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O2 SKIN OXIDATION PROTECTION
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• Vitamin C
• Vitamin D
• Vitamin E
• Folate-Folic Acid
• Omega-3 and Omega-6

PHENOTYPE NAME	GENETIC RESULTS	PAGE #	GENE/LOCUS	MARKER	GENOTYPE	GENE/LOCUS	MARKER	GENOTYPE
SKIN PHOTOAGING								
WRINKLES AND COLLAGEN DEGRADATION	**INCREASED RISK**	P.8	*MMP1*	rs1799750	TC/T	*STXBP5L*	rs322458	C/C
TANNING RESPONSE	**REDUCED**	P.9	*EXOC2* *HERC2* *intergenic* *intergenic* *IRF4* *MC1R* *MC1R* *NCOA6*	rs12210050 rs12913832 rs1015362 rs4911414 rs12203592 rs1805007 rs1805008 rs4911442	C/C A/G C/C G/G C/C C/C C/C A/A	*SLC24A5* *SLC24A5* *SLC45A2* *SLC45A2* *TYR* *TYR* *TYR*	rs1426654 rs2555364 rs26722 rs16891982 rs1042602 rs1126809 rs1393350	A/A G/G C/C G/G A/A G/G G/G
SUN SPOTS (LENTIGINES)	**NORMAL RISK**	P.9	*IRF4* *MC1R* *MC1R* *MC1R* *MC1R*	rs12203592 rs885479 rs1110400 rs1805005 rs1805006	C/C G/G T/T G/G C/C	*MC1R* *MC1R* *MC1R* *MC1R* *MC1R*	rs1805007 rs1805008 rs1805009 rs2228479 rs11547464	C/C C/C G/G G/G G/G
FRECKLES (EPHELIDES)	**NORMAL RISK**	P.10	*intergenic* *intergenic* *IRF4* *MC1R* *MC1R*	rs1540771 rs4911414 rs12203592 rs1805007 rs1805008	C/C G/G C/C C/C C/C	*MC1R* *MC1R* *NCOA6* *TYR* *TYR*	rs1805009 rs11547464 rs4911442 rs1042602 rs1393350	G/G G/G A/A A/A G/G
SKIN TEXTURE AND ELASTICITY								
CELLULITE	**INCREASED RISK**	P.10	*ACE*	rs4646994/ rs1799752	D/D	*HIF1A*	rs11549465	C/C
STRETCH MARKS (STRIAE DISTENSAE)	**INCREASED RISK**	P.11	*ELN* *HMCN1*	rs7787362 rs10798036	T/C C/G	*SRPX* *TMEM18*	rs35318931 rs7594220	G/G A/A
VARICOSE VEINS	**INCREASED RISK**	P.11	*MTHFR*	rs1801131	T/G	*MTHFR*	rs1801133	G/G
SKIN INFLAMMATION AND ALLERGY RISK								
ROSACEA	**INCREASED RISK**	P.12	*intergenic*	rs763035	A/A	*intergenic*	rs111314066	A/A
CONTACT DERMATITIS	**NORMAL RISK**	P.12	*FLG*	rs61816761	G/G	*FLG*	rs558269137	CACTG/ CACTG
GENERALIZED PSORIASIS	**HIGH RISK**	P.13	*HLA-C* *HLA-C* *IL12B* *IL13*	rs1265181 rs12191877 rs2082412 rs20541	C/C C/C G/G G/G	*IL23R* *MTHFR* *TNFAIP3* *TNIP1*	rs2201841 rs1801133 rs610604 rs17728338	A/G G/G G/T G/G
ECZEMA (ATOPIC DERMATITIS)	**NORMAL RISK**	P.13	*FLG* *FLG* *FLG* *FLG* *FLG* *FLG*	FLG:1249insG FLG:S2889X rs61816761 rs121909626 rs138726443 rs150597413	A/A TGG/TGG G/G G/G G/G G/G	*FLG* *FLG* *FLG* *FLG* *FLG*	rs200519781 rs374588791 rs397507563 rs558269137 rs761212672	CT/CT C/C AC/AC CACTG/ CACTG G/G

PHENOTYPE NAME	GENETIC RESULTS	PAGE #	GENE/LOCUS	MARKER	GENOTYPE	GENE/LOCUS	MARKER	GENOTYPE
SKIN MOISTURE FACTOR								
DRY SKIN (XEROSIS AND ICHTHYOSIS)	**NORMAL RISK**	P.14	*FLG* *FLG* *FLG*	rs61816761 rs138726443 rs150597413	G/G G/G G/G	*FLG* *FLG* *FLG*	rs200519781 rs397507563 rs558269137	CT/CT AC/AC CACTG/CACTG
O_2 SKIN OXIDATION PROTECTION								
ANTIOXIDATION RESPONSE	**NORMAL**	P.15	*CAT* *GPX1* *NQO1*	rs1001179 rs1050450 rs1800566	C/T G/A G/A	*NQO1* *SOD2*	rs2917666 rs4880	C/G A/G
SKIN GLYCATION								
GLYCATION PROTECTION	**REDUCED**	P.15	*AGER* *AGER* *AGER*	rs1800624 rs1800625 rs2070600	A/A G/G C/C	*GLO1* *GLO1*	rs1049346 rs1130534	G/G T/T
SKIN NUTRITIONAL NEEDS								
VITAMIN A DEFICIENCY	**INCREASED RISK**	P.16	*BCMO1*	rs7501331	C/T	*BCMO1*	rs12934922	A/T
VITAMIN B2 DEFICIENCY	**NORMAL RISK**	P.17	*MTHFR*	rs1801133	G/G			
VITAMIN B6 DEFICIENCY	**INCREASED RISK**	P.17	*NBPF3*	rs4654748	C/T			
VITAMIN B12 DEFICIENCY	**INCREASED RISK**	P.18	*FUT2*	rs602662	G/A			
VITAMIN C DEFICIENCY	**INCREASED RISK**	P.18	*SLC23A1*	rs33972313	C/T			
VITAMIN D DEFICIENCY	**NORMAL RISK**	P.19	*GC*	rs2282679	T/T			
VITAMIN E DEFICIENCY	**NORMAL RISK**	P.19	*intergenic*	rs12272004	C/C			
FOLATE-FOLIC ACID DEFICIENCY	**INCREASED RISK**	P.20	*MTHFR*	rs1801131	T/G	*MTHFR*	rs1801133	G/G
OMEGA-3 AND OMEGA-6 DEFICIENCY	**INCREASED RISK**	P.20	*FADS1*	rs174547	C/C			

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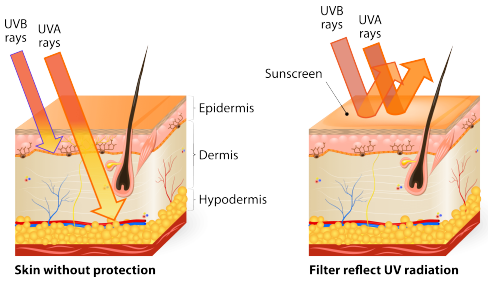
Healthy Skin From Within™

The following pages contain information about the skin phenotypes on the left side, along with your patient's result on the right side. For the tested phenotypes, multiple available treatment options are listed as a reference.

These recommendations can be taken in consideration together with the patient's medical history when you make individual recommendation for your patient.

A Product Glossary is provided at the end of the report for Physician Use.

SKIN PHOTOAGING



Photoaging refers to aging of the skin as a result of exposure to ultraviolet (UV) radiation over a person's lifetime. Although photoaging is affected by extrinsic (environmental) factors like gravity or smoking, all skin is susceptible to photoaging with UV exposure. Intrinsic factors, including skin pigmentation and genetics, can also affect the individual's response to extrinsic factors, thus affecting the degree and type of potential photoaging. Different types of skin, for example, respond differently to tanning, impacting the likelihood of developing sun spots or freckles, while vitamins and nutrition can affect collagen production and the ability of skin cells to repair damage sustained from UV exposure. The best defense against photoaging is to understand individual risk factors, maintain proper nutrition and limit UV exposure.

