Day/Part One Background Information for Teaching

Statistics and numbers in the curriculum are taken from the American Cancer Society (ACS), American Academy of Dermatology (AAD), Centers for Disease Control (CDC), Surveillance Epidemiology and End Results (SEER), National Cancer Institute (NCI) and the Skin Cancer Foundation websites, unless otherwise noted.

Getting to the School
Many schools have strict security and keep all doors except the main entrance locked during school hours. Park in the appropriate lot (not the bus lanes) and go to the main office. Place your signature on the sign-in sheet in the school's main office, make out a name tag, and put it on your shirt. Have the secretary contact the teacher or education administrator who will take you to the classroom, gym, or auditorium. It is a good idea to contact the teacher(s) or educational coordinator(s) the day before by email or phone to remind them you will be coming, and to let them know what equipment you will need [PowerPoint projector, laptop, screen, Smartboard, small table (for the demonstration box) and a tall cart or table with two chairs and an extension cord for the Skin Analyzer Machine (SAM)].

Setting up Presentation
It is best to arrive 10 minutes early on the first day. This will allow you time to check the equipment set-up. Some schools have a technology person who does the set-up, so teachers may be limited on their knowledge of the equipment. Others have the set-up as standard equipment in their room. Many schools use Smartboards which link a computer to a large dry-erase screen that is controlled by touch. Emailing the educators (or outside speaker coordinators) the day before will often eliminate problems and time delays. Finally, be ready to give the lecture without the CD in case of equipment failure. This is when the games and hands-on demonstrations come in very handy.

Day/Part One – Focus on Early Detection
Introduction
Class lengths generally run 55 or 90 minutes. Our curriculum runs 50 or 85 minutes. This gives you five minutes to set up while the teacher is doing initial class duties. After the first bell rings, the teacher will get all the students seated, take attendance and then introduce you as the invited speakers. Introduce yourselves by name to the class. Tell them what school you attend. This lets them know you are students, too.

Explain that you are there to speak about early detection of skin cancer and sun protection in a one or two-day program that includes short lectures, games, a video, handouts, a skin analyzer machine, and two surveys for educational purposes (if using surveys). Tell them you want to have an open discussion and encourage them to raise their hands with any questions. Try to end your class at least 3-5 minutes before the next bell to allow the students time to collect their things and get ready to walk to their next class.

Game
Break the ice by choosing one of the following, playing the game, and then discussing their responses and the correct answers.
- Risk game
- Myths/Facts game (included in lecture)
**Interactive Lecture : Background Information for Teaching**

**Hints for Teaching Teenagers**

When instructing teenagers, it is often best to infuse your lectures with visual material, and supplement with pictures, demonstrations, and **hands-on activities**. The human brain prefers visual, rather than auditory input. The brain’s visual processing center occupies approximately 30% of the cerebral cortex, whereas the auditory system is only delegated three percent. Additionally, each of the two optic nerves contains one million nerve fibers while each auditory nerve contains only 30,000 fibers, giving the human visual system 27% more brain space and 67 times the carrying capacity of the auditory system.

Section four of the manual contains information about practical and easy-to-use techniques to increase adolescents’ learning. Take some time to review this section before your first classroom session. It will help you understand how to better teach your teenage audience. Have the students volunteer to do some of the hands-on demonstrations of sun products. Asking questions of the student audience will help retain their attention and increase their participation, while simultaneously giving you the feedback necessary to correct any misconceptions. It also encourages debate, strengthens abstract thinking, and increases knowledge retention.

**Lecture Sections**

- **Who We Are**

  We are students studying medicine, nursing and public health who are interested in bringing information about sun protection to young people in an effort to increase their sun protection knowledge and decrease their risk of getting skin cancer.

- **Why We Are Here (Statistics)**

  Skin cancer affects more people annually than breast, lung, prostate, and colon cancer combined. One out of every two newly diagnosed cancers will be a skin cancer. Over one million cases of skin cancer will be diagnosed annually in the US. Unfortunately, the incidence of skin cancer is increasing disproportionately to the rise in population: while the US population increased by 10% from 1980-2000, skin cancer in adults increased by 83%. From the years 1976-2003, in women under the age of 40 years, the incidence of basal cell and squamous cell carcinoma doubled in the U.S.

  In 1935, the lifetime prevalence of melanoma was one out of every 1500 people. In 1960, the prevalence was one out of every 800 people, in 1980, it was one out of every 250 people, and by the year 2002, the prevalence had increased to one out of every 67 people. It is predicted that one out of every 50 people will have melanoma by the year 2010. Caucasian melanoma incidence has more than tripled in the past twenty years. Skin cancer is now twice as likely in a person under the age of 40 years as it was 30 years ago, and this is not the result of better screening or living longer. It is a substantial increase, especially in women.
Disturbingly, the past 20 years have also shown an increase in the number of young adult and pediatric melanoma cases.\textsuperscript{8-11} An analysis of SEER data for children with melanoma displayed an increase of 2.9% every year from 1973-2001.\textsuperscript{12}

Melanoma, once a cancer of older people, has become a serious problem in American youth: it is the most rapidly increasing malignancy among young people today. If a melanoma is ignored until it reaches the metastatic stage, it is largely refractory to conventional medical treatments and survival plummets. Early detection of melanoma has survival rates in the upper 90th percentiles, but this falls to 15-65% with more advanced spread.\textsuperscript{13} This is why SPOTS was created - to teach teenagers and medical/allied health students how to identify (early detection) and prevent (protective methods) skin cancer.

