1996

Pilot study to evaluate the impact of an educational video about melanoma on knowledge and behavior

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PILOT STUDY TO EVALUATE THE IMPACT OF AN EDUCATIONAL VIDEO ABOUT MELANOMA ON KNOWLEDGE AND BEHAVIOR

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Pilot Study
To Evaluate the Impact of an Educational Video About Melanoma On Knowledge and Behavior

A Thesis Submitted to the
Yale University School of Medicine
In Partial Fulfillment of the Requirements for the
Degree of Doctor of Medicine

By Sepideh Bagheri
1996

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PILOT STUDY TO EVALUATE THE IMPACT OF AN EDUCATIONAL VIDEO ABOUT MELANOMA ON KNOWLEDGE AND BEHAVIOR. Sepideh Bagheri, Jean Bolognia, Marianne Berwick. Department of Dermatology, Yale University, School of Medicine, New Haven, CT.

Background: Given the growing worldwide incidence and mortality of melanoma, and a lack of cure at more advanced stages of the disease, much effort has focused on educational and screening interventions aimed at diagnosing lesions at earlier, potentially more curable stages. Skin self-examination (SSE) has received increasing attention as a means of complementing public screenings; however, to date, there have been no trials specifically aimed at promoting SSEs among targeted populations.

Objective: In this pilot study, an educational video on melanoma was prepared to not only raise subjects' awareness about melanoma epidemiology, but also prompt regular SSEs (including the use of a "buddy").

Methods: Of the 94 participants recruited, 86 completed the study: 33 melanoma, 33 atypical nevus, and 21 control subjects. Each subject received a total skin examination by a physician and viewed the ten-minute video, in addition to completing pre- and a post-intervention questionnaires (on knowledge and SSE practices).

Results: Of note, the melanoma and nevus groups had the highest knowledge scores, while the controls had the greatest improvement. The controls were as knowledgeable of melanoma risk factors (like red hair and blue eyes) as the melanoma and nevus subjects, but less knowledgeable about melanoma signs and symptoms. One of the greatest post-intervention changes related to awareness of such methods as the "buddy" system to examine

difficult-to-see areas. Other significant predictors of knowledge increase included the hair color, the objective (physician-determined) and subjective (self-reported) nevus counts, and the presence of atypical nevi. Regarding SSE practices, the melanoma and nevus subjects were significantly more likely to perform SSEs (at least once per year) as compared to the controls. However, there were no significant predictors of either "optimal" SSEs (defined as once per month to once every four months) or improved SSEs.

Conclusion: Based on our results, a video on melanoma appears to be an excellent educational medium, as observed by the significant improvements in knowledge among the controls. However, more intensive and continuous interventions may be needed in order to affect behavioral change.

Acknowledgements:

I would like to thank all the people who helped me successfully complete this project through their precious time and valuable insights: foremost, Dr. Bolognia, whose ceaseless support, unending dedication, and unwavering high standards allowed the study to soar to new heights; Dr. Berwick, who guided me through the world of SAS and offered her statistical expertise in moments of despair; Dr. Pawelek, for the kindness and generosity of reading and critiquing my written thesis; Donna Carroll, in the Dermatology Departmental office, for the never-ending stream of copy cards, stamped envelopes, return questionnaires, and gentle reminders on my answering machine; and finally, the nurses and staff-members of the dermatology clinic at the Yale Physician's Building, who made the seemingly unending hours of chart reviews both pleasant and productive.

Introduction:

Cutaneous melanoma is the most aggressive of all skin cancers. It is a malignancy of melanocytes, the dendritic cells within the epidermis whose principal function is to synthesize melanin and distribute it to surrounding keratinocytes. The melanin serves several functions, including the protection of keratinocytes and melanocytes against ultraviolet radiation [110, 182].

One of the most alarming features in the epidemiology of melanoma is the dramatic rise in its worldwide incidence, along with a more modest rise in the associated mortality. In 1994, an estimated 32,000 new cases of melanoma and 6900 (4300 men and 2600 women) deaths due to melanoma were reported in the United States alone [6, 38].

From the 1960's to the 1980's, the incidence of melanoma increased by 3.5 times in men and 4.6 times in women [129]. Moreover, the incidence of melanoma appears to be doubling every one to two decades [43, 115, 230, 231, 253, 333, 335]. (Such trends are cause for even more concern given that the true incidence rates are probably under-estimated with the ever-increasing trend toward out-patient diagnosis and treatment, resulting in incomplete registration [36, 174, 183, 343]). For instance, the Connecticut Tumor Registry, which has been tracing the number of melanomas diagnosed since 1935, quoted an age-adjusted melanoma incidence of $1.0/10^5$/year for its first 5 years; this incidence had risen to $12.4/10^5$/year by 1989 [355]. These trends have been corroborated by the cancer registries of Denmark [166], other Scandinavian countries [43, 230, 231, 333, 335], Australia and New

Zealand [37, 47, 107, 132, 215], Canada [120], Europe [34, 123, 238, 265], and additional States within the United States [50, 129, 268], including Hawaii [156].

Melanoma mortality rates have been rising as well, although not as rapidly as the incidence rates [262]. This has been reported by countries such as the United States, the United Kingdom, Canada, Sweden, Australia, New Zealand, and Japan [102, 115, 202, 253]. In the United States, melanoma mortality has had a faster rate of increase than that for any other cancer (except for lung cancer in women) [115, 246]. Mortality rates have been increasing by approximately 2-3% annually in Caucasian populations [355]. However, two recent studies from the United States have projected a future downward trend in melanoma mortality [106]. Using birth cohort analysis of melanoma cases and deaths from the Connecticut Tumor Registry, one projected a downward trend in melanoma mortality for both men and women [306]. Using SEER and National Center for Health Statistics data (1973-1984), the other projected that the current upward trend in age-adjusted mortality rates will taper off, leading to a maximum death rate in the near future, followed by a reduction in melanoma deaths by the second decade of the 21st century [313]. Cohort analyses of mortality rates in the United States revealed the highest mortality rates among men born around 1950, and among women born between 1930 and 1950, while demonstrating lower mortality rates among later-born cohorts [306]. Given the continued rise in incidence rates, the investigators in this study attributed these findings to earlier detection. In Canada, the mortality rates among women continued to rise through the mid-1980's, showing some decline thereafter; however, among men, the upward trend in mortality has continued [105]. In Sweden, melanoma mortality rates had been rapidly rising since 1953 in all but men under

50 years of age, whose rates had shown no increase. Since 1978, however, the mortality rates have been stabilizing among Swedish women, with slowing of the rate of increase among men [336]. In Scotland, melanoma mortality rates have been declining among women since 1987, with no change in the rate among men [225]. This trend has been attributed to the intensive public education campaigns, which has resulted in increased proportions of thinner melanomas among women.

Given the growing threat of melanoma as a public health hazard and our increased knowledge regarding its risk factors, education would represent an obvious means for its containment, through both primary prevention (i.e., sun exposure precautions), and secondary prevention (i.e., early detection) [144]. To date, most efforts have focused on earlier diagnosis, in hopes of intervening at a potentially more curable stage in the cancer's natural history, i.e., the precursor or radial growth phase.[2] In this regard, melanoma holds the unique advantage of being amenable to easy detection due to its visible cutaneous location.

Early detection assumes great importance when one considers that the Breslow depth (measured in millimeters from the stratum granulosum to the lower most portion of the tumor) is the single-most important prognostic indicator for survival [45]. There is a direct correlation between the tumor thickness and the probability of developing metastases. Therefore, thinner melanomas are associated with significantly higher survival rates: lesions less than 0.76 mm in Breslow depth are associated with five-year survival rates of 93% to 100%, as compared to less than 50% survival rates for lesions greater than 3 mm in Breslow depth [14, 19]. Also, those with localized disease have long-term survival rates of 75% to

over 80%, as compared to only 15%-20% for patients with regional spread, and rare survival in those with distant metastases (with median survivals of around 6 months) [355]. These data have served as rationale for advocating skin self-examination (SSE) in the general public, and especially for high risk groups, in order to detect early, thinner melanomas [144].

Risk Factors:

In preceding decades, much effort was concentrated on delineating possible risk factors for the development of melanoma, with the dual goals of not only identifying high risk groups as targets for early intervention through screening programs, but also disseminating knowledge about the risk factors through public education campaigns. To date, the following have been identified as melanoma risk factors: (1) melanocytic nevi--the presence of either atypical nevi or greater-than-average numbers of common acquired melanocytic nevi; (2) phenotypic characteristics--color of eyes, hair, and skin type (tendency to burn and inability to tan); (3) family history of melanoma; (4) personal history of melanoma; (5) history of sunburns or chronic sun-damage (as in patients with lentigo maligna melanoma); and (6) the presence of precursor lesions such as congenital nevi.

I. Nevi:

Over the past decade, there has evolved growing awareness of the association between melanocytic nevi and melanoma. Case-control studies report that the presence of either atypical nevi or higher-than-average numbers of common acquired melanocytic nevi to be the strongest, independent risk factors for melanoma [109].

Common Acquired Melanocytic Nevi (CAMN) versus Atypical Nevi: CAMN are benign neoplasms of melanocytes, with a clinical and histologic appearance, distribution, and developmental history which differs in several ways from that of atypical nevi. (As in any biological system, however, there is some overlap). Clinically, the initial CAMN lesions present as 2-4 mm macules (or slightly elevated papules), with a symmetric shape, well-

circumscribed borders, and a uniform tan to dark brown color. At its initial developmental stage, it is termed a "junctional nevus", characterized histologically by the formation of melanocytic nests--confined to the dermo-epidermal junction--at the tips of the rete ridges, and variable lentiginous hyperplasia. Junctional nevi can remain completely stable over time, or gradually undergo a lateral expansion phase, during which they may enlarge to a maximal size of 4-6 mm.

Again, at the completion of this lateral expansion phase, they may remain stable, or begin a vertical growth phase, during which the melanocytes migrate into the dermis, and thereby evolve into "compound nevi". Clinically, compound nevi are distinguished from junctional nevi by their morphology as 1-7 mm, symmetrical, well-circumscribed, tan-brown papules. Their elevation is due to the symmetric dermal invasion. Meanwhile, compound nevi display all the histologic features of junctional nevi, in addition to the formation of melanocytic nests and cords within the dermis.

With time, these nests and cords undergo a maturation process, whereby they penetrate deeper and deeper into the dermis and its underlying connective tissue while at the same time progressively losing their epidermal components [91, 216, 252]. At its extreme, this process leads to the formation of "dermal nevi". Clinically, dermal nevi appear as 2-7 mm pink or skin-colored papules, and histologically, they are characterized by nevus cells which are completely confined to the dermis, with only mature intradermal melanocytic nests and cords. Moreover, by this later developmental stage, the melanocytes have frequently lost their ability to synthesize melanin, which accounts for their non-pigmented, pink- or skin-colored appearance.

In their anatomic distribution, CAMN tend to primarily occur on sun-exposed areas of the body, such as the upper trunk and the extensor surfaces of the upper extremities. CAMN are absent at birth, first appearing on the skin during infancy and early childhood, gradually growing in both number and size through to middle adult life. The average individual develops an estimated 15-30 nevi by early adulthood [254], but this number gradually declines in later years due to the natural regression of CAMN in late adulthood [88, 216 252, 254].

In contrast, atypical nevi (AN) are neoplasms of melanocytic origin, with clinical and histologic characteristics intermediate between those of CAMN and melanoma [56, 88, 141, 142, 194, 286, 287, 290]. Clinically, they are characterized by their larger size (frequently greater than 5 mm in diameter, sometimes up to 12 mm), asymmetric shape, notched or ill-defined borders, uneven or cobblestoned surface (with the papular component frequently embedded within the macular portion), and variegated tan-red-brown color [350]. They are frequently distinguished by an inflammatory component, as evidenced by a pink hue, either at the periphery or throughout. Histologically, atypical nevi are lentiginous junctional or lentiginous compound nevi with architectural disorder, as evidenced by scattered melanocytes and disarrayed nests, with bridging of adjacent nevus nests, formation of nests at rete edges or in inter-rete spaces (as opposed to the tips of the rete ridges only), and expansive nests which may focally erode upwards into the epidermal layer. The individual nevus cells can display cellular atypia, as manifested by enlarged nuclei, angulated contours, dense hyperchromasia, and at times, an over-abundant yet coarsely-granulated cytoplasm. Other histologic components of an atypical nevus include stromal change: lamellar

fibroplasia, vascular proliferation, melanin pigment incontinence, and a patchy lymphocytic infiltrate [18, 59, 252]. Here, erythema, nevus size, and border irregularity are the strongest clinical correlates of histologic atypia [18]--usually classified as either mild, moderate, or severe, where mild is insignificant and severe is significant, as it borders on melanoma in situ [350]. Although the lesions may remain stable over time, or regress, in general, the degree of atypia serves as a marker for the risk of malignant transformation, which may be heralded by the clinical signs of increased size, asymmetry, irregularity, or color variegation, scaling, crusting, bleeding, and sensations like tingling, itching, and pain [350].

In their distribution, atypical nevi mimic CAMN in that they also primarily occur sun-exposed areas such as the upper trunk and the extensor surfaces of the upper extremities; however, they differ from CAMN in that they also appear on doubly-shielded, non-sun-exposed areas such as the genitals, breasts (in women), scalp, and buttocks [195].

Atypical nevi are also distinguished from CAMN by their unique developmental history. Like CAMN, atypical nevi are absent at birth [141, 216]. At approximately 5 to 8 years of age, affected individuals begin to develop an increased number of morphologically normal nevi as well as a few larger nevi that are often located on the scalp or upper trunk. At puberty, these nevi may undergo significant changes in number, size, shape, and pigmentation. Although, like CAMN, individual lesions may regress in later years, unlike CAMN, new lesions may continue to develop throughout life, such that their total count may never decrease [16, 350].

Common Acquired Melanocytic Nevi (CAMN) and Melanoma: In the last two decades, there has been a growing awareness of CAMN as determinants of melanoma risk.

In biopsy specimens of cutaneous melanoma, remnants of junctional and compound nevi have been observed in histologic contiguity with the malignant cells. Reports of the frequency of this phenomenon have ranged anywhere from 18% to 72%, with estimates in the 30% to 50% range being most common [33, 56]. In a recent review, for instance, more than 50% of primary melanomas were in histologic contiguity with a precursor nevus, and this percentage rose to 64% when only thin tumors (<1.5 mm), with a lower likelihood of obliteration of the pre-existing nevus, were considered [309]. Moreover, as many as 80% of melanoma patients report changes in a pre-existing mole [88]. (It is important to note, however, that patients can mistakenly confuse an early melanoma arising *de novo* with a changing mole, due to their similar clinical morphology).

A number of case-control studies have shown the presence of greater-than-average CAMN counts to be the single most important determinant of melanoma risk, with greater risks with higher total CAMN counts [144, 361] (Table 1). The magnitude of melanoma risk associated with increased numbers of CAMN has varied considerably, ranging from 5 to 65 [361]; this wide range is partially due to differences in the definition of CAMN, the areas of the body examined, and the method used to count the CAMN. A review of nine case control studies examining the relation between CAMN and melanoma risk found an extreme risk of melanoma (RR=63.8) in subjects with more than 50 nevi greater than 2 mm in diameter, with even greater risks in the presence of clinically atypical nevi [329, 330]. In other studies, excess numbers of arm nevi [100, 135, 138, 161, 329] or total body nevi [82, 158, 200, 258, 303, 329, 330, 356] were associated with an at least 3-fold increase in melanoma risk [27, 68, 82, 86, 100, 108, 135, 138, 158, 159, 161, 200, 258, 280, 303, 320, 328, 329, 330, 356].

The Western Australia Melanoma Study reported a multi-variate odds ratio of 10.35 in the presence of 10 or more palpable nevi on the arms [161], while the Queensland Melanoma Study documented a multi-variate odds ratio of 30.1 for the presence of *any* arm nevi [135, 161] (Table 1).

Multiple case control studies have noted a gradient in melanoma risk as a function of the nevus count, with increased risk with higher nevus counts [99, 135, 159, 200, 328, 330]. In such studies, the estimated melanoma risk for the highest mole categories has ranged from 9.8 (for greater than 100 CAMN on the entire body) [159] to 133.4 (for the presence of >40 nevi on the trunk) [200][109, 135, 143, 159 356, 200, 330] (Table 1). The San Francisco Melanoma Study, counting nevi greater than 2 mm in diameter on the entire body area, documented the following gradient in melanoma risk: 0-10 nevi, RR=1.0, 11-25 nevi, RR=1.6; 26-50 nevi, RR=4.4; 51-100 nevi, RR=5.4; > 100 nevi, RR=9.8 [159] (Table 1). A population-based melanoma study in Denmark reported similar results [259]. Here, the melanoma risk associated with the nevus count was found to be independent of all other risk factors, accounting for an estimated 29-79% of all melanomas [304].

Given the significance of CAMN as not only predictors of melanoma risk, but also lesions from which melanoma may arise, much effort has been devoted to determining their etiology, so as to delineate the links common to the pathogenesis of CAMN and melanoma, and thereby possible means of prevention. At present, the etiology of CAMN is wrought with controversy. Some studies suggest that the total nevus count is determined genetically [142], while others implicate sunlight and sun-exposure (as contributors to their phenotypic expression) as major determinants [192].

The distribution of nevi on the body has been used to support the role of sunlight and sun-exposure as determinants of the total nevus count. For instance, both melanocytic and atypical nevi tend to occur in greater proportions on the lateral, more sun-exposed aspects of the arm, as compared to the medial, more sun-protected surfaces [192, 193]. However, one study refuted such a correlation between total numbers of nevi, whether benign or atypical, and ultraviolet exposure [271].

There is also evidence to support the role of sunlight in the etiology of CAMN, and therefore (possibly) melanoma: body sites with higher mole counts--which are also sites of greatest intermittent sun exposure, as explained above--have been shown to be preferential sites for the development of melanoma [121, 200]; meanwhile, sun-shielded regions such as the genitals, breasts (in women), scalp, and buttocks have shown the lowest incidences of not only melanoma, but also (melanocytic and atypical) nevi [5, 121, 167, 192, 193, 200, 270]; and migrant studies correlate higher nevus counts with a younger age at the time of migration to a sunny climate [4, 139]. Such data implicate the presence of common stimuli in the pathogenesis of CAMNs and melanoma. Either UV radiation induces the expression of (melanocytic and atypical) nevi which later act as targets for other promoters, or UV radiation acts as a promoter on these unstable target lesions, i.e. nevi, to induce melanoma [111, 361].

Atypical (AN) and Melanoma: From an historic standpoint, interest in atypical nevi (AN) as a risk factor for melanoma dates back to 1976, at which time a National Cancer Institute (NCI)/University of Pennsylvania (UPenn) team started to examine a melanoma-prone kindred, known as Family K. The presence of numerous clinically atypical nevi on

the body of many members of this family, with both morphologic [278] and histologic [55] similarities to early cutaneous melanoma ignited interest in the possible association between the two [201]. Thus, it was in the context of melanoma-prone kindreds that the association between atypical nevi and melanoma was first defined [141]. Later, the NCI/UPenn group conducted a prospective surveillance of 14 such melanoma-prone kindreds, and in the process, charted the development of new cutaneous melanomas in only those family members with AN--i.e., none developed amongst relatives with clinically normal skin [141, 142]. In these cases, the histologic contiguity of the melanoma lesions with atypical nevi demonstrated that, in at least the familial melanoma setting, atypical nevi serve as not only *markers* of increased risk of *de novo* melanoma formation, but also the structural *precursors* from which melanoma may arise [141, 350]. Another study following a larger cohort of families, for a longer period, obtained similar results [144, unpublished].

Later, it was recognized that atypical nevi also occur in melanoma patients without a family history of melanoma [87], and even in individuals with neither a personal nor a family history of melanoma [74]. Such findings led to the recognition that atypical nevi (AN) can occur in either familial or sporadic settings. The familial atypical nevus-melanoma syndrome is characterized by a increased numbers of CAMN, along with the presence of a multitude of atypical nevi as compared to the average population. Affected individuals are considered to have at least two blood relatives with both atypical nevi and a prior history of melanoma. In contrast, the sporadic cases may or may not have numerous CAMN, and may involve only a few AN [350]. These findings have stimulated interest in the epidemiology of atypical nevi, as well as their possible relationship with CAMN.

Epidemiologic studies have estimated that atypical nevi affect approximately 5% of the white population, of whom 5% will develop melanoma over their lifetime [17]. Moreover, atypical nevi increase melanoma risk in both the familial and the sporadic settings, appearing on 35% of all melanoma patients, and on 100% of all familial melanoma patients [17]. Careful histologic studies have revealed remnants of atypical nevi at the margins of invasive melanoma lesions in about one third of unselected melanoma cases [33]. Furthermore, case-control studies involving skin examinations have yielded AN prevalence estimates ranging from 6% to 55% (34% median) among melanoma cases, and from 0% to 17% (median 7%) among controls [27, 68, 74, 108, 147, 149, 159, 258, 304, 328]. Similar estimates were attained upon excluding all subjects with a family history of melanoma [149, 304].

Linkage studies have revealed the familial atypical nevus-melanoma syndrome to follow an autosomal dominant inheritance pattern, with variable penetrance and expressivity [29, 141, 142, 176, 221]. The familial atypical nevus-melanoma syndrome accounts for an estimated 5% to 10% of all cases of melanoma [91], and affected individuals are at 100% lifetime risk of developing melanoma by age 70 [142, 194]. In this setting, not only do melanomas occur at a younger age than in the general public [221], but also the survivors are at high risk for additional primaries [142, 257]. Indeed, in the NCI study of the 14 atypical nevus-melanoma syndrome kindreds, the prospective relative risk of melanoma among family members with *no* prior melanoma was *150*, while the relative risk for melanoma among family members *with* a prior melanoma was *500* [142]. Also, this study revealed that melanomas developed in only family members with atypical nevi. Such findings signal the

need for close lifelong surveillance of affected individuals by a dermatologist [142, 257]. And, in fact, prospective studies entailing careful longitudinal surveillance of such individuals have documented a shift in the Breslow depth of newly diagnosed melanomas toward thinner, better-prognosis lesions [141, 237, 344]. Such interventions may ultimately affect the melanoma mortality rates in these populations.

Similar estimates of melanoma risk in the sporadic, non-familial AN setting were controversial for some time, mainly due to the lack of definitive histopathologic criteria for the evaluation of the involved specimens. Recent studies, however, have shown acceptable degrees of interpathologist concordance following adherence to the criteria for both architectural and cytologic atypia [35, 59]. The World Health Organization has documented a concordance rate of 88% [59], while a Dutch study has reported a sensitivity of 92%, a specificity of 98%, a positive predictive value of 89%, and a negative predictive value of 98%, using the outlined criteria [321].

Subsequent to these reports, an estimated 29% to 49% of *non*-familial melanomas were attributed to AN as a risk factor [304]. Furthermore, reported relative risks of melanoma in the setting of non-familial atypical nevi have ranged between a 7-fold increased risk, as based on estimates [196], and a 22-fold increased risk, as based on prospective measurements [295], as compared to controls. (The latter value was considered unstable due to the limited number of individuals enrolled in the study: 281 subjects with non-familial AN and no personal history of melanoma were followed over 27 months, during which time 3 *in situ* melanomas and 1 invasive melanoma were detected, representing a 16-fold increased risk of invasive melanoma [295]. Nonetheless, this study was of critical

importance, in that it unequivocally documented the occurrence of both *in situ* and invasive melanomas in subjects with non-familial AN, thereby signaling the need for lifelong surveillance in this population.)

Independent studies aimed at discerning the relationship between AN and CAMN have reported not only higher CAMN counts among subjects with AN [149, 159, 258, 305], but also higher AN counts among subjects with increased numbers of CAMN [258, 266, 302]. Furthermore, analyses aimed at separating the contribution of these two classes of nevi to the overall risk of melanoma suggest them to be independent risk factors [305]. Notably, these studies verify the interesting clinical observation that these independent (though critically significant) risk factors often occur in the same individuals [159].

II. Phenotypic Characteristics:

The variation in melanoma rates by race and pigmentary skin characteristics is well-established. Melanin, the pigment whose form and concentration determines skin type and skin color, protects the melanocytes' DNA from the damaging effects of ultraviolet radiation [297]. Given that melanocytes contain little melanin themselves, they are normally protected from UV radiation by the melanin in the keratinocytes surrounding them. This protection is less efficient in fair-skinned individuals, who have lower concentrations of melanin, as compared to more pigmented individuals [91]. Moreover, molecular studies have revealed red-haired individuals to have pheomelanin [264], a form of melanin which exacerbates the effects of UV radiation through the generation of potentially genotoxic and mutagenic free radicals [90, 264]. Therefore, it is not surprising that the degree of skin pigmentation directly correlates with the risk of melanoma [297].

Case-control studies have identified a cluster of phenotypic traits, such as blue or green eyes, red or blond hair, light complexion, freckles, "poor tanning ability", and a "tendency to sunburn" that are associated with an increased risk of melanoma [109, 161, 240]. Moreover, it appears that the entire cluster of traits (as a whole) is more important than any single trait alone. And as these phenotypic traits are determined genetically, the risk of melanoma also becomes partially defined by genetic factors [188].

A skin classification scheme separates whites into four groups on the basis of their "tendency to burn" and "inability to tan". Skin phototypes I and II burn or develop a light tan, respectively, whereas skin phototypes III and IV develop a medium or dark tan, respectively. Case control studies from Europe, the United States, Canada, and Australia have established a 2-3-fold increase in melanoma risk with skin phototypes I and II [63, 98, 260, 353, 364]. Other studies have documented a two-fold increase in melanoma risk among Caucasians with "fair" skin, as compared to those with "olive" skin [27, 98, 131, 135, 138, 161]. Meanwhile, for all skin types, melanoma patients have increased light sensitivity, as evidenced by the significantly lower minimal erythema dose of their skin, as compared to controls [22]. A review of case-control studies revealed the "inability to tan" to be a significant risk factor in 15 of 16 studies, and the "tendency to sunburn" to be a significant risk factor in 21 of 24 studies [109].

Freckling, as a marker for the "inability to tan" in response to longterm sun exposure, has been associated with a two-fold increase in melanoma risk [27, 78, 98, 131, 135, 138, 161, 280]. One study, which documented a 29-fold increase in melanoma risk in the presence of both CAMN and freckles, as compared to when both traits were absent [138],

postulated a possible association between CAMN and sun-induced freckles. The same study quoted a 51-fold increase in melanoma risk in the presence of both high nevus counts and a tendency to sunburn (and not suntan), as compared to when both traits were absent. Upon adjusting for total lifetime sun exposure, this study found a relative risk of 30 for high nevus counts, 5.9 for red hair, and 3.5 for the tendency to sunburn [138].

Skin characteristics such as the "tendency to burn" and the "inability to tan" appear to be independent of such melanoma risk factors as nevi and "excessive" sun exposure [100, 135, 138]. Moreover, the tendency to burn easily and tan poorly is a stronger predictor of melanoma risk than the actual frequency of sunburns recalled; also the increased risk of heavy vacation and recreational sun exposure appears to be independent of skin pigmentation and reaction to the sun [98, 133, 163]. There also appears to be no clear relationship between (recalled) anatomical sites of sunburns and anatomic sites of melanoma--this may be due to recall bias [138].

In regard to other phenotypic features, the literature commonly reports relative risks of 2 to 4 in association with blond and red hair [27, 131, 135, 161, 280], although one study attributed a relative risk of 74 to blond (as compared to brown or black) hair [23]. Many studies have found red hair to be the strongest *phenotypic* risk factor for melanoma [27, 97]. As mentioned before, the melanocytes of red-haired individuals have been documented to contain pheomelanin, a form of melanin believed to exacerbate the effects of UV light by generating free radicals which cause mutagenesis [91, 264]. Also, case-control studies have reported 1.5- to two-fold increased risk of melanoma in association with blue, green, gray, and hazel eyes [27, 131, 135, 161, 280].

III. Family History of Melanoma:

Studies have documented a strong and statistically significant association between the risk of melanoma (RR=5) and a positive family history of melanoma [135, 142, 161, 194, 289, 290]. This risk appears to be independent of such inherited (and confounding) risk factors as pigmentation and nevus count, although these may still play a role. In one study, the risk of melanoma (in the presence of a positive family history) was unrelated to the degree of *closeness* of the affected relative--i.e., it made no difference whether the melanoma occurred in a first- or second-degree relative [138]. However, in other studies, the association between melanoma risk and a positive family history disappeared with regression analysis, suggesting that the increased risk relates more to phenotypic characteristics, sun-exposure habits, [134, 138] and a propensity for higher nevus counts [87]. Meanwhile, those with an affected first-degree relative have a melanoma relative risk of 2 to 10 [84, 135, 161, 319].

IV. Personal History of Melanoma:

Individuals with a prior history of melanoma are at a significantly high risk for additional primaries; however the estimates of this risk have varied greatly, from a five-fold to a nine-fold increased risk [161, 339]. The most accurate estimate has been derived from a Connecticut study which followed a population of 4693 individuals with a diagnosis of melanoma for the development of subsequent primaries from 1935 to 1982 [339]. In this population, 30 second primaries were detected, translating into an overall relative risk of 8.5; however, there was a significantly higher relative risk of 23 for those less than 40 years of age at the time of the first melanoma diagnosis. Another follow-up study of 384 consecutive

melanoma patients at the MD Anderson Hospital during 1969-1970 documented relative risks of 22 and 45 for second primaries--with the variance in the documented relative risks depending on the reference population used to generate the expected number of melanomas [144, unpublished]. Moreover, the diagnosis rate of second primaries is highest within one year of the original diagnosis (RR=24), but continues to remain substantially elevated long afterward [339]. These figures help identify a group at high risk for melanoma, which would benefit from preventive and interventional measures [144].

V. Sunlight and Ultra-Violet (UV) Radiation:

Retrospective case-control studies, epidemiologic studies on variations in melanoma as a function of race, phenotype, latitude of residence, migration (and age at the time of migration), body site distribution (of both melanomas and CAMN), occupation and socioeconomic status, and studies of melanoma in the setting of xeroderma pigmentosum and lentigo maligna melanoma have all helped document a role for sunlight and UV radiation in the pathogenesis of melanoma [181, 188].

Latitude Studies: Worldwide latitude gradients in melanoma incidence and mortality provided the first crude evidence for the role of sunlight in the etiology of melanoma [181]. Studies of melanoma incidence among Caucasians in five continents have documented an inverse relationship between melanoma incidence and latitude, and therefore, solar UVR exposure. This relationship is roughly linear up to a 50-degree latitude, and parabolic thereafter [251]. Others have described this relationship as a quadratic equation [251].

Specifically, in nations with homogeneous, predominantly fair-skinned populations, as in Australia, North America, England, Wales, Norway, and Sweden, melanoma incidence

and mortality rates increase with proximity to the equator [85, 91, 93, 96, 203, 204, 221, 251]. In Norway, the melanoma incidence is three times higher in the southern areas, as compared to more northern areas [229, 230]. In the United States, the melanoma rate among Connecticut residents was 3 times lower than that of Arizona residents (9 versus 26 per 10^5) [311]. Queensland, Australia has the highest worldwide melanoma incidence, at 30.9 per 10^5 men and 28.5 per 10^5 women, as compared to that of Connecticut, at 8.4 per 10^5 men and 7.7 per 10^5 women during 1978-1982 (all rates per year). The Queensland rates are four times higher than the U.S. rates--which are, in turn, double the Canadian and triple the U.K. figures [228, 251, 294, 358].

However, not all areas demonstrate consistent latitude gradients. For instance, the rates for Scandinavian countries are higher than those of Mediterranean countries [181]; and Finland and Western Australia lack such a latitude gradient [164]. Such findings re-emphasize the important interplay between sunlight and such factors as sex, age, ethnic and phenotypic heterogeneity, customs, socioeconomic status, geography and climate, vacation habits, and migration effects [5, 164, 212].

Another geographic factor related to latitude that has received considerably less publicity is altitude. With the same logic, high altitude locales should experience higher melanoma rates given their greater UV radiation exposure [181]. This might explain why Tucson, Arizona, a southern city with an elevation of 2400 feet and a UVB intensity 4% higher than that of a site at sea-level but the same latitude, has one of the highest U.S. melanoma rates [311].

Racial Differences: The geographic variation in melanoma incidence largely derives from an interplay between ethnic composition and latitude, the former an indicator of sun sensitivity and the latter an indicator of ultraviolet flux, and therefore, the intensity of sun exposure. The ethnic and racial composition of a population influences its susceptibility to melanoma through phenotypic features such as the a tendency to burn, the inability to tan, and hair and eye colors [355]. The melanin concentration in the keratinocytes overlying the melanocytes determines their (the melanocytes') degree of protection from the harmful effects of UVR and its free-radical by-products. Also, dark-skinned individuals tend to have lower total nevus counts as compared to fair-skinned individuals [318].

