Chapter 5
Tools for Assessing Skin, Hair & Nails

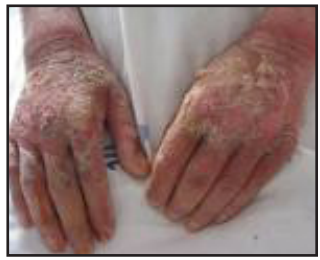
During a general survey of overall appearance, observe the skin on the face, arms and hands, nails and hair. The characteristics the RD is looking for are signs that might suggest a nutrient deficiency or toxicity. Patients often notice changes in their skin, hair or nails before the clinician does. Some questions to consider include:

Skin:

- Have you noticed any changes in your skin?
- Does the skin have an increased (brownness) or decreased pigmentation?
- Does the skin have a red cast or tone?
- Does the skin have a blue cast or tone?
- Does the skin have a yellow cast or tone?

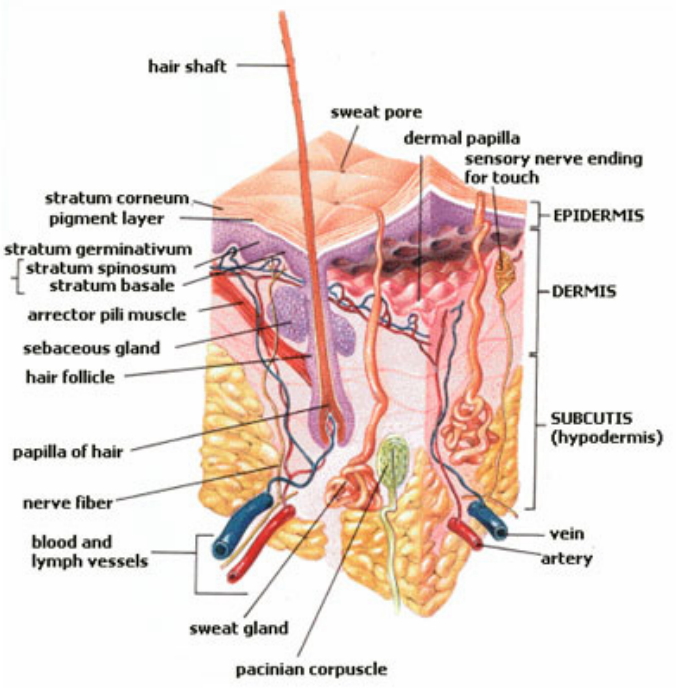
Figure 5.1. Follicular Hyperkeratosis (Vitamin A deficiency)

Figure 5.2. Pellagra (Niacin deficiency)



- Evaluate the moisture of the skin.
- Does the skin look dry and scaly, or sweaty or oily?
- Is the skin warm or cold to the touch?
- Is the skin rough or smooth?
- Are there lesions or rash on the skin?
- Does the patient have acute surgical wounds?
- Does the patient have a history or skin breakdown?
- Does the patient have nutrition risk factors for skin breakdown such as low BMI, unplanned weight loss, low nutrient density intake?
- Is the patient at increased risk for prolonged pressure on bony prominences or other areas of the skin?
- Does the patient have skin breakdown?

Figure 5.3. Anatomy of Skin



The skin is the largest organ in the body that contributes about 10 percent of the total body weight. The skin is a bilayered organ that serves as the first line of defense against a variety of assaults. The epidermis is the thin outer layer and the dermis is the thicker inner layer.

Skin changes over time with age, exposure to sun, hydration, medications and nutrition. It is often ignored until it is injured. Skin and nails are thinner at birth, increase in density through adolescence. Once adulthood is reached, the dermis decreases in thickness by about 20 percent, but the epidermis is about the same thickness. However, the epidermal turnover time increases making wound healing a longer process. In young adults the epidermal turnover is about 21 days, however, by age 35 the turnover time is more than 40 days.

The skin of an older adult is more easily injured due to changes in the epidermal-dermal junctions. Skin elasticity also decreases with age. Collagen fibers appear to unwind and elastin fibers breakdown. The degradation of elastin begins to occur around age 30, but is markedly observed by age 70 (Braverman, 1986).

The barrier function of the skin decreases with age making skin irritations more common. Protection against ultraviolet radiation is diminished with age due to a reduced number of melanocytes which in turn contributes to wrinkling and sagging skin. The sagging is due in part to the loss of underlying tissue and the loss of elastin and collagen. Wrinkles are due to an increase in dryness of the skin.