- **What is Skin Cancer?**

Cancer, simply defined, is a cell that divides abnormally. Cancer cells continue to multiply out of control until they form a mass of cells known as a tumor. Any cancer located anywhere in the body behaves in this way, whether it is a breast cell in breast cancer or a lung cell in lung cancer.

The skin is composed of three main layers -- epidermis, dermis and subcutaneous tissue. The most common skin cells that can become cancerous (basal cells, squamous cells, and melanocytes) reside within the top layer of the skin (epidermis). The thickness of the human epidermis ranges from 0.05mm (eyelids) to 1.5mm on the thickest surfaces (palms and soles).\textsuperscript{14} In relation, a piece of notebook paper is 0.1mm in thickness.\textsuperscript{15} This thin barrier of skin is all that protects people from the damaging effects of ultraviolet radiation (UVR). Once a skin cancer grows through the thin epidermal layer and reaches the dermis, it can more easily spread through the lymphatic and circulatory channels that are contained within the dermal layer. These channels are the body’s "highway" system; they transport cancer cells from the primary site of origin (the skin) to a new or secondary site. This dissemination of cancer cells is known as metastatic spread.

- **Skin Cancer Types**

The three most common types of skin cancer are basal cell carcinoma, squamous cell carcinoma, and melanoma. These three are grouped into the non-melanoma skin cancers (NMSC) or the melanoma skin cancers. The non-melanoma skin cancers include basal cell carcinoma (BCC) and squamous cell carcinoma (SCC). Melanoma skin cancer is also called malignant melanoma (MM). Each of these skin cancers has multiple subtypes. The more common subtypes of melanoma include superficial spreading (70%), nodular (15%), lentigo maligna melanoma (5%), and acral lentiginous (2-8%).\textsuperscript{16}

- **Incidence**

Schofield states that "melanoma is largely a disease of a modern lifestyle" with the rising incidence due to the way people spend their leisure time, the kind of clothes they wear, and their attitudes toward suntanning.\textsuperscript{17} The same can be said for basal and squamous cell skin cancers. Current thoughts as to why there is an increasing annual incidence of melanoma include that there are increases in the diagnosis of thinner lesions (< 1.0 mm) and increases in intermittent
sun exposure especially among higher socioeconomic persons.\textsuperscript{18} Behaviors, social norms, the media, and influential people (parents and peers) all contribute to the incidence of skin cancer, and they are all modifiable.

The most common skin cancer is basal cell carcinoma, composing approximately 75\% of all skin cancers\textsuperscript{19} and 33\% of all cancers. Squamous cell carcinoma comprises around 20\% of skin cancers,\textsuperscript{19} melanoma constitutes around four percent, all other skin cancers comprise the remaining one percent. While melanomas constitute a small percentage, they are responsible for 74\% of all deaths from skin cancer.\textsuperscript{7} Persons who have had one basal cell carcinoma diagnosed have a 20\% chance that a second one will develop within a year\textsuperscript{20} and a 45\% chance of recurrence within five years.\textsuperscript{21} The lifetime risk of Caucasians acquiring skin cancer is 30\% for basal cell carcinoma,\textsuperscript{22} 10\% for squamous cell carcinoma,\textsuperscript{23} and 3\% for malignant melanoma (including in situ cases).

In Caucasians, skin cancer occurs approximately 11 times more often than in African-Americans and Asians, and 5-6 times more often than Hispanics.\textsuperscript{24-26} Unfortunately, in Hispanics, Blacks, and Asians, melanomas are both found by the patient and diagnosed by physicians later, have a greater mortality rate, and thus a poorer prognosis.\textsuperscript{27} This may be due to the fact that melanomas in people of color tend to be found on non-sun exposed areas (palms, soles, subungual) and occur less frequently. Because of this, they may arouse less suspicion on the part of both patients and physicians when a new or changing lesion occurs.\textsuperscript{28}

- **Location**

Basal cell carcinomas are located primarily on sun-exposed areas of skin, most often the head and neck (85\%) with 25-30\% located on the nose.\textsuperscript{29,30} They are also found to a lesser degree on non-sun-exposed body parts.

Squamous cell carcinomas are found not only on the skin, but also in other organs, such as the lungs, cervix, and oral cavity. Squamous cell skin cancer grows almost exclusively on sun exposed surfaces and is found in the largest numbers on the head and neck.