WRINKLES AND COLLAGEN DEGRADATION

Wrinkles can range in severity from fine lines to deep furrows in the skin. Wrinkling is a sign of skin aging and is caused by both *intrinsic factors* (e.g. genetics, hormonal state and skin pigment) and *extrinsic factors* (e.g. the passage of time, gravity, chronic ultraviolet exposure, alcohol abuse and smoking) *(1)*. These factors can cause damage to skin cells and breakdown of supportive structures called collagen in the dermis of the skin. *(2,3)*. Wrinkling tends to occur after the age of 30, and deep wrinkling is more likely to occur in individuals with darker skin *(1)*.

Genetic variants in the *MMP1* and *STXBP5L* genes have been associated with increased susceptibility to severe wrinkling *(1,3)*.

PATIENT RESULTS & TREATMENT OPTIONS

INCREASED RISK

Based on the patient's genetics, the risk of developing skin wrinkles is considered INCREASED.

ORAL: Antioxidants, such as vitamin C + E, zinc, alpha-lipoic acid, green tea, resveratrol, proanthocyanidins (French maritime bark), soy isoflavones, carotenoids, such as lycopene, beta-carotene and vitamin A (also isotretinoin), have shown considerable efficacy as oral supplementation for skin health *(4-12)*. In addition, botanicals red ginseng, chlorophyll, aloe and algae astaxanthin all improve skin integrity *(13-16)*. Hyaluronic acid peptides + glucosamine + CoQ10 are newer oral products that have been shown to significantly improve skin photoaging damage *(17)*.

PROCEDURE: For moderate-extensive skin photoaging, many dermatology groups are now using fractional non-ablative/ablative resurfacing or Q-switched laser treatments for removing skin wrinkling or other photo-damage issues *(18-23)*. Radiofrequency and ultrasound therapies have also shown the ability to tighten the skin *(24,25)*. In addition, common alternatives used are wrinkle-relaxing injections (Botox®) and soft tissue filler treatments *(26,27)*.

TOPICAL: Sunscreens (SPF 30+, UV/IR protection) and antioxidant creams or serums (containing vitamin C + E , CoQ10, green tea + ginko, resveratrol, alpha-lipoic acid, coffee/cherry fruit and others) are baseline defenses for the patient's skin type *(28-35)*. Subsequent treatments are exfoliation, moisturization and fine-line reduction with retinoids (vitamin A, tretinoin, isotretinoin, retinol, retinyl palmitate and retinyl proprionate + niacinamide), hydroxy acids and DMAE/dimethylaminoethanol *(36-46)*. Hyaluronic acids are increasingly popular skin-firming and plumping agents *(47,48)*. There are topical formulations that provide skin health benefits by reducing cellular structural damage, such as products containing synthetic peptides (e.g. argireline + aloe), multiple peptides (+/- urea) and collagen peptides. In addition, there are newer topical products containing growth factors (e.g. TGF + FGF), DNA repair enzymes and stem cells *(49-59)*.

Refer to Product Glossary **A** on page 21 of this report

TANNING RESPONSE

Tanning is the production of melanin by the skin in response to ultraviolet radiation resulting in increased pigmentation. Tanning response varies among individuals, and can have both positive and negative effects on skin health. Those who have difficulty tanning are at higher risks of sunburn, sun spots, wrinkles, folate loss and melanoma *(60-63)*, while individuals who tan easily are at risk of vitamin D deficiency as they may derive less vitamin D from sun exposure *(64,65)*.

Multiple risk alleles in the genes, such as *MC1R, SLC45A2, SLC24A5,* and *TYR* are associated with decreased tanning response *(65-69)*. Genetic variants in the *MC1R* gene have the strongest effect, and individuals carrying these variants tend to exhibit fair, difficult to tan skin, red hair and often, freckles *(65,70)*.

PATIENT RESULTS & TREATMENT OPTIONS

REDUCED

Based on the patient's genetics, the tanning response is considered REDUCED, with the potential for increased inflammation and burning.

ORAL: Green tea with polyphenols EGCG, probiotics, vitamin A supplements and/or omega-3/omega-6 fatty acids are all known to help decrease skin inflammatory response *(8,9,71-73)*.

PROCEDURE: With more extensive or spot photoaging, some dermatology groups are now using broadband light, pulsed light, fractional non-ablative/ablative resurfacing or Q-switched laser treatments for removing sun damage *(18-23)*.

TOPICAL: Along with standard over-the-counter sunscreen products (SPF 30+) and EGCG creams, effective results can also be achieved with products containing bemotrizinol or bisoctrizole *(74)*. In addition, many newer products with allantoin have shown moisturizing effects on dry or burned skin.

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SUN SPOTS (LENTIGINES)

Sun spots, or solar lentigines, are pigmented spots that range from millimeters to centimeters in diameter and can appear light yellow to brown. They appear on areas frequently exposed to the sun, such as face, arms and back of the hands *(75,76)*. Sun spots are found more frequently in females and typically appear after the age of 50 *(75,76)*. They are caused by a local growth of pigment-producing skin cells in response to ultraviolet radiation *(76,77)*. Sun spots are a sign of skin damage and aging, and are associated sometimes with melanoma *(70,78)*.

Genetic variants in the *MC1R* and *IRF4* genes analyzed in this test have been associated with an increased risk of sun spots *(68,70)*. Multiple risk alleles for sun spots exist in the *MC1R* gene.

PATIENT RESULTS & TREATMENT OPTIONS

NORMAL RISK

Based on the patient's genetics, the risk of developing sun spots is considered NORMAL/AVERAGE.

ORAL: Astaxanthin derived from microalgae and proanthocyanidins (French maritime bark or grape seed extracts) have been shown to decrease the frequency and size of sun spots *(11,12,15,16)*.

PROCEDURE: Broadband and intense pulsed light have been demonstrated to diminish and often eliminate sun spots and other damage caused by photoaging. Q-switched and fractional laser treatments are similarly effective in reversing sun spots and rejuvenating sun damaged skin *(18-23)*.

TOPICAL: Creams with ingredients, such as hydroquinone, low dose tretinoin/vitamin A derivatives, alpha hydroxy acids, licorice root extract + glabridin, azaleic acid or 1% retinol are effective at sun spot removal *(41,79-84)*. Kojic acid and topical vitamin C are also mild and well tolerated for spot removal *(85,86)*.

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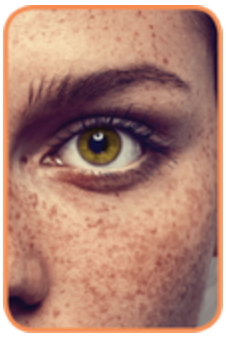
FRECKLES (EPHELIDES)



Freckles, also known as ephelides, are harmless hyperpigmented spots with distinct borders appearing most often on the face, neck, chest and arms. Freckles are a result of increased production of melanin pigment in the skin. They typically appear early in childhood but diminish with age, and they can also darken seasonally with sun exposure *(75,87)*. Freckles are common in Caucasian populations, and more frequent in fair-skin individuals with red or blond hair *(70,88)*. History of freckling is associated with fair skin, reduced tanning response, higher likelihood of sunburn and sun spots (solar lentigines) later in life *(70,77,87)*, as well as possibly malignant melanoma and non-melanoma skin cancers *(63,66,70)*.

Freckling is most strongly associated with genetic variants in the *IRF4* and *MC1R* genes *(68,70,76,89)*. *MC1R* gene is also the largest contributor to a red haired, fair skinned appearance. The degree of freckling often corresponds to the number of *MC1R* variants that the individual carries *(70)*.