The contribution of latitude, and hence sun exposure, to the development of melanoma has been differentiated from that of ethnicity by comparing melanoma rates in similar ethnic groups residing at different latitudes, such as the white populations in the United Kingdom versus Australia, and those within the United States, Canada, Australia, and Scandinavia [203, 355]. Studies have also compared melanoma rates in various ethnic groups within the same geographical region--and therefore, the same environmental exposures. From this perspective, investigations on the epidemiology of melanoma in the United States have been most interesting, given that it is a geographically unique area with widely divergent racial populations living in close proximity, not only exposed to the same environmental risk factors for melanoma, but also covered by the same population-based cancer registries--thereby facilitating the comparison of trends. The Surveillance, Epidemiology, and End Results (SEER) program is the broadest such registry in the United States, combining nine population-based registries that together cover approximately 10%

of the entire population [363]. According to the SEER, the 1987-1988 melanoma incidence was $10.9/10^5$/year for whites and $0.9/10^5$/year for blacks [294]. Meanwhile, the 1972-1982 Los Angeles County registry incidence rates for Anglos, Hispanics, and African-Americans (per 10^5/year) were 12.1, 3.3, and 1.1 among men, and 10.0, 3.6, and 1.0 among women, respectively [223]. Similarly, for the years 1978-1981, the SEER registry quoted the following rates (per 10^5/year) in various regions: in New Mexico, 12.1 and 1.7 among Anglos and Hispanics, respectively; in San Francisco, 10.0 and 1.0 among whites and blacks, respectively, and <1.0 among the Chinese, Japanese, and Filipinos; and in Hawaii, 23.0 among whites, but only 1.7 among Japanese-Hawaiians [165]. Earlier data from Hawaii [155, 156], Texas [222], South Africa [145], Germany [123], Puerto Rico [28, 345], and western Canada [98] have reported similar trends. Thus, in the United States, the risk of melanoma is 8-19 times higher in whites as compared to blacks [1, 279, 160]. Similar trends are reported worldwide. Meanwhile, a 1986 survey of 614 U.S. hospitals found 98% of the 4545 melanoma cases to have occurred in a white or Caucasian individual [12].

Of note, when melanoma *does* develop in blacks and Asians, populations with normally low incidences of melanoma, it has a predilection for subungual and palmoplantar surfaces, and mucous membranes, which are relatively sun-protected areas secondary to either their location, or their thick overlying stratum corneum [71, 73, 113, 127, 145, 202, 213, 279, 326]. The high *relative* frequency of such melanomas in these groups is precisely due to the relative absence of melanomas at other sites in these populations; the absolute incidence of palmoplantar and subungual melanomas varies little among races [31, 63, 102, 294, 314, 324, 345].

Given that incidence rates are highest among whites, it is not surprising that mortality rates are also highest among whites. In the United States, during the years 1987-1988, the overall mortality (per 10^5/year) was 2.4 among whites and 0.4 among blacks [294]. However, case fatality rates are much higher among blacks as compared to whites [294].

Migration and "Critical Period" Studies: Migration and "critical period" studies have attempted to not only differentiate the roles of environmental and genetic factors in the etiology of melanoma, but also delineate specific childhood events that increase melanoma risk later in life [65, 160, 250]. Migration studies within the United States [223, 352] and within Australia and New Zealand [65, 160, 250], as well as those of immigrants from Europe to Israel [2] and from the United Kingdom to Australia [160] have all shown higher rates of melanoma in immigrants to sunnier climates, as compared to genetically similar populations remaining in the native homelands. Also, the melanoma risk appears to increase with earlier arrival and longer residency in the new country, such that it eventually approaches that of life-long residents in the sunnier climate. Australian investigators have observed an increasing melanoma risk among immigrants to the sunny climate of Western Australia with increasing duration of residence (especially if they have resided there for more than 40 years [138]) [163]. However, as compared to native-born Australians, these immigrants are at a substantially lower *overall* risk for melanoma, regardless of the country of birth [3, 161, 164].

Other than the duration of residency, one of the main determinants of risk among immigrants' is their age of arrival to Australia [161]. Australian investigators have described a "critical period" for immigration which appear to determine later susceptibility to

melanoma [160]. Specifically, arrival before age 10 appears to be associated with the highest melanoma risk, similar to that of native-born Australians. In contrast, those arriving after age 15 have one-fourth the risk of those arriving prior to age 10 [4, 139, 160, 163]. Interestingly, these investigators have also noted a correlation between the total nevus count--which, as explained before, is the single-most significant determinant of melanoma risk--and the age of immigration to the sunnier climate, and therefore, sun-exposure [4, 139, 163]. Higher nevus counts were recorded among those who had migrated during early childhood. Such findings lend more support to the etiologic role of sunlight in the induction of both benign nevi and melanoma: early childhood is a crucial age for establishing lifetime melanoma risk, and this risk is connected to the induction of nevi [163]--i.e., early exposure is more successful at invoking a large pool of "potential precursor" lesions or CAMN [91].

Other investigators have focused on childhood, adolescence, and early adulthood as periods of potentially intense sun exposures [234]. Australian investigators evaluating sun exposure by age, have reported an association between increased melanoma risk and *high-intensity* exposure during the age periods of 10 to 24 years and 25 to 39 years [163]. (Solar exposure was estimated in terms of mean annual sunshine hours at each residence.) In contrast, the *total* outdoor sun-exposure (recreational or occupational) was not associated with melanoma risk in the cases as compared to local controls in this sun-loving Australian community. (However, some studies do not corroborate these findings [97, 163]). Meanwhile, studies from western Canada have found recreational, and not occupational, sun exposure to be associated with increased melanoma risk, even after factoring in skin

pigmentary characteristics [98]. This further supports the link between intermittent sun exposure in young adult life and melanoma.

Other studies have documented a clear association between increased melanoma risk and sunburns and "poor tanning" during adolescence, or "long vacations in sunny climates" during childhood [133, 137, 211, 224, 281]. U.S. investigators have correlated a significantly increased melanoma risk with a history of 2 or more blistering sunburns between the ages of 15 and 20 years, as opposed to beyond 30 years of age [353]. Others, following restratification of the data by sex and tanning ability, have implicated mere erythema, without pain or blistering, as a possible risk factor [211].

Concomitantly, studies from the United States examining the two factors of latitude and age simultaneously, have reported the risk to rise inversely with the latitude of one's residence during ages 15 to 20 years, while finding no such association for ages greater than 30 years [353]. These findings are crucial in pinpointing the etiologic significance of sun exposure that occurs decades prior to the peak onset of melanoma, in defining melanoma risk, an association which is consistent with melanoma's long latency period [91].

Body Site Distribution: The body site distribution of melanoma differs drastically from that of non-melanoma skin cancers (NMSC)--basal cell and squamous cell carcinomas--which are related to cumulative, low-grade lifetime sun exposure. This difference implicates the significant etiologic role of intense, intermittent sun exposure--as through leisure activities--in the development of melanoma [5, 181]. Whereas NMSC have a predilection for areas of greatest cumulative, low-grade sun exposure, such as the face, ears, neck, and the dorsum of the hands, melanoma predominates on intermittently exposed areas of the

body, such as the trunk and the lower limbs [15, 77, 181, 207, 248, 362], with a tendency to spare doubly-shielded areas such as the buttocks, genitalia, breasts (in women), and scalp (in women) [212]. Furthermore, there are distinct gender-based differences in the anatomic distribution of melanoma, with both men and women affected frequently on the back, but women affected on the lower extremities, especially the calves (a low-incidence site among men) [72]. Studies have revealed a 40-fold variation in the frequency of melanoma per unit body surface area at different anatomic locations, the frequency of which correlates with patterns of intermittent sun exposure [94].

Temporal trends in melanoma incidence over a 20-year period further substantiate this theory, given that the incidences have changed at different rates for different anatomic sites of the body. While the incidence of limb and trunk melanomas rose dramatically during this time period, that of head and neck melanomas remained virtually unchanged [93, 95, 129, 166, 230, 231, 323, 333]. Also, on the extremities, there was relatively little change in the incidence of hand melanomas, as compared to melanomas on the remainder of the arm [83]. Moreover, over this time period, there were increased rates of lower limb melanomas in females and of trunk melanomas in males [93]. Factors that may explain these temporal trends include changed fashion styles (with exposure of female legs by short skirts, and male ears and scalp by shorter haircuts), thinner clothing, and the growing popularity of bathing suits and sun-related activities such as sun-bathing. All of these factors may contribute to greater sun exposure to previously shielded areas [61, 163, 175, 209, 231, 332].

Occupation and Socioeconomic Status: Epidemiologic studies comparing the incidence of melanoma among indoor and outdoor workers substantiate the intermittent

exposure theory of melanoma. Professionals, office workers, and indoor technical workers have higher incidences of melanoma when compared to outdoor workers like farmers and construction workers, while the reverse holds true for non-melanoma skin cancers [5, 76, 97, 99, 342]. Others have reported a direct relationship between melanoma risk and measures of socioeconomic status [109]. One study reported the melanoma risk to increase significantly with increasing salary among men [266]. Still other studies have found no association between melanoma and outdoor occupational sun exposure [8]. Investigators have documented an excess risk of melanoma on the trunk and limbs (normally covered areas) but not the head and neck of indoor office workers, as compared to those with outdoor occupations [25]. Of note, one study found that the risk of melanoma was actually significantly reduced in the presence of a permanent, deep, and even tan [99]. Also, the highest melanoma mortality rates have been documented in professional, managerial, and clerical workers [64, 208].

All of the above evidence further supports the role of intermittent, intense sun-exposure in the etiology of melanoma, given that the population of indoor workers has greater susceptibility to sunburns as a result of its normally untanned skin. An additional factor could relate to their higher incomes, which allows therm greater opportunities for outdoor leisure acitivities and tropical vacations [266]. For instance, in Sweden, persons of higher SES were shown to be more likely than low SES individuals to visit southern Europe during vacations [42]. Notably, the association between melanoma risk and socioeconomic status, as defined by either occupation or income, has held true for men, but not women; this was explained by the observation that, most of the women in the reported studies were

married, with lower-paying occupations than their husbands, but most probably the same recreational activities (and sun exposure) as their husbands [266]. This theory was tested in the population of England and Wales, where the women, after being classified according to their husbands occupations, were documented to have the same melanoma risk as men in the same SES [208].

Some investigators have offered an alternate explanation for the observed associations between melanoma risk and SES, stating that those with more disposable income may have greater awareness of health issues, and consequently, a greater likelihood of detecting possible lesions [266]. In a survey of persons attending the Melanoma/Skin Cancer Screening sponsored by the American Academy of Dermatology, it was found that most attendants were well-educated [184]. Similarly, an Australian study found a correlation between higher education levels and greater probabilities of periodic screenings for melanoma [128].

Physical, Non-Solar Ultra-Violet Radiation: UVR from a variety of non-solar sources has been related to increased risk of melanoma. Artificial sources of light differ in their spectral distribution of UVA and UVB, with UVB considered to have the greatest mutagenic and carcinogenic potential [219, 256]. Sunbeds and sunlamps have been the best-publicized artificial lighting source, associated with a relative risk of 2.9, which becomes even higher in the case of (primarily UVB-emitting) domestic tanning devices [119, 162, 260, 331, 348]. Nearly all U.S. tanning salons have UVA-emitting devices.

Personal History of Non-Melanoma Skin Cancer: Personal history of non-melanoma skin cancer, related to cumulative sun damage, has also been shown to be significantly

associated with higher melanoma risks [136, 159, 160]. A population-based survey of 1973 patients with basal cell carcinoma known to the Swedish Cancer Registry, documented significantly elevated relative risks of melanoma in both men (RR=6.8) and women (RR=4.2) [214]. Also, a review of 3,260 patients revealed a 7-fold increase in melanoma risk in those with a prior personal history of basal cell carcinoma. Likewise, actinic keratoses, also related to cumulative sun exposure, are related to a 3- to 5-fold increase in melanoma risk [133, 138, 160, 232, 280].

Xeroderma Pigmentosum: Xeroderma pigmentosum is a rare autosomal recessive disease, characterized by defective repair of DNA damaged by UVB. Thus, the disease is characterized clinically by an excessive sensitivity to the sun, and biologically, by cellular hypersensitivity to the mutagenic effects of UVB. Patients have a 2000-fold risk of developing melanoma as compared to the general population [58, 197].

VI. Other Pigmented Lesions:

Congenital Melanocytic Nevi: Congenital melanocytic nevi (CMN) are distinguished from common acquired melanocytic nevi (CAMN) by their presence at birth. They are further subclassified into three categories on the basis of their size: small CMN are less than 1.5 cm in diameter, while medium and large (or giant) CMN measure 1.5-20.0 cm, and greater than 20.0 cm in diameter, respectively [144]. The melanoma risk, and therefore, the management issues surrounding each CMN category varies substantially [1, 188, 205, 298, 349].

Giant congenital melanocytic nevi (GCMN) are considerably more rare, as compared to small CMN, occuring in approximately 1 in 20,500 live births by some estimates [52], and

1 in 100,000 to 1 in 500,000 (depending on racial factors and size criteria used) by others [173, 205, 276, 277, 282, 285, 290, 291, 307, 338, ;KK,14-15]). In terms of their clinical appearance, they have a characteristic morphology, comprised of a grossly irregular surface, marked by hyperpigmentation with varying shades of brown and hypertrichosis [233]. The risk of melanoma in GCMN has been a matter of much controversy [89, 169, 269, 284], estimated at 5%-20% over the lifetime--which constitutes a greater than 17-fold increased risk of melanoma over the general population [220, 255]. However, a research team at NYU is conducting a prospective melanoma surveillance in a group of patients with GCMN, which should yield a more precise estimate of melanoma risk in such persons [124].

In terms of their natural history, about half of all melanomas associated with GCMN are diagnosed within the first 3 to 5 years of life, with an additional 10%-20% occuring before puberty [220, 277, 307]. Of note, 40% of all childhood melanomas arise from giant congenital nevi [170]. Concurrently, melanomas associated with GCMN carry a graver prognosis as a result of several intrinsic features. First, they develop deep in the dermis and subcutaneous tissue, as well as in the central nervous in the setting of neurocutaneous melanosis, which renders them difficult to detect at an early stage. Second, the respective melanoma cells are highly anaplastic, with a greater propensity toward early and rapid metastases [220, 276, 282, 350]. These considerations have led to the recommendation that all such nevi be excised as early as technically feasible; however, with up to 50% of the melanomas developing in the CNS or as unknown primaries, this has been questioned [173, 205, 276, 282, 285, 291, 338].

Small- and medium-sized CMN (SCMN) are more prevalent than giant ones, appearing in approximately 1% of newborns [52]. SCMN also predispose to melanoma, although at lower rates than GCMN. Moreover, the actual estimates for the rate of transformation of these lesions into melanoma remains a matter of controversy, because most estimates are based on histologic analyses, and there are no definitive histologic criteria for distinguishing between acquired melanocytic nevi and small congenital nevi [169]. In a histologic survey of 234 melanoma specimens, 64 (27.4%) were contiguous with a dermal nevocellular nevus, of which 19 (8.3% of all the melanomas) contained histologic markers of congenital nevi [284]. Such studies attribute an estimated cumulative incidence risk of 2.6%-4.9% to SCMN up to age 60, an 18-fold increase in risk over the general population [283]. (Again, such analyses may over-estimate the actual risk, given that there is no histologic means of differentiating acquired from congenital nevi [288]).

In terms of their natural history, unlike GCMN, SCMN rarely progress to melanoma before 12 years of age, and consequently, most specialists recommend that they be carefully observed until puberty, at which time they may be removed under local (and not general) anesthesia, given the greater likelihood of patient cooperation at that point. However, others advocate early surgical excision for immediate assurance, as well as freedom from the need for life-long follow-up (and its added economic burden) [89, 165, 169, 188, 283, 284, 301]. In a survey, 50% of the physicians opted for early excision, while 27% recommend long-term observation [89].

Interestingly, the presence of "congenital-looking nevi" (large in size but not "atypical" in appearance) in the familial melanoma setting was associated with an 8.5-fold increased risk, as compared to those with only atypical nevi [195].

Lentigo Maligna (Melanoma): Lentigo maligna (LM), which represents *in situ* melanoma, is related to cumulative exposure to ultra-violet radiation [209], thus sharing the same epidemiologic distribution as basal cell and squamous cell carcinomas--i.e., it frequently appears on the face and other chronically sun-exposed skin surfaces of middle-aged and elderly individuals [21, 171, 241].

Clinically, LM is a large, haphazardly pigmented, irregularly bordered, macular lesion, ranging in color from tan to brown to dark brown to black. At times, the lesions may show signs of regression, presenting as "lightened" or "normal-looking" areas within the pigmented lesion. Over time, these lesions tend to expand slowly, become more haphazardly pigmented, and may undergo invasive change, heralded by the appearance of focal indurated papules or a diffuse elevation of the entire lesion [144, 290]. If not recognized and treated, the invasive form can lead to metastases and death. The interval for this invasive transformation has been estimated to be very long, at least 5-15 years, as based on patient reports [249].

Histologically, LM is marked by the hyperplasia of a single layer of melanocytes, with a hyperpigmented basal epidermal layer, where the pigment density is independent of the amount of sun-exposure--i.e., it does not increase following sun exposure [138]. The melanocytes are atypical in appearance, with a disordered proliferation within an atrophic epidermis, which overlies a sun-damaged dermis--i.e., solar elastosis [54, 160, 177].

Lentigo maligna melanoma (LMM) accounts for only 5% of all cases of melanoma [290]. Each individual LM lesion has an unclear risk of progressing to invasive melanoma [177, 242, 267, 352]. Estimates of the incidence of invasive transformation vary between sources, ranging from 5%-10% to 25%-50%, depending on the study [171, 351, 352]. Thus, LM is considered to be associated with a 10-fold increase in melanoma risk up to age 75 [352]. Even though LM is generally considered to be a slow-growing lesion, there have been occasional reports of rapidly-growing cases [171, 245]. When controlled for Breslow depth, LMMs have the same prognosis as other forms of melanoma. Surgical excision, usually the preferred form of treatment [245], may not be practical or acceptable, given the high associated morbidity of facial scars [188]; so, when the lesions are large in size or the patient is very old, alternate therapies include radiation therapy and cryosurgery.

Materials and Methods[1]:

From June to November of 1992, 94 individuals were enrolled in the study after receiving an explanation of the protocol, and giving oral consent to participate in it. The subjects were recruited from three Yale University dermatology clinics: 69 from the Pigmented Lesion Clinic[2] (PLC), 18 from a university general dermatology clinic (UGDC), and 7 from a hospital general dermatology clinic (HGDC) (Table 2).

Of the original 94 subjects, 86 finally qualified for the study (66 PLC, 17 UGDC, and 3 HGDC), while 8 subjects (3 PLC, 1 UGDC, and 4 HGDC) were disqualified, as they failed to complete the follow-up questionnaire (Table 2). Of the 86 subjects, 33 had a personal history of cutaneous melanoma [M], 22 with and 11 without atypical nevi; 33 had atypical nevi only [N]; and 20 served as controls [C] (Table 4). All of the melanoma patients and 31 (94%) of the atypical nevus patients were recruited from the PLC; and 2 (10%) of the controls were enrolled through the PLC: one attended the PLC because of *falsely* believing in having atypical nevi due to a history of non-melanoma skin cancer in his father, while the other attended the clinic due to a history of melanoma in her husband. The subjects ranged in age from 18 to 85 years (mean=45), with a mean age of 47, 38, and 54 years for the melanoma, nevus, and control groups, respectively. The control and melanoma groups were significantly older than the nevus group ($p=0.001$); (Table 3).

All subjects with a history of melanoma had histologic confirmation of their diagnosis. Of the atypical nevus subjects, 25 had histologic confirmation of their diagnosis [257], with biopsy specimens demonstrating architectural disorder, with or without cellular

atypia, while 8 were diagnosed clinically [142], based on the appearance of their nevi as evaluated by a dermatologist (Table 4).

At the initial evaluation, all subjects received a total body skin examination by the dermatologist, which included a complete nevus count, removal of all lesions suspicious for early melanoma, and photography of the most atypical pigmented lesions, if any. Following the skin examination, all subjects completed a questionnaire which assessed baseline knowledge of clinical characteristics and risk factors for cutaneous melanoma, as well as general health-seeking attitudes and behavior. The questionnaire was divided into three sections, each of which evaluated a different area of knowledge regarding melanoma: (1) clinical signs, e.g., irregularities of color or shape, the presence of bleeding, itching, or tenderness, and increase in thickness; (2) melanoma facts, e.g., curability with early surgery and familial predisposition, and (3) risk factors, e.g., phenotypic characteristics like skin type, blue eyes, and red hair, presence of many nevi or atypical nevi, history of sunburns, and personal or family history of melanoma. In order to assess each subject's general health-seeking practices, the questionnaire also inquired about his/her body awareness, frequency of skin self-examination, and the number of health visits in the previous year. Information was also obtained regarding each subject's sun-exposure habits, use of sunscreens, self-estimate of nevi, and personal and/or family histories of melanoma and non-melanoma skin cancers.

Moreover, each subjects was screened for the presence of various melanoma risk factors. These included the total nevus count--both objectively, as ascertained by a physician[3], and subjectively, as reported by the subject him/herself--a subject's own

impression of "having more nevi than the average individual", a subject's personal history of atypical nevi and non-melanoma skin cancer, and finally, his/her family history of melanoma and non-melanoma skin cancers.

The questionnaire was followed by a ten-minute educational video that reviewed recent epidemiologic trends in melanoma incidence and mortality, melanoma risk factors and clinical characteristics, while also emphasizing sun-safe behavior, regular skin self-examinations, and the use of a "buddy" for examination of *difficult-to-see* regions of the body. The video, prepared through the collaboration of Yale University's Dermatology, Epidemiology and Public Health, and Audiovisual departments, combined a factual and didactic tone with the emotional story of a family's loss to melanoma, in order to further reinforce its messages.

The follow-up questionnaires were completed by either mail (n=66), telephone (n=11), or during return clinic visits (n=9); (Table 2). The time period between the preliminary questionnaire and the follow-up questionnaire ranged between 4 and 22 months, with 94% completed within 14 months.

Analysis: The analyses were performed using the SAS statistical analysis program, with a comparison of knowledge and SSE practices by group and clinic at both baseline and follow-up. Both the *degree* and *proportion* of improvement were analyzed as a function of the various variables--the former through the *mean change*, and the latter as the *proportion* of subjects to progress from an incorrect response at baseline to a correct one at follow-up.

[1]All the work for the study--the recruitement of subjects, completion of post-questionnaires, chart reviews, data analysis, and literature review--were performed by the student. The questionnaire assessing the subjects' knowledge-base was adopted from a previous pilot study (performed with an educating nurse as opposed to

an educational video), with only minor modifications to the sections on subjects' skin self-examination practices. Dr. Bolognia, M.D. was the dermatologist who examined all the subjects, while Dr. Berwick, Ph.D. was the epidemiologist providing advice regarding the analyses.

[2] On their first visit to the Pigmented Lesion Clinic, patients received a folder with information on melanoma risk factors (including common acquired melanocytic nevi, atypical nevi, sun exposure, and sunburns), the signs and symptoms of melanoma and non-melanoma skin cancers, precautions regarding sun exposure, and finally, the importance of early detection through regular skin self-examinations. These pamphlets included the following: *Why You Should Know About Melanoma*; *the ABCDs of Moles and Melanomas*; *Dysplastic Nevi and Malignant Melanoma: a Patient's Guide*; *What You Need to Know About Moles and Dysplastic Nevi*; *What You Need to Know About Skin Cancer*; *Fry Now, Pay Later*; *For Every Child Under the Sun: A Guide to Sensible Sun Protection*; *Facts About Sunscreens*; *Sunless Self-Tanners: the Safer Tan*; *Solumbra Sun Protective Clothing and Accessories*; and finally, some general information on melanoma prevention and the Pigmented Lesion Clinic at Yale.

[3] One control subject did not receive an objective, physician-determined total body nevus count, being that she was recruited from the UGDC of a second dermatologist and refused a total body skin examination by the dermatologist involved in the study. She did return all the follow-up questionnaires by mail.

Results:

I. Demographic and Phenotypic Characteristics:

The demographic characteristics of the patient population are shown in table 3. There was a predominance of females in all the groups, in approximately a 3:2 to 3:1 ratio, with no statistically significant differences between the groups (Table 3). (Of note, the dermatologist following the Pigmented Lesion Clinic patients and one of the University General Dermatology Clinics from which most of the control subjects were recruited is also female.)

The mean age for the melanoma and the control groups (47 [A] and 54 [A] years old, respectively) was significantly higher than that for the atypical nevus group (38 [B] years old). The largest proportion of nevus patients was in the 18 to 35 year-old age-group, (n=15 (45%)), as compared to the melanoma and control groups, in which the highest proportion of subjects were greater than 51 years old (n=14 (42%) and 13 (65%), respectively) (p=0.001); (Table 3).

Data on the highest educational level attained by each subject was not available, so the occupational scores were used as a proxy for the socioeconomic status. There were no statistically significant differences in socioeconomic status among the groups (Table 3).

Data regarding phenotypic characteristics such as hair and eye color, and skin susceptibility to burns and ability to tan, were based on participants' subjective self-reports. There were no statistically significant differences between the groups with respect to these phenotypic characteristics (Table 3).

In the melanoma group, approximately one-third of subjects had blond/red, light brown, and brown/black hair, each. In contrast, in the nevus and control groups, the highest proportions of subjects had brown/black hair (n=21 (64%) and 11 (55%), respectively). These differences were not significant (Table 3).

Most melanoma subjects (n=21 (64%)) had blue/green eyes, as compared to the nevus and control subjects, who mostly had blue/green or light/dark brown eyes (n=15 (45%) and 13 (39%) for the nevus group and 8 (40%), each, for the controls). These differences were not statistically significant (Table 3).

To define the skin types, two characteristics were emphasized: (1) the predisposition to sunburns in reaction to acute sun-exposure and (2) the ability to tan in response to chronic sun-exposure. These two characteristics, related to inherent skin pigmentation, influence individuals' risk of melanoma, as described in the introduction. Interestingly, in all three groups, the majority of subjects reported experiencing painful sunburns, or mild sunburns followed by a slight tan (n=30 (90%), 30 (91%) and 14 (70%) for the melanoma, nevus and control groups, respectively). Also, none of the melanoma subjects, as compared to 1 (3%) nevus and 2 (10%) control subject(s), reported tanning without a sunburn in response to acute sun-exposure. These differences were not significant (Table 3).

The majority of subjects in all three groups reported a history of either mild tans with several peelings, or of moderate tans, in response to chronic sun-exposure (n=21 (64%), 19 (58%), and 9 (45%), for melanoma, nevus, and control groups, respectively). Also, only 2 (6%) melanoma subjects, as compared to 5 (15%) nevus and 4 (21%) controls reported developing a deep tan (Table 3).

II. Risk Factors:

The objective, physician-determined total body nevus count was significantly different between the groups (p=0.001). The majority of the melanoma subjects had less than 33 nevi (n=20 (61%)), and only 3 (9%) subjects had greater than 67 nevi. In the nevus group, most subjects had either 0-33 or 34-66 nevi (n=12 (36%) each), while 9 (27%) subjects had greater than 67 nevi. In contrast, all control subjects had 0-33 nevi (Table 4).

There were statistically significant differences in the groups' subjective, self-reported total body nevus counts (p=0.001). The majority of melanoma subjects (n=19 (58%)) reported total body nevus counts of 0-33, while the majority of nevus subjects believed they had greater than 67 nevi (n=16 (48%)). Most controls estimated their total body nevus counts to be less than 33 (n=17 (85%)). These self-assessed total nevus counts were correct in 17 (52%) melanoma, 18 (55%) nevus, and 17 (85%) control subjects. These results had borderline statistical significance (p=0.051); (Table 4).

Each participant's subjective impression of having "more-than-average numbers of nevi" was also assessed. The fact that 12 (36%) melanoma, 23 (70%) nevus, and 4 (20%) control subjects reported having greater-than-average nevus counts was not significant (Table 3). Given that the average individual has a total nevus count of approximately 20-30, the proportion of subjects who correctly believed in having (or not having) more-than-average numbers of nevi was calculated. The objective, physician-determined nevus count was used as the gold standard. Twenty-four (73%) melanoma, 27 (82%) nevus, and 14 (70%) control subjects correctly estimated their nevus counts to be either average or greater-than-average, with no significant differences between the groups (Table 4).

There were significant differences in the group incidences of atypical nevi (AN), with either a clinical or a histologic diagnosis in 22 (67%) melanoma, 33 (100%) nevus, and 1 (5%) control subject(s) (p=0.001); (Table 4).

The three groups reported similar incidences of a personal history of non-melanoma skin cancer, with positive self-reported histories in 5 (15%) melanoma, 5 (15%) nevus, and 4 (20%) control subjects (Table 4).

Nine (27%) melanoma, 14 (42%) nevus, and 3 (15%) control subjects reported a family history of malignant melanoma. Of these subjects, 6 (67%) melanoma, 10 (71%) nevus, and 0 control subjects had histologic documentation of that family history in the medical records (Table 4).

Sixteen (48%) melanoma, 11 (33%) nevus, and 4 (20%) control subjects related a family history of non-melanoma skin cancer; however, histologic documentation of these self-reported cases were not sought (Table 4).

III. Knowledge Scores on Individual Questions:

The baseline and follow-up questionnaires contained the same series of questions so as to enable the evaluation of subjects' baseline melanoma knowledge, as well as the improvement in that knowledge as a result of the intervention. The knowledge scores were analyzed by both group and clinic--i.e., for part of the analysis, the melanoma and nevus groups were combined into a single "Pigmented Lesion Clinic" or "PLC" group, which was then compared to the controls. Of note, our analyses (as shown in Tables 5 & 6) revealed that in many instances, the melanoma and nevus groups performed similarly on the questions--with at times even superior performance on individual questions among nevus

subjects. In these instances, an increased significance (lower p-value) emerged when the PLC group was compared to the controls, as opposed to when all the groups were compared to one another. Thus, when the melanoma and nevus groups had similar knowledge levels, the "PLC vs. control" data were reported; this also simplified the discussions. However, the tables for both methods of analysis are included.

Clinical Characteristics of Melanoma were emphasized in the video, because to be an effective (skin) self-examiner, each individual needs a working knowledge of melanoma signs and symptoms, so as to be able to identify a suspicious lesion and bring it to his/her dermatologist's attention. In the questionnaires, the subjects' awareness of the signs and symptoms of melanoma were explored through nine questions, which are discussed below.

- 66 (100%) PLCs and 18 (90%) controls correctly identified a change in nevus shape, color, or size as a potential marker for melanoma at baseline (p=0.01). The proportion with the correct answer--i.e., 63 (95%) and 19 (95%), respectively--was the same post-intervention (Table 6).

- 65 (98%) PLCs and 13 (65%) controls correctly identified abnormal shape as a potential sign of melanoma at baseline (p=0.001). This proportion was 64 (97%) and 16 (80%), respectively, at follow-up (p=0.009). There was a significantly higher proportion of controls (n=5 (25%)) who learned this fact post-intervention, as compared to the PLCs (n=1 (2%)); (p=0.001); (Table 6).

- 62 (94%) PLCs, as compared to 13 (65%) controls, correctly identified dark and variegated color as a potential marker for melanoma at baseline (p=0.001). This proportion was 64 (97%) and 17 (85%), respectively, at follow-up (p=0.046). The proportion of

controls (n=5 (25%)) who had learned this fact was significantly higher than that of the PLCs (n=4 (6%)); (p=0.016); (Table 6).