Bowen’s disease is a type of in situ (non-invasive) squamous cell skin cancer that is often misdiagnosed as warts, psoriasis, or eczema due to both its appearance (a round, raised, rough plaque that bleeds if irritated) and because its location can be on the hands and subungual areas.\textsuperscript{31} However, most Bowen’s lesions are found on the scalp and ears in men and the lower limbs in women.\textsuperscript{29} Invasive later stage squamous cell skin cancers can also look like warts, and they frequently bleed.

Melanoma is most commonly found on the trunk in both men (especially the upper back) and women (especially the chest).\textsuperscript{32} This is a change in anatomic distribution for women since the 1970’s when melanomas were most often found on the legs.\textsuperscript{32} There has also been a significant increase in head and neck melanomas in the last three decades. However, melanoma can occur anywhere, even on non-sun-exposed areas. Less than 10\% of melanomas arise in these non-sun-exposed areas (ocular-pigmented retinal areas, the mucous membranes of the oral cavity, anal canal and vulva).
In African-Americans and Asians, melanoma usually occurs on the acral surfaces (palms of the hands, soles of the feet), subungual areas (nailbeds) and the mucosal surfaces (inside the mouth). Its appearance is typically a brown-black lesion with irregular borders. Although melanoma is not common in African-Americans and Asians, the prognosis is worse when it does occur because it is usually detected at a later stage.

- **Metastatic Rates of Non Melanoma Skin Cancer (NMSC)**

  Basal cell carcinomas (BCC) rarely metastasize before they are discovered and removed (cited metastatic rates run from 0.0028-0.1%). However, tissue destruction can be large and the risk of having another BCC arise is 45% in the five years following diagnosis. Squamous cell carcinomas arising from actinic keratoses (pre-cancerous lesions) have a slightly higher overall rate of metastasis (0.5-3.7%). This rate can exceed 20% in certain patients, depending on the location and subtype of the cancer, and host immunosuppression. There is one NMSC (Merkel cell carcinoma) which is highly and rapidly metastatic. Fortunately, it is also very rare (1.4/100,000 US Caucasians).

- **Metastatic Rates of Malignant Melanoma (MM)**

  Rates of metastasis for malignant melanoma vary with the different subtypes. Superficial spreading and lentigo maligna melanoma both have in situ phases and spread horizontally first, making them slower growing. In contrast, nodular and desmoplastic melanomas have no in situ phase, have a rapid vertical growth phase, and are therefore usually diagnosed at a later stage. Acral lentiginous melanomas are of a moderate growth rate, but have a worse prognosis due to late detection. Prognosis in melanoma is predominantly based on the stage of tumor at diagnosis, subtype, presence of ulceration, and thickness of lesion as determined by Breslow’s Criteria. Advanced stage (stages III and IV), ulcerated, and thick tumors have a poorer prognosis.

- **Skin Cancer and Sun Exposure**

  Squamous cell skin cancer is caused by chronic UVR exposure and is almost always found on sun-exposed surfaces. Basal cell carcinoma and melanoma are the result of intermittent or cumulative UVR exposure and can be found on both sun-exposed and non-sun-exposed areas. Sun exposure during childhood and the teenage years (15-19 years) is a major risk factor for development of basal cell carcinoma. Parental protection of children is paramount. Less than half the parents in one study reported regular use of sunscreen on their children and even fewer used other means of protection (hats, shade, clothing). Their primary reason for using sunscreen was to prevent sunburn, not decrease sun exposure. 

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Nevus or Mole

Nevi (plural of nevus) are either congenital (present at birth) or acquired (appearing after 6 months of age and increasing in size and number through the third and fourth decades of life). Acquired nevi tend to be less than 6mm, flat (junctional) or slightly raised (dermal), evenly pigmented (light brown, brown-black, flesh-colored), symmetric in shape (round, oval) and have well-circumscribed borders. They are named based on the location of the melanocytic nests. Junctional nevi have nests of melanocytes at the dermoepidermal junction. Compound nevi have melanocytic nests that have also migrated to the dermis so they are a combination of dermoepidermal and dermal nests. Intradermal nevi have melanocytic nests completely within the dermis and no junctional component.

Studies have shown that nevi increase in size and number during childhood and adolescence and these changes correlate with increased sun exposure, number of severe sunburns, and tendency of the skin to burn. Broad-spectrum sunscreen (SPF 30) use and coverage with clothing in white, freckled children has been shown to attenuate the number of nevi developed. Congenital nevi tend to be of greater diameters (1.5 to over 20 cm), are of lighter color (early in life) often changing to a darker pigmentation and developing hair outgrowths, and when large (>20 cm) have an increased risk of melanoma. It is common for most Caucasian adults to have about 30 nevi.