PATIENT RESULTS & TREATMENT OPTIONS

NORMAL RISK

Based on the patient's genetics, the risk of developing freckles is considered NORMAL/AVERAGE.

ORAL: N/A

PROCEDURE: With mild skin freckling, or individual spots, many dermatology groups are now using broadband light, pulsed light, fractional non-ablative/ablative resurfacing or Q-switched laser treatments for removing pigmentary skin defects *(18-23)*.

TOPICAL: Sunscreen (SPF 30+) should be applied daily. Kojic acid and topical vitamin C are mild and well tolerated for freckle removal *(85,86)*. Licorice and niacinamide products brighten the skin and even out skin tone *(90)*. In addition, applied home remedies, such as lemon juice (+/- honey), sour cream paste, vitamin E mask and others, have shown some efficacy towards lightening freckles *(91)*. Over-the-counter skin whitening creams containing hydroquinone and oxybenzone are very effective, but are harsher and could cause damage to the skin *(79)*.

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CELLULITE

Cellulite, also known as orange peel skin, refers to the bumpy appearance of skin due to uneven fibrous tissue and fat build-up (subcutaneous fat) underneath the upper skin layers. Cellulite mainly appears on the thighs, hips and buttocks, and is present in about 85% of women over the age of 20 *(92)*. Caucasian women are more prone to cellulite than Asian women, partly due to the differences in diet. Genetic predisposition, hormonal changes, gender, ethnicity, age and weight changes contribute to risks of developing cellulite. Cellulite poses major cosmetic and psychological issues for women. Certain anti-cellulite creams, weight loss diets, direct massage and spa treatments are sometimes beneficial in treating cellulite *(93)*. Other treatments, such as mesotherapy (injecting a substance to breakdown fats) and laser treatments, are medical procedures that could be harmful to some individuals *(92-94)*.

Genetic variants in the *ACE* and *HIF1A* genes have been associated with the risks of developing cellulite *(94,95)*.

PATIENT RESULTS & TREATMENT OPTIONS

INCREASED RISK

Based on the patient's genetics, the risk of developing cellulite is considered INCREASED.

ORAL: Recent evidence has shown efficacy of products containing bioactive collagen peptides (BCP) and/ or chokeberry juice on the cellulite treatment of normal weight and overweight women *(96,97)*.

PROCEDURE: Skin smoothing procedures, such as FDA-approved subcision + microcannula systems (e.g. Cellfina™ by Ulthera), subcision + cannula/laser-assisted procedures (e.g. Cellulaze™ by Cynosure), radiofrequency and optical technologies, have shown good efficacy in reducing “orange peeling” and increasing skin smoothness in moderate cases of cellulite *(98,99)*.

TOPICAL: Most current topical products in use include 3.5% caffeine *(100-103)*. Newer anti-cellulite treatments include caffeine + tetrahydroxypropyl ethylenediamine (THPE) + retinol and/or red algae + retinol + glaucine *(92,93)*.

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STRETCH MARKS (STRIAE DISTENSAE)

Stretch marks, also known as striae distensae, appear initially as red or purple lines on the skin and later as white or silver lines *(104)*. Mechanical stretching of the skin due to weight loss-regain, obesity, hormonal changes and pregnancy can cause stretch marks *(104-108)*. Stretch marks frequently affect lower back and knees in adolescent males, while they are more common on thighs and calves in adolescent females. During pregnancy, abdomen and breasts are areas prone to developing stretch marks *(106-108)*. African-American women have a significantly higher risk of developing stretch marks than white women in the same geographic region *(106)*.

Genetic factors, such as variants in the *ELN, SRPX, HMCN1* and *TMEM18* genes, are also associated with risks of developing stretch marks *(104)*. Other factors associated with increased risks of stretch marks include Cushing syndrome, Marfan syndrome, diabetes mellitus and long-term systemic or topical steroid use *(108,109)*.

PATIENT RESULTS & TREATMENT OPTIONS

INCREASED RISK

Based on the patient's genetics, the risk of developing stretch marks is considered INCREASED.

ORAL: N/A

PROCEDURE: Most advanced treatment of stretch marks includes either non-ablative fractional resurfacing or pulsed-dye laser technologies *(110-113)*.

TOPICAL: There are very few clinical trials of topical agents showing positive results in preventing stretch marks. One such study, however, looked at the ingredients hydroxyprolisilane-C + rosehip oil + vitamin E + centella, and obtained significant results *(114)*.

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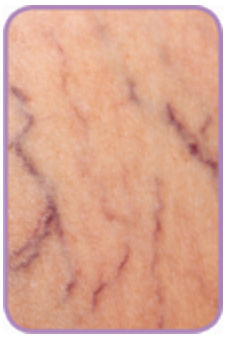
VARICOSE VEINS



Varicose veins are dark purple to blue veins under the skin on the back of the legs that often appear twisted and bulged like cords. Varicose veins affect more than a third of the world population *(115)* and approximately 23% of US adults *(116)*. Varicose veins can cause pain, aches and itching, or more severe conditions, such as venous ulceration and venous thrombosis *(116,117)*.

Varicose veins are often genetically inherited. Genetic variants in the *MTHFR* gene have been associated with an increased risk of developing varicose veins *(118-120)*. Other non-genetic factors include obesity, age, standing and walking upright for long time and hormonal changes *(115,119,121,122)*.

PATIENT RESULTS & TREATMENT OPTIONS

INCREASED RISK

Based on the patient's genetics, the risk of developing varicose veins is considered INCREASED.

ORAL: There is some evidence that oral CoQ10 decreases symptoms of varicose veins *(123)*.

PROCEDURE: Foam sclerotherapy and RF catheter insertion are newer technologies increasing in popularity for vein ablation *(124,125)*. Many laser systems, such as pulsed dye, endovenous, KTP and Nd:YAG, are also used for ablation *(126-131)*. Older technologies, such as ambulatory phelbectomy, are still used for minor cases of venous distension *(132,133)*.

TOPICAL: N/A

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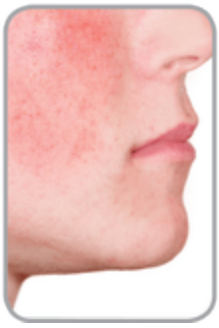
ROSACEA



Rosacea is a long-lasting skin disorder characterized by recurrent episodes of inflammation, redness and acne-like skin eruptions on the cheeks, nose, chin, forehead, or eyelids *(134)*. Rosacea usually appears in the second decade of life and affects 10% of the world population *(135,136)*. Women and fair-skin individuals of northern European and Celtic heritage are more prone to rosacea *(134,137-141)*. Although rosacea is relatively harmless, there are significant morbidities associated with it, including quality of life and psychological well-being *(142,143)*. Many factors contribute to rosacea and the root cause is still not well understood *(144)*. There is a wide range of causal hypotheses including vascular abnormalities *(145-147)* and genetic predispositions *(148-150)*.

Two genetic variants in the intergenic regions analyzed in this test are associated with an increased risk of rosacea occurrence *(148)*. Rosacea is worsened by triggers, including lifestyle changes (stress), environmental factors (heat, sunlight), food, alcohol, skin microorganisms and *Demodex* parasitic mites *(136,151)*.

PATIENT RESULTS & TREATMENT OPTIONS

INCREASED RISK

Based on the patient's genetics, the risk of rosacea is considered INCREASED.

ORAL: Oral supplementation with omega-3 fatty acids and gamma-linolenic acid and/or a reduction in the dietary glycemic load for 10 weeks can result in improvement in flaring and rosacea symptoms *(152)*. Oral antibiotics tetracycline or doxycycline are also commonly prescribed to patients with moderate rosacea. Isotretinoin is infrequently prescribed for severe and resistant rosacea *(134)*.