- Twenty-seven (82%) melanoma, 31 (94%) nevus, and 14 (70%) control subjects correctly identified an increasing nevus thickness as a potential sign of melanoma at baseline (p=0.021); the post-intervention proportion, at 27 (82%), 27 (82%), and 17 (85%), respectively, was not statistically significant. However, that 5 (25%) controls, as compared to 5 (15%) melanoma and 1 (3%) nevus subject(s) had learned this fact post-intervention, was significant (p=0.018); (Table 5).
- There were no significant differences at either baseline or follow-up in the proportion of subjects correctly identifying a non-healing scab/lesion as a potential melanoma: 54 (82%) PLCs and 17 (85%) controls at baseline, as compared to 54 (82%) PLCs and 19 (95%) controls at follow-up (Table 6).
- The analysis, by group, of bleeding as a sign of melanoma yielded significant differences at both baseline and follow-up. Twenty-four (73%) melanoma, 29 (88%) nevus, and 9 (45%) control subjects correctly identified bleeding as a potential marker for melanoma at baseline (p=0.001). The post-intervention proportion was 23 (70%), 31 (94%), and 14 (70%), respectively (p=0.021). The controls displayed not only a significantly higher mean change in knowledge [A], but also a significantly higher proportion of intervention-related incorrect-to-correct answers--n=6 (30%) controls as compared to no melanoma and 2 (6%) nevus subjects; (p=0.013); (Table 5).
- 46 (70%) PLCs and 9 (45%) controls correctly identified tenderness in a nevus as a potential sign of melanoma at baseline (p=0.045). This proportion--43 (64%) and 15 (75%),

respectively--was similar post-intervention. Here, the mean learning was significantly higher among the controls [A]. Also, a significantly higher proportion of controls (n=7 (35%)), as compared to PLCs (n=5 (8%)), changed from an incorrect to a correct answer post-intervention (p=0.002); (Table 6).

- The analysis, by group, of itching as a potential sign of melanoma yielded significant differences at both baseline and follow-up. Eighteen (55%) melanoma, 25 (76%) nevus, and 4 (20%) control subjects were knowledgeable of this fact at baseline (p=0.001). This proportion had increased to 19 (58%), 27 (82%), and 7 (35%), respectively, at follow-up (p=0.001). That 7 (35%) controls learned this fact, as compared to 8 (12%) PLCs, was statistically significant (p=0.019); (Table 6).
- The analysis, by group, of nevus "size" as a marker of melanoma also pinpointed significant differences between the groups at both baseline and follow-up. Twenty-three (70%) nevus, as compared to 13 (39%) melanoma and 3 (15%) controls correctly identified large size as a potential sign of melanoma at baseline (p=0.001). This proportion--15 (45%), 22 (67%), and 8 (40%), respectively--was of borderline significance post-intervention (p=0.045). The mean increase in knowledge was significantly higher among the controls [A], while the proportion of changes from an incorrect to a correct answer was also higher among the controls (n=5 (25%)), as compared to the melanoma (n=5 (15%)) and nevus (n=1 (3%)) groups (Table 5).

In this section, the PLC population most frequently missed questions about itchiness, tenderness, and size in melanoma. Meanwhile, the controls were most suspicious of *non-healing* lesions, or lesions *changing* in thickness, color, shape, or size, and least suspicious

of factors such as bleeding, tenderness, itching, size, abnormality in shape, and darkness and variegation in color. Also, the controls, with the worst baseline performances, displayed the most significant improvements in knowledge.

The Survey of Back Nevi was one of the points emphasized in the video. Given that a significant proportion of melanomas originate on the upper back--the most common site in males, and the second-most common site in females--it was important to raise participants' awareness of this difficult-to-self-examine area of the body, and educate them about alternative examination methods, such as using a mirror or a "buddy". In this respect, 33 (50%) PLCs as compared to 6 (30%) controls were aware of these examination methods at baseline. This proportion increased to 44 (67%) PLCs and 16 (80%) controls at follow-up, with the improvement in the control group [A] being statistically significantly higher than that of the PLCs [B]. Moreover, a significantly higher proportion of controls improved (n=11 (55%)) as compared to the PLCs (n=19 (29%)); (p=0.032); (Table 6).

Factual Knowledge of melanoma characteristics, risk factors, and treatment/cure was tested through six true-false questions, as discussed below.

- 64 (97%) PLCs as compared to 18 (90%) controls correctly identified a history of severe childhood sunburns as a significant risk factor for melanoma. This proportion--65 (98%) PLCs and 19 (95%) controls--was almost unchanged at follow-up (Table 6).
- At baseline, 62 (94%) PLCs as compared to 18 (90%) controls recognized that the only cure for melanoma is early surgical excision--not statistically significant. This proportion--64 (97%) and 17 (85%), respectively--had borderline significance at follow-up (p=0.046). However, the *change* from baseline to follow-up was not significant (Table 6).

- 57 (86%) PLCs as compared to 16 (80%) controls correctly identified a positive family history of melanoma as a significant risk factor for melanoma at baseline. At follow-up, this proportion had increased to 62 (94%) PLCs and 17 (85%) controls, respectively. However, the change was not statistically significant (Table 6).
- Almost identical proportions of PLCs and controls (n=51 (77%) and 15 (75%), respectively) recognized the statement "An itchy mole could not be melanoma" as false. There was almost no change in the proportions at follow-up--52 (79%) PLCs and 14 (70%) controls (Table 6).
- 58 (88%) PLC as compared to 10 (50%) controls recognized that a black and hairy mole could *not* be melanoma at baseline (p=0.001). This proportion was almost unchanged at follow-up--58 (88%) PLCs and 12 (60%) controls (p=0.005). However, that 5 (25%) controls, as opposed to only 3 (5%) PLCs learned this fact was statistically significant (p=0.006); (Table 6).
- Few subjects recognized that a disappearing mole could potentially represent melanoma, with very little improvement at follow-up--5 (15%) melanoma, 14 (42%) nevus, and 1 (5%) control subject(s) at baseline (p=0.001), and 9 (27%) melanoma, 16 (48%) nevus, and 3 (15%) controls at follow-up (p=0.009); (Table 5).

In this section, the most frequently missed question in all groups related to a disappearing nevus, as a possible natural history for a melanoma. Among controls, there was a high awareness of risk factors such as childhood sunburns and a positive family history, as well as of cure through early excision. These high levels of baseline knowledge among

controls probably reflects knowledge already disseminated through various public media, especially in view of the lack of significant change at follow-up.

Melanoma Risk Factors were also emphasized in the video. To raise an individual's awareness of melanoma risk factors is an empowering act, allowing that individual to assess his/her own relative risk of melanoma, which could in turn influence his/her implementation of skin self-examination recommendations and sun-safe practices. Thus, the groups' knowledge about melanoma risk factors was explored in the third section of the questionnaire, through ten clinical scenarios which were categorized as high- or low-risk.

- All groups well-recognized a changing nevus as a high-risk lesion--65 (98%) PLCs and 20 (100%) controls at both baseline and follow-up (Table 6).
- Both clinic populations were highly aware of such phenotypic characteristics as blue eyes and red hair as significant melanoma risk factors--65 (98%) PLCs and 19 (95%) controls at baseline, and 65 (98%) PLCs and 20 (100%) controls at follow-up (Table 6).
- Both clinic populations were highly aware of a positive family history as a risk factor--65 (98%) PLCs and 19 (95%) of controls at baseline, and 64 (97%) PLCs and 19 (95%) controls at follow-up (Table 6).
- Both clinic populations were highly aware of the predisposition to melanoma in an individual who burns, never tans--65 (98%) PLCs and 18 (90%) controls at baseline, as compared to *all* the subjects (100%) at follow-up (Table 6).
- Both clinic populations were highly aware of high melanoma risks in individuals with many nevi and both family and personal histories of melanoma--64 (97%) PLCs and 19 (95%) controls at baseline, as compared to *all* the subjects (100%) at follow-up (Table 6).

- Both clinic populations were highly aware of the greater susceptibility to melanoma in the presence of a positive personal history--64 (97%) PLCs and 19 (95%) controls at baseline, and 64 (97%) PLCs and 20 (100%) controls at follow-up (Table 6).

- At baseline, there was a statistically significant difference in the groups' awareness of the low melanoma risk in individuals with no nevi and dark hair--64 (97%) PLCs and 15 (75%) controls (p=0.002). However, *all* subjects (100%) answered the question correctly post-intervention. The controls had not only significantly higher mean changes in knowledge [A], but also significantly higher proportions of incorrect-to-correct answers post-intervention--5 (25%)) controls as compared to 2 (3%) PLCs (p=0.002); (Table 6).

- Both clinic populations were highly aware of the association between large and oddly-shaped nevi and melanoma--63 (95%) PLCs and 17 (85%) controls at baseline, and 64 (97%) PLCs and 18 (90%) controls at follow-up. The controls had higher proportions of incorrect-to-correct answers compared to the PLCs (3 (15%) versus 2 (3%), respectively); (p=0.046); (Table 6).

- Both clinic populations were highly aware of higher-than-average (> 50) nevus counts as melanoma risk factors--61 (92%) PLCs and 19 (95%) controls at baseline, and 62 (94%) PLCs and 18 (90%) controls at follow-up (Table 6).

- All groups appeared to be equally aware of the low-risk status of black skin--54 (82%) PLCs and 16 (80%)controls at baseline, and 55 (83%) PLCs and 16 (80%) controls at follow-up (Table 6).

In this section, which focused on melanoma risk factors, the performance of the three groups was nearly identical, with no significant change in most questions from baseline to

follow-up, even among the controls. This might perhaps reflect the recent media focus on melanoma as a serious public health hazard, with concomitant campaigns aimed at increasing the general awareness of those risk factors.

IV. Knowledge Scores as Stratified into Percentiles:

The knowledge scores were stratified into percentiles--defined as 0%, 1%-37%, 38%-74%, and 75%-100%--for each section of the questionnaire, as well as for the total questionnaire. Then, the analysis entailed determining the proportion of subjects in each group who responded correctly to greater than 75% of the tested material. The scores themselves were labeled as score1 (corresponding to the first part of the *pre*-questionnaire), score1a (corresponding to the first part of the *post*-questionnaire), etc., up to score4a (corresponding to the post-questionnaire *in its entirety*). [Again, the sections of the questionnaire examined the awareness of melanoma signs and symptoms, melanoma facts (in a true-false format), and melanoma risk factors, respectively.] With respect to almost all scores, there were significant differences in the proportions in each group attaining a greater-than-75% sum. Again, when the performances of the melanoma and nevus groups were similar, they were combined into a single PLC group in order to simplify the discussions.

- In the first section of the questionnaire--on the clinical characteristics of melanoma--37 (56%) PLCs, but only 1 (5%) control attained baseline sums greater than 75% (p=0.001). Of note, most controls (n=16 (80%)) correctly identified only 38%-74% of the characteristics. This difference (*by clinic*) was no longer significant at follow-up, with 34 (52%) PLCs and 5 (25%) controls attaining a sum greater than 75%. (In the analysis of the

percentiles *by group*, there was *reduced* significance, marked by a change in the p-value from 0.001 to 0.036); (Table 7).

- In the second section of the questionnaire--testing knowledge of various melanoma facts--29 (44%) PLCs, as compared to only 4 (20%) controls, attained baseline sums greater than 75% (p=0.027). Again, most controls (n=12 (60%)) were knowledgeable about only 38%-74% of those facts. At follow-up, these proportions had increased in both populations, with 39 (59%) PLCs and 6 (30%) controls attaining sums greater than 75% of the total (p=0.005). However, most controls (n=11 (55%)) still scored in the 38%-74% range (Table 7).
- The third section of the questionnaire--examining knowledge of melanoma risk factors--revealed no significant differences between the groups, except for a difference of borderline significance between the PLC and control baseline sums. Thus, at baseline, 60 (91%) PLCs as compared to 14 (70%) controls attained sums greater than 75% (p=0.052); this proportion was 61 (92%) and 18 (90%), respectively, post-intervention. In both populations, most subjects were highly aware of potential melanoma risk factors (Table 7).
- The baseline and follow-up total sums were significantly different between the three groups. The nevus group had the highest proportions of greater-than-75% sums (at both baseline and follow-up). Meanwhile, despite having the lowest proportions of greater-than-75% sums at baseline, the control group resembled the melanoma group in its post-intervention performance: at baseline, 21 (64%) melanoma and 27 (82%) nevus, but only 6 (30%) controls attained a sum greater than 75% (p=0.001); the post-intervention proportions were 20 (61%) melanoma, 29 (88%) nevus, and 13 (65%) controls (p=0.04). Of

note, most controls knew 38%-74% of the material at baseline (n=13 (65%)), but greater than 75% of the material at follow-up (n=13 (65%)); (Table 7).

V. Baseline, Follow-up, and Improvement in Knowledge Scores by Group, Demographic & Phenotypic Characteristics, and Melanoma Risk Factors:

The means for the baseline and follow-up knowledge scores, along with the mean changes, were analyzed with respect to the defined demographic, phenotypic, and high risk variables, so as to determine which ones relate to significant increase in knowledge. This is especially important in educational interventions, where the main objective is to change knowledge, and therefore, behavior. Therefore, a deeper understanding of the factors affecting motivation could benefit the intervention's design, tailoring the strategies used to each sub-population's needs or psychobiological framework.

For the analysis, the knowledge variables were modeled after the questionnaire format, with score1 denoting the section on the clinical characteristics of melanoma, score2 delineating the section on melanoma facts, score3 designating the section on melanoma risk factors, and score4 representing the total sum. The analysis involved a comparison of the means by both group and clinic, and then, within each group, by the various variables. The same approach was used to determine the proportion of subjects in each group whose scores increased.

- The analysis of knowledge scores by group revealed the melanoma and nevus groups to be highly knowledgeable about melanoma, with usually significantly higher scores as compared to the controls. However, the controls had significantly higher improvements in knowledge. At baseline, the melanoma and nevus groups' score1 means were 6.9 [A] and

7.8 [A], respectively, as compared to 5.0 [B] for the controls. (The classification into groups A vs. B vs. C indicates the presence of statistically significant differences). At follow-up, given the significant increase in knowledge among the controls, the performance of the melanoma group resembled that of the controls--with score1 means of 6.8 [B] and 6.6 [B] for the melanomas and the controls, respectively, as compared 7.8 [A] for the nevus group. The mean change in score1 was significantly higher among the controls, at 1.6 [A], as compared to the almost unchanged scores of the melanoma and nevus groups, at -0.1 [B] and 0 [B], respectively. Moreover, 13 (65%) controls had increased score1s, as compared to 11 (33%) melanoma and 6 (18%) nevus subjects (p=0.001); (Table 8a).

Again, with respect to score2, (melanoma facts), the melanoma and nevus groups had significantly higher means at both baseline and follow-up: 5.1 [A], 5.3 [A], and 4.2 [B] at baseline, and 5.6 [A], 5.6 [A], and 4.9 [B] at follow-up, for the melanoma, nevus, and control groups, respectively. There were no significant differences among the groups in either the mean change, or the proportion of subjects with increased score2s, even though both were higher among controls; (Table 8a).

With respect to score3 (risk factors), there were no significant differences among the groups in the either baseline or follow-up means, the degree of change, or the proportion of subjects with increased scores. However, the melanoma and nevus groups tended to have higher means, while the controls had the highest mean change as well the greatest proportion of subjects with increased scores; (Table 8a).

The score4 means were again significantly higher in the melanoma and nevus groups, as compared to the controls, at 21.6 [A], 22.6 [A] as compared to 18.3 [B] at baseline, and

22.0 [B], 23.1 [A], as compared to 21.1 [C] at follow-up, for the melanoma, nevus, and controls, respectively. The controls had the significantly higher mean improvements, at 2.8 [A], as compared to 0.4 [B] and 0.5 [B] for the melanoma and nevus groups, respectively. Even though a greater proportion of controls had increased scores, this was not significant; (Table 8a).

- The analysis of knowledge as a function of sex *within* each group--comprised of the melanoma, nevus, control, PLC, and *all* (the study subjects) groups--revealed generally higher knowledge scores among females, as compared to males. However, these differences were not statistically significant. Likewise, the tendencies toward higher *proportions* of females improving their scores, as well as greater *degrees* of improvement among females, were not statistically significant. The only exceptions involved follow-up score2s for *all* subjects (5.6 [A] for females and 5.1 [B] for males), follow-up score4s for PLC subjects (23.0 [A] for females and 21.5 [B] for males), and the follow-up score4s for *all* subjects (22.6 [A] for females and 21.2 [B] for males); (Table 8b).

- The analysis of knowledge as a function of age revealed different patterns for different groups. In the melanoma group, there was generally greater knowledge with increasing age, but greater improvements in the youngest age group. However, none of the differences were statistically significant. Nor were there significant differences between the age groups in the proportion of melanoma patients with increased scores (Table 8c).

In the nevus group, by comparison, the younger age groups had significantly higher knowledge means as compared to the oldest one. The score1 means were 8.2 [A], 7.8 [A], and 6.2 [B] at both baseline and follow-up, for the 18-35, 36-50, and 51-85 age groups,

respectively. The score2s were 5.8 [A], 5.2 [B], and 4.2 [C] at baseline, and 5.8 [A], 5.8 [A], and 4.4 [B] at follow-up, for the 18-35, 36-50, and 51-85 age groups, respectively. The score3s were 9.8 [A], 9.7 [A], and 8.4 [B] at baseline, and 9.9 [A], 9.8 [A], and 9.0 [B] at follow-up for the 18-35, 36-50, and 51-85 age categories, respectively. Finally, the score4s were 23.8 [A], 22.7 [A], and 18.8 [B] at baseline, and 23.9 [A], 23.5 [A], and 19.6 [B] at follow-up for the 18-35, 36-50, and 51-85 age categories, respectively. Concurrently, the older age groups tended to have greater improvements as well as higher proportions of individuals with increased scores, although none of these differences were significant (Table 8c).

The controls displayed yet another pattern, with the highest means in the youngest and/or the oldest age groups. The score1s were 6.7 [A], 3.5 [C], and 5.1 [B] at baseline, and 7.0 [A], 4.8 [B], and 7.1 [A] at follow-up, for the 18-35, 36-50, and 51-85 age groups, respectively. The score2s followed the same pattern, but were not significant. For score3, only the follow-up means were significant, at 9.0 [B], 8.8 [C], and 9.9 [A], for the three respective age groups. Finally, the score4 means were 19.7 [A], 14.3 [C], and 19.2 [B] at baseline, and 21.0 [B], 18.3 [C], and 21.9 [A] at follow-up, for the three respective age groups. Again, even though the middle age group had both greater proportions of individuals with increased scores, and higher mean improvements, none of the differences were statistically significant (Table 8c).

In the PLC group, the highest means were observed in the youngest age group, with statistically significant differences in the follow-up score2s only, at 5.6 [B], 5.8 [A], and 5.3 [C] for the 18-35, 36-50, and 51-85 age groups, respectively. Even the two older age groups

had higher degrees of improvements, as well higher proportions of individuals with increased scores, none of the differences were significant (Table 8c).

When *all* the subjects were combined, the highest mean scores were seen in the youngest age group, however with only a few significant differences: baseline score1s, at 7.5 [A], 6.8 [B], and 6.3 [C], and baseline score4s, at 22.4 [A], 21.0 [B], 20.4 [C] for the respective age categories. Like the PLCs, even though the two older age groups had greater degrees of improvement and higher proportions of individuals with increased scores, none of the differences were significant (Table 8c).

- The analysis of knowledge as a function of the phenotypic marker *hair color* revealed different patterns within each group. The overall trend *within* both the melanoma and nevus groups--and by extension, the PLC group--was for higher baseline and follow-up means (for all the scores) for subjects with light brown and/or brown/black hair. This was significant only with respect to the melanoma baseline score2s, where subjects with blond/red and light brown hair had significantly higher means--at 5.3 [A] and 5.5 [A], respectively--as compared to those with brown/black hair--at 4.4 [B]. The mean change was not higher for any predominant hair color. Also, those with blond/red and light brown hair had the greatest proportions of individuals with increased scores. In the melanoma group, 7 (58%) blond/red haired, as compared to 3 (27%) and 1 (10%) light brown and brown/black haired individuals, respectively, had improved score1s ($p=0.017$); only 3 (25%) blond/red haired, as compared to 5 (45%) and 7 (70%) light brown and brown/black haired individuals, respectively, had increased score2s ($p=0.038$); and 9 (75%) blond/red haired, as compared to 6 (55%) and 3 (30%) light brown and brown/black haired subjects, respectively, had improved score4s

(p=0.038). In the PLC population, 9 (45%) blond/red haired, as compared to 4 (27%), and 4 (13%) light brown and brown/black haired subjects, respectively, had increased score1s (p=0.011); and 14 (70%) blond/red haired, as compared to 8 (53%), and 12 (39%) light brown and brown/black haired subjects, respectively, had increased score4s (p=0.03); (Table 8d).

Among controls and *all* the subjects, the follow-up means were consistently highest in those with blond/red hair, sometimes with statistical significance. Among controls, the follow-up score1s were 8.5 [A] as compared to 6.2 [B] and 6.1 [B], and the follow-up score4s were 24.3 [A], as compared to 20.4 [B] and 20.2 [B], for subjects with blond/red, light brown, and brown/black hair, respectively. Those with blond/red hair also had the highest proportion of subjects with improved scores--except in relation to score3, where their *perfect* mean score of 10 (out of 10) at both baseline and follow-up precluded further improvement. These difference were significant in regards to score1, only, where 4 (100%) blond/red haired, as compared to 4 (80%) and 5 (45%) light brown and brown/black haired subjects, respectively, had increased scores. This trend also applied when *all* the subjects were combined together. In the *all* group, 13 (54%) blond/red haired, as compared to 8 (40%), and 9 (21%) light brown and brown/black haired subjects, respectively, had increased score1s (p=0.007); and 13 (75%) blond/red haired, as compared to 12 (60%), and 18 (43%) light brown and brown/black haired subjects, respectively, had increased score4s (p=0.011); (Table 8d).

- The analysis of knowledge as a function of the phenotypic variable *eye color* yielded less significant results as compared to the variable *hair color*. *Within* the melanoma and

nevus--and by extension, the PLC--groups, there was a tendency toward higher means among, first, light/dark brown eyed, and then, hazel/grey eyed individuals. These differences were significant only in the nevus group's baseline score2s, where light/dark brown eyed subjects had the highest mean, at 5.6 [A], as compared to 5.3 [B] and 4.4 [C] for those with blue/green and hazel/grey eyes, respectively. The mean improvement in knowledge, and the proportion of individuals with increased scores, also tended to be highest in those with light/dark brown or hazel/grey eyes. In the PLC group, the mean change was greatest among hazel/grey eyed subjects, at 1.0 [A], as compared to 0.2 [C] and 0.3 [B] for blue/green and light/dark brown eyed ones, respectively (Table 8e).

Among controls, there were higher means and degrees of change in association with either blue/green or hazel/grey eyes. These were significant in the control follow-up score2s, where blue/green eyed and hazel/grey eyed subjects had means of 5.6 [A] and 5.3 [A], respectively, as compared to 4.3 [C] for those with light/dark brown eyes. Also, among controls, the mean score2 increase was highest among blue/green eyed subjects, at 1.5 [A], as compared to 0 [B] and 0.5 [B] for hazel/grey and light/dark brown eyed subjects. Controls with blue/green eyes also tended to have the greatest proportion of increased scores, although these were not significant (Table 8e).

Among *all* the subjects, the follow-up means, the mean increases, and the proportion of subjects with increased scores tended to be highest in hazel/grey and light/dark brown eyed individuals, however without any statistical significance (Table 8e)

- The analysis of knowledge with respect to the self-reported skin reaction to acute sun-exposure revealed almost no significant differences *within* groups. Melanoma, control, and

PLC subjects with a tendency to burn had, in general, higher baseline and follow-up means, as well as greater proportions of subjects with increased scores. None of these differences were significant, however, other than the follow-up score2s in the control group, where those who burned had significantly higher means, at 5.6 [A], as compared to those who tanned, at 4.6 [B]. In contrast, among nevus and *all* subjects, those who tanned had a tendency toward higher means and greater proportions of knowledge increase (Table 8f).

- The analysis of knowledge as a function of the self-reported skin reaction to chronic sun-exposure revealed more consistent, though still mostly insignificant, trends. In all the subject groupings and with respect to all scores (1-4), those who reported to freckle or develop a mild tan after several peelings tended have higher means, as compared to those who reported a moderate to deep tan. Among controls, the baseline score4s were significantly higher among frecklers, at 20.4 [A], as compared to the tanners, at 18.1 [B]. Also, in the *all* group, the baseline score3s and score4s were significantly higher among the frecklers--at 9.9 [A] and 22.3 [A], respectively--as compared to the tanners--at 9.3 [B] and 21.0 [B], respectively. In contrast, the tanners tended to have higher proportions of subjects with increased scores throughout, other than in score1. This might be related to the higher baseline and follow-up scores in the frecklers, which left greater room for improvement among the tanners. However, none of these were significant. Also, the mean change in the scores was usually higher among tanners, except again for score1, where the frecklers had greater mean improvements in knowledge. However, none of these differences were significant, other than the mean change in score3 in the *all* group--0 [B] for frecklers and 0.2

[A] for tanners. In this case, the frecklers had nearly perfect baseline and follow-up scores, which might contribute to their minimal improvement (Table 8g).

- The analysis of knowledge as a function of the objective, physician-determined nevus count revealed very consistent patterns *within* all the groups. Although not always significant, the highest means were usually observed among those with higher nevus counts (34-66 and greater than 67). The degree of improvement did not follow a specific pattern, being at times higher among those with 0-33 nevi, and at other times, higher among those with greater than 34 nevi. In contrast, in most cases--except for score4--those with 0-33 nevi had the highest proportion of subjects with increased scores (Table 8h).

None of these differences were significant in the melanoma group. In the nevus group, there were significant differences in the follow-up score1s and score4s, at 8.4 [A] and 8.4 [A], as compared to 6.6 [B] (for the score1s), and 23.8 [A] and 23.9 [A] as compared to 21.7 [B] (for the score4s), for those with 34-66, greater than 67, and 0-33 nevi, respectively. In the nevus group, the mean changes in score1 and score2 were also significant at 0.9 [A] and -0.1 [B] as compared to -0.8 [C] (for the score1s), and -0.2 [C] and 0.1 [B] as compared to 0.9 [A] (for the score2s) in those with 34-66, greater than 67, and 0-33 nevi, respectively (Table 8h).

In the PLC group, both the baseline and the follow-up score1s and score4s were significantly higher among those with greater than 34 nevi. The actual means were 7.4 [B] and 8.3 [A] as compared to 6.9 [C] (for the baseline score1s), 7.9 [A] and 8.2 [A] as compared to 6.5 [B] (for the follow-up score1s), 22.7 [B] and 23.3 [A] as compared to 21.2 [C] (for the baseline score4s), and finally, 23.5 [A] and 23.5 [A] as compared to 21.5 [B] (for

the follow-up score4s), for those with 34-66, greater than 67, and 0-33 nevi, respectively. Also, in the PLC group, the mean score1 increase was significantly higher among those with greater nevus counts--0.5 [A] and -0.2 [B] as compared to -0.4 [C], for those with 34-66, greater than 67, and 0-33 nevi, respectively (Table 8h).

In the *all* group, as well, both the baseline and follow-up score1s and score4s were significantly higher among those with greater than 34 nevi. The actual means were 7.4 [A] and 8.3 [A] as compared to 6.2 [B] (for the baseline score1s), 7.9 [A] and 8.2 [A] as compared to 6.5 [B] (for the follow-up score1s), 22.7 [A] and 23.3 [A] as compared to 20.1 [B] (for baseline score4s), and finally, 23.5 [A] and 23.5 [A] as compared to 21.2 [B] (for follow-up score4s), for those with 34-66, greater than 67, and 0-33 nevi, respectively. In contrast, in the *all* group, 27 (53%) subjects with 0-33 nevi, as compared to 8 (36%) and 3 (25%) of those with 34-66 and greater than 67 nevi, respectively, had increased score1s ($p=0.051$--borderline significance); (Table 8h).

- The analysis of knowledge as a function of the subjective, self-reported nevus count revealed fewer significant differences *within* the groups. In general, for all the groups, those with reportedly 34-66 nevi most often had the highest knowledge means. Those who reported greater than 67 nevi had the highest means second most frequently. Also, those who recounted greater than 67 nevi predominantly had the greatest mean improvements. Moreover, those who believed in having either 34-66 or greater than 67 nevi tended to have the largest proportion of subjects with increased scores. In most instances, however, these differences were not significant (Table 8i).

In the melanoma group, mean score2 improvements were significantly higher in those reporting greater than 67 nevi, at 1.1 [A], as compared to 0.4 [B] and 0 [C] for those reporting 0-33 and 34-66 nevi, respectively. In the nevus group, the mean score1 improvements were significantly higher in those reporting greater than 67 nevi (0.7 [A]), as compared to those reporting 0-33 (-0.8 [C]) and 34-66 nevi (-0.3 [B]). Also, in the nevus group, those reporting greater than 67 nevi had the highest proportions of increased score1s and score4s: 5 (31%) as compared to 0 and 1 (25%) for score1, (p=0.034) and 11 (69%) as compared to 4 (31%) and 1 (25%) for score4, (p=0.041) in subjects with reportedly greater than 67, 0-33, and 34-66 nevi, respectively (Table 8i).

- The analysis of knowledge as a function of the self-reported impression of "having more-than-average numbers of nevi" yielded *no* significant differences between those who held such a belief, and those who either did not have such a belief or did not know. In the melanoma group, there was no prevailing pattern, in that the means were at times higher among the "believers", and at other times higher in the other groups. However, the total score4 means were highest among the "believers" at baseline, and highest among the "uncertain" ones at follow-up: those who were "uncertain" also had the greatest mean score4 improvements, while the "believers" had the greatest proportion of individuals with increased score4s. However, none of these differences were significant (Table 8j).

In the nevus group, the highest baseline and follow-up scores were among those who were "uncertain" about having greater than average nevus counts, while the greatest mean improvements and the greatest proportion of individuals with increased scores were

predominantly in the "believer" and "uncertain" categories. Again, none of these differences were significant (Table 8j).

Among the controls, the "believers" tended *not* to have the highest scores, at either baseline or follow-up, but they *did* have the highest mean improvements, as well as the greatest proportion of subjects with increased scores. None of these trends were significant (Table 8j).

In the PLC group, the greatest mean scores, at both baseline and follow-up, and the greatest mean improvements tended to occur among those who were "uncertain". However, greater proportions of subjects who believed in having more nevi than average had increased score4s. The *all* group displayed a similar pattern (Table 8j).

- The analysis of knowledge with respect to the personal history of atypical nevi (AN) revealed some statistically significant results. In the melanoma group, those with AN tended to have the highest baseline and follow-up knowledge scores, along with the highest mean improvements and the greatest proportion of individuals with increased scores. However, none of these differences were significant (Table 8k).

The results for the nevus group could not be analyzed, given that all subjects in this group had a positive history of AN (Table 8k).

In the control group, there were significant differences between the *one* subject with a documented atypical nevus, and the rest who lacked such a history. Overall, those with a negative AN history had higher knowledge scores at both baseline and follow-up; however, the subject with positive history of AN had the greatest increase in knowledge. These differences were significant with respect to score1 means (0 [B] and 5.3 [A] at baseline, and

3.0 [B] and 6.8 [A] at follow-up, for those *with* and those *without* AN, respectively), and baseline score3s and score4s (6.0 [B] and 9.2 [A] (score3s) and 8.0 [B] and 18.8 [A] (score4s), for those *with* and those *without* AN, respectively). Also, the mean score3 improvements were also significantly higher in the subject *with* positive AN (3.0 [A]), as compared to those *without* AN (0.4 [B]); (Table 8k).

In the PLC group, the follow-up score1s and score4s were significantly higher in those *without* AN (at 7.5 [A] and 22.8 [A], respectively), as compared to the subject *with* AN (at 6.1 [B] and 21.0 [B], respectively); (Table 8k).

In the *all* group, again, those *with* AN had significantly higher baseline and follow-up means as compared to those *without* AN. The actual means were 7.4 [A] and 5.7 [B] (for the baseline score1s), 7.4 [A] and 6.5 [B] (for the follow-up score1s), 5.2 [A] and 4.6 [B] (for the baseline score2s), 5.6 [A] and 5.1 [B] (for the follow-up score2s), 22.1 [A] and 19.5 [B] (for the baseline score4s), and finally, 22.7 [A] and 21.2 [B] (for the follow-up score4s) for those *with* AN those *without* AN, respectively. Also, those *without* AN had the greatest mean improvements, significantly so with respect to only score2 (0.1 [B] for those *with* AN, as compared to 0.8 [A] for those *without* AN). Also, those *without* AN (n=15 (50%)) had significantly higher proportions of subjects with increased scores, as compared to those *with* AN (n=15 (28%)); (p=0.032); (Table 8k).

- The analysis of knowledge as a function of a personal history of non-melanoma skin cancer (NMSC) yielded some significant differences. In the melanoma group, those with a positive NMSC history had higher (baseline and follow-up) means, as well as greater score

increases and higher proportions of subject with increased scores. However, none of these differences were significant (Table 8l).