The sensory receptors in the skin are diminished in capacity with aging. The risk of being burned or injured is more

common due to a decreased sensation. Vitamin D production in the skin decreases with age and supplementation of vitamin D may be necessary for individuals with limited exposure to sunlight.

Nutrient deficiencies and toxicities can result in changes in skin color and texture and changes in the mucous membranes. While nutrient deficiencies are rare, these are reported in adults with malabsorptive disorders and following weight loss surgery. Toxicities are reported in individuals taking megadoses of vitamin mineral supplements.

Numerous medications affect the skin as noted on Table 5.1 (Potts, 1990). Corticosteroids are known to interfere with epidermal regeneration and collagen synthesis (Ehrlich, 1968).

Table 5.1. Categories of Medications that can Affect Skin

• Analgesics	• Diuretics
• Antibacterials	• Hypoglycemic agents
• Antihypertensives	• Oral contraceptives
• Antihistamines	• Sunscreens
• Antineoplastic agents	• Tricyclic antidepressants
• Antipsychotic agents	

Skin & Nutrient Deficiencies

The skin color, texture, dryness, temperature, abnormal mucous membranes and presence of lesions or injuries are all characteristics to note in NFPA. Abnormalities should be corroborated with data from lab test results, anthropometric data, dietary history, vital signs and other clinical assessment data noted by the other medical team members

Review the medical history for contributors to impaired nutrient utilization.

Practical Applications for NFPA: Color

The color of normal skin depends primarily on four pigments as noted in Table 5.2.

Table 5.2. Skin Pigments

Pigments	Color
Melanin	Brown
Carotene	Golden yellow
Oxyhemoglobin	Bright red
Deoxyhemoglobin	Blue

Changes in skin color may also be due to a number of medical conditions. Inspect the skin for abnormal color. Compare the findings to other evidence in the medical records that suggest a change in medical condition or functional status. The nutrition professional is looking for changes that suggest impaired ability to prepare meals, impaired functional status to eat, nutrient deficiency or nutrient overload.

Increased Brownness of Skin

The amount of melanin in the skin is genetically determined. It can also be increased with exposure to sunlight. Individuals with areas of darker, thick, velvety skin in body folds and creases may have acanthosis nigricans. It is more often seen in patients of African and Hispanic/Latino descent. Acanthosis nigricans usually appears slowly and doesn't cause any symptoms other than skin changes. It results in velvety light-brown to black markings in body creases such as in the armpits, neck folds, and over the joints of the fingers and toes. Refer to Figures 5.4.-5.6.

Acanthosis nigricans is seen in patients with obesity, insulin resistance, metabolic syndrome, hypertension, dyslipidemia, Addison's disease, disorders of pituitary gland, hypothyroidism and growth hormone therapy. It has been shown to be a reliable early marker for metabolic syndrome in children (Otto, 2010).

Weight management and improved glucose control can help prevent acanthosis nigricans as well as improve medical management of endocrine disorders (Kong, 2010). Patients with lymphoma, cancers of the gastrointestinal or genitourinary tracts can also develop severe cases of acanthosis nigricans.

Occasionally acanthosis nigricans is medication-induced. Medications associated with acanthosis nigricans, include nicotinic acid, insulin, pituitary extract, systemic corticosteroids, diethylstilbestrol, triazinate, oral contraceptives, fusidic acid, and methyltestosterone (Miller, 2011, Otto, 2010).

Increased Yellow Cast to Skin

Jaundice or icterus occurs when excess amounts of serum bilirubin dissolve in the subcutaneous fat, causing a yellowish appearance of the skin and the whites of the eyes. The yellowing may extend to other tissues and body fluids as well as turn the urine a darker color. Jaundice is not a disease, but a sign of a variety of medical conditions. It can occur when the normal process of destruction of red blood cells and elimination of bilirubin is interrupted. This occurs when there is excessive hemolysis, liver disease that reduces the ability of the liver to remove and modify bilirubin, or obstruction to the flow of bile into the intestine (Bickley, 2009). Refer to Figure 5.7.

A lemon-yellow–tinged pallor to the skin is reported in pernicious anemia. Other physical signs and symptoms include raw-beefy red tongue lacking filiform papillae, paresthesia (sensation of pins and needles) in extremities, loss of sense of touch, changes in gait, stiffness in arms and legs, dementia, hallucinations and paranoia (Bickley, 2009). Refer to Figure 5.15 and Chapter 9.