PROCEDURE: Pulsed dye laser and pulsed light treatments (broadband light) are used to treat the continual redness and noticeable blood vessels on the face, neck and chest. Photodynamic therapy (PDT) is one of the regularly performed treatments. PDT uses a topical photosensitizer liquid (aminolevulonic acid) that is applied to the skin and a light to activate the sensitizer *(153-157)*.

TOPICAL: Topical brimonidine tartrate gel and calcineurin inhibitors are newer topical therapeutic agents. Antibiotics, such as metronidazole, applied one to two times a day after cleansing may significantly improve rosacea *(158-161)*. Other topical treatments include erythromycin, clindamycin and azelaic acid. Mild steroids in desonide lotions or hydrocortisone creams (0.5% or 1%) can be applied sparingly to irritated areas. Green tea soaks to the face may also help decrease the redness and inflammation, as can compounds containing caffeine, zin, bisabolol, hyaluronic acid and EGF + aloe *(82,84,162-164)*. Sodium sulfacetamide is also known to help reduce inflammation. Tretinoin, tazarotene or adapalene are used for more advanced cases *(165-167)*.

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CONTACT DERMATITIS

Contact dermatitis (CD) is the most common work-related skin disease resulting in work disability and decreased quality of life *(168)*. There are two main types of CD: allergic (ACD) and irritant (ICD) *(169)*. A high proportion of individuals affected by CD are industrial workers in health, skin-care and beauty, food industry and metal-related occupations *(170-175)*. Differences in prevalence also depend on age and gender *(176,177)*. Although exposure to environmental insults is a major cause for CD, there is a strong link between CD and genetic variations in the *FLG* gene.

Certain *FLG* variants lead to a decrease or loss of the filaggrin protein, resulting in increased susceptibility to chronic ICD *(170,178)* and contact sensitization to metals such as nickel *(179-181)*. Filaggrin is important in maintaining the structure of the skin's outermost layer called the epidermis, and it is also involved in inflammatory response *(168,169,174)*.

PATIENT RESULTS & TREATMENT OPTIONS

NORMAL RISK

Based on the patient's genetics, the risk of contact dermatitis (CD) is considered NORMAL/AVERAGE.

ORAL: Oral antihistamines, such as fexofenadine and cetirizine, can help relieve any associated itching. Generally, prevention and protection from re-exposure to an allergen is the best treatment for CD.

PROCEDURE: N/A

TOPICAL: Vitamin C + E or green tea creams have shown efficacy in relieving symptoms for mild CD cases *(30,86,182)*. Low-potency hydrocortisone creams are also effective for minor localized use *(183)*.

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GENERALIZED PSORIASIS

Psoriasis is a chronic inflammatory genetic disorder primarily affecting the skin and joints *(184)* representing as raised red skin with white scales. Psoriasis is a worldwide disease observed in 2% of the population in Europe and North America *(184-188)*. Males are more prone to severe forms of psoriasis than females *(184,189)*. Psoriasis poses a significant healthcare burden due to physical and psychological challenges, including visible disfiguration, disability and depression *(190-196)*.

Genetic variants in the *HLA-C, IL12B, IL13, IL23R, MTHFR, TNIP1* and *TNFAIP3* genes analyzed in this test are strongly associated with psoriasis *(191,194,197,198)*. Genetic predisposition is the underlying cause of psoriasis *(191,197-202)*. In addition, extrinsic factors, such as scratching, medications (e.g beta-blockers and nonsteroidal anti-inflammatory drugs), infections (e.g. HIV and streptococcal pharyngitis), cold temperature and ultraviolent exposure, may worsen or trigger psoriasis outbreaks *(191)*.

PATIENT RESULTS & TREATMENT OPTIONS

HIGH RISK

Based on the patient's genetics, the risk of psoriasis is considered HIGH.

ORAL: Oral vitamin D, vitamin B12, selenium and omega-3 fatty acids in fish oils are increasingly being used to manage psoriasis. Evidence of the benefits of omega-3 fatty acids supplementation is strong and well established *(203,204)*.

PROCEDURE: UVB phototherapy for mild psoriasis cases is often the first choice therapy *(205,206)*. Newer excimer devices (laser) facilitate localized treatment *(206-208)*. Psoralen photochemotherapy (PUVA) combines the use of psoralen (P) and long-wave UV radiation (UVA), and is used for severe diseases *(206)*.

TOPICAL: Topical corticosteroids remain the mainstay of psoriasis treatment. Combinations of topical clobetasol and either calcipotriene or tazarotene, and UVB phototherapy + tar are routinely used for most cases *(205,206,208-211)*. Topical tacrolimus or pimicrolimus are used as corticosteroid sparing agents. For scalp psoriasis, the steroids betamethasone or fluocinolone are commonly prescribed *(212-214)*. Hydration, keratolytics, emollients and urea are valuable adjuncts to psoriasis treatment *(82,215,216)*. Severe psoriasis is treated by phototherapy and/or systemic therapies, such as retinoids, methotrexate or cyclosporine. In addition, biological treatments for psoriasis include anti-TNF agents (adalimumab, etanercept and infliximab) and anti-IL-12/IL-23 agents (ustekinumab) *(217-222)*.

Refer to Product Glossary **C** on page 21 of this report

ECZEMA (ATOPIC DERMATITIS)

The word eczema comes from a Greek word that means to effervesce or bubble or boil over. Eczema, or atopic dermatitis (AD), is a chronic inflammatory skin disease characterized by acute itching and a persistent red rash on creases of elbows or knees. Other symptoms include sleep deprivation and decreased psychosocial well-being. AD often starts in infancy, affecting 15-30% of children and 5-10% of adults *(223,224)*. In Europeans, 80-90% of AD cases are inherited *(225,226)*.

Genetic variants in the *FLG* gene are the strongest risk factors for AD and allergic sensitizations *(227)*.

Certain *FLG* variants lead to a decrease or loss of the filaggrin protein, resulting in dry and fissured skin on the hands and elbows *(226,228-231)*. Filaggrin is important in maintaining the structure of the skin's outermost layer called the epidermis.

PATIENT RESULTS & TREATMENT OPTIONS

NORMAL RISK

Based on the patient's genetics, the risk of atopic dermatitis is considered NORMAL/AVERAGE.

ORAL: Multiple clinical trials have identified vitamin A, vitamin D and zinc supplementation as effective treatment of atopic dermatitis. *(5,9,232,233)*. In addition, probiotics have shown efficacy in decreasing symptoms of AD, particularly in children *(234)*.

PROCEDURE: Newer excimer devices (laser), which are FDA approved, facilitate localized treatment *(206)*. Narrow-band UVB light treatment is also effective for larger lesions.

TOPICAL: Moisturizers/emollients increase hydration of affected skin and are first choice therapies *(235-237)*. Newer moisturizers that include ceramides and/or filaggrin breakdown products, green tea creams *(162,238)*, collodial oatmeal and chamomile salves have also shown efficacy *(82,239-241)*. Oil (rose, avocado, shea, myrrh, coconut) and glycerin products have varying levels of success for AD, more as skin "hydration and calming" agents *(242)*.

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SKIN MOISTURE FACTOR

If skin cells are not properly hydrated, dry skin (or xerosis) can develop reducing skin elasticity and creating wrinkles, cracks or flakiness. Usually dry skin is the result of environmental factors like exposure to sun, dry conditions, harsh soaps, hot water or chlorine, but can also be influenced by biological factors like genetic predisposition and nutrition. Dry skin can be uncomfortable and contribute to wrinkle development. The symptoms of dry skin can be alleviated by limiting environmental exposure, proper moisturization and optimizing collagen growth. Occasionally, an inherited group of more serious dry skin disorders can develop, known as ichthyosis, which can cause severe cracking, redness, flaking desfiguration and psychological stress.