In the nevus group, by contrast, the highest means occurred in those with a negative NMSC history, with significant differences in the baseline score1s (6.5 [B] and 8.0 [A] for those *with* and those *without* prior NMSCs, respectively), and baseline score3s (8.7 [B] and 9.7 [A] for those *with* and those *without* prior NMSCs, respectively). Meanwhile, those *with* prior NMSC had not only greater degrees of score increase, but also greater proportions of individuals with increased scores. Notably, 3 (50%) subjects *with*, as compared to 3 (11%) subjects *without* prior NMSC had increased score1s (p=0.028); (Table 8l).

In the control group, those *with* prior NMSCs tended toward higher means at both baseline and follow-up, while those *without* prior NMSCs had higher proportions of subjects with increased scores. However, none of the differences were significant (Table 8l).

In the PLC group, those *without* prior NMSC tended to have higher baseline and follow-up means, but those *with* prior NMSC had greater degrees of improvement. Also, those *with* prior NMSCs had higher proportions of subjects with increased scores. Namely, 6 (50%) subjects *with* prior NMSC, as opposed to only 11 (20%) subjects without prior NMSC, had increased score1s (p=0.035); (Table 8l).

In the *all* group, those *with* prior NMSC usually had higher means at both baseline and follow-up. There were no specific patterns to describe the mean score increases or the proportion of subjects with increased scores. None of the differences were significant (Table 8l).

- The analysis of knowledge as a function of the family history of melanoma also revealed some significant differences. Among melanoma subjects, those who were "uncertain" had the highest baseline and follow-up means--significantly so in the follow-up score2s (6.5 [A] for those who were "uncertain", as compared to 7.4 [B] and 6.8 [C] for those with a positive and negative family history, respectively). The score3 trends were an exception to this rule, given that those with a positive family history had higher score3 means--significantly so at baseline (9.8 [A], 9.5 [A], and 8.5 [B] for those reporting positive, negative, and "uncertain" family histories of melanoma, respectively). Also, those who were "uncertain" of a family history of melanoma had higher degrees of score increase, as well as higher proportions of subjects with improved scores. However, none were significant (Table 8m).

In the nevus group, the highest means and the greatest score increases occurred in those with either a positive or an "uncertain" family history of melanoma; however, the overall sum (score4) was highest in those *with* a positive family history. By comparison, those without a family history of melanoma had higher proportions of subjects with increased knowledge. None of these findings were significant (Table 8m).

Among the controls, those *with* a family history of melanoma *never* had higher baseline knowledge scores, but at times, had greater degrees of improvement, resulting in higher follow-up scores. The overall score (score4) was, nonetheless, higher in those who were "uncertain". The follow-up score1s were significantly higher in "uncertain" subjects, at 9.0 [A], as compared to 6.0 [B] and 6.2 [B] for those *with* and *without* family histories,

respectively. Also, subjects *with* a family history of melanoma, or "uncertain' of it, had higher proportions of individuals with increased knowledge (Table 8m).

Likewise, in the PLC group, the highest means, the greatest mean improvements, and the greatest proportions of subjects with increased knowledge were associated with either positive or "uncertain" family histories of melanoma. However, none of the differences were significant. The same pattern also prevailed in the *all* group, in which the "uncertain" subjects had significantly higher follow-up score1s (8.3 [A], as compared to 7.4 [B] and 6.8 [C] in those *with* and *without* such family histories, respectively), mean improvements (1.6 [A], as compared to 0.1 [B] and 0.2 [B] in those *with* and *without* such family histories, respectively), and proportions of subjects with increased score1s (6 (24%), as compared to 6 (24%) and 18 (35%) of those reporting positive and negative family histories, respectively); (p=0.025); (Table 8m).

- The analysis of knowledge as a function of the family history of non-melanoma skin cancers (NMSC) revealed, again, unexpected trends. In the melanoma group, the highest (baseline and follow-up) scores, mean improvements, and proportions of subjects with improved scores were observed in those reporting an "uncertain" family history of NMSC. There were significant differences in the mean score1s (8.2 [A], as compared to 6.8 [B] and 6.3 [C] at baseline, and 8.7 [A], as compared to 6.6 [B] and 6.0 [B] at follow-up, for those reporting "uncertain", positive, and negative family histories of NMSC, respectively), follow-up score2s (6.2 [A], as compared to 5.6 [A] and 5.4 [A] for those reporting "uncertain", positive, and negative family histories of NMSC, respectively), and follow-up score4s (24.7 [A], as compared to 21.9 [B] and 20.6 [B], for those reporting an "uncertain",

positive, and negative family histories of NMSC, respectively). The mean improvements for score3 and score4 were significantly higher in those with an "uncertain" and/or positive family history of NMSC (score1 improvements of 0.3 [A] and 0.3 [A] for those with "uncertain" and positive family histories of NMSC, as compared to -0.5 [B] for those reporting no such family history, and score4 improvements of 1.5 [A], as compared to 0.5 [B] and -0.3 [C], for those reporting "uncertain", positive, and negative family histories of NMSC, respectively). Finally, all 6 (100%) subjects with an "uncertain" family history of NMSC, as compared to 10 (63%) and 2 (18%) of those *with* and *without* such a history, respectively, had increased score4s (p=0.038); (Table 8n).

In the nevus group, the patterns were not as consistent, given that the means were mostly, but not always, higher in those with an "uncertain" history. All these findings were nonsignificant, other than the baseline score1s, which were significantly higher in those *without* a family history of NMSC (at 8.1 [A]), as compared to those *with* such a history or "uncertain" of it (at 7.5 [B] and 6.3 [C], respectively). The mean changes in knowledge were also usually higher in those with an "uncertain" family history of NMSC, as compared to the others. These were nonsignificant, except for the mean score1 changes, which were significantly higher (at 1.3 [A]) in those with an "uncertain" history, as compared to 0.5 [B] and -0.5 [C] for those with positive and negative family histories, respectively. The highest proportions of increased scores also usually occurred in those with an "uncertain" family history of NMSC, however without statistical significance (Table 8n).

Among the controls, in contrast, those with a positive family history of NMSC tended to have the highest knowledge means, the greatest mean knowledge improvements, and the

greatest proportions of subjects with increased scores. These differences were only significant in relation to follow-up score2s (at 6.3 [A] for those with a positive family history, as compared to 4.8 [B] and 4.2 [B] for those with negative and "uncertain" family histories, respectively); (Table 8n).

Among PLCs, again, those with an "uncertain" family history of NMSC usually had the highest means, mean improvements, and proportions of individuals with increased scores. These were significant only for the mean improvements in score1 and score4--0.8 [A] as compared to 0 [B] and -0.4 [B] (for the score1s) and 1.4 [A] as compared to 0.6 [B] and 0 [C] (for the score4s) for those with "uncertain", positive, and negative family histories of NMSC, respectively. Finally, all 9 (100%) subjects with an "uncertain" family history, as compared to only 15 (56%) and 10 (33%) subjects with positive and negative such histories, respectively, had increased score4s (p=0.006); (Table 8n).

In the *all* group, those with either positive or "uncertain" family histories of NMSC had higher means, mean improvements, and proportions of subjects with increased scores. The score1 improvements (1.1 [A] as compared to 0.3 [B] and 0 [C]), and the proportion of subjects with increased score1s and score4s (9 (60%) as compared to 12 (39%) and 9 (23%) for the score1s, (p=0.047), and 13 (87%) as compared to 19 (61%) and 16 (40%) for the score4s (p=0.02) for those with "uncertain", positive, and negative such histories, respectively) were statistically significant (Table 8n).

- There were *no* significant differences in knowledge between new and return pigmented lesion clinic patients. The return patients had higher baseline and follow-up score1s, while the new patients had greater degrees and proportions of subjects with score1

improvements. The new patients, however, had higher baseline and follow-up score2s and score4s, along with greater degrees and proportions of subjects with improvements. Finally, the melanoma and PLC (baseline and follow-up) score4s were higher among return patients, while this was reversed in the nevus group--higher (baseline and follow-up) score4s among new patients. However, in all three groups, the new patients had higher degrees and proportions of improved score4s (Table 8o).

VI. Skin Self-Examination Practices as a Function of Demographic and Phenotypic Characteristics and of Melanoma Risk Factors:

Skin self-examination (SSE) was analyzed first, by comparing the group SSEs to one another, and second, by analyzing the variations in SSE *within* each group as a function of demographic, phenotypic, and high-risk variables. Again, the *subjects* were analyzed by both group and clinic, while the *SSEs* were analyzed in the three different steps to be explained. The first step entailed comparing the number of times per year that each subject (in each group) reported to examine his/her skin--from never (0 times per year) to everyday (365 times per year)--and determining what variables, if any, promoted change in each group. In the second step, given that at the two extremes, *no* skin self-examinations and *daily* ones were both judged to be ineffective at detecting new melanoma lesions, an "optimal" skin self-examination was defined to range from once per month to once every 4 months. Thus, the second step entailed analyzing the proportion of "optimal" SSEs *within* each group, and determining what variables, if any, promoted more "optimal" SSEs. The third step entailed determining differences (*between* and *within* groups) in the proportion of

subjects who *never* performed SSEs, and what variables, if any, increased the likelihood of change.

- First, the SSE practices were compared by both group and clinic population. The SSE means (per year) were significantly different between the groups at both baseline and follow-up, at 49.6 [B], 18.0 [C], and 77.2 [A] at baseline, and 52.3 [B], 15.2 [C], and 76.8 [A] at follow-up, for the melanoma, nevus, and control groups, respectively. (As observed, the SSE (per year) means are significantly higher among the controls, but closest to "optimal" in the nevus group). The mean change in SSE was minimal, with no significant differences in either the degree of change, or the proportion of increased SSEs between the groups. The results were similar for the PLC versus control populations (Table 9a).

Significantly higher proportions of controls *never* performed SSEs at both baseline and follow-up: 4 (12%) melanoma and 3 (9%) nevus, as compared to 6 (30%) controls at baseline (p=0.054), and 4 (12%) melanoma, 1 (3%) nevus, as compared to 5 (25%) controls at follow-up (p=0.017). (The *same* melanoma subjects performed *no* SSEs at both baseline and follow-up). This trend also pertains to the comparison of the PLC and control populations, where 7 (11%) PLCs as compared to 6 (30%) controls at baseline (p=0.035), and 5 (8%) PLCs as compared to 5 (25%) controls at follow-up (p=0.034) *never* performed SSEs. Also, higher proportions of melanoma, nevus--and by extension, PLC--subjects performed "optimal" SSEs at both baseline and follow-up; however, these differences were not significant. Also, there were no significant differences between the groups or clinics in the proportion of subjects with either change from "non-optimal" to "optimal" SSEs, or change from *no* SSEs to +SEEs (Table 9a).

- The analysis of SSE practices as a function of sex yielded almost no significant differences. In general, except in the controls, the males had higher SSE means (per year), but the female SSE means were closer to "optimal". Only the nevus follow-up SSEs were significantly different, at 12 [B] for females and 25 [A] for males. None of the mean SSE changes were significant *within* the various groups (Table 9b).

Meanwhile, except in the nevus group where *all* males reported +SSEs, lower proportions of females performed *no* SSEs at follow-up (the baseline proportions were mixed). None of these differences were significant. Nor were the changes from *no* SSEs to +SSEs significant (Table 9b).

In the melanoma and control groups, more females than males performed "optimal" SSEs, while in the control and *all* groups, more males than females performed "optimal" SSEs, at both baseline and follow-up. Only the controls' baseline "optimal" SSEs were significant, where no females, as compared to 3 (50%) males performed "optimal" SSEs ($p=0.005$). None of the changes from "non-optimal" to "optimal" SSEs were significant (Table 9b).

- There were *no* significant age-related differences in SSE practices *within* the groups. The 18-35 year old melanoma and PLC subjects, and the greater-than-36 year old nevus subjects had lower, but closer to "optimal", SSE means (per year); however, this was not significant. Also, the degrees and proportions of SSE increase *within* the groups were not statistically significant (Table 9c).

In all the groups, at both baseline and follow-up, those in the 36-50 year old age group had the lowest proportions of *no* SSEs. Neither these, nor the changes therein, were statistically significant in any of the groups (Table 9c).

Also, there were no significant age-related differences in the proportions of "optimal" SSEs, or changes therein, *within* the groups. Nonetheless, in general, the greatest proportions of "optimal" SSEs, and changes from "non-optimal" to "optimal" SSEs, were observed in the 18-35 or the 51-85 age groups (Table 9c).

- The analysis of SSE practices as a function of hair color revealed few significant differences. In all the groups, blond/red or light brown haired subjects had higher SSE means (per year). However, the SSE means (per year) were close to "optimal" in only the nevus group. In all the groups, higher proportions of subjects with blond/red or brown/black hair had increased SSEs; however, this was not significant (Table 9d).

In all the groups, at baseline, higher proportions of subjects with either blond/red or brown/black hair reported performing *no* SSEs. These had all decreased by follow-up, except among the controls. Significantly, in the melanoma group, at both baseline and follow-up, *no* blond/red, as compared to 1 (9%) light brown and 3 (30%) brown/black haired subjects reported to *never* perform SSEs ($p=0.036$). *Within* the groups, there were no significant differences relating to the color of the hair in the proportion of subjects with change from *no* SSEs to +SSEs (Table 9d).

At baseline, in all but the melanoma group, those with light brown hair had the highest proportions of "optimal" SSEs. At follow-up, in all but the controls, those with either blond/red or light brown hair had the highest proportions of "optimal" SSEs. These

differences were significant in the PLC group, where 13 (65%) blond/red and 9 (60%) light brown, as compared to 12 (39%) brown/black haired subjects had follow-up "optimal" SSEs (p=0.059, borderline significance). Also, in all but the control group, those with blond/red hair had higher proportions of individuals with change from "non-optimal" to "optimal" SSEs. This was significant in the PLC group, where 10 (50%) blond/red, as compared to 3 (20%) light brown and 7 (23%) brown/black haired subjects changed from "non-optimal" to "optimal" SSEs (p=0.051); (Table 9d).

- The analysis of SSE with respect to eye color yielded virtually no significant trends. In all groups but the *all* group, those with either hazel/grey or light/dark brown eyes had the highest SSE means (per year). In the *all* group (consisting of *all* subjects in the study), those with blue/green eyes had the highest baseline and follow-up SSE means (per year). However, only the baseline and follow-up SSE means of light/dark brown eyed melanoma and PLC subjects, all nevus subjects, and hazel/grey-eyed control subjects were close to "optimal". Also, in all but the melanoma group, light/dark eyed subjects had the greatest proportions of individuals with increased SSEs. However, none of these differences were significant (Table 9e).

Within most groups, at both baseline and follow-up, lower proportions of subjects with either hazel/grey or light/dark brown eyes reported to *never* perform SSE. However, none of these differences were significant. Nor were there significant differences relating to the color of the eyes in the proportions of individuals with change from *no* SSEs to +SSEs (Table 9e).

Within all the groups, the eye color category with the highest proportions of "optimal' SSEs shifted from a darker category of eye color at baseline, to a lighter one at follow-up. Among PLCs, for instance, those with hazel/grey eyes had the greatest proportion of baseline "optimal" SSEs, which by follow-up had shifted to both hazel/grey and blue/green eyes. These differences were significant in only the melanoma baseline SSEs, where 4 (67%) light/dark brown, as compared to 4 (19%) blue/green and 1 (17%) hazel/grey eyed subjects performed "optimal" SSEs ($p=0.044$). Meanwhile, in all but the control group, subjects with hazel/grey eyes had the highest proportions of change from "non-optimal" to "optimal" SSEs; however, none were significant (Table 9e).

- The analysis of SSE as a function of self-reported skin reaction to acute sunlight yielded *no* significant differences between those who burn and those who tan. In the melanoma, control, and *all* groups, the tanners had higher baseline and follow-up SSE means (per year), while in the nevus and PLC groups, the reverse was true. However, of all subjects, only nevus patients who burned had close to "optimal" SSE means. In all but the control and *all* groups, those who burned had higher proportions of increased SSEs as compared to the tanners. However, none of the above differences were significant (Table 9f).

In all but the melanoma group, higher proportions of burners had reported to *never* perform SSEs at baseline. However, by follow-up, in the melanoma, nevus, and PLC groups (but not the control and *all* groups), lower proportions of burners reported performing *no* SSEs. However, neither the baseline and follow-up differences, nor the proportions of subjects with change from *no* SSEs to +SSEs, were statistically significant (Table 9f).

In the melanoma and PLC groups, higher proportions of burners performed "optimal" SSEs at both baseline and follow-up, while the reverse held true for the nevus, control, and *all* subjects. Also, in all but the controls, those who burned had greater proportions of change from "non-optimal" to "optimal" SSEs. However, none of these differences were significant (Table 9f).

- The analysis of SSEs as a function of the self-reported inability to tan also revealed no significant differences. In all the groups, at both baseline and follow-up, those who could not tan in response to chronic sun-exposure (i.e., those who freckled or developed a mild tan following several peelings) had higher SSE means (per year). However, only nevus subjects (both frecklers and tanners) had close to "optimal" SSE means. Meanwhile, in all the groups, the tanners had higher proportions of increased SSEs. Again, none of these differences were significant (Table 9g).

In all but the baseline nevus and follow-up control groups, lower proportions of frecklers than tanners reported *never* performing SSEs. However, neither these baseline and follow-up proportions, nor the proportions of change from *no* SSEs to +SSEs were significant (Table 9g).

At baseline, in all but the melanoma group, higher proportions of tanners performed "optimal" SSEs; however, at follow-up, in all but the control group, higher proportions of frecklers performed "optimal" SSEs. In all but the controls, the frecklers had higher porportions of change from "non-optimal" to "optimal" SSEs. However, none of these differences were significant (Table 9g).

- The analysis of SSEs as a function of the objective, physician-determined nevus count revealed some significant differences. In all the applicable groups--melanoma, nevus, PLC, and *all*, but not control, given that all controls had 0-33 nevi--subjects with greater than 67 nevi had the highest SSE means (per year) at both baseline and follow-up. However, only the melanoma baseline SSEs were significantly different, at 46.9 [B], 27.4 [C], and 141.3 [A] for those with 0-33, 34-66, and greater than 67 nevi, respectively. Also, the only close to "optimal" SSE means pertained to melanoma subjects with 34-66 nevi at follow-up, nevus subjects with 0-66 nevi at both baseline and follow-up, PLC subjects with 34-66 nevi at follow-up, and *all* subjects with 34-66 nevi at follow-up. However, none of these differences were significant. Meanwhile, in all the groups, subjects with 0-33 nevi had the greatest proportions of increased SSEs, however, without statistical significance (Table 9h).

In all groups (but the nevus group at follow-up), at both baseline and follow-up, a higher proportion of subjects with 0-33 nevi reported *never* performing SSEs. These differences were significant in the nevus group, where 3 (25%), 0, and 0 (at baseline, p=0.040), and 0, 0, and 1 (11%) (at follow-up, non-significant p) subjects with 0-33, 34-66, and greater than 67 nevi, respectively, reported *never* performing SSEs. In this group, the proportions who changed from *no* SSEs to +SSEs were significant: 3 (25%), 0, and 0 subjects with 0-33, 34-66, and greater than 67 nevi, respectively (p=0.040). Also, the baseline proportions of PLC and *all* subjects who reported *never* performing SSEs were significantly different, at 7 (22%), 0, and 0 (for the PLCs, p=0.011), and 13 (25%), 0, and 0 (for the *all* group, p=0.004) for those with 0-33, 34-66, and greater than 67 nevi,

respectively. However, the change from baseline to follow-up was not significant for these groups (Table 9h).

In all but the nevus group (at follow-up), subjects with 34-66 nevi had the highest proportions of "optimal" SSEs at both baseline and follow-up. Also, in all but the nevus group, those with greater than 67 nevi had the greatest proportions of change from "non-optimal" to "optimal" SSEs. In the nevus group, those with 0-33 nevi had the greatest proportions of change from "non-optimal" to "optimal" SSEs. However, none of these differences were significant (Table 9h).

- The analysis of SSE as a function of the subjective, self-reported nevus count yielded some significant differences. Melanoma, PLC, and *all* subjects reporting 34-66 nevi, and nevus subjects reporting greater than 67 nevi had the highest SSE means (per year). Meanwhile controls who recounted 0-33 and greater than 67 nevi at baseline, and those who reported 0-33 nevi at follow-up, had the highest SSE means (per year). There were significant differences in the melanoma and PLC follow-up SSEs as a function of the subjective nevus count: the SSE means (per year) were 15.3 [C], 131.4 [A], and 73.7 [B] (for melanoma), and 13.9 [B], 89.8 [A], and 34.5 [B] (for PLC) subjects reporting 0-33, 34-66, and greater than 67 nevus categories. These SSE means were close to "optimal" in only melanoma and PLC subjects reporting 0-33 nevi (at both baseline and follow-up), almost all nevus subjects (at both baseline and follow-up), and controls reporting greater than 34 nevi (at follow-up only). However, none of these were significant. Meanwhile, in all but the control group, the mean increase in SSE was highest in those recounting 34-66 nevi, with significant differences in the *all* group, at 0.8 [B], 23.6 [A], and -13.4 [C] for controls

reporting 0-33, 34-66, and greater than 67 nevi, respectively. Also, in all groups, those reporting either 0-33 or 34-66 nevi had the greatest proportions of increased SSEs. These were significant only in the nevus group, where 8 (62%), 1 (25%), and 4 (25%) subjects reporting 0-33, 34-66, and greater than 67 nevi, respectively, had increased SSEs (p=0.051); (Table 9i).

In all but the control group, those recounting 0-33 nevi had higher proportions of subjects who *never* performed SSEs at baseline. This was significant in the nevus and *all* groups, where 3 (23%), 0, and 0 (nevus, p=0.036) and 11 (22%), 1 (8%), 1 (4%) (all, p=0.031) subjects reporting 0-33, 34-66, and greater than 67 nevi *never* performed SSEs. By follow-up, this significance had dissipated in both groups; however, only the change from *no* SSEs to +SSEs in the 3 (23%), 0, and 0 atypical nevus subjects who reporting 0-33, 34-66, and greater than 67 nevi, respectively, was significant (p=0.036). Moreover, at follow-up, only those who reported 34-66 nevi had a 100% +SSE rate (Table 9i).

In all but the control group, those reporting 34-66 nevi had the highest proportions of "optimal" baseline SSEs. At follow-up, in all groups, subjects recounting greater than 67 nevi had increased proportions of "optimal" SSEs; however, subjects reporting 0-33 nevi had the greatest proportion changes from "non-optimal" to "optimal" SSEs--however, none of these findings were significant (Table 9i).

- The analysis of SSEs as a function of the subjective impression of having more-than-average nevi yielded some significant trends. In general, those who believed in having more-than-average nevi had the highest SSE means (per year), at both baseline and follow-up. These SSE means were close to "optimal" in almost all nevus subjects (at both baseline and

follow-up), "uncertain" control and *all* subjects (at baseline), and "non-believing" and "uncertain" PLCs (at follow-up). None of these findings were significant. Also, in all but the melanoma group, "uncertain" subjects had the greatest proportions of increased SSEs, with significant differences in the nevus group, where 7 (30%), 4 (40%), and 2 (100%) subjects believing in, not believing in, or "uncertain" of having more-than-average nevi had increased SSEs (p=0.051, borderline significance); (Table 9j).

Subjects "uncertain" of having more-than-average nevi had significantly higher proportions of *no* SSEs, while the "believers" had significantly lower proportions of *no* SSEs: 0 as compared to 2 (13%) and 2 (40%) melanomas (at both baseline and follow-up, p=0.029), 1 (4%) as compared to 1 (13%) and 1 (50%) nevus (at baseline, p=0.032), 1 (3%) as compared to 3 (13%) and 3 (43%) PLCs (at baseline, p=0.002), and 1 (3%) as compared to 2 (8%) and 2 (29%) PLCs (at follow-up, p=0.021), 2 (5%) as compared to 7 (18%) and 4 (44%) of *all* (at baseline, p=0.004), and 2 (5%) as compared to 5 (13%) and 3 (33%) of *all* (at follow-up, p=0.021) subjects who believed in, did not believe in, or were "uncertain" of having more-than-average nevi reported to *never* perform SSEs. From baseline to follow-up, there was a loss of significance in the nevus group, and a reduction in significance in the PLC and *all* groups. However, only in the nevus group were the proportions of changes from *no* SSEs to +SSEs significant: 1 (4%), 1 (13%), and 1 (50%) subjects in the respective groups (p=0.032); (Table 9j).

At baseline, in all the groups, subjects reporting either average nevus counts, or "uncertainty" about having more-than-average nevi had the highest proportions of "optimal" SSEs. By follow-up, those reporting average nevus counts had the highest proportions of

"optimal" SSEs (all groups but the controls), and the highest porportions of change from "non-optimal" to "optimal" SSEs (all groups). However, none of these differences were significant (Table 9j).

- The analysis of SSEs with respect to the personal history of atypical nevi (AN) yielded some significant results. In all but the *all* group, subjects *with* AN had higher SSE means (per year) at both baseline and follow-up. In the control group, the SSE means (per year) were significantly higher for the *one* subject with AN (at 365 [A] at both baseline and follow-up) as compared to those without AN (at 62 [B] at both baseline and follow-up). However, only the nevus (baseline and follow-up) SSE means approached an "optimal" SSE. Also, in all groups (except for the controls), the mean increase in SSE and the proportions of increased SSEs were greatest in those *with* AN. However, none of these differences were significant (Table 9k).

In all the groups, those *with* AN had lower proportions of subjects who *never* performed SSEs. These were significant for the baseline and follow-up SSEs in the PLC and *all*groups: 4 (7%) AN as compared to 3 (27%) non-AN PLCs (at baseline, p=0.051), 2 (4%) AN as compared to 3 (27%) non-AN PLCs (at follow-up, p=0.007), and 4 (7%) AN as compared to 9 (30%) non-AN *all* subjects (at baseline, p=0.005), and 2 (4%) AN and 8 (27%) non-AN *all* subjects (at follow-up, p=0.002) reported *never* performing SSEs. As observed, the differences *within* these groups are heightened from baseline to follow-up (as suggested by the lower, more significant p-value). However, the proportion of changes from *no* SSEs to +SSEs were not significant in any of the groups (Table 9k).

In all but the control group, those *with* AN had higher proportions of "optimal" SSEs at both baseline and follow-up, as well as greater proportions of change from "non-optimal" to "optimal" SSEs. However, none of these differences were significant (Table 9k).

- The analysis of SSEs with respect to the personal history of non-melanoma skin cancer (NMSC) revealed almost no significant differences. Melanoma, nevus, and therefore, PLC subjects *without* prior NMSC, and control and *all* subjects *with* prior NMSC had the highest baseline SSE means (per year). By follow-up, in all the groups, those *with* prior NMSC had the highest NMSC means (except in the nevus group), and the greatest mean SSE increases (except in the control group). However, in all groups, those *without* prior NMSC had the greatest proportions of increased SSEs. However, none of these findings were significant (Table 9i).

Melanoma and PLC subjects *with* prior NMSC, and control and *all* subjects *without* prior NMSC had higher proportions of subjects who *never* performed SSEs, at both baseline and follow-up. In the nevus group, subjects *with* prior NMSC at baseline, and those *without* prior NMSC at follow-up, had higher proportions of *no* SSEs. Neither these differences, nor the proportions of changes from *no* SSEs to +SSEs, were significant (Table 9l).

In all but the melanoma and control groups, those *with* prior NMSC had higher proportions of "optimal" SSEs. The controls *with* prior NMSC, however, had significantly lower proportions of optimal SSEs: 0 controls *with*, as compared to 2 (13%) controls *without* prior NMSC performed baseline "optimal" SSEs ($p=0.015$). This significance had dissipated by follow-up. Also, higher proportions of melanoma and control subjects *with*, and nevus,

PLC, and *all* subjects *without* prior NMSC changed from "non-optimal" to "optimal" SSEs, however, none with statistical significance (Table 9l).

- The analysis of SSEs as a function of the family history of melanoma yielded few significant findings. Surprisingly, in all but the nevus group, those with either a negative or an "uncertain" family history of melanoma (FHM) had higher (baseline and follow-up) SSE means (per year). In the nevus group, in contrast, those with a +FHM had higher (baseline and follow-up) SSE means (per year). The SSE means were close to an "optimal" SSE in melanoma subjects with +FHM (both baseline and follow-up), almost all nevus subjects, regardless of FHM, PLC and *all* subjects with +FHM (at follow-up only), and control, PLC, and *all* subjects with an "uncertain" FHM (at follow-up only). However, none of these findings were significant. Also, in most groups, those with a negative FHM had the highest mean SSE increases--with significant differences in the *all* group, where the mean SSE changes were -3.4 [B], 6.5 [A], and -29.1 [C] for subjects with a positive, negative, and "uncertain" FHMM, respectively. Also, in all the groups, those with +FHMs had higher proportions of increased SSEs. These were significant in the PLC group (12 (52%) subjects with positive, as compared to 13 (35%) and 0 subjects with negative and "uncertain" FHMs, respectively (p=0.026)) and the *all* group (13 (52%) subjects with positive, as compared to 17 (33%) and 1 (11%) subjects with negative and "uncertain" FHMs, respectively had increased SSEs (p=0.054, borderline significance)); (Table 9m).

Except in the nevus and control groups, those with a +FHM had lower proportions of *never* performing SSEs at baseline. Interestingly, at follow-up, none of the subjects with

a +FHM reported *never* performing SSEs. However, neither these findings, nor the proportions of changes from *no* SSEs to +SSEs were significant (Table 9m).

At baseline, in only the melanoma group did those with a +FHM have the highest proportion of baseline "optimal" SSEs. At follow-up, in all but the nevus group, those with a +FHM had the highest proportions of "optimal" SSEs. Also, in all but the melanoma and nevus groups, those with a +FHM had the greatest proportions of change from "non-optimal" to "optimal" SSEs. However, none of these findings were significant (Table 9m).

- The analysis of SSEs as a function of the family history of non-melanoma skin cancer (FHNMSC) yielded a few significant differences. In the melanoma, nevus, and PLC groups, those with a +FHNMSC had higher (baseline and follow-up) SSE means (per year), while in the control and *all* groups, higher means were observed in those with "uncertain" FHNMSCs. SSE means close to an "optimal" were observed in melanoma subjects with an "uncertain" FHNMSC (at follow-up), almost all nevus subjects (at both baseline and follow-up), controls with +FHNMSC (at follow-up), and PLCs with an "uncertain" FHNMSC (at follow-up). However, none of these findings were significant. In only the melanoma and PLC groups did those with a +FHNMSC have the greatest SSE increases. Meanwhile, controls with a negative FHNMSC had significantly higher mean increases in SSE, at 22.6 [A], as compared to -58.8 [C] and 0 [B] for those with positive and "uncertain" FHNMSCs, respectively. Meanwhile, in all but the melanoma group, those with negative FHNMSCs had the highest proportions of increased SSEs. These proportions were significantly different in the melanoma and *all* groups (8 (50%), 4 (36%), and 0 melanoma (p=0.036), and 12

(39%), 17 (43%), and 2 (13%) *all* (p=0.054, borderline significance) subjects with positive, negative, and "uncertain" FHNMSCs had increased SSEs (Table 9n).

In all groups, lower proportions of subjects with +FHNMSCs reported performing *no* SSEs at both baseline and follow-up. However, neither these differences, nor the proportions of changes from *no* SSEs to +SSEs were significant (Table 9n).

In only the control group did subjects with +FHNMSCs have the highest proportions of "optimal" SSEs (at both baseline and follow-up). Moreover, melanoma and control subjects with +FHNMSCs, and nevus, PLC, and *all* subjects with negative FHNMSCs had the highest proportions of change from "non-optimal" to "optimal" SSEs. However, none of these findings were significant (Table 9n).

- Analysis of SSEs with respect to the number of previous visits to the pigmented lesion clinic yielded interesting results. Return melanoma, but new nevus patients had the highest SSE means (per year) at both baseline and follow-up. These were significant in the nevus baseline SSEs, at 50 [A] for new, and 20 [B] for return subjects. Only the return nevus patients had close to "optimal" SSE means. Meanwhile, higher proportions of new melanoma and return nevus patients had increased SSEs. However, none of these findings were significant (Table 9o).

In both groups, lower proportions of return patients reported *never* performing SSEs by follow-up. These were significant in the nevus group, where 1 (17%) new, as compared to *no* return patient reported *never* performing SSEs (p=0.034). The changes from *no* SSEs to +SSEs were not significant (Table 9o).

In both groups, higher proportions of return patients reported "optimal" SSEs (at both baseline and follow-up), while greater proportions of new patients changed from "non-optimal" to "optimal" SSEs. However, none of these findings were significant (Table 9o).

