana, raspberry, cherry, cranberry, blackberry, avocado, banana, apple, peach, grape and pear), nuts (betel nut, kola nut, walnut, cashew, peanut, pistachio), beverages (coffee, tea, cocoa, beer, wine, soft drinks), spices (ginger, ginseng, garlic, red chillies, asafetida, coriander, cumin, black pepper, ajwain) and food additives [52].

Dermatitis Herpetiformis

It is a well-known gluten intolerance disease and is controlled by a gluten-free diet. A gluten-free diet (GFD) is the mainstay of treatment in celiac disease [54].

After following 133 DH patients who consumed a GFD, Garioch et al. [55] reported that the related advantages are the need for medication is reduced or abolished, there is resolution of enteropathy, a general feeling of wellbeing and protective effect against development of lymphoma. Patients with DH often have malabsorption. A GFD improves absorption of essential nutrients and prevents alimentary deficiencies of iron, vitamin B12 and folate [54].

Food stuffs having gluten, and hence to be avoided, are wheat, rye, oats and barley. Rice, corn and potatoes are safe for ingesting. Iodine-containing food (fish, kelp, iodized salt and vitamin) may be avoided in patients who do not respond to a GFD, as iodides worsen DH by local chemotaxis and stimulating neutrophil migration [56].

Psoriasis

A low-calorie and low-protein diet has been recommended in the treatment of psoriasis [1]. Psoriasis is an inflammatory condition that appears to be aggravated by an inflammatory diet. An inflammatory diet may consist of a food allergen or a diet with imbalanced ω 6 and ω 3 fatty acids [57].

Many psoriatic patients show increased sensitivity to gluten and their symptoms improve on a gluten-free diet [58]. A vegetarian-based diet may put an individual at a risk of eating high amounts of vegetable oils and soy products, and low amounts of fish, which can tip the balance toward a proinflammatory state [1].

Low serum calcium and zinc during pregnancy is known to cause pustular psoriasis. Supplementations of food containing calcium and zinc have been suggested in such situations [3]. Iodide can precipitate

pustular psoriasis. Seafood and iodized salt are rich sources of iodine [59,60].

PUFA intake in Psoriasis

Daily intake (170 g) of oily fish EPA/DHA 1.8 g/day or fish oil (10 capsules three times a day) supplementation [61] parenteral infusion of EPA and DHA 4.2 g/day useful in acute guttate psoriasis [62]. Combined ω 3 and ω 6 fatty acid supplementation (rationale: low concentration of PUFA in membrane phospholipids, increased saturated fatty acids and decreased ω 6 fatty acid in psoriatic arthritis, high doses of linoleic acid suppressing LTB₄ production) [63].

The consumption of fresh fruits and vegetables, such as carrots and tomatoes, may be beneficial in psoriasis because of their high content of carotenoids, flavonoids and vitamin C [64]. A sufficient status of antioxidants (e.g., vitamin C, vitamin E, β -carotene and selenium) may be helpful to prevent an imbalance of oxidative stress and antioxidant defence in psoriasis [65].

Alcohol stimulates histamine release and may thereby worsen skin lesions [66]. The intake of alcohol is associated with a concomitant increase in the intake of fatty foods and reduced consumption of fresh vegetables and fruits. Hence, alcohol intake should be restricted in psoriasis.

Acne

American Academy of Dermatology published recommendations in 2007 suggesting that caloric restriction has no benefit in the management of acne and that there is insufficient suggestion to link the consumption of certain "food enemies" to acne. On the other hand, recent studies have suggested a rather close relationship between diet and acne [67,68].

Bulkeley's 1887 book [69] discussed the subject and up until the 1950s, in America, restrictions on various foods, most often dairy products, were presented in dermatology textbooks as part of acne therapy [70].

A positive association was made between dairy products (particularly skim milk) and acne [71-73]. A high glycemic load diet, processed cheese, a high-fat diet, and iodine play a role in the exacerbation of acne in Koreans [74]. Ludwig [75] and Liu and co-workers [76] found that chronic ingesting of high glycemic load carbohydrates may cause long-term hyperinsulinemia and insulin resistance. Insulin influences circulating concentrations of free insulin-like growth factor I (IGF-1) and insulin-like growth factor binding protein 3 (IGFBP-3), which in turn directly regulate keratinocyte proliferation and apoptosis [77].