DRY SKIN (XEROSIS AND ICHTHYOSIS)

Dry skin, also known as xerosis, is a condition of rough, itchy (occasionally painful) skin with fine scaling and small cracks *(231)* that occurs at all ages *(243)*. Xerosis may be caused by environmental factors, including dry/cold weather, frequent bathing/removing skin lipids, malnutrition and certain medical conditions *(244)*. More severe forms of dry skin may be inherited and appear during early childhood. The most common of these disorders is ichthyosis vulgaris (IV), or fish scale disease, where dead skin cell accumulation results in thick, dry scales on the skin's surface *(245)*.

Certain *FLG* variants lead to a decrease or loss of the filaggrin protein *(246,247)*. Filaggrin is important in maintaining the structure of the skin's outermost layer called the epidermis. The same *FLG* variants are found in individuals presenting skin inflammatory disorders, including atopic and contact dermatitis *(229,231,245,248,249)*.

▶ PATIENT RESULTS & TREATMENT OPTIONS ◀

NORMAL RISK

Based on the patient's genetics, the risk of dry skin is considered NORMAL/AVERAGE.

ORAL: Vitamin C and/or collagen hydrosylate supplementation has shown good efficacy in augmenting skin hydration and elasticity in patients with mild skin dryness *(5,250)*.

PROCEDURE: N/A

TOPICAL: For mild dryness, over-the-counter therapy includes standard moisturizing creams *(235-237,244)*. Glycerol and ceramide formulations are also widely used *(238)*. For more moderate dryness, lactic/glycolic acids (AHA) or AHA plus ceramides or urea are used *(251)*. Coconut/mineral oils have shown good results as moisture enhancers *(252)*; and many newer products with allantoin have shown moisturizing effects on dry or burned skin *(253)*.

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ANTIOXIDATION RESPONSE

Antioxidant response is our body's natural ability to detoxify and counteract harmful agents like ultraviolent (UV) rays, environmental pollutants, and toxins produced by the body. Oxidative stress occurs when the antioxidation response is weakened, and is a major factor in skin aging. Oxidative stress leads to breakdown of the collagen that provides structural support to the skin, alters cycles of cell regeneration, and causes DNA damage thats trigger skin inflammation. Foods containing antioxidants like tocopherols, polyphenols, aloe vera, red ginseng, coenzyme Q10, lycopene, carotenoids, vitamin E and vitamin C *(13)*, as well as their topical applications, are beneficial in preventing the UV-induced oxidative damage *(254,255)*.

Genetic variants coding for the antioxidant enzymes including *SOD2, GPX1, CAT* and *NQO1* have been associated with an increased risk of oxidative stress-induced skin aging or a decreased antioxidation response *(256-258)*.

PATIENT RESULTS & TREATMENT OPTIONS

NORMAL

Based on the patient's genetics, the antioxidation response is considered NORMAL/AVERAGE.

ORAL: Vitamin C + E, alpha-lipoic acid, CoQ10, resveratrol, green tea/EGCG, mushrooms and coffee berry polyphenols have all shown efficacy as antioxidants and free-radical scavengers for skin protection *(4,7,17,32,35,259)*. Micronutrients lycopene (tomatoes), omega-3 fatty acids (fish oils) and combinations like soy isoflavone + vitamin E are all active compounds that help enhance skin oxidation protection *(6,35,73,260)*.

PROCEDURE: N/A

TOPICAL: Vitamin C (ascorbic acid), vitamin E, alpha-lipoic acid and niacinamide are the mainstays of topical antioxidant therapy and are components of many creams and serums. Combinations of vitamin C + E (+/- ferulic acid) are also effective treatments. Topical resveratrol (+/- vitamin E and baicallin) and other flavonoids (green tea, caffeic acid) also have excellent antioxidant properties *(37,38,261-266)*. Newer topical products, such as astaxanthin, apple stem cells + urea + peptides and kinetin, are all active compounds with generally mild effects *(16,37,51,267,268)*.

Refer to Product Glossary **E** on page 22 of this report

GLYCATION PROTECTION

Glycation is a process by which sugar molecules are chemically linked to proteins (e.q. collagen and elastin), lipids and nucleic acids in skin cells *(269-272)*. These glycation products are referred to as advanced glycation end products (AGEs), and they are implicated in accelerated skin aging and inflammation *(270)* leading to loose, cracked and thinned skin *(269)*. AGEs accumulation increases with age and become more harmful in combination with UV exposure *(271,273)*. Glycation stress, and subsequently skin aging, may be reduced by managing levels of blood glucose, low-density lipoprotein cholesterol, and triglycerides through an appropriate diet *(270)*.

Genetic variants in the *AGER* and *GLO1* genes have been associated with increased AGEs level in both healthy individuals and those with diabetes *(274-278)*.

PATIENT RESULTS & TREATMENT OPTIONS

REDUCED

Based on the patient's genetics, the glycation protection is considered REDUCED.

ORAL: Niacinamide, carnosine and green tea have all been implicated in reducing advanced glycation end products (AGE) in skin *(8,37,264,265,279,280)*.

PROCEDURE: N/A

TOPICAL: Topical carnosine and/or niacinamide creams have been shown to decrease the dullness and sallowness of skin from glycation end products (AGEs), and increase collagen production *(37,38,264,265,280)*. Combining products with milk thistle silibinin and alpha-lipoic acid have recently demonstrated anti-glycation activity. In addition, truffles and mushrooms have increased antioxidant capability and are now used in high-end topical anti-glycation serums and creams *(281-285)*.

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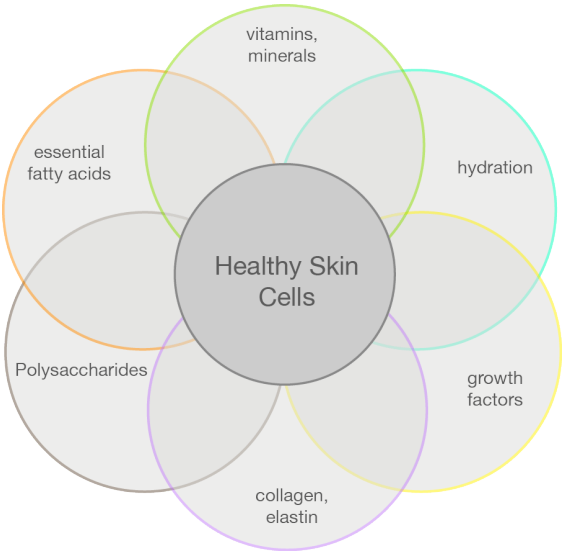
SKIN NUTRITIONAL NEEDS

Vitamins and minerals are known to play an integral role in the skin's health and complexion. Similarly, new research has shown the importance of antioxidants in general health and specifically their critical role in the way the skin looks, feels and how fast it ages *(286,287)*. A balance diet in combination with appropriate antioxidants and vitamin supplementation can help maintain health and slow down the appearance of signs of aging skin *(288,289)*. Moreover, preventing chronic sleep deprivation, improving sleep quality, and avoiding risky practices (smoking, alcohol abuse and indoor tanning), can help guard against skin damage *(290-296)*.



VITAMIN A DEFICIENCY

Vitamin A and its related compounds (beta-carotene, retinol, and retinoic acid) are important for skin growth and damage prevention *(297-300)*. Deficiency in vitamin A is associated with various skin conditions, including dry skin, abnormal thickening of the skin, atopic dermatitis and delayed wound healing *(300,301)*. Both dietary intake and genetic factors influence vitamin A metabolism. The recommended daily vitamin A intake for most adults is 700 to 900 micrograms *(302)*. In addition, topical application of vitamin A derivatives, such as retinol and retinoic acid, have been shown to reduce signs of photoaging including wrinkles, hyperpigmentation and skin roughness *(303)*.

Vitamin A is converted from beta-carotene in the body. Two genetic variants in the *BCMO1* gene have been associated with decreased ability to convert beta-carotene to vitamin A in women *(304)*.