Atypical (Dysplastic) Nevi

The presence of large numbers of nevi (>100), and atypical nevi (irregular borders/shapes), larger size (5-15 mm), and variation in color (shades from tan to dark brown to pink) increase the risk of melanoma in one's lifetime to approximately ten percent. Large numbers of atypical nevi tend to run in certain families, are an inherited trait, and comprise a syndrome known as atypical mole syndrome or dysplastic nevus syndrome. The three classic presentations of atypical mole syndrome are: 50-100 or more melanocytic nevi, one or more melanocytic nevi with a diameter of 8mm or greater, and one or more melanocytic nevi with atypical ABCD features. Familial atypical mole malignant melanoma syndrome (FAMM) adds a first degree
relative with melanoma to the previous criteria. Research has shown that dysplastic nevus cells exposed to UVR have a greater sensitivity and tendency to mutation. People with atypical nevus syndrome or FAMM have a greater incidence of multiple primary melanomas.

Most adolescent students are not familiar with dermatologic medical terminology (lesions, moles, and nevi). Explain that you will be using the term “spots” to define the circular skin colorations. Most of the spots on their bodies are the result of sun exposure and can be affected by UVR. Students in this age group often don’t know that freckles are a result of sun damage or that nevi can be changed by UVR exposure. Many teenagers consider a mole different than a nevus, although the two are actually synonymous. They think of a mole as a very large, ugly, raised bump similar to the ones on the noses of Halloween witches. They don’t even know the word nevus. It would be difficult, time-consuming, and unnecessary for you to define nevus, so resist the urge to use the medical term. Show them a typical nevus (mole) on your arm and explain that this is the "spot" we will be concentrating on.

- **Freckles**

You may receive some questions regarding freckles. Freckles are skin spots that are caused by sun exposure and first appear in early childhood. They may initially come and go (present during periods of more intense UVR exposure in the summer and then fade in the winter) when young and may become permanent with age or continued exposure. Freckles tend to be flat, approximately the size of a nail head, irregularly shaped, and have a transparent color quality (red or light tan). They are known medically as an ephelis. Several studies have shown a correlation between increased numbers of nevi and freckles, lower/fairer skin types, and greater sun exposure in children and young adults.

- **Lentigines**

Lentigines (plural of lentigo) tend to be darker in color (tan, brown or black) than ephelides (freckles) and do not fade in the winter. They appear with increasing age and are the result of cumulative sun damage. The bottom line is that all spots on their skin, even freckles, can be affected by UVR. The more UVR exposure they receive, the greater the number of spots. Any spot on their skin that changes shape, grows vertically or horizontally, has an unusual color, is 6mm or larger, doesn’t heal, or consistently feels different (burns, bleeds, itches, is rough to touch, sandpapery, or tender) should be checked by a medical professional.
- **What Causes Skin Cancer?**

The main cause of skin cancer is exposure to UVR. UVR can come from either natural outdoor sunlight or artificial radiation from indoor tanning beds. UVR damages the DNA of skin cells. Long-term UVR exposure is the cause of squamous cell skin cancer, whereas both melanoma and basal cell carcinoma can be the result of cumulative and/or intermittent UVR. Ultraviolet radiation is believed to contribute to the development of approximately 65-90% of melanomas.

A family history of skin cancer is another risk factor. Genetic syndromes that are linked to skin cancer development include basal cell nevus syndrome and xeroderma pigmentosa. Heredity is what you are genetically composed of -- half of your mother's and half of your father's DNA. If a person's parents have a hereditary disease, such as heart disease, hypertension, or cancer, that person may have inherited the ability to contract the same condition later in life because they are comprised of the same genetic material. Thus, a family history of skin cancer raises the odds of occurrence. People only have control of one of the two possible causes of skin cancer: you can’t change your genes, but you can modify your exposure to UVR.

- **Warning Lesions**

Squamous cell skin cancers tend to have “early warning spots” that present as an *in situ* or precursor lesion known as an actinic keratosis. These are easily treated with cryotherapy (liquid nitrogen), medicated creams (5-FU, imiquimod), or photodynamic therapy in an office or clinic setting. Neither basal cell carcinomas or melanomas have warning lesions with the exception of the subtype lentigo maligna (*in situ*), which is a precursor lesion to lentigo maligna melanoma (invasive). The good news is that most NMSC’s are generally slow-growing, visible to the naked eye, easy to surgically excise, and 90-95% curable. The most common subtype of cutaneous melanoma is the superficial spreading type. It also tends to enlarge slowly due to its horizontal growth pattern and is easily identified and removed. In the majority of cases, skin cancer is one of the few cancers that can actually be seen by the naked eye. Exceptions include ocular melanomas (non-cutaneous) and vaginal melanoma tumors. Increasing public awareness of detectable changes or warning signs, therefore, should become a teaching priority.

- **Genetic Mutations**

If you are teaching in an advanced science class, the students may have more detailed questions about genetic mutations. A concise, but direct answer is best. There are many genetic factors that are still being researched, but we do know that two genes are commonly mutated in melanoma: CDKN2A and CDK4. CDKN2A is a tumor suppressor gene that normally prevents cells from becoming cancerous, but is inactivated in melanoma. CDK4 is a proto-oncogene which, when mutated, becomes an oncogene (a gene that causes cancer). Melanocortin-1 receptor gene variations (related to skin type) also increase the risk of melanoma (low penetrance) as do p53 gene mutations (in certain genetic syndromes).
• What Does Skin Cancer Look Like?