Clement et al., in their studies have shown a positive association between the intake of skim milk and acne [78] Robyn N Smith et al., have recommended that nutrition-related lifestyle factors may play a role in the pathogenesis of acne [67]. The role of chocolate and other dietary factors in acne development has also been reported [79].

In the 1969 experiment by Fulton and co-workers 65 subjects (14 adolescent boys, 16 adolescent girls, and 35 young adult male prisoners) consumed either a 112-g bittersweet chocolate bar enriched with chocolate liquor and cocoa butter or a 112-g control bar without chocolate liquor and cocoa butter once a day for 4 weeks in a single-blind crossover design with a 3-week washout period. Authors concluded "ingestion of high amounts of chocolate did not materially affect the course of acne vulgaris or the output or composition of sebum [80]."

Herpes

Herpes is a viral infection of the skin. A study suggested that a mix of nutrients, such as those found in fruits and vegetables, act together to sustain immune health, rather than individual dietary intakes of vitamins A, B6, C, and E, and of folic acid, zinc, and iron [81].

Scleroderma

It is an autoimmune disease of the connective tissue, characterized by fibrosis and thickening of various tissues. Avoidance of high-fiber diet is advised to patients with scleroderma. Improvement in the skin of scleroderma on vitamin E supplementation has been reported [82,83].

Atopic Dermatitis

Foods such as chocolate, cheese, coffee, yogurt, and some Japanese foods such as glutinous rice cake, soy sauce, and fermented soybeans are stated to play an important role in unpredictable, uneven aggravation of skin lesions in patients with atopic dermatitis [84].

Arguments in favor of the role of diet in AD include the fact that some foods provoke AD, an elimination diet can heal AD, diet manipulation can prevent allergy in newborns at risk for atopy, presence of specific serum immunoglobulin (Ig) E for food allergens [85].

Food allergy plays a role in 20% of children under the age of 4 years with AD. A direct effect on eczema is observed in four of 10 children with AD and proven food allergy [86]. Ninety percent of food allergy is caused by six foods such as wheat, milk, soy, fish, eggs and peanut [87].

A recent Cochrane review of nine randomized controlled trials of food allergy in patients with AD showed that there seems to be no benefit of an egg and milk free diet in unselected participants with atopic eczema [88].

Treatment of AD can be supported by supplementation of 'probiotic' intestinal bacteria [89]. A probiotic is currently defined as a live microbial food supplement with a recognized beneficial effect on human health [90]. However, a recent cochrane Intervention Review suggests that probiotics are not an effective treatment for eczema and may, in fact, carry a small risk of adverse events such as infections and bowel ischemia [91].

Cow's milk containing penicillin spores, chocolates, food additives, citrus fruits, fish, shell fish, cheese, eggs, meat, nuts, alcohol, caffeine, tomatoes and wheat are known to aggravate atopic dermatitis [6].

Urticaria

Adverse reactions to food are a frequently discussed cause of urticaria. In acute urticaria, 63% of patients suspect food as the eliciting factor [3]. Penicillin can cause urticaria after food ingestion when present in beef, frozen meats and soft drinks, in penicillin sensitive individuals [92]. Food additives producing urticaria due to tartrazine dye has been reported [93]. Food additives most commonly concerned are azo-dyes, tartrazine, amaranth, sunset yellow and carmosine.

Allergic Contact Dermatitis

Approximately 30-50% of individuals who are allergic to natural rubber latex show a related hypersensitivity to some plant derived foods, especially freshly consumed fruits. This association of latex allergy and allergy to plant derived foods is called latex fruit [85].

The oral intake of nickel can induce systemic contact dermatitis in nickel sensitive individuals. A flare up of recurrent vesicular hand eczema is the most common clinical manifestation of systemic nickel contact dermatitis [94].