VII. Optimal Skin Self-Examination of Various Body Zones & Examination with a "Buddy":

The final stage of analysis entailed comparing the groups' examination of different body zones and the use of a "buddy" (Table 10). The subjects were, again, analyzed by both group and clinic; however, given that only the clinic-related results revealed significant differences, only those were included in the table. Also, an "optimal" examination frequency was defined, given that both extremes of SSE (either *no* or *daily* SSEs) would be undesirable. An "optimal" SSE ranged in frequency from once per month to once every 4 months, except in relation to non-traditional areas such as the scalp and genitals, for which the "optimum" was defined to range from once per month to once per year--given that they would probably be examined less frequently, given their difficult-to-examine locations. Thus, for the genitals and scalp to be self-examined at least once per year was considered to be a significant indicator of skin-awareness (Table 10).

There were no significant differences between the proportions in each clinic "optimally" using a "buddy" to examine difficult-to-see areas. There were, however, significant differences in the proportions in each clinic population performing "optimal" SSE of the front (30 (45%) PLCs as compared to 4 (20%) controls, $p=0.043$), sides (32 (48%) PLCs as compared to 3 (15%) controls, $p=0.008$), and the back (30 (45%) PLCs as compared to 6 (30%) controls, $p=0.043$). There differences in the proportions in each clinic population

performing "optimal" SSEs of areas such as the scalp and genitals were not significant. The controls' SSE means were significantly higher than that of the PLCs with respect to the examination of the sides (127 [A] as compared to 48 [B]) and such areas as the scalp and genitals (84 [A] as compared to 15 [B]). However, these PLC SSE means were closer to the defined "optimum" (Table 10).

Conclusion:

I. Discussion of Results:

Potential Sources of Bias: The principle task in interpreting the study results would be to identify all possible sources of bias. Some of the biases in this study pertained to the patient population, while others related to the questionnaire design. These biases will be systematically addressed in the following section.

- Biases in the Patient Population: First, the study participants were primarily recruited through university dermatology clinics at Yale. Most of the subjects were of middle- to upper-middle class SES, and therefore, higher educational levels, given that greater than 70% of them had (at least) semi-professional careers. This was expected from a population of Connecticut residents being followed by academic physicians at a university-based medical center. As this was a potential source of bias--given the documented association between high education levels and better health awareness and health practices--it was attempted to rectify the situation by recruiting patients through a hospital-based dermatology clinic (with mostly Medicaid patients). However, the enrollment efforts met with very low yield at the hospital-based clinic, given that most of the approached patients refused to participate; this differed starkly from the university-based dermatology clinics, where almost all the approached patients readily consented to partipating in the study. Moreover, of the three hospital general dermatology clinic patients who *did* consent to participating in the study (*and* completed the study), one reported a family history of melanoma and one was

consulting about pregnancy-related changes in pre-existing melanocytic nevi. These raise the issue of self-selection bias, which is inherent to this study design.

Second, the study controls were a population of *general dermatology* patients. In retrospect, this probably biased the analysis of skin self-examination (SSE) practices, given that, in this population, there was no means of differentiating between skin examination for signs of dermatologic diseases like psoriasis and sarcoidosis, and signs of potential melanoma lesions. (Perhaps the anticipation of this dilemma at the onset would have allowed the modification of the questionnaire to elicit such differences, or else, the selection of a general medicine patient population, with no confounding dermatologic diseases, as controls. But as such was not the case, the effects of this bias would remain unknown).

Third, given that this was a pilot study, with a consequently small population size, variables which could have been significant predictors of change in knowledge and SSE with a larger sample size, may have appeared to be non-significant in our analyses.

- Questionnaire-Based Biases: Knowledge of the subjects' risk factor profiles relied solely on their self-reports. Whereas in many cases (such as with eye and hair colors) the categories were straight-forward, and therefore, not subject to recall or self-report bias, in other cases (such as acute and chronic skin reactions to sunlight), this might not have been so. For example, subjects might have reported not burning in reaction to acute sun-exposure because of wearing sunscreen or avoiding excessive sun-exposure, as opposed to basing their responses on their natural skin tendencies.

Second, the questionnaires examined the subjects' awareness of several melanoma facts that were not addressed in the educational video. The rationale behind incorporating

these questions into the questionnaires, despite our awareness of this flaw, was that this pilot study had been performed in the past with an educating nurse as opposed to the video, and therefore, it was important to maintain as much constancy as possible between the studies, in order to be able to compare their efficacy of the two educational media or tools in promoting change in knowledge and behavior.

Third, there was no means of verifying the self-reported SSE frequencies, which could have been inflated due to an awareness of the study messages regarding melanoma and the importance of early detection through regular SSEs. Of note, the follow-up questionnaire had two questions on the frequency of general body SSEs, one at the beginning and another at the end of the questionnaire. Lower proportions of individuals reported *never* performing SSEs in response to the *second* question, as compared to the *first* one. Since the responses to the first question were more likely to be accurate, they were used in the analyses so as to minimize bias.

Finally, in both the pre- and post-questionnaires, the subjects were asked whether they conducted careful, directed examinations of their skin, in order to assess their SSE practices. In retrospect, it would have been useful to also ascertain *how carefully* they performed SSEs--i.e., what they meant by a "careful and directed" exam. In one study, four definitions of self-surveillance were used, consisting of (1) a general awareness, both cosmetic and medical (although it might be more useful to separate these); (2) purposeful examination of *regions* of the body (which was addressed in our study at follow-up); (3) the observation of a particular mole; and (4) surveillance by a spouse or "buddy" [30].

Discussion of Results: Having a grasp of the areas of melanoma awareness versus deficit in each population would be critical to the design of any educational interventions and campaigns. The melanoma and nevus (or PLC) groups represent a population which, given its high risk of melanoma, has been actively targeted by physicians with not only regular surveillance, but also education and behavior modification interventions. Therefore, such a population would have a much higher understanding of melanoma risk factors and characteristics. In contrast, the control group, a population at lower risk for melanoma, with probably no prior history of active interventions against melanoma, would constitute a group whose knowledge base would most likely reflect public education efforts through the popular media. These differences in knowledge would by necessity translate into different needs, which would have to be addressed in any intended intervention--i.e., the campaign focus for each population should be guided by its knowledge deficits.

In this study, the controls were highly suspicious of changing (whether in color, size, shape, or thickness) and non-healing lesions, and those that were dark or variegated in color. That these melanoma signs would appear most alarming to a lay person makes sense. The popular media has already identified melanoma as being black or dark in color; and *change* (implying active growth) and poor healing are probably features frequently associated with cancer. Meanwhile, in all the groups, there was an overall low awareness of the somatic symptoms (like itching and tenderness) and size criteria of early melanoma. These melanoma signs and symptoms represent potential areas of deficit for focusing future educational interventions, especially where it concerns the early self-detection of melanoma.

There was also a generalized lack of awareness about alternate methods of examining difficult-to-see areas of the body, as in using a "buddy" or a mirror. This is an area of growing importance given the recent trends in melanoma epidemiology--for instance, the back is the *most* common and the *second-most* common site for development of melanoma in men and women, respectively. It assumes even greater significance in view of the finding that women tend to find their own lesions regardless of the location, whereas men rarely discover a lesion on the back [185]. In our study, at baseline, all groups (and especially the controls) were poorly aware of how to survey difficult-to-see skin surfaces, like the back. However, the intervention significantly improved subjects' awareness regarding this area. Thus, it appears that this is also an important area for focusing future public intervention efforts.

Meanwhile, all three groups had nearly identical knowledge levels about melanoma risk factors, perhaps reflecting the growing media focus on melanoma, concomitant with the AAD screening campaigns over the past decade. Given this context, the subjects' high awareness of high-risk phenotypes like red hair, blue eyes, and a tendency to burn, were understandable. However, the high awareness of the association between melanoma and either atypical nevi or high numbers of CAMN was surprising, given that they have received relatively little media attention. This seemingly high awareness of high nevus counts and atypical nevi as melanoma risk factors might be *either* true, *or* subtly (and unpredictably) influenced by cues in the phrasing of the questions (as in "*odd*-shaped moles"), leading the subjects to the correct answer. Regardless of this point, future public education interventions *should* devote greater attention to these significant melanoma risk factors, so that at-risk

individuals can appropriately ascertain their own risk, and (hopefully) attend AAD annual screenings and adopt more self-screening and sun-safe behaviors.

The data revealed the subject *group* to be one of the most important predictors of knowledge levels, as well as knowledge improvement: despite lower scores, the controls had the greatest knowledge improvement. The intervention either directly caused this increased awareness, or raised the subjects' awareness of melanoma as a public health hazard, thereby indirectly leading them to pay greater attention to public health messages about it in the media. Regardless of whether the increase in knowledge was achieved directly or indirectly, one of intervention's goals--i.e., to raise awareness about melanoma--was accomplished. This is significant, given that it documents the efficacy of such targeted educational interventions in raising public awareness about melanoma risk factors and clinical characteristics. A video such as the one used in this study could be distributed to physicians everywhere--including pediatricians, internists, surgeons--and shown in the waiting rooms, to various industries for employee education, etc. Moreover, in such small targeted populations, the educational tools such as the video may be tailored to the special needs and circumstances of the population, which would probably result in even greater yields. For instance, a video distributed to pediatricians, in emphasizing the role of early childhood and adolescence sunburns in determining later risk of melanoma, would (inadvertently) alter their *own* attitudes toward and behaviors in the sun.

Other important predictors of knowledge scores and/or knowledge increase included hair color (significantly higher mean improvements and proportions of increased scores in those with red or blond hair); the objective, physician-determined nevus count (significantly

higher scores in those with either 34-66 or greater than 67 nevi); the personal history of atypical nevi (with usually significantly higher post-intervention knowledge means in those *with* AN); and surprisingly, the family history of non-melanoma skin cancer (with a tendency toward significantly higher knowledge scores and proportions of score increase in those with either a positive or an uncertain family history of non-melanoma skin cancer). In terms of knowledge score percentiles, of course, the pigmented lesion clinic population consistently had significantly higher proportions of individuals answering greater than 75% of the questions correctly at both baseline and follow-up.

With respect to the reported SSE practices, I found the absolute frequency of SSE (which ranged from 0 for *no* exams, to 365 for *daily* ones) reported by the subjects less useful a guide to their skin-awareness and self-screening. In general, however, the atypical nevus group's SSE means were more consistently near the defined "optimal" range of once per month to once every 4 months. Nonetheless, a subject's group (or clinic affiliation) was a significant predictor of whether he/she performed *no* SSEs. Red or blond hair color was a significant predictor of both "optimal" and +SSEs, as well as of *change* from "non-optimal" to "optimal" SSEs. Both the objective, physician-determined and the subjective, self-reported nevus counts were significant predictors of +SSEs (given that almost all PLCs physician- or self-reported nevus counts exceeding 33 nevi reported +SSEs at baseline). Yet another predictor of +SSEs was the self-reported impression of having more-than-average nevus counts (this subgroup had significantly higher proportions of +SSEs at both baseline and follow-up). Finally, the presence of atypical nevi was also a significant predictor of +SSEs.

The lack of significant differences with respect to the rate of "optimal" SSEs, as well as other SSE-related measures, might reflect the simple truth about the complexities of behavioral modification. It is well-recognized that inducing behavioral change is a much more demanding task than that of enhancing knowledge. It may be that an intervention much more intensive than our ten-minute instructional video was needed to motivate individuals to perform monthly SSEs.

II. Discussion of Strategies:

Theoretical Considerations in Education and Screening Campaigns:

Given the discussed trends in the epidemiology of melanoma and the body of information available on its risk factors, our first strategy toward melanoma control must be primary prevention through education and behavior modification, so as to reduce melanoma incidence (and therefore, mortality) through risk factor modification. The next strategy would be secondary prevention through early detection and diagnosis, in hopes of reducing melanoma mortality by intervening at an earlier, potentially more curable stage in its natural history [144]. As discussed earlier, one major advantage of melanoma, as compared to other malignancies, pertains to its cutaneous, readily-visible location, which allows for ready detection of thinner, prognostically more favorable lesions [144]. Moreover, given its uniquely visible, cutaneous location, one may consider its early detection to be inextricably intertwined with education about it [189], given that greater melanoma awareness could increase the likelihood of participation in screening efforts--including self-screening--while engaging in various screening efforts would heighten one's awareness about melanoma.

Theoretically, education and early detection should decrease melanoma morbidity and mortality [182]. However, as of yet, no formal prospective, randomized trials (which would constitute the gold standard for evaluating such interventions) have attempted to test the efficacy of such programs in reducing melanoma mortality [144, 187]. Moreover, that such a trial would ever be undertaken seems very unlikely, given that it would present many logistic and practical difficulties [190]. Nonetheless, melanoma prevention and control efforts have begun in many nations around the world over the past decade [70, 80, 101, 199, 235, 261]. These programs have varied in scope and intensity among nations, and include nationwide public and professional education campaigns focusing on sun-safe behavior in Australia; regional professional and public education programs in Italy, the United Kingdom, and Austria; local screening efforts in the Netherlands and New Zealand; and national screening efforts in the United States [190].

Complicating the evaluation of these efforts are data from a collaborative study of cases from Alabama, U.S.A. and New South Wales, Australia [13], along with data from the Netherlands [81], Germany [122], Israel [315], and the United Kingdom [360], which suggest a trend toward improved survivals and reduced mean Breslow depths at diagnosis, with the trends predating the interventional efforts in these countries [190]. For instance, the evaluation of the King College Hospital 1986-1987 public education campaign [360], which entailed reviewing all melanoma specimens from 1970 to 1987, traced the greatest decrease in tumor thickness to the late 1970's, when the median Breslow depth fell from approximately 4 mm to 1.5 mm. This was accompanied by a concomitant increase in the proportion of thin lesions (<1.5 mm) and a decrease in both the proportion and the absolute

number of thick lesions (>3.5 mm), while the proportion of lesions of intermediate Breslow depth (1.5 mm to 3.49 mm) remained relatively stable. Also, the investigators noted an increase in the proportion of superficial spreading melanomas and a decrease in the proportion of nodular melanomas throughout the same 18-year period, with little alteration in this pattern during the campaign years [360].

Another critical issue complicating the evaluation of these education and screening interventions, and ultimately, of melanoma control, relates to the questionable accuracy of the documented melanoma diagnoses and incidences. This problem, which results from the increasing trend toward diagnosis and treatment of melanoma on an outpatient basis, has been escalating over the previous years. Increasingly, cases are not reported to central registries, leading to falsely lower incidence rates [190]. Two studies from Massachusetts and western Washington State indicated under-reporting rates of 12%-19% and 21%, respectively [174, 183].

Despite these limitations, and given the obstacles against a prospective, randomized trial, by necessity, the focus has shifted onto intermediate-term outcome measures as means of evaluating education and screening programs [308]. To this end, some of the intermediate-term measures for monitoring the efficacy of melanoma *education* could include: change in knowledge among target populations; increased public awareness of significant risk factors and one's own susceptibility; skin self-examination (SSE) rates among high risk individuals; requests for physician skin examinations; and practitioners' knowledge of melanoma risk [190]. In this respect, it is hoped that a change in knowledge would translate into improved attitudes and promote behavioral change through both primary

prevention (as with altering one's sun exposure behavior), and early detection outside of organized screening (as with increased skin self-examination (SSE)) [190].

For instance, a recent U.S. study of 502 whites older than 50 years in age revealed an inadequate frequency of SSE among the subjects, despite high awareness of melanoma and its risk factors in 74% of the subjects [247]. The same study revealed both knowledge and preventive practices to be significantly worse among men and those *without* higher education. Only 19% of those with just a high school degree or less, as opposed to 29% of those with a college degree, had ever requested a physician skin examination ($p < 0.05$). Likewise, only 46% of those with just a high school degree or less, as compared to 65% of those with a college degree had ever received a physician skin examination ($p < 0.05$).

Professional education of medical providers should also help optimize screening and thereby enhance early melanoma detection. Few medical professionals receive specific education and training in the early detection of melanoma. In one study, only 12% of non-dermatologists and 69% of dermatologists could correctly identify at least five of six melanoma lesions [51]. To this end, major professional education efforts have been implemented in Australia, Austria, and Scotland [190]. Australians have targeted medical school curricula, emphasizing primary care practitioners' ability to detect high risk individuals within routine medical practice, implement opportunistic screening, and develop targeted screening for high risk persons [316]. In Austria (1988), all surgeons (n=900), dermatologists (n=350), and general practitioners (n=9000) received illustrated folders and publications about pigmented lesions [261]. In Scotland (1985) media and poster/leaflet

publicity efforts were preceded by preparing general practitioners with specially prepared booklets entitled "An Illustrated Guide to Early Malignant Melanoma" [80].

Intermediate measures of successful *screening* efforts could include: lower numbers and proportions of thick tumors in screened populations; thinner tumors in the screened populations as compared to controls--and whether the above two measures result in ultimately lower morbidity and mortality rates in the screened populations; greater proportions of high risk individuals attending the screenings; and increased frequencies of SSE's [190]. In this respect, attempts at evaluating mass screenings have focused on evaluating the cancers found during the screenings, the access to the screenings, the risk profile of the screened participants, the educational effects of the screening programs, and the evaluation of the visual examination as a screening tool [190].

Regarding the cancers found in the screenings, studies from New York and Massachusetts have reported melanoma yields of 14 per 2239 and 9 per 2560, respectively [179, 296]. Also, evaluations of nationwide screenings in the United States have documented Breslow depths of less than 1.50 mm in almost 99% of the screen-detected melanomas [182]. In this respect, these screenings (conducted by the American Academy of Dermatology (AAD)) appear to detect early melanomas, with stage and thickness distributions comparing favorably with those of the SEER registry [325]. For instance, of individuals diagnosed with melanoma in the 1992-1993 AAD screening, 261 melanomas were histologically confirmed among 257 individuals, with all but four persons having localized disease [325]. When compared to the 1990 SEER population-based data, these figures were far more favorable, given that they had fewer advanced melanomas. However,

there is not only a possibility for self-selection bias [178, 325], but also an inability to predict improved mortalities [325].

Screening programs also have the additional benefit of providing access to medical care to such populations as the poor [62]. In the United States, the AAD screenings represent some groups' sole opportunity for receiving skin cancer examinations [190]. Among those screened in the 1992 and 1993 campaigns, over 75% did not have a regular dermatologist, 47% would not have seen their physician without the screen, and 9% had no health insurance. Also, 80% of the participants were attending their first screening, implicating screenings as many individuals' first opportunity for expert skin examination [184, 325].

Regarding the risk profile of the participants, a Massachusetts study revealed a tendency toward self-selection among the participants, given that greater than 86% had at least one, and 78% had at least two melanoma risk factor(s) [184]. Also, data from Massachusetts and Rhode Island document that high risk individuals tend to appropriately self-select for screening, even though they have not been specifically targeted as a group [184, 354].

With respect to education, various measures ranging from prescreening publicity, education during the screenings, and postscreening efforts have been used, aimed at heightening public awareness of melanoma risk factors and warning signs, along with disseminating information on sun protection, excessive sun exposure, tanning parlors, guides to SSE, and the importance of early detection [190]. For instance, a Swedish study of 190 individuals attending a melanoma screening clinic documented increased attention to nevi among the participants: those who were concerned about their nevi were reassured, while

those who were not so interested learned to pay more attention to their nevi [44]. However, the long-term effects of such interventions still remain to be investigated [190].

The evaluation of the visual skin examination as a screening tool would entail determining its sensitivity, specificity, and positive predictive value. As mentioned earlier, the estimated sensitivity, specificity, and positive predictive value of the visual skin examination range from 73% to 99%, 92% to 99%, and 35% to 80%, respectively [20, 178, 179, 191, 244, 327]. A study of university-based dermatologists with a special interest in melanoma documented estimates of 77%, 99%, and 80%, respectively, for the three parameters [191]. Meanwhile, the AAD screenings, more representative of the real world, have yielded a sensitivity of 97% and a positive predictive value of 35%-40% in Massachusetts, and a positive predictive value of 13%-24%, nationally [179, 180]. (The study design had not allowed the calculation of specificity values.)

Also in an effort to evaluate the efficacy of the visual skin examination as a tool for detecting melanoma, a number of reports have documented the accuracy of dermatologists' diagnosis of melanoma in the clinical setting: one study recorded an accuracy rate of 64% [191]; another study noted improvement in the clinical diagnosis of melanoma from 1955 to 1982, with a sensitivity of 84.5% and a specificity of 72.4% in the most recent period (1974-1982) [146]; still another study revealed variations in clinical diagnosis with differences in tumor thickness--58% of lesions smaller than 2 mm, and 51% of those greater than 2 mm were correctly diagnosed [272]; and finally, one study reported significant inter-dermatologist differences in the assessment of skin images, when measures of diagnostic sensitivity, reproducibility, and reliability were evaluated [263].

Public information campaigns have increasingly encouraged SSE as a means of screening for early melanoma lesions, with detailed directions for the proper examination, including the use of mirrors or "buddies" for otherwise difficult-to-see areas [117]. However, there are few, if any, quantitative data regarding the utility or efficacy of self-examinations as a screening technique [144, 190]. A prospective case-control study in Connecticut, hypothesizing that SSE reduces mortality through early detection of melanoma, will provide the first real information in this regard [30].

A study evaluating the SSE practices of 1344 Australians documented that 48% either self-screened or had their skin checked by another lay person [128]. In particular, men, those of lower occupational and educational status, the unemployed, the infirm, and those with only basic medical insurance were found to have poor self- and medical-screening practices. In the United Study, only one in five subjects with prior melanoma were found to practice SSE [185]. In another study, 61% of self-selected screenees reported performing SSE at least once during the previous year, whereas only 20% reported performing monthly SSE, and only a minority were knowledgeable about most of the recommended SSE steps [118]. Likewise, in a population of 874 dermatology patients, only 6% were found to follow all the recommendations for monthly SSEs, yearly professional examinations, and sun protection [206], which led the investigators to recommend a thorough skin examination for all new patients, and annual skin examinations for all return patients (given that most do not adopt the SSE recommendations).

One study, evaluating general dermatology patients' self-assessment of pigmentary risk factors for melanoma, recorded the number of freckles on the right forearm, the number

of palpable arm nevi, and the number of nevi greater than 5 mm in diameter on the entire body as determined by first the patients and then a dermatologist. Using the physician counts as the standard, the investigators reported a specificity of 83% to >95% for the three cutaneous markers, leading them to propose a tiered approach to screening: self-assessment of risk by SSE, followed by physician confirmation of that risk, and finally, more regular SSE and surveillance of those deemed to be high risk [148].

It is hoped that screening and close surveillance of high risk individuals would lead to earlier detection of malignant lesions, before the highly lethal stages are reached. Also, targeted screenings (of higher risk persons) should improve the predictive value of the exam [178]. Risk factors used to identify high risk individuals could include high nevus counts (or the presence of atypical nevi), red hair, freckling, and burning following repeated sun exposures [236, 340, 344], a changing nevus, and a family history of melanoma [292].

As discussed earlier in the introduction, the NCI/UPenn prospective surveillance of multiple kindreds with the atypical nevus-melanoma syndrome led to the detection of significantly thinner lesions [237]. In this study, the average Breslow depth of the melanoma lesions were 0.52 mm for the 28 surveillance incident melanomas, 0.55 mm for the 64 non-surveillance incident melanomas, and 1.44 mm for the 48 index lesions ($p < 0.001$). Also, the investigators have noted a significant decline in the proportion of tumors presenting in the vertical growth phase, from 80% to 41% [237]. A similar study in the Netherlands involving the surveillance of nine kindreds with the atypical nevus-melanoma syndrome revealed similar results [344]. The average Breslow depth of the melanoma lesions discovered prior to the study (n=19), at the initial examination *for* the study (n=11), and

detected during the routine follow-up in the course of the study (n=20) was 1.75 mm, 0.80 mm, and 0.54 mm, respectively (p=0.001). These results have been reported by multiple other studies following individuals with atypical nevi [227, 295, 340], pinpointing the beneficial effects of surveillance and education in such high risk populations [237].

The increased incidence of melanoma among workers at the Lawrence Livermore National Laboratory--who were found to have a four-fold increased incidence of melanoma as compared to individuals from the surrounding community in the early 1980s--also prompted the prospective surveillance of this population. The intervention mounted for the early detection and treatment of melanoma adopted a multi-tiered strategy of educating employees, management, and local practitioners; promoting skin self-examinations and mole counting; and an on-site melanoma clinic for dermatologic examinations and treatment. Since this project's onset, all the melanoma diagnoses have been thin (<0.76 mm) and *in situ* lesions, with an anticipated survival of 95%-100%, a median tumor thickness of *zero* (i.e., most lesions were *in situ*). Also, the median Breslow depth of melanomas in these workers declined significantly more rapidly as compared to that of the reference community population [7, 310].

Middle-aged and older men (50 years of age and above) might constitute another appropriate target group for education and early detection, given that they have the highest melanoma mortality rates--documented by data from the United States, Australia, and New Zealand [37, 66, 126, 152, 186, 346]--along with disproportionately lower levels of participation in mass screenings (which depend on patient self-referral). Lower socioeconomic groups may also constitute another appropriate target population, given that

a recent study reported that lower socioeconomic persons are more likely to die from their cancer than are individuals of higher socioeconomic status [190 (Geller et al., 1995)].

Review of International Screening and Education Efforts in Melanoma:

Even though many of these efforts have been alluded to in previous sections, they will be discussed more formally in the following section.

Australia: The Queensland Melanoma Project, which began in 1963, is the forefather of all population-wide screening efforts in melanoma [80, 317]. The project initially began with a population-based retrospective survey of melanoma incidence and mortality in Queensland, spanning the years 1945-1963, which formally documented Queensland's high melanoma incidence (the highest in the world), and lay the foundation for an ongoing statewide melanoma registry capable of providing up-to-date morbidity and mortality data. Then, the project launched an educational campaign targeting both professionals and the public through modifying medical school curricula, entering the school systems so as to reach children at an earlier, perhaps more impressionable age, and finally, utilizing all media forms, from publications and brochures, to television advertising and programing. Also, since 1985, these efforts have been complemented by screening caravans (known as "battle stations") which examine individuals at such public locations as beaches and public squares, and refer those with suspicious lesions for further treatment [104]. In other instances, these large community-based public health campaigns have been supplemented by industry-based programs (initiated by the industries themselves, targeting their workers' specific needs), with favorable preliminary results [39].

In terms of televised programing, for instance, "Good-bye Sunshine" was the most notable one, consisting of three televised "episodes" about melanoma spaced over a 2-year period, featuring the story of a 26 year old man diagnosed with invasive melanoma [334]. The episodes chronicled first, the impact of the melanoma--metastatic at the time of diagnosis--on the man and his girlfriend, his crusade against melanoma on beaches and other public settings, and finally, his death, interspersing emotional and tearful scenes with information on the signs and symptoms of melanoma and the importance of prevention and early detection. The program was shown to affect not only the viewers' knowledge levels, but also their sun-related preventive practices. Also, the program improved viewers' skin awareness and skin self-examination behavior, in that more than half reported examining their skin for "suspicious" spots as depicted in the program. In the meantime, there was a 143% and a 100% increase in the number of diagnosed melanomas in the two consecutive years following this program's broadcast. Also, the mean Breslow depth of the melanoma lesions decreased significantly from 1.59 mm to 0.91 mm, while the proportion of tumors $\leq$0.75 mm in Breslow depth increased significantly, from 50% to 74% [334].

Another component of the campaigns consisted of a three-year SunSmart melanoma prevention campaign, aimed at promoting melanoma prevention by reducing sun-exposure, and more specifically, sunburns [153, 154]. The behavior changes induced by the campaign significantly reduced the frequency of sunburns over the period. There were also significant shifts in public attitudes: there was a markedly changed public perception of susceptibility to skin cancer, with significantly fewer individuals denying their risk of developing skin cancer; there was a markedly changed attitude towards suntans, with an increase from 39%

to 51% of individuals not desiring any degree of suntan. Also, the campaign significantly increased the use of protective measures, such as wearing hats and applying sunscreens [153, 154].

These educational efforts have remained continuous and ongoing in Australia, given that decreases in these efforts were associated with reductions in melanoma awareness [144]. These activities have had favorable results: the proportion of *in situ*, stage I melanomas (tumor cells confined to the epidermis) rose from 9% in the early years of the project (1963-1969) to 26% in 1977; there was a 25% increase in the proportion of stage II melanomas (tumor cells invading the papillary dermis); and the five-year mortality decreased from 41% to 26% [243]. During 1963-1969 period, nearly three quarters of males and two thirds of females had primary tumors of Clark levels III, IV, or V (tumor cells expanding the papillary dermis, invading the reticular dermis, and invading subcutaneous fat, respectively), whereas by 1977 only half of males and just under half of females had such tumors [243]. Also, even though melanoma incidences continued to rise in the same period (from 16 per 10^5 in 1963 to 34 per 10^5 in 1979 [317]), the melanoma mortality rates stabilized [243]. In fact, due to these interventions, despite having the highest melanoma incidence rates, Australia has higher survival rates than North America and Europe [11, 151].

New Zealand: The New Zealand Cancer Society has sponsored "skin check" days, attended by more than 12,000 individuals in the 1988-1989 period [104, 190]. The screening efforts have been complemented by intensive advertising through both television and printed material. Moreover, in the Wellington region, the 1985-1992 public education campaigns led to significant increases in not only the total number of treated melanomas, but also the

number and proportion of thin (<0.75 mm in Breslow depth) invasive melanomas, from 30% in 1985 to 70% in 1992 [337].

Italy: A public education campaign was launched in the province of Trentino, Italy, targeting both professional and non-professional audiences, while also establishing free clinics for the expeditious referral of patients with suspicious lesions [70]. The investigators compared the standardized mortality rates for Trentino with those of three neighboring regions where no campaigns were carried out, over time. The campaign resulted in a four-fold increase in patient consultations, tripling of excisions, and doubling of melanoma diagnoses, and an eight-fold increase in the proportion of lesions <0.76 mm in Breslow depth, from 2.5% to 21% [70]. Also, the campaign resulted in a decline in melanoma mortality rates in women, and a mild increase in melanoma mortality rates in men in Trentino, in comparison with the mortality rates in the surrounding regions, which continued to rise at the high pre-campaign rates [70].

United Kingdom: Perhaps the most thorough (in both design and execution) of contemporary melanoma screening programs was conducted in the west of Scotland. The discovery that nearly one third of Scottish patients were presenting with melanoma lesions >3.5 mm in Breslow depth (with an associated five-year survival of 38%) prompted a survey to assess the reason underlying this delay in diagnosis. The survey attributed the delay in diagnosis to patients' lack of knowledge about melanoma characterisitics and failure to appreciate the significance of changing pigmented lesions, and not to shortcomings in the health care system [79]. This led to the design of a public education campaign to inform the public about the features of early melanoma, and to encourage them to seek treatment [80].

Moreover, the six-month period preceding the public phase of the campaign was set aside for professional education and update, accomplished through the mailing of a detailed booklet on melanoma, with color illustrations of early lesions, along with problem-solving case histories and an educational video. These were complemented by a series of educational meetings for both physicians and nurses. Also, a weekly pigmented lesion clinic was established for the immediate care of referred patients. The public education phase of the campaign involved posters, brochures, and a press release for the local and national press, radio, and television [80].

The campaign resulted in a 278% increase in the number of referrals to the specialty pigmented lesion clinic; an increase in the mean number of melanomas diagnosed per month, from 12 to 21; a 23% rise in the total number of melanomas diagnosed; and a significant (16%) rise in the proportion of thin melanomas (<1.5 mm in Breslow depth). By three years, the campaign had resulted in a significant decline in the proportion of thick tumors (≥3.5 mm in Breslow depth) among the females only, which translated into a decreasing mortality rate in this group--the first direct evidence of such an effect ever noted in a melanoma screening program [225].

A few years after the Scottish experience, public education campaigns were launched in seven additional regions throughout the United Kingdom [92]. In the region served by the King's College Hospital (described earlier), the evaluation entailed reviewing all melanoma specimens from 1970 to 1987. This study traced the greatest decrease in tumor thickness to the late 1970's, from a median Breslow depth of 4 mm to 1.5 mm, along with an increase in

the proportion of thin lesions (<1.5 mm) and a decrease in both the proportion and absolute number of thick lesions (>3.5 mm). This pattern altered little during the campaign years [360].