▸ PATIENT RESULTS & TREATMENT OPTIONS ◂

INCREASED RISK

Based on the patient's genetics, the risk of vitamin A deficiency is considered INCREASED.

ORAL: Increase consumption of foods containing high concentrations of vitamin A, including sweet potatoes, spinach, carrots, pumpkin and cantaloupes. Alternatively use one of many types of vitamin A supplements available *(305)*.

PROCEDURE: N/A

TOPICAL: There are many serums and creams that exclusively include vitamin A as the primary ingredient. Higher potency vitamin A serums, and many with retinol, are available for over-the-counter use *(303,306,307)*.

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VITAMIN B2 DEFICIENCY

Vitamin B2 (riboflavin) is critical in carbohydrate, fat and protein metabolism *(308)*. Vitamin B2 deficiency can lead to skin conditions, such as angular cheilitis (inflammation of the corners of the mouth) and seborrheic dermatitis *(309)*. Riboflavin acts to improve the secretion of mucus in the skin, which may help to clear up rosacea. It is also used to prevent and treat skin problems, such as dermatitis and eczema. Low vitamin B2 levels have been associated with elevated homocysteine, which subsequently may affect skin aging by degrading skin collagen, fibrillin and elastin, as well as by inducing injury to skin blood vessels *(310)*. Both dietary intake and genetic factors can influence vitamin B2 levels in the body. The recommended daily vitamin B2 intake for most adults is 1.1-1.3 milligrams *(311)*. Vegetarians, vegans, pregnant and lactating women are at a higher risk of vitamin B2 deficiency *(311)*.

A genetic variant in the *MTHFR* gene has been associated with individuals who tend to have elevated homocysteine levels and are more sensitive to changes in vitamin B2 levels *(312,313)*.

▶ PATIENT RESULTS & TREATMENT OPTIONS ◀

NORMAL RISK

Based on the patient's genetics, the risk of vitamin B2 deficiency is considered NORMAL/AVERAGE.

ORAL: Maintain consumption of foods containing high levels of vitamin B2, including dairy products, broccoli, eggs, and tuna *(314,315)*.

PROCEDURE: N/A

TOPICAL: As vitamin B2 is water-soluble, it is usually combined with other micronutrients in topical products, such as moisturizing creams.

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VITAMIN B6 DEFICIENCY

Vitamin B6 (pyridoxine) is involved in a variety of functions, such as protein and sugar metabolism, neurological development, immune function, and hemoglobin formation *(316-318)*. Deficiency in vitamin B6 is associated with various skin disorders, such as pellagra-like dermatitis, stomatitis and seborrheic dermatosis *(317,319,320)*. Vitamin B6 deficiency can also lead to vitamin B3 deficiency causing a thick, scaly pigmented rash on skin exposed to sunlight *(309)*. Both dietary intake and genetic factors can influence vitamin B6 levels in the body. The recommended daily vitamin B6 intake in most adults is 1.3 milligrams *(318)*. Individuals with alcohol dependency, pregnant women and pregnant women with preeclampsia or eclampsia may be at risk for vitamin B6 deficiency *(320)*. However, excessive vitamin B6 (>100 milligrams/day) can lead to skin lesions, dermatitis, and photosensitivity *(320,321)*.

A genetic variant near the *NBPF3* gene has been associated with reduced levels of vitamin B6 *(322,323)*.

▶ PATIENT RESULTS & TREATMENT OPTIONS ◀

INCREASED RISK

Based on the patient's genetics, the risk of vitamin B6 deficiency is considered INCREASED.

ORAL: Increase consumption of foods containing high levels of vitamin B6, including chickpeas, poultry, tuna, bananas, avocado and Brussels sprouts. There are also many types of vitamin B6/B complex supplements available *(324)*.

PROCEDURE: N/A

TOPICAL: Most vitamin B6 creams and serums are B-complex products, but single compound vitamin B6 sprays have been used to some effect for seborrheic dermatitis *(325,326)*. Vitamin B6 by itself has poor light stability, and therefore, are prone to cause skin irritation.

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VITAMIN B12 DEFICIENCY

Vitamin B12 (cobalamin) plays an important role in the optimal neurological function. It is essential for proper red blood cell formation and cellular DNA synthesis *(327,328)*. Both vitamin B12 and folate aid in lowering homocysteine blood level. Elevated homocysteine blood level is associated with cardiovascular diseases, psychiatric disorders, as well as skin disorders *(329-331)*. Specifically, deficiency in vitamin B12 is associated with various skin conditions, such as oral atrophy and skin hyperpigmentation *(329,332)*. Both dietary intake and genetic factors can influence vitamin B12 levels in the body. The daily recommended vitamin B12 intake for adults is 2.4 micrograms. Older adults and individuals who have limited consumption of animal products (vegans and vegetarians) are at a higher risk of B12 deficiency *(327,331)*.

A genetic variant in the *FUT2* gene has been associated with lower vitamin B12 levels in the blood *(322,333-335)*.

▶ PATIENT RESULTS & TREATMENT OPTIONS ◀

INCREASED RISK

Based on the patient's genetics, the risk of vitamin B12 deficiency is considered INCREASED.

ORAL: Increase consumption of foods containing high levels of vitamin B12, including clams, beef, liver, mackeral, dairy products and eggs *(324)*.

PROCEDURE: N/A

TOPICAL: Vitamin B12 creams and serums have been shown to be effective against eczema and atopic dermatitis *(336-338)*.

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VITAMIN C DEFICIENCY

Vitamin C (L-ascorbic acid) must be acquired from dietary sources, as humans are unable to synthesize the molecule. Deficiency in vitamin C leads to scurvy *(300)*. Skin conditions associated with scurvy include abnormal thickening of the outer layer of skin, inflammation, delayed wound healing and dry, rough skin *(339)*. Both dietary intake and genetic factors can influence vitamin C levels in the body. The recommended daily vitamin C intake for most adults is 75-90 micrograms *(340)*. Vitamin C topical application has also been widely used to improve signs of photoaging, including wrinkles, skin roughness and laxity. Vitamin C also promotes skin hydration and collagen production *(300,301)*.

Multiple studies reported a genetic variant in the *SLC23A1* gene, which is associated with decreased levels of circulating vitamin C *(341-343)*.

▶ PATIENT RESULTS & TREATMENT OPTIONS ◀

INCREASED RISK

Based on the patient's genetics, the risk of vitamin C deficiency is considered INCREASED.

ORAL: Increase consumption of foods containing high levels of vitamin C, including oranges, kiwi, cantaloupe, grapefruit, strawberries and tomatoes *(13,344)*.

PROCEDURE: N/A

TOPICAL: Topically applied vitamin C is highly effective as a rejuvenation therapy inducing significant collagen synthesis and photoprotection in all age groups. It also works synergistically with vitamin E and hyaluronic acid *(13,30,345)*. There are many creams and serums available with high concentrations of vitamin C, and/or vitamin E and hyaluronic acid. Patients at risk of vitamin C deficiency should try combination products with vitamin E and hyaluronic acid, which are more effective *(13,30,345)*.

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VITAMIN D DEFICIENCY

Vitamin D is produced in the skin following exposure to ultraviolent (UV) B light or from food consumption *(346,347)*. Excessive exposure to UV radiation accelerates skin aging, while deficiency in vitamin D is associated with various skin conditions, such as psoriasis, atopic dermatitis, vitiligo and ichthyosis *(348-351)*. Having sufficient vitamin D in the skin helps minimize acne, boost elasticity and skin immunity, stimulate collagen production, enhance radiance, and lessen lines and appearance of dark spots *(352)*. Both dietary intake and genetic factors can influence vitamin D levels in the body. The recommended vitamin D intake for most adults is 15 micrograms/day (600 IUs/day); however, the American Academy of Dermatology recommends 25 micrograms/day (1000 IUs/day) for individuals who have an increased risk of vitamin D deficiency *(346,353)*. Individuals with dark skin, limited sun exposure, older adults and those who choose to photoprotect using sunblocks may be at risk.