Certain foods are usually high in nickel content, such as cocoa and chocolate, soya beans, oatmeal, nuts and almonds, and fresh and dried legumes [95]. Food items most usually mentioned by patients as producing aggravation of dermatitis due to balsam of Peru are wine, candy, chocolate, cinnamon, curry, citrus fruit, tomatoes and flavorings. Avoidance of these food stuffs would constitute a low balsam diet and may alleviate contact dermatitis to balsam of Peru [96,97]. Nickel, cobalt and chromium allergies frequently coexist and patients sometimes respond to dietary restrictions of all three metals [98].

Fixed Drug Eruption

Artificial flavors, colors and preservatives in foods as well as dyes in medications can rarely be culprits in classic fixed drug eruptions [99,100].

Homocystinuria

A low methionine diet is mandatory. Forbidden foods contain milk and milk products, meat and fish, wheat, maize, rice, pulses, legumes, nuts and dried fruits. Fruits and vegetables may be consumed in moderate amounts. Foods that need not be controlled are sago, arrowroot, corn flour, custard, sugars, fats, tea and coffee [101].

Galactosemia

Dietary elimination of galactose and lactose is necessary throughout childhood. Nutritionally satisfactory galactose/lactosefree milk should be used during infancy. In later childhood, occasional lactose free milk and calcium and vitamin supplements may suffice [101].

Vitiligo

Childhood Vitiligo has been related to malnutrition and intake of junk food [102]. Food containing antioxidants like citrus fruits, carrots and tomatoes (Vit A), sour yoghurt, sour pickles etc. is contraindicated in patients with vitiligo [3]. The simultaneous consumption of milk and fish is also discouraged.

It has been found, however, that oral supplementation with antioxidants containing alfa-lipoic acid and vitamin B12 before and during NB-UVB broadband UVB significantly improves the clinical effectiveness of phototherapy [103,104].

Rosacea

Consumption of coffee, tea, other hot drinks, tobacco, alcoholic beverages, and spicy foods is known to precipitate rosacea [10] hence it should be avoided [105].

Refsum's Disease

Vegetables and fruits rich in phytanic acid are contraindicated [106].

Phytanic acid is almost exclusively of exogenous origin and dietary restriction reduces plasma and tissue levels. Fish, beef, lamb and dairy products should be avoided. The average daily intake of phytanic acid is 50-100 mg/day, which should ideally be reduced to 10-20 mg/day [85].

Nutritional Ichthyosis

Topical application of sunflower or safflower oil and systemic supplementation of fish, which are rich in linoleic acid (essential fatty acids), are useful [107].

Phrynoderma

Green leafy vegetables, carrots, tomatoes, milk, eggs, fish (containing essential fatty acids), soyabean, and sunflower oil are useful [3,108].

Xanthomas

Xanthelasma may be associated with hyperlipidemia [108]. In addition to specific therapy with lipid-lowering agents, patients need lifestyle modifications, in which foods containing high concentrations of cholesterol, such as fat, eggs, meat and dairy products are to be avoided [109,110].

Gout

Patients with gout is the avoidance of foods with a high purine content, such as organ meat (liver, kidney), selected fish and shellfish, meat and yeast extract brewer, baker's yeast, pulses, certain vegetables (spinach, asparagus) and fermented milk products. Studies have observed an increased risk of gout among those who consumed large quantities of meat, seafood and alcohol. Although limited by confounding variables; low-fat dairy products, ascorbic acid and wine consumption appear to be protective for the development of gout [111].

Porphyrias

High-fibre diet of natural vegetable/fruit products with a daily caloric content of 1676 kJ/day has been assessed for porphyria cutanea tarda and is found to be useful. In addition, patients with porphyria may use from dietary supplementation of carotene-containing foods, such as carrots and green leafy vegetables.

Conclusion

Relation between diet and dermatological conditions is well established. Dietary changes or modification might help to prevent recurrences of many skin diseases. Diet which we are presented in this paper show indication that there is link between nutrition and skin condition. Evidence from literature review as well as published works show that appropriate nutritional supplementation is beneficial in the prevention of the harmful effect of UV exposure, in the management of skin aging and of reactive skin, as well as for limiting hair loss. Altogether, the data show that a balanced diet represents a globalized approach for improving skin health.

