A) Atopic Dermatitis

Atopic dermatitis, also called atopy, is a chronic non-contagious multifactorial skin disorder. Symptoms of atopy include itching, swelling (caused by scratching), cracking, weeping of clear fluid followed by crusting and scaling. Atopy is becoming more common in developed countries and has been associated with pollution, infections, housing and diet (Denburg et al. 2005).

 Evaluations of dietary habits in atopic and non-atopic men and women that were age and sex matched have demonstrated that there is no association between dietary habits and clinical status of atopy (Solvoll et al. 2000). An epidemiological study in Denmark involving the comparison of fatty acid composition of breast milk in atopic and non-atopic mothers while taking into account their dietary habits has shown that the breast milk of mothers in all atopic groups had significantly lower concentrations of 20:5n-3 eicosapentanoic acid (EPA) and other long chain polyunsaturated fatty acids (PUFAs), as well as significantly higher concentrations of 22:5n-6 docosapentanoic acid than the breast milk of non-atopic mothers (Lauritzen et al. 2006). It was concluded that the PUFA composition differences of breast milk were due to the differences in the diet of the mothers and that the acid composition of breast milk is not affected by atopy, and that it does not vary in mothers with different forms of atopic disease. Another breast milk fatty acid composition study performed in Finland found that infants with atopy had consumed breast milk with a higher ratio of saturated to polyunsaturated and less omega-3 fatty acids (Hoppu et al. 2005). The authors concluded that low breast milk omega-3 fatty acid levels may be a risk factor for atopy; furthermore, they concluded that stearic acid in breast milk is related to the development of atopic dermatitis.

 A randomized control trial was performed with 83 atopic pregnant women supplemented with either 3.7 g omega-3 PUFAs/ day from fish oil or placebo for 20 days before gestation (Dunstan et al. 2003). It was determined that there was no difference in the frequency of atopy in the infants at one year of age in the fish oil supplemented group, however the infants in the fish oil group had less severe signs of the disease. Furthermore, all neonatal cytokines (associated with inflammation) examined (IL-5, IL-13, IL-10, and IFN-gamma) were lower in the fish oil group, IL-10 in response to cat being the only cytokine that was statistically significant (p<0.05). The cord blood from these same women was also analyzed for hemopoietic progenitors with respect to the development of atopy in high risk infants (Denburg et al.)
2005). It was concluded that while supplementation with n-3 fatty acids could alter the expression of hemopoietic progenitor cells, the study lacked the sample size to determine whether supplementation of pregnant women with n-3 fatty acids could affect the development of atopy in infants.

A meta-analysis of five studies involving oral fish oil supplementation as treatment for atopy showed no statistically-significant improvements in any of the evaluation components (area, pruritus, erythema, lichenifications, dryness or scaling)(Van Gool et al. 2004). The capsules used in these studies contained both EPA and docosahexaenoic acid (DHA) in daily doses. The doses ranged from 204mg to 3060mg of EPA and 132mg to 1920 mg for DHA, the period of supplementation ranged from 12 to 16 weeks.

B) Psoriasis

Psoriasis is an inflammatory skin disorder that is characterized by fibroblast activation, leukocyte infiltration, increased vascularization of the skin, and hyperproliferation of keratinocytes (Mayser et al 2002).

Four uncontrolled crossover studies comparing the consumption of 170g of white fish or oily fish daily for four weeks showed an increase in the plasma EPA concentrations and “modest significant clinical improvement” in the oily fish group only (Wolters 2005). Four other uncontrolled studies involving supplementation with EPA/DHA daily with intakes ranging from 2 g to 12 g omega-3 fatty acids also showed improvements in psoriasis severity. However the results are less promising in randomized controlled trials, with only one of the fours studies performed reporting positive results with daily oral omega-3 supplementation (3 g omega-3, predominantly EPA).

Further randomized controlled trials in which subjects with acute-exanthematic psoriasis were intravenously infused with either a 50 ml omega-3 fatty acid emulsion or an omega-6 emulsion for 10 days showed a decrease in disease severity in the omega-3 group; however, the symptoms returned 1-2 weeks after terminating lipid infusions (Mayser et al 2002).

C) Skin Cancer
Skin cancer is the most common form of cancer in the white Caucasian population. (Rhodes et al. 2003) This increase in incidence of skin cancer is due to increased exposure of the skin to ultraviolet radiation (UVR); UVR causes DNA damage in the skin and ultimately leads to skin cancer. Skin cancer has many different forms, the most common of which is basal cell carcinoma (BCC) followed by squamous cell carcinoma (SCC). The less common form but with the highest mortality rate is malignant melanoma (MM).

Initial animal studies involving supplementation of omega-3 PUFAs indicated a protective affect against skin cancer at promotion and initiation stages of cancer development although the mechanism is not known (Rhodes et al. 2003).

In humans, an epidemiological case-control study has shown that increased fish consumption is related to a decreased incidence of skin cancer (Black and Rhodes 2006 from Kune et al. 1992). A prospective study found that higher intakes of omega-3 fatty acids were associated with lower BCC risk. However, when non-white males were removed the association weakened (Black and Rhodes 2006 from van Dam et al. 2000). Another population-based study was able to demonstrate a lower risk of SCC with higher intakes of omega-3 fatty acids (Black and Rhodes 2006 from Hakim et al. 2000).

A double-blind randomized study supplemented 42 subjects daily with 4 g of purified omega-3 PUFA (EPA) or a monounsaturated fatty acid, oleic acid (OA) for 3 months (Rhodes et al. 2003). The EPA supplemented group displayed an 8-fold increase from baseline of bioavailable EPA content at the skin level. When exposed to UVR sunburn sensitivity was reduced and early markers of damages (P-53) was reduced. Significant changes were not seen in the OA supplemented group in any of the tested parameters. The authors state that these results indicate that dietary EPA protects against acute UVR induced genotoxicity and that longer term supplementation might reduce skin cancer in humans.