A genetic variant in the *GC* gene has been associated with decreased blood levels of vitamin D *(354-357)*.

▶ PATIENT RESULTS & TREATMENT OPTIONS ◀

NORMAL RISK

Based on the patient's genetics, the risk of vitamin D deficiency is considered NORMAL/AVERAGE.

ORAL: Maintain consumption of foods containing high levels of vitamin D, including fatty fish, vitamin D-fortified orange juice and milk, eggs and mushrooms. There are also many types of vitamin D supplements available *(358,359)*.

PROCEDURE: N/A

TOPICAL: Although the benefits of many topical products containing vitamin D in the market remains controversial, the use of topical vitamin D derivatives should be considered in the treatment of psoriasis *(360)*.

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VITAMIN E DEFICIENCY

Vitamin E refers to a group of eight antioxidant molecules, of which alpha-tocopherol is the most abundant in the body. Vitamin E functions to promote a strong immune system and protects the skin from ultraviolent radiation and inflammation *(300,361,362)*. Deficiency in vitamin E may lead to skin ulcers and increase collagen breakdown *(300)*. Both dietary intake and genetic factors can influence vitamin E levels in the body. The recommended daily vitamin E intake for most adults is 15 milligrams *(363)*. Several studies have demonstrated that when vitamin E and vitamin C are taken together as oral supplements, they reduce UV-induced skin inflammation and decrease the skin's susceptibility to sunburn *(13,289,364-366)*.

A genetic variant near the *APOA5* gene has been associated with increased plasma levels of alpha-tocopherol or reduced risk of vitamin E deficiency *(367)*.

▶ PATIENT RESULTS & TREATMENT OPTIONS ◀

NORMAL RISK

Based on the patient's genetics, the risk of vitamin E deficiency is considered NORMAL/AVERAGE.

ORAL: Maintain consumption of foods containing high levels of vitamin E, including sunflower seeds, almonds, sweet potato, asparagus and wheat germ *(363)*. There are also many vitamin E supplement products available.

PROCEDURE: N/A

TOPICAL: There are many creams and serums available with high concentrations of vitamin E. However, combination products containing vitamin E, vitamin C, vitamin A and vitamin B3 are more effective *(368)*.

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FOLATE-FOLIC ACID DEFICIENCY

Folate (folic acid) works synergistically with vitamins B6 and B12 in nucleic acid synthesis and amino acid metabolism *(369)*. Deficiency in folate can increase the risk of skin conditions, including psoriasis, deep venous thrombosis, oral atrophy and skin aging. Individuals with these conditions may benefit from increase uptake of folate rich foods or folic acid supplementation *(310,329,330,370,371)*. Increased level of homocysteine, a signature of folate deficiency, is associated with skin aging caused by collagen, fibrillin and elastin degradation in the skin *(310)*. Folic acid helps improve the firmness of the human skin and also helps reduce the signs of skin aging *(372)*. Both dietary and genetic factors can influence folate levels in the body. Folate is obtained from food or synthetically as folic acid supplement. The recommended daily folate intake for most adults is 400 micrograms (600 micrograms for pregnant women) *(373)*.

Genetic variants in one or more risk alleles in the *MTHFR* gene are associated with individuals presenting low plasma folate levels. In addition, these variants have also been associated with varicose vein disorder *(119,374)*.

▶ PATIENT RESULTS & TREATMENT OPTIONS ◀

INCREASED RISK

Based on the patient's genetics, the risk of folate deficiency is considered INCREASED.

ORAL: Increase uptake of foods that contain high amounts of folate, including black-eyed peas, asparagus, spinach, broccoli, lima beans and eggs *(373)* and/or supplements containing folic acid.

PROCEDURE: N/A

TOPICAL: Topical folate (+ creatine) helps with skin firmness by exerting sustained effects on collagen metabolism *(372,375)*. It is found in many combination cream/serum products.

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OMEGA-3 AND OMEGA-6 DEFICIENCY

Omega-3 and omega-6 fatty acids are polyunsaturated fatty acids (PUFAs) important for heart and brain health, anti-inflammatory response and aging *(376-380)*. Both omega-3 derivative alpha-linolenic acid (ALA) and omega-6 derivative linoleic acid (LA) are essential fatty acids that must be acquired from dietary sources *(381,382)*. In the body, ALA is further converted to eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), while LA is converted to arachidonic acid (AA). Deficiencies in these fatty acids can lead to various skin problems, including dermatitis, acne, scaling, dry skin, and psoriasis *(383)*. These skin conditions can be alleviated by omega fatty acids supplementation 384,379,380,385,386,6). Omega-3 fatty acids protect against UV-induced skin damage and reduce inflammation *(13,387)*. Both dietary intake and genetic factors can influence fatty acid levels in the body. Most western diets contain sufficient omega-6 but insufficient omega-3, thus additional omega-3 intake may be beneficial *(381)*.

A genetic variant in the *FADS1* gene has been associated with decreased blood levels of EPA (omega-3) and AA (omega-6) *(388,389)*.

▶ PATIENT RESULTS & TREATMENT OPTIONS ◀

INCREASED RISK

Based on the patient's genetics, the risk of omega-3 and omega-6 deficiency is considered INCREASED.

ORAL: Increase uptake of foods that contain high amounts of omega-3, including flaxseed, walnuts, fatty fish, marine fish oils, salmon, DHA fortified eggs and milk. Sources of omega-6 are found in olives, nuts, poultry, evening primrose and borage oils *(390)*. There are also many supplements containing omega-3/ omega-6, but products that are derived from fish oils are recommended.

PROCEDURE: N/A

TOPICAL: Omega fatty acids are found in many combination creams and serums. In addition to sunscreen use, omega-3 PUFAs are promising candidates to protect the skin from UVR damage *(386,391)*.

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*Ingredients are listed followed by product names and content will look like this: "**Ingredient**: Product Name".*

A - Skin Photoaging

Oral:

Alpha-Lipoic acid: DermaVite®
Astaxanthin: AlgaLife®
Hyaluronic acid + Glucosamine + CoE Q10: Viscoderm Pearls®
Isotretinoin: Accutane®
Probiotics: Lactinex®
Proanthocyanidins: Grape seed extract/TerraVita®
Resveratrol: Nutricost™

Topical:

Alpha Hydroxy Acids: AHA Targeted/Spot Light®
Alpha-Lipoic acid: QAL-100/Jabu'she®
Azelaic acid: Azelex/Finacea®
Bemotrizinol: Tinosorb®S
Bisoctrizole: Tinosorb®M
Coffee/Cherry Fruit: Coffeeberry®, Supremya/Sisley®
Collagen Peptides: Verisol®, Pro-Collagen Marine Cream/Elemis®
CoQ10: Night Cream/Acure Organics®
DMAE: Firming Fluid/Reviva Labs®
DNA Repair Enzymes: DNA EGF Renewal®
EGCG Creams: Camilla Care™, Green Tea Skin/Life flo®
Glycolic acid: AHA/AHA Soufflé®, AHA/Spot Light®
Green Tea + Ginko: Ascoderm® Serum/biopelle®
Growth Factors: hGF Regeneration Booster/Jan Marini®, TGF + FGF Anti-Aging Serum/Reluma®, Skin Serum/AQ Skin Solutions®
Hyaluronic acids: Hydra-5 B-Complex/Cellex-C®, 14 doses Filler Treatment/Fillerina®, Voluma® XC/Juvéderm®, Restylane®
Hydroquinone: Meladerm Skin Lightening/Civant®, White Lucent/Shiseido®
Hydroxy acids/Glycolic acids: AHA/AHA Soufflé®
Kojic Acid: Kojic Acid Skin Lightening Cream/Supplement Spot®
Licorice + Niacinamide: Lytera® Skin Brightening Complex/SkinMedica®
Licorice root + Glabridin: Glabridin A71/ETAT PUR®
Niacinamide: Fair & Lovely®
Peptide Argireline + Aloe: Face Whisperer®/Sublime Beauty®, Instantly Ageless™/Jeunesse®
Peptides (+/-) Urea: Anti-Aging Rapid Response/La Prairie®, ybf:Control/Your Best Face™, Ureadin Fusion®/ISDIN®
Resveratrol: Pure Super Grape/M&S®
Retinol: Glow/Dr. Brandt®, ZO Skin Health®, Age Smart/Dermalogica®
Retinol 1%: Retinol Complex 1.0/SkinMedica®, Retinol 1.0/SkinCeutials®