References

1. Basavaraj KH, Seemanthini C, Rashmi R (2010) Diet in dermatology: present perspectives. *Indian J Dermatol* 55: 205-210.
2. Rushton DH (2002) Nutritional factors and hair loss. *Clin Exp Dermatol* 27: 396-404.
3. Hanumanthappa H (2001) Diet in dermatology. *Indian J Dermatol Venereol Leprol* 67: 284-286.
4. Lazzari P, Sanna A, Mastinu A, Cabasino S, Manca I, et al. (2011) Weight loss induced by rimonabant is associated with an altered leptin expression and hypothalamic leptin signaling in diet-induced obese mice. *Behav Brain Res* 217: 432-438.
5. Liu T, Howard RM, Mancini AJ, Weston WL, Paller AS, et al. (2001) Kwashiorkor in the United States: fad diets, perceived and true milk allergy, and nutritional ignorance. *Arch Dermatol* 137: 630-636.
6. Purba MB, Kouris-Blazos A, Wattanapenpaiboon N, Lukito W, Rothenberg EM, et al. (2001) Skin wrinkling: can food make a difference? *J Am Coll Nutr* 20: 71-80.
7. Cosgrove MC, Franco OH, Granger SP, Murray PG, Mayes AE (2007) Dietary nutrient intakes and skin-aging appearance among middle-aged American women. *Am J Clin Nutr* 86: 1225-1231.
8. Boelsma E, van de Vijver LP, Goldbohm RA, Klöpping-Ketelaars IA, Hendriks HF, et al. (2003) Human skin condition and its associations with nutrient concentrations in serum and diet. *Am J Clin Nutr* 77: 348-355.
9. Roe DA (1986) *Nutrition and the skin*. New York: Liss.
10. Garrow JS, James WP, Ralph A (2000) *Skin, hair and nails: Human nutrition and dietetics*. (10th edn) Churchill Livingstone 731-746.
11. Bender DA (1999) Pellagra. In: Sadler MJ, Strain JJ, Caballero B, Academic Press; 1999. pp. 1298-1302.
12. Ziboh VA, Miller CC, Cho Y (2000) Metabolism of polyunsaturated fatty acids by skin epidermal enzymes: generation of antiinflammatory and antiproliferative metabolites. *Am J Clin Nutr* 71: 361S-6S.
13. Tobin DJ (2006) Biochemistry of human skin--our brain on the outside. *Chem Soc Rev* 35: 52-67.
14. Burton JL (1989) Diet and dermatology. *BMJ* 298: 770-771.
15. Boelsma E, Hendriks HF, Roza L (2001) Nutritional skin care: health effects of micronutrients and fatty acids. *Am J Clin Nutr* 73: 853-864.
16. Alexander JM *Micronutrients and Skin Health*: Linus Pauling Institute Oregon State University.
17. Freinkel RK (2001) Metabolism of skin. In: Freinkel RK, Woodley D. *The biology of the skin*. New York: Parthenon Pub. Group pp.191-199.
18. Van Hattem S, Bootsma AH, Thio HB (2008) Skin manifestations of diabetes. *Cleve Clin J Med* 75: 772, 774, 776-777 passim.
19. Nagata C, Nakamura K, Wada K, Oba S, Hayashi M, et al. (2010) Association of dietary fat, vegetables and antioxidant micronutrients with skin ageing in Japanese women. *Br J Nutr* 103: pp 1493-1498.
20. Chia MW, Tay YK, Liu TT (2008) Phrynoderma: a forgotten entity in a developed country. *Singapore Med J* 49: e160-162.
21. Fielding JM, Rowley KG, Cooper P, O' Dea K (2005) Increases in plasma lycopene concentration after consumption of tomatoes cooked with olive oil. *Asia Pac J Clin Nutr* 14: 131-136.
22. Unlu NZ, Bohn T, Clinton SK, Schwartz SJ (2005) Carotenoid absorption from salad and salsa by humans is enhanced by the addition of avocado or avocado oil. *J Nutr* 135: 431-436.
23. Brown MJ, Ferruzzi MG, Nguyen ML, Cooper DA, Eldridge AL, et al. (2004) Carotenoid bioavailability is higher from salads ingested with full-fat than with fat-reduced salad dressings as measured with electrochemical detection. *Am J Clin Nutr* 80: 396-403.
24. Harrison EH (2005) Mechanisms of digestion and absorption of dietary vitamin A. *Annu Rev Nutr* 25: 87-103.
25. Mulokozi G, Hedrén E, Svanberg U (2004) In vitro accessibility and intake of beta-carotene from cooked green leafy vegetables and their estimated contribution to vitamin A requirements. *Plant Foods Hum Nutr* 59: 1-9.
26. Ribaya-Mercado JD1 (2002) Influence of dietary fat on beta-carotene absorption and bioconversion into vitamin A. *Nutr Rev* 60: 104-110.
27. Griffiths WA (1980) Pityriasis rubra pilaris. *Clin Exp Dermatol* 5: 105-112.
28. David L, *Healthy nutrition and your skin*
29. Amirul IM *Carbohydrates, Proteins, Vitamins and Minerals*.
30. Das R, Parajuli S, Gupta S (2006) A rash imposition from a lifestyle omission: a case report of pellagra. *Ulster Med J* 75: 92-93.
31. Thami GP, Kaur S, Kanwar AJ (2002) Delayed reactivation of haloperidol induced photosensitive dermatitis by methotrexate. *Postgrad Med J* 78: 116-117.
32. Puizina-IviA N (2008) Skin aging. *Acta Dermatovenerol Alp Pannonica Adriat* 17: 47-54.