Increased Yellow to Orange Cast to Skin

Carotene exists in subcutaneous fat. Hypercarotenemia or carotenemia is characterized by a yellow to orange skin color. It is due to consuming too much beta carotene supplements or eating too many dietary sources of beta carotene, (orange juice, carrots, carrot juice or pumpkin) In hypercarotenemia, the whites of the eyes remain white, unlike jaundice who have a yellowish tinge to the eyes (Schwartz, 2011). Refer to Figure 5.8.

Increased Bluish Cast of Skin

The form of hemoglobin that circulates without oxygen is called deoxyhemoglobin. Increased concentration in cutaneous blood vessels gives skin a bluish cast called cyanosis. There are two forms of cyanosis. If the arterial blood oxygen level is low, cyanosis is central. Central cyanosis is best identified in the lips, oral mucosa and tongue. In dark skinned individuals, look at the palms, nails and soles of feet. If the arterial blood oxygen level is normal, cyanosis is peripheral. Peripheral cyanosis occurs when the cutaneous blood flow decreases or slows and tissues extract more oxygen than usual from the blood. It is a normal response to anxiety or a cold environment.

Cyanosis that is seen in only one part of the body may be due to a blood clot. It is often seen in patients with chronic

heart or lung problems (Bickley, 2009). Refer to Figure 5.9.

Decreased Color of Skin

The form of hemoglobin that circulates carrying oxygen is called oxyhemoglobin. Very pale skin or pallor and pale conjunctivae suggest a lack of oxygen possibly due to a nutritional anemia. See anemia section. Refer to Figures 5.10.-5.11.

Increased Redness of Skin

An increased redness of skin is caused by an increase in blood flow through the arteries to the capillaries causing a reddening of the skin. Blushing is a normal body response to strong emotion. Flushing of the face may be related to high fever, menopause, hyperthyroidism, rosacea, intense exercise, spicy foods, caffeine, niacin supplements, or medications. Increased erythema in other parts of the body may suggest inflammation, tissue injury, rash, skin disorder or infection (Bickley, 2009).

Clinical Nutrition Pearls: Skin Color

Diseases and disorders associated with changes in skin are medical diagnoses. The nutrition professional is identifying nutrition diagnoses. PES statements may incorporate NFPA observations specific to skin color, medical signs and symptoms related to changes in skin color, dietary data and anthropometric data. Nutrition diagnoses that may apply include altered nutrition-related lab test results, excessive or suboptimal intake of micro or macronutrients, excessive bioactive substance intake or harmful beliefs about food. Use NFPA findings as a benchmark to monitor and evaluate effectiveness of nutrition interventions.

Practical Applications for NFPA: Nutritional Anemias

Anemia is symptomatic of a disease and is a biomarker for increased morbidity, hospitalizations, mortality and increased healthcare costs. The prevalence of anemia increases with each decade of life over age 70 and is associated with both frailty and mobility impairment (AMDA, 2007).

A very pale skin color and pale conjunctivae are two signs of anemia. Anemia may be caused by acute or chronic blood loss, deficient erythropoiesis, or excessive hemolysis. There are four common types of nutrition related anemias;

- iron deficiency anemia
- pernicious anemia
- megaloblastic anemia
- anemia of chronic and inflammatory diseases

Other rare types of nutritional anemias include vitamin B_6 deficiency, an overload of zinc resulting in a copper deficiency or lead toxicity causing iron deficiency anemia.

Common symptoms of nutritional anemias include fatigue, unusually rapid heart rate, shortness of breath with exercise, difficulty concentrating, dizziness, leg cramps and insomnia. Individuals with iron deficiency may also have pica, soreness of the mouth with stomatitis and koilonychia (spooned nails). Iron deficiency anemia results from insufficient dietary intake or impaired utilization or a combination of factors (Santo, 1991)

Physical signs uniquely associated with pernicious anemia include lemon-yellow–tinged pallor with raw-beefy red tongue lacking filiform papillae (Figure 5.15), paresthesia

in extremities, loss of sense of touch, changes in gait, stiffness in arms and legs, dementia, hallucinations and paranoia. Pernicious anemia results from insufficient dietary intake of vitamin B12 or impaired utilization of vitamin B12 or insufficient intrinsic factor required for absorption or a combination of factors.