A - Skin Photoaging

Retinyl Palmitate: Boots No. 7/Boots®
Retinyl Propionate + Niacinamide: Pro-X/Olay®
Soy: Fresh® Soy Face/Sephora®
Stem Cells: Intense Stem Cell Skincare/Dr. LEVY®, Radiance Lift/EVE LOM®, Eye Wonder™/Oskia®
Sunscreens: Blue Lizard®, CeraVe®, Badger Balm®, Neutrogena®
Tretinoin: Tretinol/glō therapeutics®
Tretinoin/Vitamin A: Tretinol/glō therapeutics®
Triple Therapy: Tri-Luma®
Vitamin A/Tretinoin/Isotretinoin: Accutane®, Avage®, Renova®/Obagi®
Vitamin C: Radiance Day Nite creams/Burt's Bees®
Vitamin C/E (+/-) Ferulic acid creams and serums: Complexion Brightening Cream/Suki®, CE Ferulic/SkinCeuticals®
Vitamin E: Argania™

B - Skin Texture And Elasticity

Oral:

ChokeBerry Juice: Superberries™

Topical:

Caffeine: Firm and Tone Serum/Murad®
Caffeine + Tetrahydroxypropyl Ethylenediamine (THPE) + Retinol:
Anti-Cellulite Intensive Cream/ROC J&J®
Red Algae + Retinol + Glaucine: Perfect Body Contour Creator/Oriflame®, Body Contour/Boots®, Cellulaze®
Centella: Hydra Zen Neurocalm™/Lancôme®
Hydroxyprolisilane-C: Lineless Cream/Dr. Brandt®
Rosehip Oil + Vitamin E: Aura Cacia®

C - Skin Inflammation And Allergy Risk

Oral:

Antihistamines: Benadryl®
Doxycycline: Oracea®
Probiotics: Lactinex®
Tetracycline: Sumycin®

Topical:

Adapalene: Differin®
Aloe: Aloe Vera®
Azelaic acid: Finacea®
Betamethasone valerate: Luxiq®
Brimonidine tartrate: Mirvaso®
Caffeine + Zinc + Bisabolol: SkinCeuticals®

C - Skin Inflammation And Allergy Risk

Calcipotriene: Calcitrene®, Dovonex®
Clindamycin: Cleocin®
Colloidal oatmeal: Avena®
Corticosteroids: Clobetasol/Temovate®, Clobex®
Desoximetasone: Topicort®
EGF + Aloe: Dermaced Redux®
Erythromycin: Erygel®
Fluocinolone: Capex®, Synalar®
Glycerin: Enkido®
Green Tea Cream: Green Tea Skin/Life-Flo®
Hyaluronic acid: Bionect®
Low potency corticosteroids: Hytone®, Texacort®, Cortaid®, Cortizone 10®
Medium/High potency corticosteroids: Trianex®, Temovate®
Metronidazole: Metrogel®
Moisturizers/emollients: Noxzema®, Ponds®, CeraVe PM®, Cetaphil®, Neutrogena®, Lubriderm®
Petroleum jelly: Vaseline®
Retinoids: Avage®, Fabior®
Sodium sulfacetamide: Klaron®
Tacrolimus: Protopic®
Pimecrolimus: Elidel®
Tazarotene: Tazorac®
Thick creams: Ponds®, Noxzema®, Lubriderm®
Tretinoin: Retin-A®
Triamcinolone: Trianex®
Urea: Gordons Urea®
Vitamin C/E creams: triple oxygen/bliss®
Adalimumab: Humira®
Etanercept: Enbrel®
Infliximab: Remicade®
Ustekinumab: Stelara®

D - Skin Moisture Factor

Oral:

Collagen Peptides: PeptanF®/Rousselot®

Topical:

AHA (+/-) Ceramides or Urea: AquaPorin Hydrating Cream/ Circadia®, Amlactin®, Ultramide 25/Baker Cummins®, Burt's Bees®
Allantoin: Flawless Skin/Laura Mercier®
Ceramide: Skin Inc/Sephora®, CeraVe®
Eucerin: Eucerin Plus Cream/Beiersdorf®
Glycerol formulations: AQUAporin Cream/Eucerin®, TonyMoly®
Moisturizing creams: Noxzema®, Ponds®, CeraVe PM®, Cetaphil®, Neutrogena®, Lubriderm®
Petroleum jelly: Vaseline 100®
Urea: Carmol®
Vitamin A: Retin-A®, Tazorac®

E - Skin Oxidation Protection

Oral:

Alpha-Lipoic acid: DermaVite®

E - Skin Oxidation Protection

Niacinamide: NOW Foods®

Topical:

Ascorbic acid/Vitamin C: Isabis™ Formulae, SkinCeuticals®
Apple stem cells + Urea + Peptides: Ureadin Fusion®/ISDIN®
Astaxanthin: Madre Labs®
Kinetin: Gentle Rejuv/Obagi®
Resveratrol + Vitamin E + Baicalin: Resveratrol B E/ SkinCeuticals®
Vitamin C + E: C E Ferulic/SkinCeuticals®

F - Skin Glycation

Oral:

Niacinamide: NOW Foods®

Topical:

Carnosine: Lineless/Dr. Brandt®, Bio Lifting/Chantecaille®
Niacinamide: InstaNatural®, Olay®, Metacell Renewal B3/ SkinCeuticals®
Silibinin + Alpha-Lipoic acid: Revitalizing Night Crème/Aubrey Organics®
Truffles/Mushrooms: ReNutriv/Estee Lauder®, Truffle Therapy™ Serum/Skin&Co Roma®

G - Skin Nutritional Needs

Topical:

Vitamin A:
Vitamin A Plus Serum/ MyChelle®, Vitamin A Serum Regenerate/ Skin Inc.®, Dermalogica®
High Potency Vitamin A: Perricone MD®, SkinCeuticals®, Alpha-H®
Retinol: Obagi®, Exuviance®
Vitamin B2:
Yu-Be®, Olay®
Vitamin B6:
Combination products: Burt's Bees®, Vitamin C Serum Rebalance/Skin Inc.®, Total Effects 7/Olay®
B6 Spray: Urban Decay®
Vitamin B12:
Maxasorb™ B12/Vita™ Sciences, Vitacream B12®, C-Serum® Seaweed Filtrate/Repechage®
Vitamin C:
Isabis™ Formulae®, ViolàVe®, 90210 Naturals®, goPure®, 100% Pure®
Vitamin D:
PureLx®
Vitamin E:
Vitamin E + Vitamin C + Hyaluronic acid: Body Shop®, Skin Inc.®, goPure®, 100%Pure®
Folate-Folic Acid:
Folate + Creatine: Thin to Thick®/JĀSÖN®, Neutrogena Naturals®
Omega-3, Omega-6:
Omega 3+6: Global Beauty®, Olay®

The scientific studies referenced in this report are provided below and can be found at www.pubmed.gov. All cited articles are published in peer-reviewed journals, U.S. government or medical associations websites. PubMed is a service managed by the National Institutes of Health (NIH), a part of the U.S. Department of Health and Human Services, and it tracks more than 19 million citations for biomedical articles and scientific research.

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