33. Heinrich U, Gartner C, Wiebusch M, Eichler O, Sies H, et al. (2003) Supplementation with beta-carotene or a similar amount of mixed carotenoids protects humans from UV-induced erythema. *J Nutr* 133: 98-101.
34. Stahl W, Heinrich U, Jungmann H, Sies H, Tronnier H (2000) Carotenoids and carotenoids plus vitamin E protect against ultraviolet light-induced erythema in humans. *Am J Clin Nutr* 71: 795-798.
35. Stahl W, Sies H (2002) Carotenoids and protection against solar UV radiation. *Skin Pharmacol Appl Skin Physiol* 15: 291-296.
36. Stahl W, Heinrich U, Wiseman S, Eichler O, Sies H, et al. (2001) Dietary tomato paste protects against ultraviolet light-induced erythema in humans. *J Nutr* 131: 1449-1451.
37. Black HS (2004) Mechanisms of pro- and antioxidation. *J Nutr* 134: 3169S-3170S.
38. Horrobin DF (1989) Essential fatty acids in clinical dermatology. *J Am Acad Dermatol* 20: 1045-1053.
39. Zouboulis CC (2001) Is acne vulgaris a genuine inflammatory disease? *Dermatology* 203: 277-279.
40. Trebble T, Arden NK, Stroud MA, Wootton SA, Burdge GC, et al. (2003) Inhibition of tumour necrosis factor-alpha and interleukin 6 production by mononuclear cells following dietary fish-oil supplementation in healthy men and response to antioxidant co-supplementation. *Br J Nutr* 90: 405-412.
41. James MJ, Gibson RA, Cleland LG (2000) Dietary polyunsaturated fatty acids and inflammatory mediator production. *Am J Clin Nutr* 71: 343S-8S.
42. Rubin MG, Kim K, Logan AC (2008) Acne vulgaris, mental health and omega-3 fatty acids: a report of cases. *Lipids Health Dis* 7: 36.
43. Llewellyn A (1967) Variations in the composition of skin surface lipid associated with dietary carbohydrates. *Proc Nutr Soc* 26: 11.
44. Macdonald I (1964) Changes in the Fatty Acid Composition of Sebum Associated With High Carbohydrate Diets. *Nature* 203: 1067-1068.
45. MacDonald I (1967) Dietary carbohydrates and skin lipids. *Br J Dermatol* 79: 119-121.
46. Lim Y, Levy M, Bray TM (2004) Dietary zinc alters early inflammatory responses during cutaneous wound healing in weanling CD-1 mice. *J Nutr* 134: 811-816.
47. Aggarwal BB, Shishodia S (2004) Suppression of the nuclear factor-kappaB activation pathway by spice-derived phytochemicals: reasoning for seasoning. *Ann N Y Acad Sci* 1030: 434-441.
48. Yu HH, Shan YS, Lin PW (2007) Zinc deficiency with acrodermatitis enteropathica-like eruption after pancreaticoduodenectomy. *J Formos Med Assoc* 106: 864-868.
49. Berger MM, Baines M, Raffoul W, Chioloro RL, Reeves C, et al (2007) Trace element supplementation after major burns modulates antioxidant status and clinical course by way increased tissue trace element concentration. *Am J Clin Nutr* 85: 1293-300.
50. Takita Y, Ichimiya M, Hamamoto Y, Muto M (2006) A case of carotenemia associated with ingestion of nutrient supplements. *J Dermatol* 33: 132-134.
51. Vasantha L, Srikantia SG, Gopalan C (1970) Biochemical changes in the skin in kwashiorkor. *Am J Clin Nutr* 23: 78-82.
52. Tur E, Brenner S (1998) Diet and pemphigus. In pursuit of exogenous factors in pemphigus and fogo selvagem. *Arch Dermatol* 134: 1406-1410.
53. Brenner S, Srebrnik A, Goldberg I (2003) Pemphigus can be induced by topical phenol as well as by foods and drugs that contain phenols or thiols. *J Cosmet Dermatol* 2: 161-165.
54. Turchin I, Barankin B (2005) Dermatitis herpetiformis and gluten-free diet. *Dermatol Online J* 11: 6.
55. Garioch JJ, Lewis HM, Sargent SA, Leonard JN, Fry L (1994) 25 years' experience of a gluten-free diet in the treatment of dermatitis herpetiformis. *Br J Dermatol* 131: 541-545.
56. Rottmann LH (1991) Details of the gluten-free diet for the patient with dermatitis herpetiformis. *Clin Dermatol* 9: 409-414.