○ Warning Signs of Basal Cell Carcinoma

Basal cell carcinoma is often ignored by the patient because it frequently does not look like a cancer to the lay person.* In addition, these skin cancers are common in the elderly who already have many skin changes (wrinkles, loss of elasticity, scars, discolorations). Therefore, skin cancer may not make a strong enough impression on the patient for them to feel the need to have it checked until the spot grows to a more advanced stage. Any skin spot that changes in size, shape, color or feel (scaliness, oozing, bleeding, persistent itching or burning) should be examined by a physician or nurse.

The common warning signs of basal cell skin cancer are:

♦ A reddish patch that can be slightly raised, itchy or non-itchy
♦ A shiny bump that is pearly in appearance
♦ A pink bump with an elevated rolled border and a depressed center
♦ A pink bump with small red blood vessels (telangiectasias) on the surface
♦ A scar-like area with poorly defined borders
♦ A persistent non-healing sore

* Many young people have thought their basal cell carcinoma was a pimple that did not heal. Any “pimples” or bumps that persist for longer than 3 weeks should be checked by a medical professional.
- A reddish patch that can be slightly raised, itchy or non-itchy

- A shiny bump that is pearly in appearance

- A pink bump with an elevated rolled border and a depressed center
♦ A pink bump with small red blood vessels (telangiectasias) on the surface

♦ A persistent non-healing sore

♦ A scar-like area with poorly defined borders
Warning Signs of Squamous Cell Carcinoma

Squamous cell carcinoma tends to grow more rapidly and is often more raised and tender to the touch than basal cell carcinoma. These lesions look more like cancer -- they are often ugly and they stand out. The common appearance is an elevated reddened growth with a central depression that bleeds.

The warning signs of squamous cell skin cancer are:
- Scaly red patches that are tender, itch or bleed
- Open sores that don’t heal within three weeks
- Wart-like growths that appear raw, red, and may bleed
- Elevated growth with a central depression that may crust and bleed
- Sores within old scars

♦ Scaly red patches that are tender, itch or bleed

♦ Open sores that don’t heal within three weeks
♦ Wart-like growths that appear raw, red, and bleed

♦ Elevated growth with a central depression that may crust and bleed

♦ Sores within old scars
ABC’s of Melanoma

In melanoma, a mnemonic is used to remember what to look for in a changing spot. This mnemonic consists of the ABC’s of melanoma. Each slide will have both a normal and abnormal spot to show the students the difference and help define the ABC’s. You may also want to explain that the black ring or purple markings they may notice around some of the spots has been drawn with a marker by the doctor or nurse to delineate the lesions that need to be checked. Some students have thought that the inked circular rings are a sign of skin cancer because they see them in a lot of skin cancer pictures. If any spot has one or more of the ABC’s, it should be examined by a medical professional. Studies have demonstrated that teaching lay people the ABC’s of melanoma helped them in their discrimination of suspicious lesions especially when paired with photographs, and may present the best opportunity for patients to find changing lesions during a skin self-exam, especially those with a family history of melanoma. Men were found to be better at detecting irregularities in border and women in finding color changes.

- **"A" stands for Asymmetry**

If you draw a line down the middle of the spot, one half will not equal the other half in size or shape. Most middle schoolers know that the word “symmetry” means same, but may not be familiar with the word “asymmetry.” Most high schoolers will be taking the college entrance exams (ACT, SAT) in which vocabulary is paramount. Sometimes it’s good to remind them that putting an “a” before a word means “not,” so if symmetry means same, then asymmetry means not the same.
"B" stands for Border

Border is the outer edge or circumference of the spot. Circumference is a term they should know well from math classes. Explain that an abnormal edge or border is irregular, notched (bumpy), or scalloped (like a seashell). It is not smooth like the border of normal spots.

"C" stands for Color

Ask the students to look at their arms and tell you the color of their spots - most of them will be brown. Brown is a normal color for spots for most skin types. Redheads may have a reddish brown or paler tint to their spots, which is also normal. Abnormal colors are red (although this can be normal if the entire spot is red, as in a cherry angioma), blue, black, blue-black, gray, or white (amelanotic or depigmented). A spot is also abnormal if it contains more than one color or shade.
"D" stands for Diameter

Diameter always needs to be defined. While the younger students seem to remember what a circumference is, the word diameter often throws them for a loop. They may confuse it with the term radius. Explain that while the border is the outer edge or ring of the circle, the diameter is a straight line across that circle. It is a measurement of the spot’s size at its widest. Any spot with a diameter of six millimeters or greater may be abnormal. Any spot the same size as, or larger than, the size of a pencil eraser is equivalent to 6mm. Take out a pencil and place the eraser on a skin spot on your body to demonstrate this concept to them. It should be noted that lesions can be smaller in diameter than 6mm and still be abnormal, especially in the case of nodular melanomas and early (in situ) lesions. Not all melanomas are large in diameter.