In Leicestershire, there were three consecutive, annual public education campaigns, resulting in the diagnosis of significantly higher numbers of melanoma immediately after the first campaign, lower numbers following the second and third years, and again, increased numbers in the fourth and fifth (non-campaign) years. Also, the campaigns led to a significant rise in the proportion of thin (<1.50 mm) lesions, and a significant decline in the proportion and absolute number of the thickest (≥3.5 mm) lesions [150]. It was reasoned that the "early" diagnosis of melanomas immediately following the first campaign led to an artifactual lowering of melanoma diagnoses in the second and third campaign years--when the "early" melanomas would have been detected otherwise. The rise in the number of melanoma diagnoses in the fourth and fifth (non-campaign) years was attributed to the "wearing off" of the impacts from the first campaign. The investigators concluded that, in low melanoma incidence regions like the U.K., pulsatile publicity may be more effective than annual or continuous ones [150].

Finally, given that childhood sunburns constitute a significant risk factor for melanoma, a health education program was developed for secondary schools, modeled after a similar program (with encouraging results) in Australia. Seven schools in various regions of England were recruited for the study, to which educational material on melanoma in the form of pamphlets, workbooks, and a video, were distributed [168]. The effects of the video

on students' knowledge, attitudes, and behavior were assessed through pre- and post-questionnaires. Despite significant differences in both knowledge and attitude between the intervention groups and the controls, there were no significant differences in behavior between the groups, except in relation to the use of sunscreen. Also, more children reported using sunscreen and wearing a hat if they traveled abroad (revealing a subjective association of sunburns with sunlight abroad, as opposed to local sunlight). Meanwhile, with respect to attitudes, those who either covered up in the sun, wore sunscreen, or sat in the shade had significantly better attitudes than those who did not behave in this way [168].

The United States: In response to the growing incidence and mortality of melanoma and the continued ignorance of the public about melanoma, the American Academy of Dermatology (AAD) initiated the National Melanoma/Skin Cancer Prevention Program in the spring of 1985 [239, 325], enlisting the volunteer services of AAD dermatologists and American Cancer Society volunteers to examine an essentially self-selected population. Concurrently, each screening was preceded by national and local media (newspapers, radio, and television) help, augmenting these efforts by publicizing the screening times, encouraging attendance [190], and disseminating information on melanoma risk factors and warning signs, the importance of sun protection and early detection--and even performing skin self-examinations [239]. Other educational materials included posters, a 15-minute skin cancer slide/script set designed for presentation to community groups and organizations, an 8-minute video on skin cancer for on-site use, and informative pamphlets for ready distribution

Thus, between 1985 to 1993, the AAD provided free skin screening to more than 600,000 Americans [325]. The screening participants first completed a standardized form on risk factors, changing moles, and family and personal history of skin cancer and melanoma, after which they were examined by the AAD physicians. Suspicious lesions were referred to the individual's primary physician for diagnosis and treatment. Of the 559 persons diagnosed with skin cancer from 1989 to 1991, 176 were melanomas: 173 (98%) were stage I [185b]; 104 (59%) were less than 1.5 mm in Breslow depth (associated with an 86% five-year survival) [10, 46].

Meanwhile, there have also been other public education and screening campaigns independent of those of the AAD. For instance, in New Mexico, following the establishment of a melanoma registry in 1980 for more consistent diagnosis and pathologic staging (e.g., in 1981, only 47% of the melanoma diagnoses had a documented Breslow depth), the proportion of Clark level I and II lesions increased from 38% in 1981 to 61% in 1985, and the proportion of melanomas with Breslow depth less than 1 mm increased from 46% in 1981 to 66% in 1985. This was attributed to the variety of educational programs instituted for both the public and various professionals, as well as the Registry's regular news letters to physicians and sporadic press releases via the public media [32].

The Arizona Cancer Center initiated the Arizona Sun Awareness Project after a 1979 report identified melanoma as a serious public health hazard in the region [312]. Over the years, this project has grown into a comprehensive, multifaceted community health promotion vehicle, using several original educational media (including a skin cancer

prevention videotape, a sun awareness brochure, a health fair display, and a puppet show accompanied by an activity booklet for children in grades kindergarten through 6) [217].

The evolution of the Arizona Sun Awareness Project has also culminated in two developmentally appropriate, age-based curricula aimed at teaching children about the benefits and dangers of the sun (given that excessive sun-exposure in early childhood has been linked to melanoma development later in adulthood) [217]. Of these programs, one, *Sunny Days, Healthy Ways* targeted elementary school children, while the other, *Be Sun Safe*, was aimed at pre-school children. The choice of the target populations was influenced by studies documenting that (1) 29-70% of adolescents never/do not regularly use sunscreen, despite an awareness of the rationale for its use [15b, 61, 147b, 258b]; and (2) even elementary school children who have received formal skin cancer prevention education, with clearly improved knowledge and attitude toward sun protection, have problems incorporating preventive behaviors into their lifestyles [48, 275]. Such data seemed to suggest the need for targeting children at younger, more impressionable ages, or initiating student-parent programs, to increase the likelihood of behavior modification [67, 218].

The *Sunny Days, Healthy Ways* curriculum, entailing a multi-disciplinary and comprehensive approach to education about the sun, skin cancer, and prevention strategies, while also attempting to instill a sense of control and responsibility for disease prevention, detection, and control, consisted of a series of five hour-long lessons in an interactive/active format for students in grades 4 through 6. Beyond the in-class lessons and activities, the project had take-home materials, a glossary, a review, and a student-parent newsletter [217]. The curriculum resulted in increased knowledge at both the immediate post-intervention

period, and eight weeks later. The lessons also reduced favorable attitudes toward tanning, while improving attitudes toward sunscreen use. Behavioral modifications were less consistent, with lower reports of suntanning, more frequent use of sunscreen among fourth graders, and increased use of protective clothing among firth and sixth graders [48]. Later, the *Sunny Days, Healthy Ways* messages were delivered to a similar population of students in a condensed, day-long *curriculum* format and a sun safety *health fair* format. The study results revealed the greatest improvement in knowledge among fourth graders in both formats. Also, not surprisingly, the day-long curriculum format was more efficacious than the health fair one, while the original (five-lesson) format was even more efficacious than these modified versions, suggesting that attitude and behavior modification require more intensive, continuous interventions [48, 217].

After the *Sunny Days, Healthy Ways* program, the investigators became curious to learn the youngest age at which sun safety education would have an effect [217]; this question led to the development of the *Be Sun Safe* project for pre-schoolers and their parents. This project emphasized developmentally appropriate, sun safety concepts such as "find shade", "cover up", and "ask for sun-safe things" (in three 45- to 50-minute units), while also trying to promote positive health habits such as self-protection from sunburns and taking care of one's body. It avoided the concept of "skin cancer", which would have been poorly understood and frightening in this age group. Thus, the curriculum included a section for teacher information, classroom activities (from engaging in theme-specific games, songs, and story-telling, a puppet show, etc.), take-home activities to do with care givers, a glossary, and a reference list of learning resources. The curriculum resulted in significantly increased

knowledge levels, with attitudes centering on not getting sunburned. However, there was no behavioral change, which had been expected by the investigators, given the target population's age [218].

The Under Cover Skin Cancer Prevention Project was a community-based program utilizing media partners (newspapers, radio, and television) in three Texas cities, to disseminate UV radiation readings (in the form of the minimal erythema dose) four times per day, along with education and behavioral change messages [41, 125]. This program documented significant changes in the behavioral practice of sun avoidance among the study sample over four months. However, it also drew attention to the need for constructs centered around peer pressure and social norms, which were documented to be key determinants of voluntary sun exposure [41].

Canada: The Canadian Dermatology Association (CDA) has been supervising the *Sun Awareness Program*, aimed at increasing public awareness of the dangers of UV radiation, as well as providing information on prevention and early detection [299]. This program entails an annual national press campaign (involving national, provincial, and community newspapers, periodicals, and French and English radio public service announcements), the distribution of educational materials (posters and brochures, including information to *all* mothers on the importance of sun-protection in newborns and children, as well as general information on protection from excessive sunlight and artificial sources of UV light), the organization of screening stations throughout the country during the "Sun Awareness Week", and finally, providing daily information on UV intensity through Environment Canada's UV index program.

To maintain public and media interest, the CDA has focused on an annual theme each year; past themes have included the importance of early detection, the importance of sun-protection during childhood, and sun protection by way of hats and proper clothing. Also, more recently (since 1992), the CDA has been launching a primary school education program targeting grades 1 through 3 (i.e., children 6 to 8 years of age). This program, entitled "Living with Sunshine", has enlisted the help of elementary school teachers. First piloted in five provinces, it is now an approved curriculum resource throughout Canada.

The CDA modeled its screening campaigns after the Australian beach "battle stations", starting in 1991 as a pilot study in Vancouver (with screenings on seven different weekends) and later expanded in scope, though limited to a single weekend. To monitor the screenings, questionnaire surveys were distributed to all participants; the surveys revealed the presence of at least one melanoma risk factor in 67% of the participants [299]. Overall, however, there is still a need for more formal and systematic evaluation of these intervention efforts.

The Netherlands: In the Netherlands, there have been public education campaigns, along with two screening programs [199, 273]. In one study, 2564 individuals were screened for skin cancer, leading to the discovery of 10 melanoma and 43 non-melanoma skin cancers [273]. In the second study, 3069 individuals were screened at four beach-side resorts, over 4 consecutive Saturdays, using a mobile trailer, leading to the diagnosis of 6 melanomas (all less than 1 mm in Breslow depth) [359].

Switzerland: The evaluation of the Swiss campaign, consisting of a national information program, revealed a doubling of the number of melanoma diagnoses in the two

months following the launch of the campaign, along with a shift in the case distribution toward younger ages (younger than 60 years of age) [49].

Austria: The first Austrian melanoma educational campaign was conducted in 1988. It involved not only professional education--through providing illustrated folders and publications on the clinical appearance and nature of pigmented lesions to all Austrian surgeons, dermatologists, and general practitioners--but also public education through the media--including articles and interviews in newspapers, broadcast television notices, and posters at physicians' offices, schools, and other public places [261]. The campaign resulted in a significant increase in the number of melanomas diagnosed, as well a significant decline in the median Breslow depth of the lesions (from 1.4 mm in the pre-campaign years, to 1.1 mm year in the year of the campaign, and 0.95 mm in the first post-campaign year). Also, there was a significant rise in the proportion of thin tumors (<0.75 mm) from the pre-campaign to the post-campaign years.

Other Methods of Melanoma Screening:

The skin cancer screening programs discussed above, exemplified by the American Academy of Dermatology, for instance, represent but one method of screening--i.e., episodic screening performed by dermatologists on self-selected populations [178]. Other methods of screening would also need to be instituted to more effectively control the escalating melanoma incidence and mortality rates.

Screening as Part of a Routine Medical Visit: Many have called for the integration of a thorough skin examination into the routine physical examinations provided by primary health practitioners [42, 75]. These serve as excellent opportunities for the early detection

of possible malignant lesions, in as much as approximately 85% of the U.S. population visits a physician every 2 years, and routine physical examinations are among the 10 most common reasons for seeing a physician [114, 157]. Also, about 7% of all outpatient visits are dermatologic in nature, while dermatologists see only a third of these disorders [322]. In one study, approximately half (53%) of the melanomas were self-detected, 26% were detected by medical providers, and 17% were detected by relatives; and of the subpopulation of melanomas discovered by a physician, only 12% were discovered by a dermatologist [185].

Experts differ in their stance regarding routine skin examinations (for early melanoma detection) in the context of medical visits. While some sources recommend routine skin examination of all adults, though more frequently for older adults, others leave it optional. Some recommend the training of all types of physicians, as well as nurses, paramedical personnel, medical students, and even, chiropractors and physical therapists, in the diagnosis of melanoma, while others fear poor sensitivity and specificity in the hands of non-dermatologists [51, 274, 347].

Meanwhile, from a practical standpoint, such a venture would face many obstacles. First, data have documented that an average visit to a family physician or general practitioner lasts 14 minutes, while the average visit to a general internist lasts 17 minutes [341]. In view of the growing demand on primary care services and the continued inadequate supply of primary care providers, an increase in the average length of time per visit, (to include a complete skin examination) would seem unlikely [357]. Second, many patients have multiple medical problems which would contribute to the lack of time for complete skin examinations [357]. Indeed, the need to review multiple active problems would detract

attention from such preventive measures as skin examination for skin cancer [24]. Third, the lack of reimbursement for preventive services represents a major obstacle to the practice of preventive skin examinations [357]. Finally, primary practitioners' lack of expertise in melanoma diagnosis and management might contribute to their unwillingness to provide complete skin examinations [357].

Self-Screening though Regular Skin Self-Examinations (SSE): There is growing publicity about SSEs (akin to the breast [210] and testicular self-examinations) for the early detection of melanoma. By performing SSEs, individuals assume a more active role in their health care, taking partial responsibility for identifying melanoma lesions at a potentially more curable stage. The proper method of performing SSEs has been detailed [116], and the AAD and the American Cancer Society provide additional educational pamphlets for patient information.

As cited earlier, in one study, more than half (53%) the melanomas were self-detected, and there was a significant association between regular SSEs and more frequent self-discovery of the melanomas ($p=0.02$) [185]. Such evidence implicates regular SSE for potential signs of melanoma as a particularly efficacious means of reducing melanoma mortality rates [30].

Others: Screening for melanoma could be organized through the workplace, for a more inclusive approach, or be targeted at high risk populations, such as those with the atypical nevus-melanoma syndrome. Examples of both have been described earlier, as with the Lawrence Livermore Laboratory and the NCI/UPenn prospective surveillance studies. Therefore, they shall not be discussed again. However, some experts [172] have indicated

their skepticism of the "target" approach to melanoma surveillance, believing the identified risks to be too small to be useful for the selection of an appropriate subpopulation, while also considering the proportion of cases in the low-risk populations to be substantial. Instead, they have called for unselective screening (while taking into account known risk factors), followed by grouping into high- and low-risk categories, which would later allow a risk-appropriate schedule of melanoma surveillance.

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Appendix

Table 1 - Case Control Studies on the Relationship Between Melanoma, and the Total Nevus Counts &/or Atypical Nevi.

Study	Country	Cases	Controls	Method of Count	Body Distribution	*Nevus* Definition	Nevus Count	RR
Reynolds, Austin, & Thomas, 1982[279b]	U.S.A.	13	Matched, 113	Interview	Unspecified	Nevi > 0.5 cm diam.	< 6 nevi ≥ 6 nevi	1.0 12.4
Beral, Evans, Shaw, & Milton, 1983[27]	Australia	287	Age-matched, 574	Interview: > average # nevi or not; Examination: by trained interviewer	Limbs	Unspecified. Average nevus count defined at 20.	Self-Report: ≤ average > average Examination: ≤ average > average	 1.0 3.5 1.0 3.7
Holman & Armstrong, 1984[161]	Australia	507	Sex-, Age-, & Area-of-Residence-Matched, 507	Examination: by interviewing nurse	Both arms, below level of axilla	Only palpable, papular nevi	None 1-4 5-9 ≥ 10	1.00 2.02 4.14 10.35
Sorahan & Grimley, 1985[320]	U. K.	58	Matched, 182 Unmatched, 151	Mailed Questionnaire	Right forearm	Unspecified	Unmatched: None 1-4 5-14 > 15 Unmtch'd/Adjst'd: None 1-4 5-14 > 15 Matched: None 1-4 5-14 > 15	 1.0 2.2 2.9 14.3 1.0 2.1 2.4 --- 1.0 1.1 2.2 4.3

Study	Country	Cases	Controls	Method of Count	Body Distribution	*Nevus* Definition	Nevus Count	RR
Green, MacLennan, & Siskind, 1985[135]	Australia	183	Sex-, Age-, & Area-of-Residence-Matched, 183	Examination by dermatologist	Left arm below tip of shoulder	Macular or papular, pigmented, ≥ 2mm	None 2-4 5-10 > 10 Any # Nevi Any # Nevi **	1.0 15.7 14.9 20.1 15.8 30.1**
							**Adjusted for total sun exposure	
Nordlund, Kirkwood, Forget, Scheibner, Albert, Lerner, & Milton, 1985[258]	Australia	103	Age-Matched, 145	Examination: by dermatologist, oncologist, & nurse, but count by only one	Total body	Macular or Papular Nevi ≥ 3 mm	0-30 > 30	1.00 1.60
Elwood, Williamson, & Stapleton, 1986[100]	U. K.	83	Sex-, Age-, & Area-of-Residence-Matched, 83	Interview: Self-estimate of # nevi; Examination	Each upper arm, to the shoulder	Only palpable, papular nevi	Self-Report: <14 ≥ 15 Examination: None 1-2 ≥ 3	 1.0 6.7 1.0 1.8 17.0
Swerdlow, English, MacKie, O'Doherty, Hunter, Clark, & Hole, 1986[329]	Scotland	180	Sex-, Age-, & Area-of-Residence-Matched, 197	Examination: by dermatolgist	Total body, except for scalp (noted presence or absence) and genitalia	Pigmented nevi, > 2 mm; any unusual features were noted	None 1-9 10-24 25-49 ≥ 50 # Nevi ≥ 7 mm: None 1-4 ≥ 5 Color Variegation Irregular Edge	1.0 0.9 4.4 8.7 63.8 1.0 5.1 6.0 29.6 72.7

Study	Country	Cases	Controls	Method of Count	Body Distribution	*Nevus* Definition	Nevus Count	RR
Dubin, Moseson, & Pasternack, 1986[82]	U.S.A.	1103	Random Sample, 585	Interview: pick most representative mole diagram &/or Examination: by melanoma fellow	Total body	Unspecified	None 1-25 26-100 > 100	0.18 1.00 1.98 3.43
Holly, Kelly, Shpall, & Chiu, 1987[159]	U.S.A.	121	Sex- & Age-Matched, 139	Examination: by a dermatologist and dermatology fellow	Total body, except for the scalp & genitals	Nevi ≥ 2 mm diam.	0-10 11-25 26-50 51-100 > 100	1.0 1.6 4.4 5.4 9.8
Cristofolini, Franceschi, & Tasin, 1987[68]	Italy	103	Sex- & Age-Matched, 205	Examination: by dermatologist	Unspecified, but total body implied.	Unspecified	None 1-4 ≥ 5	1.00 1.50 1.20
Osterlind, Tucker, Hou-Jensen, Stone, Engholm, & Jensen, 1988[259]	Denmark	474	Sex- & Age-Matched, 926	Examination: by trained interviewer	Arm nevi, below level of axilla	Palpable, pigmented nevi, ≥ 5 mm or < 5 mm	Total nevi: None One 2-4 ≥ 5	 1.0 1.5 2.3 5.4
							Nevi < 5 mm: None One 2-4 ≥ 5	 1.0 1.6 2.5 5.0
							Nevi >= 5 mm: None One ≥ 2	 1.0 1.8 3.6
Elwood, Whitehead, Davison, Stewart, & Galt, 1990[103]	U. K.	195	Sex-, Age-, & Area-of-Residence-Matched, 195	Examination: by trained interviewer	Both arms, from wrist to shoulder	Pigmented Nevi ≥ 3 mm	None One 2-4 5-9 10-14 > 15	1.00 1.30 1.60 3.00 3.30 5.20

Study	Country	Cases	Controls	Method of Count	Body Distribution	*Nevus* Definition	Nevus Count	RR
Grob, Gouvernet, Aymar, Mostaque, Romano, Collet, Noe, Diconstanzo, & Bonerandi, 1990[147]	France	207	Sex- & Age-Matched, 295	Two separate nevus counts, once by a specially trained resident, once by a dermatologist	Total body, with special attention to buttocks (sun-shielded) & left forearm (sun-exposed) counts	All darkly pigmented nevi > 1 mm	Total # Nevi:	
							0-10	1.00
							11-20	1.30
							21-40	2.05
							41-80	2.53
							81-120	3.94
							> 120	9.53
							Nevi 1 to 5 mm:	
							0-10	1.00
							11-20	1.33
							21-40	1.91
							41-80	2.64
							81-120	3.89
							> 120	10.89
							Nevi 5 to 10 mm:	
							None 1	1.00
							4 ≥	1.08
							5	8.52
							Buttocks Count:	
							None	1.00
							One	0.97
							2-4	2.65
							≥ 5	10.86
							Forearm Count:	
							None	1.00
							1-3	1.06
							≥ 4	2.28
							Clinical AN:	
							None	1.00
							One	1.20
							> 1	4.05
Weiss, Bertz, & Jung, 1991[356]	Germany	204	Sex- & Age-Matched, 200	Examination	Total body	Nevi > 2 mm diam.	< 10	1.0
							10-50	4.3
							> 50	14.9

Study	Country	Cases	Controls	Method of Count	Body Distribution	*Nevus* Definition	Nevus Count	RR
Kruger, Garbe, Buttner, Stadler, Guggenmoos-Holzmann, & Orfanos, 1992[200]	Germany	200	Sex- & Age-Matched, 200	Examination: by two dermatologists	Total body, separated into 5 regions, excluding the scalp & genitals	Pigmented Nevi ≥ 2 mm	Head & Neck [M]:	
							≤ 4	1.00
							> 4	2.30
							Head & Neck [F]:	
							≤ 4	1.00
							> 4	4.70
							Arms [M]:	
							0-4	1.00
							5-10	1.30
							11-20	8.00
							> 20	19.0
							Arms [F]:	
							0-4	1.00
							5-10	1.50
							11-20	2.30
							> 20	5.20
							Legs [M]:	
							0-4	1.00
							5-10	3.30
							11-20	9.20
							> 20	27.40
							Legs [F]:	
							0-4	1.00
							5-10	1.70
							11-20	9.30
							> 20	14.90
							Trunk [M]:	
							0-4	1.00
							5-10	2.90
							11-20	5.50
							> 20	32.60
							(> 40)	(133.40)
							Trunk [F]:	
							0-4	1.00
							5-10	0.80
							11-20	3.80
							> 20	6.10

Table 2- Clinics Through Which Subjects were Recruited, and the Subjects' Methods of Follow-up.

		GROUPS				
		MELANOMA	NEVUS	CONTROL	TOTAL	TOTAL
TOTAL # PTS INITIALLY	PLC				69 (73%)	
	UGDC				18 (19%)	94
	HGDC				7 (7%)	
TOTAL # PTS AT COMPLETION	PLC	33 (100%)	31 (94%)	2 (10%)	66 (77%)	
	UGDC	-	2 (6%)	15 (75%)	17 (20%)	86
	HGDC	-	-	3 (15%)	3 (3%)	
METHOD OF COMPLETION	MAIL	29 (88%)	27 (82%)	10 (50%)	66 (77%)	
	CLINIC	2 (6%)	3 (9%)	4 (20%)	9 (10%)	86
	PHONE	2 (6%)	3 (9%)	6 (30%)	11 (13%)	

Table 3 - Demographic and Phenotypic Characteristics

GROUPS		MELANOMA	NEVUS	CONTROL	P-VALUE
CLINICAL CHARACTERISTICS					
SEX	FEMALE	20 (61%)	25 (76%)	14 (70%)	
	MALE	13 (39%)	8 (24%)	6 (30%)	-
AGE	18-35	6 (18%)	15 (45%)	3 (15%)	
	36-50	13 (39%)	13 (39%)	4 (20%)	0.001
	51-85	14 (42%)	5 (15%)	13 (65%)	
AGE, MEAN		47 (A)	38 (B)	54 (A)	
OCCUPATIONAL SCORE, MEAN		6 (A)	7 (A)	6 (A)	
	MAJOR/INTRMED PROFESS'L	6 (18%)	9 (27%)	4 (21%)	
	MINOR/SEMI- PROFESS'L	18 (55%)	18 (55%)	10 (53%)	
	CLRK/SALES/SKILLED + SEMI-	5 (15%)	1 (3%)	2 (10%)	-
	UNSKILLED/MENIAL SERVICE	1 (3%)	0	1 (5%)	
	UNKNOWN	3 (9%)	5 (15%)	2 (10%)	
HAIR	BLOND/RED	12 (36%)	8 (24%)	4 (20%)	
	L. BROWN	11 (33%)	4 (12%)	5 (25%)	-
	BROWN/BLACK	10 (30%)	21 (64%)	11 (55%)	
	BLUE/GREEN	21 (64%)	15 (45%)	8 (40%)	
EYES	HAZEL/GREY	6 (18%)	5 (15%)	3 (15%)	
	L./D. BROWN	6 (18%)	13 (39%)	8 (40%)	-
	DK	0	0	1 (5%)	
SKIN TYPE** BURNABILITY	BLISTER'G SUNBURN	3 (9%)	2 (6%)	2 (10%)	
	PAINFUL SUNBURN	15 (45%)	13 (39%)	7 (35%)	
	MILD BURN W/ LITTLE TAN	15 (45%)	17 (52%)	7 (35%)	-
	TAN W/O SUNBURN	0	1 (3%)	2 (10%)	
	DK	0	0	2 (10%)	
SKIN TYPE** INABILITY TO TAN	FRECKLES/NO TAN	3 (9%)	3 (9%)	2 (10%)	
	MILD TAN W/ H/O PEELING	7 (21%)	5 (15%)	3 (15%)	
	MODERATE TAN	21 (64%)	19 (58%)	9 (45%)	-
	DEEP, BROWN TAN	2 (6%)	5 (15%)	4 (21%)	
	DK	0	1 (3%)	2 (10%)	

** Based on a patient's own *subjective* assessment of his/her acute and chronic reaction to sunlight.

Table 4 - Malignant Melanoma Risk Factors of Subjects in the Study.

GROUPS		MELANOMA	NEVUS	CONTROL	P-VALUE
MM RISK FACTORS					
OBJ # NEVI	**0-33**	20 (61%)	12 (36%)	19 (100%)[1]	
	34-66	10 (30%)	12 (36%)	0	0.001
	67-100 & >100	3 (9%)	9 (27%)	0	
SUBJ # NEVI	**0-33**	19 (58%)	13 (39%)	17 (85%)	
	34-66	7 (21%)	4 (12%)	1 (2%)	0.001
	67-100 & >100	7 (21%)	16 (48%)	2 (10%)	
% Correct		17 (52%)	18 (55%)	17 (85%)	0.051
MORE NEVI?	**YES**	12 (36%)	23 (70%)	4 (20%)	
	NO	16 (48%)	8 (24%)	14 (70%)	-
	DK	5 (15%)	2 (6%)	2 (10%)	
% Correct		24 (73%)	27 (82%)	14 (70%)	-
PHx A. NEVI	**YES**	22 (67%)	33 (100%)	1 (5%)[2]	
	NO	11 (33%)	-	19 (95%)	0.001
PHx NMSCa	**YES**	5 (15%)	5 (15%)	4 (20%)	
	NO	28 (85%)	27 (82%)	15 (75%)	-
	DK	-	2 (6%)	1 (5%)	
FHx MM	**YES**	9 (27%)	14 (42%)	3 (15%)	
	BY SELF-REPORT	3 (33%)	4 (29%)	3 (100%)	
	DOCUMENTED	6 (67%)	10 (71%)	-	-
	NO	22 (67%)	15 (45%)	14 (70%)	
	DK	2 (6%)	4 (12%)	3 (15%)	
FHx NMSCa	**YES**	16 (48%)	11 (33%)	4 (20%)	
	NO	11 (33%)	19 (58%)	10 (50%)	-
	DK	6 (18%)	3 (9%)	6 (30%)	

1-One subject lacks an objective nevus count as a result of being recruited through a dermatologic surgery UGDC.

2-A control, followed in the university general dermatology clinic for the diagnoses of lupus and psoriasis, incidentally documented to have an AN.

Table 5 - Analysis of Baseline & Follow-up Knowledge of Specific Melanoma Facts By Group.

	BEFORE				AFTER				SIGNIFICANT CHANGE?			SIGNIFICANT CHANGE?			
GROUPS	MELANOMA	NEVUS	CONTROL	P-VAL	MELANOMA	NEVUS	CONTROL	P-VAL	MELNM	NEVUS	CONTRL	MELNM	NEVUS	CONTRL	P-VALUE
KNOWLEDGE QUESTIONS									MEANS	MEANS	MEANS	INCREASE	INCREASE	INCREASE	
CHNG SHAP/COLR/SIZE	33 (100%)	33 (100%)	18 (90%)	0.034	32 (97%)	31 (94%)	19 (95%)	-	/	/	/	0	0	1 (5%)	-
ABNL SHAPE	33 (100%)	32 (97%)	13 (65%)	0.001	32 (97%)	32 (97%)	16 (80%)	0.033	/	/	/	0	1 (3%)	5 (25%)	0.007
DARK/VAR COLOR	32 (97%)	30 (91%)	13 (65%)	0.018	33 (100%)	31 (94%)	17 (85%)	-	/	/	/	1 (3%)	3 (9%)	5 (25%)	-
INCR'D THICKNESS	27 (82%)	31 (94%)	14 (70%)	0.021	27 (82%)	27 (82%)	17 (85%)	-	/	/	/	5 (15%)	1 (3%)	5 (25%)	0.018
NON-HEALING LESION	24 (73%)	30 (91%)	17 (85%)	-	23 (70%)	31 (94%)	19 (95%)	-	/	/	/	3 (9%)	1 (3%)	3 (15%)	-
BLEEDING	24 (73%)	29 (88%)	9 (45%)	0.001	23 (70%)	31 (94%)	14 (70%)	0.021	B	B	A	0	2 (6%)	6 (30%)	0.013
TENDERNESS	23 (70%)	23 (70%)	9 (45%)	-	19 (58%)	24 (73%)	15 (75%)	-	B	B	A	0	5 (15%)	7 (35%)	-
ITCHING	18 (55%)	25 (76%)	4 (20%)	0.001	19 (58%)	27(82%)	7 (35%)	0.001	/	/	/	4 (12%)	4 (12%)	7 (35%)	0.053
LARGE	13 (39%)	23 (70%)	3 (15%)	0.001	15 (45%)	22 (67%)	8 (40%)	0.045	B	C	A	5 (15%)	1 (3%)	5 (25%)	0.018
SURVEY BACK NEVI	18 (55%)	15 (45%)	6 (30%)	-	25 (76%)	19 (58%)	16 (80%)	-	B	C	A	10 (30%)	9 (27%)	11 (55%)	0.056
CHILDHOOD SUNBURNS	32 (97%)	32 (97%)	18 (90%)	-	32 (97%)	33 (100%)	19 (95%)	-	/	/	/	1 (3%)	1 (3%)	2 (10%)	-
CURE BY EARLY DISC	33 (100%)	29 (88%)	18 (90%)	-	33 (100%)	31 (94%)	17 (85%)	-	/	/	/	0	2 (6%)	1 (5%)	-
FHx MM	30 (91%)	27 (82%)	16 (80%)	-	32 (97%)	30 (91%)	17 (85%)	-	/	/	/	3 (9%)	3 (9%)	3 (15%)	-
ITCHY MOLE	22 (67%)	29 (88%)	15 (75%)	-	23 (70%)	29 (88%)	14 (70%)	-	/	/	/	4 (12%)	2 (6%)	3 (15%)	-
?MM=BLACK & HAIRY?	29 (88%)	29 (88%)	10 (50%)	0.003	31 (94%)	27 (82%)	12 (60%)	-	/	/	/	2 (6%)	1 (3%)	5 (25%)	0.013
DISAPP'ING MOLE	5 (15%)	14 (42%)	1 (5%)	0.001	9 (27%)	16 (48%)	3 (15%)	0.009	/	/	/	6 (18%)	5 (15%)	3 (15%)	-
CHANGING MOLE	33 (100%)	32 (97%)	20 (100%)	-	33 (100%)	32 (97%)	20 (100%)	-	/	/	/	0	0	0	-
BLUE EYES/RED HAIR	33 (100%)	32 (97%)	19 (95%)	-	33 (100%)	32 (97%)	20 (100%)	-	/	/	/	0	0	1 (5%)	-
FHx MM x2	33 (100%)	32 (97%)	19 (95%)	-	33 (100%)	31 (94%)	19 (95%)	-	/	/	/	0	0	0	-
BURNS, NEVER TANS	32 (97%)	33 (100%)	18 (90%)	-	33 (100%)	33 (100%)	20 (100%)	-	/	/	/	1 (3%)	0	2 (10%)	-
F/PHx & MANY MOLES	32 (97%)	32 (97%)	19 (95%)	-	33 (100%)	33 (100%)	20 (100%)	-	/	/	/	1 (3%)	1 (3%)	1 (5%)	-
PHx MM	31 (94%)	33 (100%)	19 (95%)	-	31 (94%)	33 (100%)	20 (100%)	-	/	/	/	1 (3%)	1 (3%)	1 (5%)	-
NO MOLES/DARK HAIR	33 (100%)	31 (94%)	15 (75%)	0.039	33 (100%)	33 (100%)	20 (100%)	-	B	B	A	0	2 (6%)	5 (25%)	0.039
LARGE/ODD MOLES	31 (94%)	32 (97%)	17 (85%)	-	31 (94%)	33 (100%)	18 (90%)	-	/	/	/	1 (3%)	1 (3%)	3 (15%)	-
>50 NEVI	30 (91%)	31 (94%)	19 (95%)	-	30 (91%)	32 (97%)	18 (90%)	-	/	/	/	2 (6%)	1 (3%)	0	-
BLACK SKIN	27 (82%)	27 (82%)	16 (80%)	-	33 (100%)	28 (85%)	16 (80%)	-	/	/	/	3 (9%)	2 (6%)	1 (5%)	-

Table 6 - Analysis of Baseline & Follow-up Knowledge of Specific Melanoma Facts By Clinic.