57. Traub M, Marshall K (2007) Psoriasis--pathophysiology, conventional, and alternative approaches to treatment. *Altern Med Rev* 12: 319-330.
58. Brown AC, Hairfield M, Richards DG, McMillin DL, Mein EA, et al. (2004) Medical nutrition therapy as a potential complementary treatment for psoriasis-five case reports. *Altern Med Rev* 9: 297-307.
59. Marfatia YS, Asmi P (2002) Diet in dermatology. *Indian J Dermatol Venereol Leprol* 68: 313.
60. Collier PM, Ursell A, Zaremba K, Payne CM, Staughton RC, et al. (1993) Effect of regular consumption of oily fish compared with white fish on chronic plaque psoriasis. *Eur J Clin Nutr* 47: 251-254.
61. Bjørneboe A, Smith AK, Bjørneboe GE, Thune PO, Drevon CA (1988) Effect of dietary supplementation with n-3 fatty acids on clinical manifestations of psoriasis. *Br J Dermatol* 118: 77-83.
62. Chalmers RJ, O'Sullivan T, Owen CM, Griffiths CE (2001) A systematic review of treatments for guttate psoriasis. *Br J Dermatol* 145: 891-894.
63. Kragballe K (1989) Dietary supplementation with a combination of n-3 and n-6 fatty acids (super gamma-oil marine) improves psoriasis. *Acta Derm Venereol* 69: 265-268.
64. Naldi L, Parazzini F, Peli L, Chatenoud L, Cainelli T (1996) Dietary factors and the risk of psoriasis. Results of an Italian case-control study. *Br J Dermatol* 134: 101-106.
65. Wolters M (2005) Diet and psoriasis: experimental data and clinical evidence. *Br J Dermatol* 153: 706-714.
66. Smith KE, Fenske NA (2000) Cutaneous manifestations of alcohol abuse. *J Am Acad Dermatol* 43: 1-16.
67. Smith RN, Mann NJ, Braue A, Mäkeläinen H, Varigos GA (2007) A low-glycemic-load diet improves symptoms in acne vulgaris patients: a randomized controlled trial. *Am J Clin Nutr* 86: 107-115.
68. Smith RN, Braue A, Varigos GA, Mann NJ (2008) The effect of a low glycemic load diet on acne vulgaris and the fatty acid composition of skin surface triglycerides. *J Dermatol Sci* 50: 41-52.
69. Bulkley LD (1885) Acne, Its Etiology, Pathology and Treatment. New York GP Putnam's Sons.
70. Fisher JK (1965) Acne Vulgaris; A Study of One Thousand Cases. In: Fisher JK.
71. Adebamowo CA, Spiegelman D, Danby FW, Frazier AL, Willett WC, et al. (2005) High school dietary dairy intake and teenage acne. *J Am Acad Dermatol* 52: 207-214.
72. Adebamowo CA, Spiegelman D, Berkey CS, Danby FW, Rockett HH, et al (2006) Milk consumption and acne in adolescent girls. *Dermatol Online J* 12: 1.
73. Adebamowo CA, Spiegelman D, Berkey CS, Danby FW, Rockett HH, et al. (2008) Milk consumption and acne in teenaged boys. *J Am Acad Dermatol* 58: 787-793.
74. Jung JY, Yoon MY, Min SU, Hong JS, Choi YS, et al. (2010) The influence of dietary patterns on acne vulgaris in Koreans. *Eur J Dermatol* 20: 768-772.
75. Ludwig DS (2002) The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA* 287: 2414-2423.
76. Liu S, Willett WC (2002) Dietary glycemic load and atherothrombotic risk. *Curr Atheroscler Rep* 4: 454-461.
77. Edmondson SR, Thumiger SP, Werther GA, Wraight CJ (2003) Epidermal homeostasis: the role of the growth hormone and insulin-like growth factor systems. *Endocr Rev* 24: 737-764.
78. Magin P, Pond D, Smith W, Watson A (2005) A systematic review of the evidence for 'myths and misconceptions' in acne management: diet, face-washing and sunlight. *Fam Pract* 22: 62-70.
79. Fulton JE Jr, Plewig G, Kligman AM (1969) Effect of chocolate on acne vulgaris. *JAMA* 210: 2071-2074.
80. Thomas SL, Wheeler JG, Hall AJ (2006) Micronutrient intake and the risk of herpes zoster: a case-control study. *Int J Epidemiol* 35: 307-314.
81. Gough A, Sheeran T, Bacon P, Emery P (1998) Dietary advice in systemic sclerosis: the dangers of a high fibre diet. *Ann Rheum Dis* 57: 641-642.
82. Gaby AR (2006) Natural remedies for scleroderma. *Altern Med Rev* 11: 188-195.