"E" stands for Evolving/Elevation

This is a new member of the ABC group advocated by many in the dermatology profession. Evolving refers to any spot that is changing in size, shape or color. Elevation refers to a spot that is growing in height (vertically). While the majority of melanomas go through a slower, horizontal growth pattern (Enlarging), the nodular subtype has a rapid vertical growth phase (Elevation) which allows it to metastasize in a shorter time period and thus makes it more dangerous. Liu suggests that change in color and size are the best criteria for lay people to differentiate between melanomas and benign skin lesions. Guibert backs this up with his study of occupational physicians who found the criteria most often selected for lesion evaluations were variations in color and enlarged diameter (> 6mm).
Other melanoma warning signs are:

- A sore that does not heal
- A new growth
- Spread of pigment from the border of a spot to the surrounding skin
- Redness or a new swelling beyond the border
- Change in sensation – itchiness, tenderness, or pain
- Change in the surface of a mole – scaliness, oozing, bleeding, or the appearance of a bump or nodule

Spread of pigment from the border of a spot to surrounding skin

Signs of Nodular Melanoma

Nodular melanoma is the second most common subtype of melanoma following superficial spreading melanoma. It is mentioned here because early nodular melanoma often does not follow the standard ABC’s. It can be symmetric in shape, regular in border, symptomatic, small in diameter (< 5mm), and even in color or amelanotic. Chamberlain found that nodular melanomas were mostly symmetric (80%), elevated (90%), and have one color (78.1%) often red or pink. Dr. John Kelly, Head of the Dermatology Unit, Victorian Melanoma Service, AU, suggests that early nodular melanoma follows the EFG’s instead.

- **EFG’s of Nodular Melanoma**
  - E -- Elevated (rapid vertical growth phase, quicker to metastasize)
  - F -- Firm to touch or palpation
  - G -- Growing progressively for greater than a month (short history of change)
  - S -- Small diameter, symmetrical, raised, even color (often red or pink)
• Who Gets Skin Cancer? [Risk Factors]

Risk factors are things that increase one’s chances of getting a disease, infection, cancer, etc. They are not causal agents. They are variables associated with, and correlating to, an increased risk. Studies have shown that personalizing the risks of UV exposure\(^71\) and educating individuals on risk information will improve sun protective behaviors.\(^72\) In addition to being educated in how to acquire healthy attitudes toward sun protection, teens and adults may be more apt to avoid risky behaviors if they are presented with the benefits rather than the negative consequences or when they feel better able to control the level of risk of a certain behavior.\(^73\)

The number one risk factor for melanoma is change in an existing mole. Change includes itching, crusting, bleeding, growth in size, or change in shape or color. This risk factor is associated with an estimated relative risk of greater than 400% association with the development of melanoma.\(^74\)

Change in an existing mole

**Low (fairer) skin type** is another risk factor. Skin types are classified into six levels (Fitzpatrick skin phenotypes) with Skin Type I being the most susceptible to skin cancer and Skin Type VI being the least susceptible. This will be further defined when we discuss prevention. People with low phenotype or Skin Type I (very fair white skin, freckling, white-blond or red hair, blue eyes) have a higher incidence of skin cancer compared to the general population\(^75\) and tend to burn and freckle easily. **Redheads** are a Skin Type I and they produce a type of melanin (the pigment that colors your skin brown or tan) called *pheomelanin* (phee is Greek for false) instead of *eumelanin* (found in greater quantities in Skin Types III and IV). Because of this, redheads never tan, they always freckle and burn. *Pheomelanins* also produce free radicals which are phototoxic. Conversely, *eumelanin* scavenges these reactive oxygen species and is photoprotective.\(^76\) White-blonds and redheads have two to four times the incidence of melanoma as the general population.

Conversely, the darker a person’s skin, the lower their chance of getting skin cancer. African-Americans and Asians have a much lower incidence of skin cancer than Caucasians, while the rate of skin cancer for Hispanics lies in the intermediate range. This is due to the increased size and number of melanosomes, and thus more protective skin of darker races. Studies have shown that lighter skinned Hispanics\(^77\) and lighter-skinned African-Americans have increased rates of skin cancer. Squamous cell carcinoma is the most prevalent skin cancer in African-Americans, but is usually found on non-sun-exposed areas, whereas in Caucasians, it is found on sun-exposed body parts. Overall, melanoma in Asians, African-Americans, and Hispanics generally presents as the subtype acral lentiginous (found on the soles of the feet, palms of the hands, and nail beds), is often detected late in the course of the disease, and therefore carries a worse prognosis with higher mortality rates.\(^26, 28\) Melanoma in Caucasians is of the superficial spreading subtype about 70% of the time, is detected earlier, and
carries a better prognosis. So, while darker skinned races develop skin cancer at a much lower rate than lighter skinned peoples, when they do get skin cancer, it is typically diagnosed at a later stage and therefore carries a worse prognosis, underscoring the need for sun protection teaching in all races.