	BEFORE			AFTER			SIGN'T CHANGE?		SIGN'T CHANGE?		
GROUPS	PLC	CONTROL	P-VALUE	PLC	CONTROL	P-VALUE	PLC	CONTRL	PLC	CONTRL	P-VAL
KNOWLEDGE QUESTIONS							MEANS	MEANS	INCREASE	INCREASE	
CHNG SHAP/COLR/SIZE	66 (100%)	18 (90%)	0.01	63 (95%)	19 (95%)	-	/	/	0	1 (5%)	-
ABNL SHAPE	65 (98%)	13 (65%)	0.001	64 (97%)	16 (80%)	0.009	/	/	1 (2%)	5 (25%)	0.001
DARK/VAR COLOR	62 (94%)	13 (65%)	0.001	64 (97%)	17 (85%)	0.046	/	/	4 (6%)	5 (25%)	0.016
INCR'D THICKNESS	58 (88%)	14 (70%)	-	54 (82%)	17 (85%)	-	/	/	6 (9%)	5 (25%)	-
NON-HEALING LESION	54 (82%)	17 (85%)	-	54 (82%)	19 (95%)	-	/	/	4 (6%)	3 (15%)	-
BLEEDING	53 (80%)	9 (45%)	0.002	54 (82%)	14 (70%)	-	B	A	2 (3%)	6 (30%)	0.001
TENDERNESS	46 (70%)	9 (45%)	0.045	43 (65%)	15 (75%)	-	B	A	5 (8%)	7 (35%)	0.002
ITCHING	43 (65%)	4 (20%)	0.001	46 (70%)	7 (35%)	0.005	/	/	8 (12%)	7 (35%)	0.019
LARGE	36 (55%)	3 (15%)	0.002	37 (56%)	8 (40%)	-	B	A	6 (9%)	5 (25%)	-
SURVEY BACK NEVI	33 (50%)	6 (30%)	-	44 (67%)	16 (80%)	-	B	A	19 (29%)	11 (55%)	0.032
CHILDHOOD SUNBURNS	64 (97%)	18 (90%)	-	65 (98%)	19 (95%)	-	/	/	2 (3%)	2 (10%)	-
CURE BY EARLY DISC	62 (94%)	18 (90%)	-	64 (97%)	17 (85%)	0.046	/	/	2 (3%)	1 (5%)	-
FHx MM	57 (86%)	16 (80%)	-	62 (94%)	17 (85%)	-	/	/	6 (9%)	3 (15%)	-
ITCHY MOLE	51 (77%)	15 (75%)	-	52 (79%)	14 (70%)	-	/	/	6 (9%)	3 (15%)	-
?MM=BLACK & HAIRY?	58 (88%)	10 (50%)	0.001	58 (88%)	12 (60%)	0.005	/	/	3 (5%)	5 (25%)	0.006
DISAPP'ING MOLE	19 (29%)	1 (5%)	0.028	25 (38%)	3 (15%)	-	/	/	11 (17%)	3 (15%)	-
CHANGING MOLE	65 (98%)	20 (100%)	-	65 (98%)	20 (100%)	-	/	/	0	0	-
BLUE EYES/RED HAIR	65 (98%)	19 (95%)	-	65 (98%)	20 (100%)	-	/	/	0	1 (5%)	-
FHx MM x2	65 (98%)	19 (95%)	-	64 (97%)	19 (95%)	-	/	/	0	0	-
BURNS, NEVER TANS	65 (98%)	18 (90%)	-	66 (100%)	20 (100%)	-	/	/	1 (2%)	2 (10%)	-
F/PHx & MANY MOLES	64 (97%)	19 (95%)	-	66 (100%)	20 (100%)	-	/	/	2 (3%)	1 (5%)	-
PHx MM	64 (97%)	19 (95%)	-	64 (97%)	20 (100%)	-	/	/	1 (2%)	1 (5%)	-
NO MOLES/DARK HAIR	64 (97%)	15 (75%)	0.002	66 (100%)	20 (100%)	-	B	A	2 (3%)	5 (25%)	0.002
LARGE/ODD MOLES	63 (95%)	17 (85%)	-	64 (97%)	18 (90%)	-	/	/	2 (3%)	3 (15%)	0.046
>50 NEVI	61 (92%)	19 (95%)	-	62 (94%)	18 (90%)	-	/	/	3 (5%)	0	-
BLACK SKIN	54 (82%)	16 (80%)	-	55 (83%)	16 (80%)	-	/	/	5 (8%)	1 (5%)	-

Table 7 - Group Knowledge Scores Stratified into Percentiles.

		MELANOMA	NEVUS	CONTROL	PLC
SCORE1	**0%**	0	0	1 (5%)	0
	1% - 37%	1 (3%)	0	2 (10%)	1 (2%)
	38% - 74%	15 (45%)	13 (39%)	16 (80%)	28 (42%)
	75% - 100%	17 (52%)	20 (61%)	1 (5%)	37 (56%)
P-value			0.001		0.001
SCORE1A	**0%**	0	0	0	0
	1% - 37%	1 (3%)	2 (6%)	1 (5%)	3 (5%)
	38% - 74%	18 (55%)	11 (33%)	14 (70%)	29 (44%)
	75% - 100%	14 (42%)	20 (61%)	5 (25%)	34 (52%)
P-value			0.036		-
SCORE2	**0%**	0	0	0	0
	1% - 37%	3 (9%)	2 (6%)	4 (20%)	5 (8%)
	38% - 74%	16 (48%)	16 (48%)	12 (60%)	32 (48%)
	75% - 100%	14 (42%)	15 (45%)	4 (20%)	29 (44%)
P-value			0.042		0.027
SCORE2A	**0%**	0	0	0	0
	1% - 37%	0	1 (3%)	3 (15%)	1 (2%)
	38% - 74%	14 (42%)	12 (36%)	11 (55%)	26 (39%)
	75% - 100%	19 (58%)	20 (61%)	6 (30%)	39 (59%)
P-value			0.02		0.005
SCORE3	**0%**	0	0	0	0
	1% - 37%	0	1 (3%)	0	1 (2%)
	38% - 74%	3 (9%)	2 (6%)	6 (30%)	5 (8%)
	75% - 100%	30 (91%)	30 (91%)	14 (70%)	60 (91%)
P-value			-		0.052
SCORE3A	**0%**	0	0	0	0
	1% - 37%	0	0	0	0
	38% - 74%	4 (12%)	1 (3%)	2 (10%)	5 (8%)
	75% - 100%	29 (88%)	32 (97%)	18 (90%)	61 (92%)
P-value			-		-
SCORE4	**0%**	0	0	0	0
	1% - 37%	0	0	1 (5%)	0
	38% - 74%	12 (36%)	6 (18%)	13 (65%)	18 (27%)
	75% - 100%	21 (64%)	27 (82%)	6 (30%)	48 (73%)
P-value			0.001		0.001
SCORE4A	**0%**	0	0	0	0
	1% - 37%	0	0	0	0
	38% - 74%	13 (39%)	4 (12%)	7 (35%)	17 (26%)
	75% - 100%	20 (61%)	29 (88%)	13 (65%)	49 (74%)
P-value			0.04		-

Table 8a - Knowledge Scores as a Function of Group & Clinic.

GROUPS	MELANOM'	NEVUS	SCORE1 CONTROL	PLC	MELANOM'	NEVUS	SCORE2 CONTROL	PLC
BEFORE	6.9 [A]	7.8 [A]	5.0 [B]		5.1 [A]	5.3 [A]	4.2 [B]	
AFTER	6.8 [B]	7.8 [A]	6.6 [B]		5.6 [A]	5.6 [A]	4.9 [B]	
IMPROVEM'T	-0.1 [B]	0 [B]	1.6 [A]		0.5 [-]	0.3 [-]	0.7 [-]	
%INCREASE	11 (33%)	6 (18%)	13 (65%)	17 (26%)	15 (45%)	12 (36%)	12 (60%)	27 (41%)
P-value		0.001		0.001		-		-

GROUPS	MELANOM'	NEVUS	SCORE3 CONTROL	PLC	MELANOM'	NEVUS	SCORE4 CONTROL	PLC
BEFORE	9.6 [-]	9.6 [-]	9.1 [-]		21.6 [A]	22.6 [A]	18.3 [B]	
AFTER	9.6 [-]	9.7 [-]	9.6 [-]		22.0 [B]	23.1 [A]	21.1 [C]	
IMPROVEM'T	0.1 [-]	0.2 [-]	0.5 [-]		0.4 [B]	0.5 [B]	2.8 [A]	
%INCREASE	8 (24%)	5 (15%)	6 (30%)	13 (20%)	18 (55%)	16 (48%)	14 (70%)	34 (52%)
P-value		-		-		-		-

Table 8b - Knowledge Scores as a Function of Sex.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		FEMALE	7.1 [-]	7.8 [-]	4.9 [-]	7.5 [-]	6.9 [-]	5.4 [-]	5.4 [-]	4.2 [-]	5.4 [-]	5.1 [-]
		MALE	6.5 [-]	7.6 [-]	5.2 [-]	6.9 [-]	6.5 [-]	4.8 [-]	4.9 [-]	4.2 [-]	4.8 [-]	4.7 [-]
AFTER	SEX	FEMALE	7.2 [-]	7.8 [-]	6.5 [-]	7.5 [-]	7.3 [-]	5.7 [-]	5.8 [-]	5.1 [-]	5.7 [-]	5.6 [A]
		MALE	6.1 [-]	7.6 [-]	6.8 [-]	6.7 [-]	6.7 [-]	5.5 [-]	5.1 [-]	4.3 [-]	5.3 [-]	5.1 [B]
IMPROVEM'T		FEMALE	0.1 [-]	0 [-]	-1.6 [-]	0 [-]	0.4 [-]	0.4 [-]	0.3 [-]	0.9 [-]	0.3 [-]	0.5 [-]
		MALE	-0.4 [-]	0 [-]	1.7 [-]	-0.2 [-]	0.2 [-]	0.7 [-]	0.3 [-]	0.2 [-]	0.5 [-]	0.4 [-]
%INCREASE		FEMALE	8 (40%)	4 (16%)	8 (57%)	12 (27%)	20 (34%)	8 (40%)	10 (40%)	9 (64%)	18 (40%)	27 (46%)
		MALE	3 (23%)	2 (25%)	5 (83%)	5 (24%)	10 (37%)	7 (54%)	2 (25%)	3 (50%)	9 (43%)	12 (44%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		FEMALE	9.6 [-]	9.6 [-]	8.9 [-]	9.6 [-]	9.4 [-]	22.1 [-]	22.8 [-]	18.1 [-]	22.5 [-]	21.4 [-]
		MALE	9.5 [-]	9.4 [-]	9.3 [-]	9.5 [-]	9.4 [-]	20.8 [-]	21.9 [-]	18.7 [-]	21.2 [-]	20.6 [-]
AFTER	SEX	FEMALE	9.8 [-]	9.7 [-]	9.7 [-]	9.7 [-]	9.7 [-]	22.7 [-]	23.3 [-]	21.4 [-]	23.0 [A]	22.6 [A]
		MALE	9.4 [-]	9.6 [-]	9.2 [-]	9.5 [-]	9.4 [-]	20.9 [-]	22.4 [-]	20.3 [-]	21.5 [B]	21.2 [B]
IMPROVEM'T		FEMALE	0.2 [-]	0.1 [-]	0.8 [-]	0.2 [-]	0.3 [-]	0.6 [-]	0.4 [-]	3.3 [-]	0.5 [-]	1.2 [-]
		MALE	-0.2 [-]	0.3 [-]	-0.2 [-]	0 [-]	0 [-]	0.2 [-]	0.5 [-]	1.7 [-]	0.3 [-]	0.6 [-]
%INCREASE		FEMALE	6 (30%)	3 (12%)	5 (36%)	9 (20%)	14 (24%)	13 (65%)	13 (52%)	10 (71%)	26 (58%)	36 (61%)
		MALE	2 (15%)	2 (25%)	1 (17%)	4 (19%)	5 (19%)	5 (38%)	3 (38%)	4 (67%)	8 (38%)	12 (44%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

Table 8c - Knowledge Scores as a Function of Age Group.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		18-35	6.0 [-]	8.2 [A]	6.7 [A]	7.6 [-]	7.5 [A]	4.5 [-]	5.8 [A]	3.7 [-]	5.4 [-]	5.2 [-]
		36-50	6.8 [-]	7.8 [A]	3.5 [C]	7.3 [-]	6.8 [B]	5.2 [-]	5.2 [B]	3.3 [-]	5.2 [-]	4.9 [-]
		51-85	7.4 [-]	6.2 [B]	5.1 [B]	7.1 [-]	6.3 [C]	5.4 [-]	4.2 [C]	4.6 [-]	5.1 [-]	4.9 [-]
AFTER	AGE	18-35	6.0 [-]	8.2 [A]	7.0 [A]	7.6 [-]	7.5 [-]	5.2 [-]	5.8 [A]	5.0 [-]	5.6 [B]	5.5 [-]
		36-50	6.7 [-]	7.8 [A]	4.8 [B]	7.3 [-]	6.9 [-]	5.8 [-]	5.8 [A]	4.8 [-]	5.8 [A]	5.7 [-]
		51-85	7.1 [-]	6.2 [B]	7.1 [A]	6.9 [-]	7.0 [-]	5.6 [-]	4.4 [B]	4.9 [-]	5.3 [C]	5.2 [-]
IMPROVEM'T		18-35	0 [-]	0 [-]	0.3 [-]	0 [-]	0 [-]	0.7 [-]	0 [-]	1.3 [-]	0.2 [-]	0.3 [-]
		36-50	-0.1 [-]	0 [-]	1.3 [-]	0 [-]	0.1 [-]	0.7 [-]	0.2 [-]	1.5 [-]	0.7 [-]	0.8 [-]
		51-85	-0.2 [-]	0 [-]	2.0 [-]	-0.2 [-]	0.7 [-]	0.2 [-]	0.7 [-]	0.3 [-]	0.2 [-]	0.3 [-]
%INCREASE		18-35	3 (50%)	2 (13%)	1 (33%)	5 (24%)	6 (25%)	3 (50%)	3 (20%)	3 (100%)	6 (29%)	9 (38%)
		36-50	3 (23%)	3 (23%)	3 (75%)	6 (23%)	9 (30%)	7 (54%)	6 (46%)	3 (75%)	13 (50%)	16 (53%)
		51-85	5 (36%)	1 (20%)	9 (69%)	6 (32%)	15 (47%)	5 (36%)	3 (60%)	6 (46%)	8 (42%)	14 (44%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		18-35	9.8 [-]	9.8 [A]	9.3 [A]	9.8 [-]	9.8 [-]	20.3 [-]	23.8 [A]	19.7 [A]	22.8 [-]	22.4 [A]
		36-50	9.5 [-]	9.7 [A]	7.5 [B]	9.6 [-]	9.3 [-]	21.4 [-]	22.7 [A]	14.3 [C]	22.0 [-]	21.0 [B]
		51-85	9.5 [-]	8.4 [B]	9.5 [A]	9.2 [-]	9.3 [-]	22.2 [-]	18.8 [B]	19.2 [B]	21.3 [-]	20.4 [C]
AFTER	AGE	18-35	9.7 [-]	9.9 [A]	9.0 [B]	9.8 [-]	9.7 [-]	20.8 [-]	23.9 [A]	21.0 [B]	23.0 [-]	22.8 [-]
		36-50	9.6 [-]	9.8 [A]	8.8 [C]	9.7 [-]	9.6 [-]	22.2 [-]	23.5 [A]	18.3 [C]	22.8 [-]	22.2 [-]
		51-85	9.6 [-]	9.0 [B]	9.9 [A]	9.4 [-]	9.6 [-]	22.3 [-]	19.6 [B]	21.9 [A]	21.6 [-]	21.7 [-]
IMPROVEM'T		18-35	-0.2 [-]	0.1 [-]	-0.3 [-]	0 [-]	0 [-]	0.5 [-]	0.1 [-]	1.3 [-]	0.2 [-]	0.3 [-]
		36-50	0.2 [-]	0.1 [-]	1.3 [-]	0.1 [-]	0.3 [-]	0.8 [-]	0.8 [-]	4.0 [-]	0.8 [-]	1.2 [-]
		51-85	0.1 [-]	0.6 [-]	0.5 [-]	0.2 [-]	0.3 [-]	0.1 [-]	0.8 [-]	2.8 [-]	0.3 [-]	1.3 [-]
%INCREASE		18-35	1 (17%)	2 (13%)	0	3 (14%)	3 (13%)	2 (33%)	5 (33%)	2 (67%)	7 (33%)	9 (38%)
		36-50	3 (23%)	1 (8%)	2 (50%)	4 (15%)	6 (20%)	9 (69%)	7 (54%)	3 (75%)	16 (62%)	19 (63%)
		51-85	4 (29%)	2 (40%)	4 (31%)	6 (32%)	10 (31%)	7 (50%)	4 (80%)	9 (69%)	11 (58%)	20 (63%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

Table 8d - Knowledge Scores as a Function of Hair Color.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		BLOND/RED	6.8 [-]	7.1 [-]	4.8 [-]	7.0 [-]	6.6 [-]	5.3 [A]	5.0 [-]	4.0 [-]	5.2 [-]	5.0 [-]
		L. BROWN	7.2 [-]	8.0 [-]	4.2 [-]	7.4 [-]	6.6 [-]	5.5 [A]	6.0 [-]	4.6 [-]	5.7 [-]	5.4 [-]
		BRWN/BLCK	6.6 [-]	8.0 [-]	5.5 [-]	7.5 [-]	7.0 [-]	4.4 [B]	5.3 [-]	4.1 [-]	5.0 [-]	4.8 [-]
AFTER		BLOND/RED	7.0 [-]	6.8 [-]	8.5 [A]	6.9 [-]	7.2 [-]	5.5 [-]	5.6 [-]	5.8 [-]	5.6 [-]	5.6 [-]
	HAIR	L. BROWN	7.2 [-]	8.0 [-]	6.2 [B]	7.4 [-]	7.1 [-]	5.9 [-]	5.5 [-]	4.6 [-]	5.8 [-]	5.5 [-]
		BRWN/BLCK	6.0 [-]	8.1 [-]	6.1 [B]	7.4 [-]	7.1 [-]	5.4 [-]	5.6 [-]	4.7 [-]	5.5 [-]	5.3 [-]
IMPROVEM'T		BLOND/RED	0.2 [-]	-0.4 [-]	3.8 [A]	-0.1 [-]	0.6 [-]	0.2 [-]	0.6 [-]	1.8 [-]	0.4 [-]	0.6 [-]
		L. BROWN	0 [-]	0 [-]	2.0 [B]	0 [-]	0.5 [-]	0.4 [-]	-0.5 [-]	0 [-]	0.1 [-]	0.1 [-]
		BRWN/BLCK	-0.6 [-]	0.1 [-]	0.6 [C]	-0.1 [-]	0.1 [-]	1.0 [-]	0.3 [-]	0.6 [-]	0.5 [-]	0.6 [-]
%INCREASE		BLOND/RED	7 (58%)	2 (25%)	4 (100%)	9 (45%)	13 (54%)	3 (25%)	5 (63%)	4 (100%)	8 (40%)	12 (50%)
		L. BROWN	3 (27%)	1 (25%)	4 (80%)	4 (27%)	8 (40%)	5 (45%)	1 (25%)	2 (40%)	6 (40%)	8 (40%)
		BRWN/BLCK	1 (10%)	3 (14%)	5 (45%)	4 (13%)	9 (21%)	7 (70%)	6 (29%)	6 (55%)	13 (42%)	19 (45%)
P-VALUE			0.017	-	0.041	0.011	0.007	0.038	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		BLOND/RED	9.6 [-]	9.1 [-]	10.0 [-]	9.4 [-]	9.5 [-]	21.8 [-]	21.3 [-]	18.8 [-]	21.6 [-]	21.1 [-]
		L. BROWN	9.4 [-]	9.5 [-]	8.8 [-]	9.4 [-]	9.3 [-]	22.1 [-]	23.5 [-]	17.6 [-]	22.5 [-]	21.3 [-]
		BRWN/BLCK	9.7 [-]	9.7 [-]	8.8 [-]	9.7 [-]	9.5 [-]	20.7 [-]	23.0 [-]	18.4 [-]	22.2 [-]	21.2 [-]
AFTER		BLOND/RED	9.7 [-]	9.3 [-]	10.0 [-]	9.5 [-]	9.6 [-]	22.2 [-]	21.6 [-]	24.3 [A]	22.0 [-]	22.3 [-]
	HAIR	L. BROWN	9.7 [-]	9.8 [-]	9.6 [-]	9.7 [-]	9.7 [-]	22.8 [-]	23.3 [-]	20.4 [B]	22.9 [-]	22.3 [-]
		BRWN/BLCK	9.4 [-]	9.9 [-]	9.4 [-]	9.7 [-]	9.6 [-]	20.8 [-]	23.6 [-]	20.2 [B]	22.7 [-]	22.0 [-]
IMPROVEM'T		BLOND/RED	0.1 [-]	0.1 [-]	0 [-]	0.1 [-]	0.1 [-]	0.4 [-]	0.4 [-]	5.5 [-]	0.4 [-]	1.3 [-]
		L. BROWN	0.4 [-]	0.3 [-]	0.8 [-]	0.3 [-]	0.5 [-]	0.7 [-]	-0.3 [-]	2.8 [-]	0.5 [-]	1.1 [-]
		BRWN/BLCK	-0.3 [-]	0.1 [-]	0.5 [-]	0 [-]	0.1 [-]	0.1 [-]	0.6 [-]	1.8 [-]	0.5 [-]	0.8 [-]
%INCREASE		BLOND/RED	3 (25%)	1 (13%)	0	4 (20%)	4 (17%)	9 (75%)	5 (63%)	4 (100%)	14 (70%)	18 (75%)
		L. BROWN	4 (36%)	1 (25%)	2 (40%)	5 (33%)	7 (35%)	6 (55%)	2 (50%)	4 (80%)	8 (53%)	12 (60%)
		BRWN/BLCK	1 (10%)	3 (14%)	4 (36%)	4 (13%)	8 (19%)	3 (30%)	9 (43%)	6 (55%)	12 (39%)	18 (43%)
P-VALUE			-	-	-	-	-	0.038	-	-	0.03	0.011

Table 8e - Knowledge Scores as a Function of Eye Color.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		BLUE/GREEN	6.8 [-]	8.0 [-]	4.8 [-]	7.3 [-]	6.8 [-]	5.0 [-]	5.3 [B]	4.1 [-]	5.2 [-]	5.0 [-]
		HAZEL/GREY	7.2 [-]	7.4 [-]	5.7 [-]	7.3 [-]	6.9 [-]	5.2 [-]	4.4 [C]	5.3 [-]	4.8 [-]	4.9 [-]
		L/D BROWN	6.8 [-]	7.6 [-]	5.0 [-]	7.4 [-]	6.7 [-]	5.3 [-]	5.6 [A]	3.8 [-]	5.5 [-]	5.0 [-]
AFTER		BLUE/GREEN	6.8 [-]	7.7 [-]	7.0 [-]	7.1 [-]	7.1 [-]	5.4 [-]	5.7 [-]	5.6 [A]	5.5 [-]	5.5 [-]
	EYES	HAZEL/GREY	6.7 [-]	7.6 [-]	6.3 [-]	7.1 [-]	6.9 [-]	6.2 [-]	5.4 [-]	5.3 [A]	5.8 [-]	5.7 [-]
		L/D BROWN	6.8 [-]	7.9 [-]	6.5 [-]	7.6 [-]	7.3 [-]	5.8 [-]	5.6 [-]	4.3 [B]	5.7 [-]	5.3 [-]
IMPROVEM'T		BLUE/GREEN	0 [-]	-0.3 [-]	2.3 [-]	-0.2 [-]	0.3 [-]	0.3 [-]	0.3 [-]	1.5 [A]	0.3 [B]	0.5 [-]
		HAZEL/GREY	-0.5 [-]	0.2 [-]	0.7 [-]	-0.2 [-]	0 [-]	1.0 [-]	1.0 [-]	0 [B]	1.0 [A]	0.8 [-]
		L/D BROWN	0 [-]	0.3 [-]	1.5 [-]	0.2 [-]	0.6 [-]	0.5 [-]	0 [-]	0.5 [B]	0.2 [C]	0.3 [-]
%INCREASE		BLUE/GREEN	9 (43%)	1 (7%)	6 (75%)	10 (28%)	16 (36%)	8 (38%)	6 (40%)	7 (88%)	14 (39%)	21 (48%)
		HAZEL/GREY	0	1 (20%)	1 (33%)	1 (9%)	2 (14%)	4 (67%)	2 (40%)	1 (33%)	6 (55%)	7 (50%)
		L/D BROWN	2 (33%)	4 (31%)	6 (75%)	6 (32%)	12 (44%)	3 (50%)	4 (31%)	4 (50%)	7 (37%)	11 (41%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		BLUE/GREEN	9.6 [-]	9.5 [-]	9.4 [-]	9.6 [-]	9.5 [-]	21.5 [-]	22.9 [-]	18.3 [-]	22.1 [-]	21.4 [-]
		HAZEL/GREY	9.5 [-]	9.4 [-]	9.0 [-]	9.5 [-]	9.4 [-]	21.8 [-]	21.2 [-]	20.0 [-]	21.5 [-]	21.2 [-]
		L/D BROWN	9.3 [-]	9.6 [-]	8.6 [-]	9.5 [-]	9.3 [-]	21.5 [-]	22.8 [-]	17.4 [-]	22.4 [-]	20.9 [-]
AFTER		BLUE/GREEN	9.5 [-]	9.6 [-]	9.9 [-]	9.5 [-]	9.6 [-]	21.6 [-]	22.9 [-]	22.5 [-]	22.2 [-]	22.2 [-]
	EYES	HAZEL/GREY	9.7 [-]	9.8 [-]	9.3 [-]	9.7 [-]	9.6 [-]	22.5 [-]	22.8 [-]	21.0 [-]	22.6 [-]	22.3 [-]
		L/D BROWN	10.0 [-]	9.8 [-]	9.3 [-]	9.8 [-]	9.7 [-]	22.7 [-]	23.3 [-]	20.0 [-]	23.1 [-]	22.1 [-]
IMPROVEM'T		BLUE/GREEN	-0.1 [-]	0.1 [-]	0.5 [-]	-0.1 [-]	0 [-]	0.1 [-]	0.1 [-]	4.3 [-]	0.1 [-]	0.9 [-]
		HAZEL/GREY	0.2 [-]	0.4 [-]	0.3 [-]	0.3 [-]	0.3 [-]	0.7 [-]	1.6 [-]	1.0 [-]	1.1 [-]	1.1 [-]
		L/D BROWN	0.7 [-]	0.2 [-]	0.6 [-]	0.3 [-]	0.4 [-]	1.2 [-]	0.5 [-]	2.6 [-]	0.7 [-]	1.3 [-]
%INCREASE		BLUE/GREEN	4 (19%)	2 (13%)	2 (25%)	6 (17%)	8 (18%)	11 (52%)	7 (47%)	7 (88%)	18 (50%)	25 (57%)
		HAZEL/GREY	1 (17%)	1 (20%)	1 (33%)	2 (18%)	3 (21%)	4 (67%)	3 (60%)	2 (67%)	7 (64%)	9 (64%)
		L/D BROWN	3 (50%)	2 (15%)	3 (38%)	5 (26%)	8 (30%)	3 (50%)	6 (46%)	5 (63%)	9 (47%)	14 (52%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

Table 8f - Knowledge Scores as a Function of Skin Susceptibility to Sunburns.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		SUNBURN	6.9 [-]	7.8 [-]	4.9 [-]	7.3 [-]	6.8 [-]	5.3 [-]	5.1 [-]	4.4 [-]	5.2 [-]	5.1 [-]
		MILD-MOD TAN	6.9 [-]	7.7 [-]	5.7 [-]	7.3 [-]	7.0 [-]	4.9 [-]	5.4 [-]	4.1 [-]	5.2 [-]	5.0 [-]
AFTER	?	SUNBURN	7.1 [-]	7.7 [-]	7.1 [-]	7.3 [-]	7.3 [-]	5.8 [-]	5.4 [-]	5.6 [A]	5.6 [-]	5.6 [-]
	N	MILD-MOD TAN	6.5 [-]	7.8 [-]	6.7 [-]	7.2 [-]	7.1 [-]	5.4 [-]	5.8 [-]	4.6 [B]	5.6 [-]	5.4 [-]
IMPROVEM'T	R	SUNBURN	0.1 [-]	-0.1 [-]	2.2 [-]	0 [-]	0.5 [-]	0.4 [-]	0.3 [-]	1.1 [-]	0.4 [-]	0.5 [-]
	U	MILD-MOD TAN	-0.4 [-]	0.1 [-]	1.0 [-]	-0.2 [-]	0.1 [-]	0.5 [-]	0.3 [-]	0.4 [-]	0.4 [-]	0.4 [-]
%INCREASE	B	SUNBURN	7 (39%)	2 (13%)	7 (78%)	9 (27%)	16 (38%)	7 (39%)	5 (33%)	6 (67%)	12 (36%)	18 (43%)
		MILD-MOD TAN	4 (27%)	4 (22%)	5 (56%)	8 (24%)	13 (31%)	8 (53%)	7 (39%)	5 (56%)	15 (45%)	20 (48%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		SUNBURN	9.6 [-]	9.6 [-]	9.4 [-]	9.6 [-]	9.5 [-]	21.8 [-]	22.5 [-]	18.8 [-]	22.1 [-]	21.4 [-]
		MILD-MOD TAN	9.5 [-]	9.5 [-]	8.9 [-]	9.5 [-]	9.4 [-]	21.3 [-]	22.7 [-]	18.7 [-]	22.0 [-]	21.3 [-]
AFTER	?	SUNBURN	9.6 [-]	9.7 [-]	9.8 [-]	9.6 [-]	9.7 [-]	22.4 [-]	22.8 [-]	22.4 [-]	22.6 [-]	22.5 [-]
	N	MILD-MOD TAN	9.6 [-]	9.7 [-]	9.3 [-]	9.7 [-]	9.6 [-]	21.5 [-]	23.3 [-]	20.6 [-]	22.5 [-]	22.0 [-]
IMPROVEM'T	R	SUNBURN	0.1 [-]	0.1 [-]	0.3 [-]	0.1 [-]	0.1 [-]	0.6 [-]	0.3 [-]	3.7 [-]	0.5 [-]	1.1 [-]
	U	MILD-MOD TAN	0.1 [-]	0.2 [-]	0.4 [-]	0.2 [-]	0.2 [-]	0.2 [-]	0.6 [-]	1.9 [-]	0.4 [-]	0.7 [-]
%INCREASE	B	SUNBURN	5 (28%)	1 (7%)	1 (11%)	6 (18%)	7 (17%)	12 (67%)	7 (47%)	7 (78%)	19 (58%)	26 (62%)
		MILD-MOD TAN	3 (20%)	4 (22%)	4 (44%)	7 (21%)	11 (26%)	6 (40%)	9 (50%)	6 (67%)	15 (45%)	21 (50%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