83. Uenishi T, Sugiura H, Uehara M (2003) Role of foods in irregular aggravation of atopic dermatitis. *J Dermatol* 30: 91-97.
84. Sowmya K, Devinder MT (2010) Diet in dermatology: Revisited Indian *J Dermatol Venereol Leprol* 76: 103-115.
85. Oranje AP, de Waard-van der Spek FB (2000) Atopic dermatitis and diet. *J Eur Acad Dermatol Venereol* 14: 437-438.
86. Krafchik BR, Halbert A, Yamamoto K, Sasaki R (2003) Eczematous dermatitis. In: Shachner LA, Hansen RC, editors. *Pediatric dermatology*. (3rd edn) London 609-642.
87. Bath-Hextall F, Delamere FM, Williams HC (2008) Dietary exclusions for established atopic eczema. *Cochrane Database Syst Rev*: CD005203.
88. Isolauri E, Arvola T, Sütas Y, Moilanen E, Salminen S (2000) Probiotics in the management of atopic eczema. *Clin Exp Allergy* 30: 1604-1610.
89. Laitinen K, Isolauri E (2005) Management of food allergy: vitamins, fatty acids or probiotics? *Eur J Gastroenterol Hepatol* 17: 1305-1311.
90. Boyle RJ, Bath-Hextall FJ, Leonardi-Bee J, Murrell DF, Tang ML (2008) Probiotics for treating eczema. *Cochrane Database Syst Rev*: CD006135.
91. Ormerod AD, Reid TM, Main RA (1987) Penicillin in milk--its importance in urticaria. *Clin Allergy* 17: 229-234.
92. Lockett SD (1959) Allergic reactions due to F D and C Yellow No. 5, tartrazine, an aniline dye used as a coloring and identifying agent in various steroids. *Ann Allergy* 17: 719-721.
93. Purello D'Ambrosio F, Bagnato GF, Guameri B, Musarra A, Di Lorenzo G, et al. (1998) The role of nickel in foods exacerbating nickel contact dermatitis. *Allergy* 53: 143-145.
94. Sharma AD1 (2007) Relationship between nickel allergy and diet. *Indian J Dermatol Venereol Leprol* 73: 307-312.
95. Veien NK, Hattel T, Laurberg G (1996) Can oral challenge with balsam of Peru predict possible benefit from a low-balsam diet? *Am J Contact Dermat* 7: 84-87.
96. Salam TN, Fowler JF Jr (2001) Balsam-related systemic contact dermatitis. *J Am Acad Dermatol* 45: 377-381.
97. Ruff CA, Belsito DV (2006) The impact of various patient factors on contact allergy to nickel, cobalt, and chromate. *J Am Acad Dermatol* 55: 32-39.
98. Ritter SE, Meffert J (2004) A refractory fixed drug reaction to a dye used in an oral contraceptive. *Cutis* 74: 243-244.