Atypical Nevi

**Frequency** refers to having a large number of nevi on your body. Studies have shown that having a high number of nevi (moles) tends to run in families. Risk factors include both a high number of normal nevi and a certain number of atypical (or dysplastic) nevi.⁴⁰-⁴¹

A **family history of skin cancer** is another risk factor. Having a primary relative (parents or sibling) with melanoma increases your risk 8-12 fold for developing the disease.⁷⁸

**Ultraviolet radiation exposure under the age of 18 years** is a critical risk factor in the development of melanoma.⁷⁹-⁸¹ Young people spend more time outdoors in long stretches (3-4 hours) during the summer,⁸² don’t heed sun protective measures, and frequent tanning beds. Research has shown that two additional risk factors in this category are **sunburns under 18 years**⁸³-⁸⁴ and **three or more outdoor summer jobs during the teenage years**.⁸⁵ While not an immediate health risk factor, Warthan estimated the yearly economic impact of sunburn in the US to be around 10 million dollars due to lost work and treatment costs.⁸⁶

**Use of indoor tanning beds** has been linked to an increase in all skin cancers.⁸⁷-⁹⁴ Veierod studied over 100,000 Norweigian women followed over eight years and found that women who used a tanning bed only twice a month had a 55% increase in skin cancer.⁹⁵ Tanning beds have a very high level of UVA radiation that penetrates deeply into the lower layers of the skin (dermis) and causes wrinkling, loss of collagen, and skin cancer.

Squamous cell skin cancer is the most common cancer in patients who have received solid **organ transplants and immunosuppressive drugs**.⁹⁶ Prednisone and cyclosporine (immunosuppressive drugs) are linked to a greater than 50% increase in squamous cell skin cancer risk.⁹⁷ Duration and intensity of immunosuppression also increase percentage of risk. In rare cases, patients have developed melanoma after receiving an organ from a donor diagnosed with melanoma.
Environmental risk factors also play a role in skin cancer development. Because of the growing hole in the ozone layer, there is a decreasing amount of atmospheric ozone to filter ultraviolet rays. Therefore, the amount of UVR that reaches the Earth’s surface is higher today than 50 years ago. Additional factors like living at high altitude (places a person closer to the sun with fewer atmospheres to protect them from UVR) or low latitude (places a person closer to the equator and increases the level of direct UVR) can contribute to the development of skin cancer.\(^9\)\(^{,}\)\(^{98}\) UVR increases by 8-10% for every 1000 feet increase in elevation.\(^99\)\(^-\)\(^{101}\) This means that at most ski resorts (5000-7000 feet elevation), UVR is increased by 40-70 percent. At the top of the ski mountain (10,000 feet elevation), this number increases to 80-100% over sea level.

Studies on professional alpine skiers and cyclists have shown the amount of UVB they receive exceeds the international UV exposure limits by 10 and 30 fold, respectively.\(^101\) High intensity marathon runners (run > 70 km/week) have been found to have a higher incidence of NMSC and MM due to a lack of protective coverage from clothing, minimal use of sunscreen, extended hours outdoors, and suppressed immune function.\(^102\) Airline pilots and flight crews have been shown to have a higher incidence of skin and breast cancer\(^103\)\(^-\)\(^{105}\) with those flying longer flights and for greater than five years having the highest incidence.\(^106\)

Occupational risk factors include being exposed to large amounts of arsenic (miners, sheep shearsers, and farmers), industrial tar, coal, paraffin, soot, radium, and certain types of oil. Exposure to these items may lead to an increased risk of squamous cell skin cancer. Arsenic-contaminated drinking water can also lead to skin malignancies.\(^107\)

Ionizing radiation treatments for other types of cancer can lead to the occurrence of basal cell carcinomas (most often) and squamous cell skin cancers primarily in the area irradiated.\(^108\) Young patients who are treated for another cancer with radiation also tend to develop secondary skin cancers at an earlier age.\(^109\)

Other diseases such as systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and human immunodeficiency virus (HIV) all have increased rates of skin cancer. Xeroderma pigmentosum and basal cell nevus syndrome are inherited skin disorders with high rates of skin cancer in affected persons.\(^110\)\(^-\)\(^{111}\)

A final risk factor is age. About half of all melanomas occur in persons aged 50 or older with the percentages increasing rapidly after age 39 and affecting men more than women.\(^112\) Sun damage is cumulative. Starting sun protection early in life is vital to preventing skin cancer.

A melanoma risk tool designed by the National Cancer Institute is available for use by health professionals. It is designed for people who have not been diagnosed with skin cancer and is available at www.cancer.gov/melanomarisktool/.
Treatment of Non-Melanoma Skin Cancer
Since this program is written for teenagers and is primarily about early detection and prevention, the section on treatment is abbreviated. Treatment of basal and squamous cell skin cancers is based upon extent of disease, location, and subtype. Standard treatments for non-melanoma skin cancer include local excision, Mohs micrographic surgery (high risk areas: face, ears, genital), cryosurgery, laser surgery, and application of topical agents (i.e., 5-FU, imiquimod, retinoids). Other treatments include electrodessication and curettage, photodynamic therapy, radiation therapy, and clinical trials. Clinical trials are the use of experimental treatments under the guidance of medical professionals.