Individuals with megaloblastic anemia may have increased weakness, impaired cognitive function, irritability and anorexia. Megaloblastic anemia results from insufficient dietary intake of folate or impaired utilization of folate or a combination of factors.

Individuals with anemias caused by inflammatory and chronic diseases may also complain of syncope, palpitations, cold intolerance, and anorexia. Chronic diseases associated with anemias include congestive heart failure, chronic kidney disease and other inflammatory disorders that result in redistribution of iron stores and poor iron utilization.

Vitamin B_6 deficiency contributes to an impaired transsulfuration of methionine to cysteine resulting in elevated levels of homocysteine. It is also required to convert folate into its active form for DNA synthesis. Vitamin B_6 deficiency is rare, but may be secondary to malnutrition, malabsorption, sickle cell anemia, alcoholism or use of pyridoxine-inactivating medications (i.e. anticonvulsants, isoniazid, cycloserine, hydralazine, corticosteroids and penicillamine). It is associated with microcytic anemia, dermatitis with cheilosis and glossitis, pellagra-like syndrome, peripheral neuropathy depression and confusion, and weakened immune function.

Signs and symptoms of anemia caused by lead poisoning

include a blue-black line on the gums (Figure 5.17. lead line), abdominal pain, constipation, nausea, vomiting changes in mood, peripheral neuropathy, memory loss and encephalopathy. Lead interferes with iron utilization and dietary supplements are ineffective (Bickley, 2009).

Copper deficiencies are rare, but can be the result of zinc overload or malabsorption. Cooper deficiencies have been reported in individuals following malabsorptive weight loss surgeries. Physical signs of a copper deficiency include numbness in lower extremities, abnormal gait, myelopathy, poor wound healing or dehiscence of wounds. Copper related anemia does not respond to iron or vitamin B12 supplementation.

When an anemia is suspected, look for other corroborating evidence in the medical record or from the interview. Review lab reports for abnormal test results for hemoglobin, hematocrit, serum iron, ferritin and MVC. Determine if the anemia is microcytic, normocytic or macrocytic. If labs suggest macrocytic anemias or vitamin B12 deficiency, additional tests are needed including serum vitamin B12, methylmalonic, acid and folate. Review medications for folate antagonists.

For individuals with suspected lead-poisoning, review lab reports for abnormal test results for hemoglobin, hematocrit, serum iron and ferritin since lead interferes with the absorption of iron. Follow up testing is needed for confirmation to include lead levels and zinc protoporphyrin.

Abnormal labs reported in copper-related anemia include very low hemoglobin, hematocrit and leukocytes. However, MCV, vitamin B12, folate, ferritin and platelet count are

usually within normal ranges. **Figures 5.11-5.18** are examples of physical signs associated with nutritional anemias.

Figures 5.4.-5.6. Acanthosis Nigricans

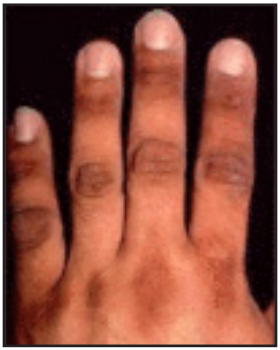


Figure 5.7. Jaundice

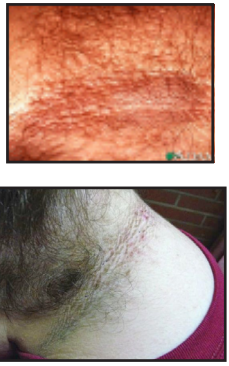


Figure 5.8. Hypercarotenemia

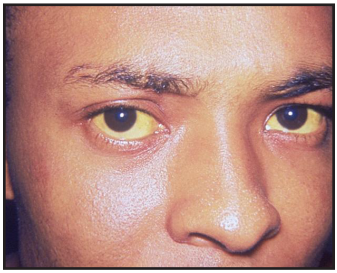


Figure 5.9. Cyanosis

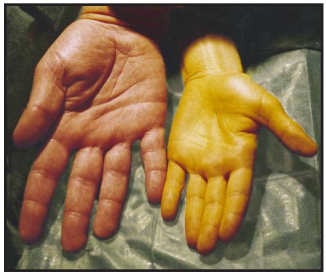


Figure 5.10. Pallor