99. Orchard DC, Varigos GA (1997) Fixed drug eruption to tartrazine. *Australas J Dermatol* 38: 212-214.
100. Kabra M (2002) Dietary management of inborn errors of metabolism. *Indian J Pediatr* 69: 421-426.
101. Behl PN, Agarwal A, Srivastava G (1999) Etiopathogenesis of vitiligo: Are we dealing with an environmental disorder? *Indian J Dermatol Venereol Leprol* 65: 161-167.
102. Dell'Anna ML, Mastrofrancesco A, Sala R, Venturini M, Ottaviani M, et al. (2007) Antioxidants and narrow band-UVB in the treatment of vitiligo: a double-blind placebo controlled trial. *Clin Exp Dermatol* 32: 631-636.
103. Don P, Iuga A, Dacko A, Hardick K (2006) Treatment of vitiligo with broadband ultraviolet B and vitamins. *Int J Dermatol* 45: 63-65.
104. Wilkin JK (1979) Heat and caffeine induced flushing in erythematotelangiectatic rosacea. *J Invest Dermatol* 73: 310.
105. Steinberg D, Vroom FQ, Engel WK, Cammermeyer J, Mize CE, et al. (1967) Refsum's disease--a recently characterized lipidosis involving the nervous system. Combined clinical staff conference at the National Institutes of Health. *Ann Intern Med* 66: 365-395.
106. Hansen AD, Wiese HF, Boesche (1963) Role of linoleic acid in infant nutrition. *Paediatrics* (supplement) 171.
107. Jain A, Goyal P, Nigam PK, Gurbaksh H, Sharma RC (2007) Xanthelasma Palpebrarum-clinical and biochemical profile in a tertiary care hospital of Delhi. *Indian J Clin Biochem* 22: 151-153.
108. Sarkany RPE, Breathnach SM, Seymour CA, Weismann K, Burns DA (2004) Metabolic and nutritional disorders. In: Burns T, Breathnach S, Cox N, Griffiths C, editors. *Rook's Textbook of Dermatology*. (7th edn). Massachusetts: Blackwell science 57: 1-124.
109. White LE (2008) Xanthomas and lipoprotein disorders. In: Wolff K, Goldsmith LA, Katz SI, Gilchrist BA, Paller AS, Leffell DJ, editors. *Fitzpatrick's Dermatology in General Medicine*. (7th edn) New York: McGraw Hill 1272-1280.
110. Lee SJ, Terkeltaub RA, Kavanaugh A (2006) Recent developments in diet and gout. *Curr Opin Rheumatol* 18: 193-198.
111. Dabrowska E, Jablonska KI, Falkiewicz B (2001) Effect of high fiber vegetable-fruit diet on the activity of liver damage and serum iron level in porphyria cutanea tarda (PCT). *Med Sci Monit* 7: 282-286.