Treatment of Cutaneous Malignant Melanoma
Treatment of cutaneous melanoma is primarily based upon the tumor stage. Staging is based on thickness of tumor (Breslow’s Criteria) and presence of nodal involvement, distant metastasis, or ulceration. Stages 0-I also use depth of invasion (Clark’s Level). Staging is classified as Stages 0-IV (0 = best prognosis, IV = worst prognosis) using the TNM classification system. The TNM (tumor, node, metastasis) classification is further subdivided based on the thickness of the Tumor and presence of ulceration, how many and what lymph Nodes are affected, and if the melanoma has Metastasized or spread. TNM, clinical, and pathological staging is described in greater detail by the National Cancer Institute at http://www.cancer.gov.

Standard treatments include surgical excision, chemotherapy, immunotherapy, and radiation therapy. Additional treatments include chemoimmunotherapy (a combination of chemotherapy and immunotherapy) and other clinical trials.

Good News, Bad News
The good news is that most skin cancers are 90-95% curable if found and treated early. It is also one of the few cancers you can see with the naked eye, making knowledge of lesion changes (ABC's of melanoma) important. The bad news is that it often grows on the face in high risk anatomical areas, including the nose, eyes, mouth, and ears. Primary treatment for skin cancer is to surgically
excise or cut it out. Facial areas often require extensive repairs to restore normal appearance due to high reconstructive needs.

Additionally, basal cell skin cancer tends to have an **iceberg effect**. The iceberg effect occurs because the basal cells are the basal or basement layer of the epidermis and can grow extensively in a horizontal direction before they change their surface presentation enough to warrant a trip to the doctor's office. Tissue damage underneath the surface of the skin can be extensive depending on the subtype and the area involved (location).

There are two lessons to be learned here. One is if you do nothing else, protect your head and neck, especially your face. The majority of skin cancers (basal and squamous cell) occur on the head and neck. The second lesson is that it is much easier to prevent than to treat skin cancer. In the next section we will discuss prevention and protection.

**Skin Analyzer Machine**

The skin analyzer machine is an important component of your teaching. It lends credibility to the program -- seeing is believing. Most teens don’t realize how much damage they have already sustained from tanning. Studies using UV photography and scanners have demonstrated that they motivate students to use positive sun protection behaviors.\textsuperscript{115-117}

**Instructions:**
Turn the machine on. Show them the outside and inside (pull back the curtain) of the machine.

Talk with the class teacher. Discuss how she would like to have the students use the machine. Some will have the students form two lines. Others will have the students come up by table, row, or in pairs to maintain some semblance of order in the classroom. One student will look through the view finder on the back of the machine and can see the student inside of the machine. The other student will place their head inside the machine, aligning their chin just above the circular mirror, far enough in so that they can see their face in the mirror, but that it doesn't touch the light bulbs. Once they have viewed their own face or seen another person’s face they can switch positions.

<table>
<thead>
<tr>
<th>Color</th>
<th>What it means</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blue-purple</td>
<td>Hydrated healthy skin</td>
</tr>
<tr>
<td>Brown-purple spots (look like freckles)</td>
<td>Sun damaged areas</td>
</tr>
<tr>
<td>White</td>
<td>Dead skin, scars, clogged pores, teeth, lint</td>
</tr>
<tr>
<td>Yellow or orange</td>
<td>Oily skin, make-up, sunscreen</td>
</tr>
<tr>
<td>Red-pink</td>
<td>Dehydrated skin, thin skin</td>
</tr>
</tbody>
</table>

Skin analyzer machine colors and what they represent
Explain what they will see when they place their head in the machine (refer to the below box). Sunscreen and most make-up will block the effects of the machine.

Respect their privacy. Some students will not want others looking through the view finder at their skin. If this is the case, place your hand over the view finder until that student is finished. Other students may not want to use the machine at all. However, we have found that most students are very enthusiastic about using the SAM and remark about how they need to better care for their skin after using the machine.

Be sure to state that this is meant to educate them about their current level of sun damage, not to scare them. If they are upset about how much damage they have, reinforce the need for sun protection, and the methods they can employ to prevent further damage.

Once again, you are not there to diagnose skin lesions. If a student or teacher asks you to look at a spot on their skin, politely explain you are still a student and suggest they visit their physician.

**Open Questions & Answers/Next Session Briefing**

Open the question and answer session by first thanking the students for listening and participating. Then ask them a question. What was new to them? Was anything confusing? What did they like the most? What made the biggest impression?

If you are presenting a two day program, briefly tell them what will be covered on Day Two: a quick review of the ABC’s of melanoma, a video of two teenagers with skin cancer, and a combined lecture/demonstration of sun protective measures (sunscreens, hats, surf shirts, sunglasses, shade), alternatives to tanning (self-tanner and powdered bronzer applications), and other protective measures, followed by open discussion.