Table 8g - Knowledge Scores as a Function of Skin Inability to Tan.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		FRCKL/PEEL+TAN	7.5 [-]	7.9 [-]	5.8 [-]	7.7 [-]	7.3 [-]	5.4 [-]	5.1 [-]	4.6 [-]	5.3 [-]	5.1 [-]
		MOD-DEEP TAN	6.6 [-]	7.7 [-]	5.1 [-]	7.1 [-]	6.7 [-]	5.0 [-]	5.3 [-]	4.2 [-]	5.2 [-]	5.0 [-]
AFTER	?	FRCKL/PEEL+TAN	7.5 [-]	8.1 [-]	7.8 [-]	7.8 [-]	7.8 [-]	5.4 [-]	5.5 [-]	5.4 [-]	5.4 [-]	5.4 [-]
	N	MOD-DEEP TAN	6.4 [-]	7.7 [-]	6.5 [-]	7.1 [-]	7.0 [-]	5.7 [-]	5.6 [-]	4.9 [-]	5.7 [-]	5.5 [-]
IMPROVEM'T	A	FRCKL/PEEL+TAN	0 [-]	0.3 [-]	2.0 [-]	0.1 [-]	0.5 [-]	0 [-]	0.4 [-]	0.8 [-]	0.2 [-]	0.3 [-]
	T	MOD-DEEP TAN	-0.2 [-]	0 [-]	1.5 [-]	-0.1 [-]	0.3 [-]	0.7 [-]	0.3 [-]	0.8 [-]	0.5 [-]	0.6 [-]
%INCREASE		FRCKL/PEEL+TAN	5 (50%)	2 (25%)	4 (80%)	7 (39%)	11 (48%)	3 (30%)	3 (38%)	3 (60%)	6 (33%)	9 (39%)
		MOD-DEEP TAN	6 (26%)	4 (17%)	8 (62%)	10 (21%)	18 (30%)	12 (52%)	9 (38%)	8 (62%)	21 (45%)	29 (48%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		FRCKL/PEEL+TAN	9.7 [-]	10.0 [-]	10.0 [-]	9.8 [-]	9.9 [A]	22.6 [-]	23.0 [-]	20.4 [A]	22.8 [-]	22.3 [A]
		MOD-DEEP TAN	9.5 [-]	9.4 [-]	8.8 [-]	9.4 [-]	9.3 [B]	21.1 [-]	22.4 [-]	18.1 [B]	21.7 [-]	21.0 [B]
AFTER	?	FRCKL/PEEL+TAN	9.8 [-]	9.9 [-]	10.0 [-]	9.8 [-]	9.9 [-]	22.7 [-]	23.5 [-]	23.2 [-]	23.1 [-]	23.1 [-]
	N	MOD-DEEP TAN	9.5 [-]	9.6 [-]	9.5 [-]	9.6 [-]	9.5 [-]	21.7 [-]	22.9 [-]	20.8 [-]	22.3 [-]	22.0 [-]
IMPROVEM'T	A	FRCKL/PEEL+TAN	0.1 [-]	-0.1 [-]	0 [-]	0 [-]	0 [B]	0.1 [-]	0.5 [-]	2.8 [-]	0.3 [-]	0.8 [-]
	T	MOD-DEEP TAN	0 [-]	0.3 [-]	0.5 [-]	0.1 [-]	0.2 [A]	0.6 [-]	0.5 [-]	2.8 [-]	0.6 [-]	1.0 [-]
%INCREASE		FRCKL/PEEL+TAN	2 (20%)	0	0	2 (11%)	2 (9%)	6 (60%)	3 (38%)	4 (80%)	9 (50%)	13 (57%)
		MOD-DEEP TAN	6 (26%)	5 (21%)	5 (38%)	11 (23%)	16 (27%)	12 (52%)	13 (54%)	9 (69%)	25 (53%)	34 (57%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

Table 8h - Knowledge Scores as a Function of Objective Physician-Determined Nevus Count.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		0-33	6.6 [-]	7.4 [-]	5.1 [-]	6.9 [C]	6.2 [B]	5.2 [-]	4.8 [-]	4.2 [-]	5.0 [-]	4.7 [-]
	VI	34-66	7.3 [-]	7.5 [-]	-	7.4 [B]	7.4 [A]	5.3 [-]	5.7 [-]	-	5.5 [-]	5.5 [-]
		67-100 & 100+	7.7 [-]	8.6 [-]	-	8.3 [A]	8.3 [A]	4.3 [-]	5.6 [-]	-	5.3 [-]	5.3 [-]
AFTER	NE	0-33	6.4 [-]	6.6 [B]	6.5 [-]	6.5 [B]	6.5 [B]	5.5 [-]	5.7 [-]	4.8 [-]	5.6 [-]	5.3 [-]
		34-66	7.3 [-]	8.4 [A]	-	7.9 [A]	7.9 [A]	5.9 [-]	5.5 [-]	-	5.7 [-]	5.7 [-]
		67-100 & 100+	7.3 [-]	8.4 [A]	-	8.2 [A]	8.2 [A]	5.3 [-]	5.7 [-]	-	5.6 [-]	5.6 [-]
IMPROVEM'T	#	0-33	-0.2 [-]	-0.8 [C]	1.4 [-]	-0.4 [C]	0.3 [-]	0.4 [-]	0.9 [A]	0.6 [-]	0.6 [-]	0.6 [-]
		34-66	0 [-]	0.9 [A]	-	0.5 [A]	0.5 [-]	0.6 [-]	-0.2 [C]	-	0.2 [-]	0.2 [-]
	J	67-100 & 100+	-0.3 [-]	-0.1 [B]	-	-0.2 [B]	-0.2 [-]	1.0 [-]	0.1 [B]	-	0.3 [-]	0.3 [-]
%INCREASE	OB	0-33	8 (40%)	0	12 (63%)	8 (25%)	20 (39%)	9 (45%)	7 (58%)	11 (58%)	16 (50%)	27 (53%)
		34-66	2 (20%)	5 (42%)	-	7 (32%)	7 (32%)	5 (50%)	3 (25%)	-	8 (36%)	8 (36%)
		67-100 & 100+	1 (33%)	1 (11%)	-	2 (17%)	2 (17%)	1 (33%)	2 (22%)	-	3 (25%)	3 (25%)
P-VALUE			-	-	-	-	-	-	-	-	-	0.051

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		0-33	9.4 [-]	9.2 [-]	9.0 [-]	9.3 [-]	9.2 [-]	21.1 [-]	21.3 [-]	18.3 [-]	21.2 [C]	20.1 [B]
	VI	34-66	9.8 [-]	9.8 [-]	-	9.8 [-]	9.8 [-]	22.4 [-]	22.9 [-]	-	22.7 [B]	22.7 [A]
		67-100 & 100+	9.7 [-]	9.8 [-]	-	9.8 [-]	9.8 [-]	21.7 [-]	23.9 [-]	-	23.3 [A]	23.3 [A]
AFTER	NE	0-33	9.5 [-]	9.4 [-]	9.5 [-]	9.5 [-]	9.5 [-]	21.4 [-]	21.7 [B]	20.8 [-]	21.5 [B]	21.2 [B]
		34-66	9.8 [-]	9.9 [-]	-	9.9 [-]	9.9 [-]	23.0 [-]	23.8 [A]	-	23.5 [A]	23.5 [A]
		67-100 & 100+	9.7 [-]	9.8 [-]	-	9.8 [-]	9.8 [-]	22.3 [-]	23.9 [A]	-	23.5 [A]	23.5 [A]
IMPROVEM'T	#	0-33	0.1 [-]	0.3 [-]	0.5 [-]	0.2 [-]	0.3 [-]	0.3 [-]	0.3 [-]	2.5 [-]	0.3 [-]	1.1 [-]
		34-66	0 [-]	0.2 [-]	-	0.1 [-]	0.1 [-]	0.6 [-]	0.9 [-]	-	0.8 [-]	0.8 [-]
	J	67-100 & 100+	0 [-]	0 [-]	-	0 [-]	0 [-]	0.7 [-]	0 [-]	-	0.2 [-]	0.2 [-]
%INCREASE	OB	0-33	7 (35%)	2 (17%)	6 (32%)	9 (28%)	15 (29%)	9 (45%)	6 (50%)	13 (68%)	15 (47%)	28 (55%)
		34-66	1 (10%)	2 (17%)	-	3 (14%)	3 (14%)	7 (70%)	7 (58%)	-	14 (64%)	14 (64%)
		67-100 & 100+	0	1 (11%)	-	1 (8%)	1 (8%)	2 (67%)	3 (33%)	-	5 (42%)	5 (42%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

Table 8i - Knowledge Scores as a Function of Subjective, Self-Reported Nevus Counts.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		0-33	6.6 [-]	8.0 [-]	5.0 [-]	7.2 [-]	6.4 [-]	5.2 [-]	5.2 [-]	4.1 [-]	5.2 [-]	4.8 [-]
	VIEW	34-66	7.6 [-]	8.3 [-]	5.0 [-]	7.8 [-]	7.6 [-]	5.6 [-]	5.5 [-]	5.0 [-]	5.5 [-]	5.5 [-]
		67-100 & 100+	6.9 [-]	7.4 [-]	5.0 [-]	7.3 [-]	7.1 [-]	4.6 [-]	5.3 [-]	5.0 [-]	5.1 [-]	5.1 [-]
AFTER		0-33	6.7 [-]	7.2 [-]	6.6 [-]	6.9 [-]	6.8 [-]	5.6 [-]	5.8 [-]	4.7 [-]	5.7 [-]	5.3 [-]
		34-66	7.3 [-]	8.0 [-]	6.0 [-]	7.5 [-]	7.4 [-]	5.6 [-]	5.5 [-]	6.0 [-]	5.5 [-]	5.6 [-]
		67-100 & 100+	6.3 [-]	8.1 [-]	7.0 [-]	7.6 [-]	7.5 [-]	5.7 [-]	5.5 [-]	6.0 [-]	5.6 [-]	5.6 [-]
IMPROVEM'T	#	0-33	0.1 [-]	-0.8 [C]	1.6 [-]	-0.3 [-]	0.4 [-]	0.4 [B]	0.5 [-]	0.6 [-]	0.5 [-]	0.5 [-]
		34-66	-0.3 [-]	-0.3 [B]	1.0 [-]	-0.3 [-]	-0.2 [-]	0 [C]	0 [-]	1.0 [-]	0 [-]	0.1 [-]
		67-100 & 100+	-0.6 [-]	0.7 [A]	2.0 [-]	0.3 [-]	0.4 [-]	1.1 [A]	0.2 [-]	1.0 [-]	0.5 [-]	0.5 [-]
%INCREASE	SUBJ	0-33	7 (37%)	0	11 (65%)	7 (22%)	18 (37%)	8 (42%)	5 (38%)	10 (59%)	13 (41%)	23 (47%)
		34-66	3 (43%)	1 (25%)	1 (100%)	4 (36%)	5 (42%)	3 (43%)	1 (25%)	1 (100%)	4 (36%)	5 (42%)
		67-100 & 100+	1 (14%)	5 (31%)	1 (50%)	6 (26%)	7 (28%)	4 (57%)	6 (38%)	1 (50%)	10 (43%)	11 (44%)
P-VALUE			-	0.034	-	-	-	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		0-33	9.5 [-]	9.2 [-]	9.1 [-]	9.4 [-]	9.3 [-]	21.3 [-]	22.5 [-]	18.1 [-]	21.8 [-]	20.5 [-]
	VIEW	34-66	9.4 [-]	10.0 [-]	9.0 [-]	9.6 [-]	9.6 [-]	22.6 [-]	23.8 [-]	19.0 [-]	23.0 [-]	22.7 [-]
		67-100 & 100+	9.7 [-]	9.7 [-]	9.0 [-]	9.7 [-]	9.6 [-]	21.1 [-]	22.4 [-]	19.0 [-]	22.0 [-]	21.8 [-]
AFTER		0-33	9.7 [-]	9.4 [-]	9.6 [-]	9.6 [-]	9.6 [-]	22.0 [-]	22.4 [-]	20.9 [-]	22.2 [-]	21.7 [-]
		34-66	9.6 [-]	10.0 [-]	10.0 [-]	9.7 [-]	9.8 [-]	22.4 [-]	23.5 [-]	22.0 [-]	22.8 [-]	22.8 [-]
		67-100 & 100+	9.4 [-]	9.9 [-]	9.0 [-]	9.7 [-]	9.7 [-]	21.4 [-]	23.5 [-]	22.0 [-]	22.9 [-]	22.8 [-]
IMPROVEM'T	#	0-33	0.2 [-]	0.2 [-]	0.5 [-]	0.2 [-]	0.3 [-]	0.7 [-]	-0.1 [-]	2.8 [-]	0.4 [-]	1.2 [-]
		34-66	0.1 [-]	0 [-]	1.0 [-]	0.1 [-]	0.2 [-]	-0.1 [-]	-0.3 [-]	3.0 [-]	-0.2 [-]	0.1 [-]
		67-100 & 100+	-0.3 [-]	0.2 [-]	0 [-]	0 [-]	0 [-]	0.3 [-]	1.1 [-]	3.0 [-]	0.8 [-]	1.0 [-]
%INCREASE	SUBJ	0-33	5 (26%)	2 (15%)	5 (29%)	7 (22%)	12 (24%)	11 (58%)	4 (31%)	12 (71%)	15 (47%)	27 (55%)
		34-66	2 (29%)	0	1 (100%)	2 (18%)	3 (25%)	3 (43%)	1 (25%)	1 (100%)	4 (36%)	5 (42%)
		67-100 & 100+	1 (14%)	3 (19%)	0	4 (17%)	4 (16%)	4 (57%)	11 (69%)	1 (50%)	15 (65%)	16 (64%)
P-VALUE			-	-	-	-	-	-	0.041	-	-	-

Table 8j - Knowledge Scores as a Function of Subjective Belief in Having More-than-Average Nevi.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		YES	6.9 [-]	7.7 [-]	3.8 [-]	7.4 [-]	7.0 [-]	5.1 [-]	5.3 [-]	4.3 [-]	5.3 [-]	5.2 [-]
	MORE NEVI ?	NO	6.9 [-]	7.8 [-]	5.4 [-]	7.2 [-]	6.5 [-]	5.1 [-]	5.1 [-]	4.1 [-]	5.1 [-]	4.7 [-]
		DK	6.8 [-]	9.0 [-]	4.5 [-]	7.4 [-]	6.8 [-]	5.4 [-]	5.5 [-]	5.0 [-]	5.4 [-]	5.3 [-]
AFTER		YES	6.7 [-]	7.9 [-]	5.8 [-]	7.5 [-]	7.3 [-]	5.8 [-]	5.4 [-]	5.5 [-]	5.5 [-]	5.5 [-]
		NO	6.6 [-]	7.0 [-]	7.0 [-]	6.8 [-]	6.8 [-]	5.5 [-]	6.0 [-]	4.6 [-]	5.7 [-]	5.3 [-]
		DK	7.4 [-]	9.0 [-]	5.5 [-]	7.9 [-]	7.3 [-]	5.6 [-]	6.0 [-]	5.5 [-]	5.7 [-]	5.7 [-]
IMPROVEM'T		YES	-0.3 [-]	0.3 [-]	2.0 [-]	0.1 [-]	0.3 [-]	0.7 [-]	0.1 [-]	1.3 [-]	0.4 [-]	0.4 [-]
		NO	-0.3 [-]	-0.8 [-]	1.6 [-]	-0.4 [-]	0.3 [-]	0.4 [-]	0.9 [-]	0.6 [-]	0.6 [-]	0.6 [-]
		DK	0.6 [-]	0 [-]	1.0 [-]	0.4 [-]	0.6 [-]	0.2 [-]	0.5 [-]	0.5 [-]	0.3 [-]	0.3 [-]
%INCREASE		YES	3 (25%)	6 (26%)	3 (75%)	9 (26%)	12 (31%)	7 (58%)	7 (30%)	3 (75%)	14 (40%)	17 (44%)
		NO	5 (31%)	0	8 (57%)	5 (21%)	13 (34%)	6 (38%)	4 (50%)	8 (57%)	10 (42%)	18 (47%)
		DK	3 (60%)	0	2 (100%)	3 (43%)	5 (56%)	2 (40%)	1 (50%)	1 (50%)	3 (43%)	4 (44%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		YES	9.6 [-]	9.5 [-]	8.3 [-]	9.5 [-]	9.4 [-]	21.6 [-]	22.5 [-]	16.3 [-]	22.2 [-]	21.6 [-]
	MORE NEVI ?	NO	9.6 [-]	9.5 [-]	9.1 [-]	9.5 [-]	9.4 [-]	21.5 [-]	22.4 [-]	18.6 [-]	21.8 [-]	20.6 [-]
		DK	9.4 [-]	10.0 [-]	10.0 [-]	9.6 [-]	9.7 [-]	21.6 [-]	24.5 [-]	19.5 [-]	22.4 [-]	21.8 [-]
AFTER		YES	9.5 [-]	9.7 [-]	9.3 [-]	9.6 [-]	9.6 [-]	21.9 [-]	23.0 [-]	20.5 [-]	22.7 [-]	22.4 [-]
		NO	9.8 [-]	9.6 [-]	9.6 [-]	9.7 [-]	9.7 [-]	21.9 [-]	22.6 [-]	21.3 [-]	22.1 [-]	21.8 [-]
		DK	9.4 [-]	10.0 [-]	9.5 [-]	9.6 [-]	9.6 [-]	22.4 [-]	25.0 [-]	20.5 [-]	23.1 [-]	22.6 [-]
IMPROVEM'T		YES	-0.1 [-]	0.2 [-]	1.0 [-]	0.1 [-]	0.2 [-]	0.3 [-]	0.5 [-]	4.3 [-]	0.5 [-]	0.8 [-]
		NO	0.2 [-]	0.1 [-]	0.5 [-]	0.2 [-]	0.3 [-]	0.4 [-]	0.3 [-]	2.6 [-]	0.3 [-]	1.2 [-]
		DK	0 [-]	0 [-]	-0.5 [-]	0 [-]	-0.1 [-]	0.8 [-]	0.5 [-]	1.0 [-]	0.7 [-]	0.8 [-]
%INCREASE		YES	2 (17%)	4 (17%)	2 (50%)	6 (17%)	8 (21%)	8 (67%)	12 (52%)	3 (75%)	20 (57%)	23 (59%)
		NO	5 (31%)	1 (13%)	4 (29%)	6 (25%)	10 (26%)	8 (50%)	3 (38%)	10 (71%)	11 (46%)	21 (55%)
		DK	1 (20%)	0	0	1 (14%)	1 (11%)	2 (40%)	1 (50%)	1 (50%)	3 (43%)	4 (44%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

Table 8k - Knowledge Scores as a Function of the Personal History of Atypical Nevi.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		YES	7.1 [-]	7.8 [-]	0 [B]	7.5 [-]	7.4 [A]	5.2 [-]	5.3 [-]	2.0 [-]	5.3 [-]	5.2 [A]
	N	NO	6.5 [-]	-	5.3 [A]	6.5 [-]	5.7 [B]	5.0 [-]	-	4.3 [-]	5.0 [-]	4.6 [B]
AFTER	A	YES	7.1 [-]	7.8 [-]	3.0 [B]	7.5 [A]	7.4 [A]	5.8 [-]	5.6 [-]	4.0 [-]	5.7 [-]	5.6 [A]
		NO	6.1 [-]	-	6.8 [A]	6.1 [B]	6.5 [B]	5.3 [-]	-	4.9 [-]	5.3 [-]	5.1 [B]
IMPROVEM'T	H x	YES	0 [-]	[-]	3.0 [-]	0 [-]	0.1 [B]	0.6 [-]	0.3 [-]	2.0 [-]	0.4 [-]	0.4 [-]
		NO	-0.4 [-]	-	1.5 [-]	-0.4 [-]	0.8 [A]	0.3 [-]	-	0.6 [-]	0.3 [-]	0.5 [-]
%INCREASE	P	YES	8 (36%)	6 (18%)	1 (100%)	14 (25%)	15 (28%)	11 (50%)	12 (100%)	1 (100%)	23 (42%)	24 (43%)
		NO	3 (27%)	-	12 (63%)	3 (27%)	15 (50%)	4 (36%)	-	11 (58%)	4 (36%)	15 (50%)
P-VALUE			-	-	-	-	0.032	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		YES	9.6 [-]	9.5 [-]	6.0 [B]	9.6 [-]	9.5 [-]	21.9 [-]	22.6 [-]	8.0 [B]	22.3 [-]	22.1 [A]
	N	NO	9.4 [-]	-	9.2 [A]	9.4 [-]	9.3 [-]	20.8 [-]	-	18.8 [A]	20.8 [-]	19.5 [B]
AFTER	A	YES	9.6 [-]	9.7 [-]	9.0 [-]	9.7 [-]	9.6 [-]	22.5 [-]	23.1 [-]	16.0 [-]	22.8 [A]	22.7 [A]
		NO	9.6 [-]	-	9.6 [-]	9.6 [-]	9.6 [-]	21.0 [-]	-	21.3 [-]	21.0 [B]	21.2 [B]
IMPROVEM'T	H x	YES	0 [-]	0.2 [-]	3.0 [A]	0.1 [-]	0.1 [-]	0.5 [-]	0.5 [-]	8.0 [-]	0.5 [-]	0.6 [-]
		NO	0.3 [-]	-	0.4 [B]	0.3 [-]	0.3 [-]	0.2 [-]	-	2.5 [-]	0.2 [-]	1.7 [-]
%INCREASE	P	YES	4 (18%)	5 (15%)	1 (100%)	9 (16%)	10 (18%)	14 (64%)	16 (48%)	1 (100%)	30 (55%)	31 (55%)
		NO	4 (36%)	-	5 (26%)	4 (36%)	9 (30%)	4 (36%)	-	13 (68%)	4 (36%)	17 (57%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

Table 8I - Knowledge Scores as a Function of the Peraonal History of Non-Melanoma Skin Cancers.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE	SC	YES	7.3 [-]	6.5 [B]	5.7 [-]	6.9 [-]	6.7 [-]	5.5 [-]	5.3 [-]	5.0 [-]	5.4 [-]	5.3 [-]
		NO	6.8 [-]	8.0 [A]	4.9 [-]	7.4 [-]	6.8 [-]	5.0 [-]	5.3 [-]	4.1 [-]	5.2 [-]	4.9 [-]
AFTER	SM	YES	7.3 [-]	7.2 [-]	7.7 [-]	7.3 [-]	7.3 [-]	5.8 [-]	5.3 [-]	5.7 [-]	5.6 [-]	5.6 [-]
		NO	6.6 [-]	7.9 [-]	6.3 [-]	7.3 [-]	7.0 [-]	5.6 [-]	5.7 [-]	4.9 [-]	5.6 [-]	5.5 [-]
IMPROVEM'T		YES	0 [-]	0.7 [-]	2.0 [-]	0.3 [-]	0.7 [-]	0.3 [-]	0 [-]	0.7 [-]	0.2 [-]	0.3 [-]
	x	NO	-0.1 [-]	-0.1 [-]	1.4 [-]	-0.1 [-]	0.2 [-]	0.5 [-]	0.4 [-]	0.9 [-]	0.4 [-]	0.5 [-]
%INCREASE	PH	YES	3 (50%)	3 (50%)	2 (67%)	6 (50%)	8 (53%)	2 (33%)	3 (50%)	1 (33%)	5 (42%)	6 (40%)
		NO	8 (30%)	3 (11%)	10 (63%)	11 (20%)	21 (30%)	13 (48%)	9 (33%)	11 (69%)	22 (41%)	33 (47%)
P-VALUE			-	0.028	-	0.035	-	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE	SC	YES	9.7 [-]	8.7 [B]	10.0 [-]	9.2 [-]	9.3 [-]	22.5 [-]	20.5 [-]	20.7 [-]	21.5 [-]	21.3 [-]
		NO	9.5 [-]	9.7 [A]	8.9 [-]	9.7 [-]	9.5 [-]	21.3 [-]	23.1 [-]	17.9 [-]	22.2 [-]	21.2 [-]
AFTER	SM	YES	9.8 [-]	9.2 [-]	10.0 [-]	9.5 [-]	9.6 [-]	23.0 [-]	21.7 [-]	23.3 [-]	22.3 [-]	22.5 [-]
		NO	9.6 [-]	9.8 [-]	9.5 [-]	9.7 [-]	9.6 [-]	21.7 [-]	23.4 [-]	20.7 [-]	22.6 [-]	22.1 [-]
IMPROVEM'T		YES	0.2 [-]	0.5 [-]	0 [-]	0.3 [-]	0.3 [-]	0.5 [-]	1.2 [-]	2.7 [-]	0.8 [-]	1.2 [-]
	x	NO	0 [-]	0.1 [-]	0.6 [-]	0.1 [-]	0.2 [-]	0.4 [-]	0.3 [-]	2.8 [-]	0.4 [-]	0.9 [-]
%INCREASE	PH	YES	1 (17%)	2 (33%)	0	3 (25%)	3 (20%)	4 (67%)	5 (83%)	2 (67%)	9 (75%)	11 (73%)
		NO	7 (26%)	3 (11%)	5 (31%)	10 (19%)	15 (21%)	14 (52%)	11 (41%)	11 (69%)	25 (46%)	36 (51%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

Table 8m - Knowledge Scores as a Function of the Family History of Malignant Melanoma.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		YES	7.1 [-]	7.9 [-]	4.5 [-]	7.6 [-]	7.3 [-]	5.6 [-]	5.4 [-]	3.0 [-]	5.5 [-]	5.3 [-]
		NO	6.7 [-]	7.8 [-]	5.0 [-]	7.1 [-]	6.5 [-]	4.9 [-]	5.1 [-]	4.4 [-]	5.0 [-]	4.8 [-]
		DK	8.0 [-]	7.3 [-]	5.3 [-]	7.5 [-]	6.8 [-]	6.0 [-]	5.5 [-]	4.0 [-]	5.7 [-]	5.1 [-]
AFTER	M	YES	6.9 [-]	7.9 [-]	6.0 [B]	7.5 [-]	7.4 [B]	6.0 [B]	5.8 [-]	6.0 [-]	5.9 [-]	5.9 [-]
	M	NO	6.5 [-]	7.6 [-]	6.2 [B]	7.0 [-]	6.8 [C]	5.4 [C]	5.5 [-]	4.8 [-]	5.4 [-]	5.3 [-]
		DK	8.5 [-]	7.8 [-]	9.0 [A]	8.0 [-]	8.3 [A]	6.5 [A]	5.3 [-]	4.7 [-]	5.7 [-]	5.3 [-]
IMPROVEM'T	x	YES	-0.2 [-]	0.1 [-]	1.5 [-]	0 [-]	0.1 [B]	0.4 [-]	0.4 [-]	3.0 [-]	0.4 [-]	0.6 [-]
	H	NO	-0.1 [-]	-0.2 [-]	1.2 [-]	-0.2 [-]	0.2 [B]	0.5 [-]	0.4 [-]	0.4 [-]	0.5 [-]	0.4 [-]
	F	DK	0.5 [-]	0.5 [-]	3.7 [-]	0.5 [-]	1.6 [A]	0.5 [-]	-0.3 [-]	0.7 [-]	0 [-]	0.2 [-]
%INCREASE		YES	2 (22%)	3 (21%)	1 (50%)	5 (22%)	6 (24%)	4 (44%)	6 (43%)	2 (100%)	10 (43%)	12 (48%)
		NO	8 (36%)	1 (7%)	9 (60%)	9 (24%)	18 (35%)	10 (45%)	6 (40%)	8 (53%)	16 (43%)	24 (46%)
		DK	1 (50%)	2 (50%)	3 (100%)	3 (50%)	6 (67%)	1 (50%)	0	2 (67%)	1 (17%)	3 (33%)
P-VALUE			-	-	-	-	0.025	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		YES	9.8 [A]	9.8 [-]	8.5 [-]	9.8 [-]	9.7 [-]	22.4 [-].	23.1 [-]	16.0 [-]	22.8 [-]	22.3 [-]
		NO	9.5 [A]	9.2 [-]	9.1 [-]	9.4 [-]	9.3 [-]	21.1 [-]	22.1 [-]	18.5 [-]	21.5 [-]	20.6 [-]
		DK	8.5 [B]	10.0 [-]	9.3 [-]	9.5 [-]	9.4 [-]	22.5 [-]	22.8 [-]	18.7 [-]	22.7 [-]	21.3 [-]
AFTER	M	YES	9.8 [-]	9.8 [-]	10.0 [-]	9.8 [-]	9.8 [-]	22.7 [-]	23.5 [-]	22.0 [-]	23.2 [-]	23.1 [-]
	M	NO	9.6 [-]	9.5 [-]	9.5 [-]	9.6 [-]	9.5 [-]	21.5 [-]	22.7 [-]	20.5 [-]	22.0 [-]	21.5 [-]
		DK	9.0 [-]	10.0 [-]	9.7 [-]	9.7 [-]	9.7 [-]	24.0 [-]	23.0 [-]	23.3 [-]	23.3 [-]	23.3 [-]
IMPROVEM'T	x	YES	0 [-]	0 [-]	1.5 [-]	0 [-]	0.1 [-]	0.2 [-]	0.4 [-]	6.0 [-]	0.3 [-]	0.8 [-]
	H	NO	0 [-]	0.3 [-]	0.4 [-]	0.2 [-]	0.2 [-]	0.4 [-]	0.5 [-]	2.0 [-]	0.5 [-]	0.9 [-]
	F	DK	0.5 [-]	0 [-]	0.3 [-]	0.2 [-]	0.2 [-]	1.5 [-]	0.3 [-]	4.7 [-]	0.7 [-]	2.0 [-]
%INCREASE		YES	1 (11%)	1 (7%)	1 (50%)	2 (9%)	3 (12%)	5 (56%)	6 (43%)	2 (100%)	11 (48%)	13 (52%)
		NO	6 (27%)	4 (27%)	4 (27%)	10 (27%)	14 (27%)	11 (50%)	8 (53%)	3 (60%)	19 (51%)	28 (54%)
		DK	1 (50%)	0	1 (33%)	1 (17%)	2 (22%)	2 (100%)	2 (50%)	3 (100%)	4 (67%)	7 (78%)
P-VALUE			-	-	-	-	-	-	-	-	-	-

Table 8n - Knowledge Scores as a Function of the Family History of Non-Melanoma Skin Cancers.

GROUPS			SCORE1 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE2 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		YES	6.8 [B]	7.5 [B]	4.8 [-]	7.1 [-]	6.8 [-]	5.1 [-]	5.2 [-]	4.5 [-]	5.1 [-]	5.1 [-]
		NO	6.3 [C]	8.1 [A]	4.9 [-]	7.4 [-]	6.8 [-]	4.9 [-]	5.3 [-]	3.9 [-]	5.2 [-]	4.9 [-]
	C	DK	8.2 [A]	6.3 [C]	5.3 [-]	7.6 [-]	6.7 [-]	5.5 [-]	5.7 [-]	4.5 [-]	5.6 [-]	5.1 [-]
AFTER	S	YES	6.6 [B]	8.0 [-]	7.0 [-]	7.1 [-]	7.1 [-]	5.6 [B]	5.4 [-]	6.3 [A]	5.5 [-]	5.6 [-]
	M	NO	6.0 [B]	7.6 [-]	6.2 [-]	7.0 [-]	6.8 [-]	5.4 [C]	5.8 [-]	4.8 [B]	5.6 [-]	5.4 [-]
	N	DK	8.7 [A]	7.7 [-]	7.0 [-]	8.3 [-]	7.8 [-]	6.2 [A]	5.3 [-]	4.2 [B]	5.9 [-]	5.2 [-]
IMPROVEM'T		YES	-0.3 [-]	0.5 [B]	2.3 [-]	0 [B]	0.3 [B]	0.4 [-]	0.2 [-]	1.8 [-]	0.3 [-]	0.5 [-]
	x	NO	-0.3 [-]	-0.5 [C]	1.3 [-]	-0.4 [C]	0 [C]	0.5 [-]	0.5 [-]	0.9 [-]	0.5 [-]	0.6 [-]
	H	DK	0.5 [-]	1.3 [A]	1.7 [-]	0.8 [A]	1.1 [A]	0.7 [-]	-0.3 [-]	-0.3 [-]	0.3 [-]	0.1 [-]
%INCREASE	F	YES	6 (38%)	3 (27%)	3 (75%)	9 (33%)	12 (39%)	6 (38%)	3 (27%)	3 (75%)	9 (33%)	12 (39%)
		NO	2 (18%)	1 (5%)	6 (60%)	3 (10%)	9 (23%)	5 (45%)	8 (42%)	7 (70%)	13 (43%)	20 (50%)
		DK	3 (50%)	2 (67%)	4 (67%)	5 (56%)	9 (60%)	4 (67%)	1 (33%)	2 (33%)	5 (56%)	7 (47%)
P-VALUE			-	-	-	-	0.047	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	NEVUS	CONTROL	PLC	ALL	SCORE4 MELANOM'	NEVUS	CONTROL	PLC	ALL
BEFORE		YES	9.4 [-]	9.8 [-]	10.0 [-]	9.6 [-]	9.6 [-]	21.4 [-]	22.5 [-]	19.3 [-]	21.9 [-]	21.5 [-]
		NO	9.7 [-]	9.4 [-]	8.7 [-]	9.5 [-]	9.3 [-]	20.9 [-]	22.8 [-]	17.5 [-]	22.1 [-]	21.0 [-]
	C	DK	9.5 [-]	9.3 [-]	9.0 [-]	9.4 [-]	9.3 [-]	23.2 [-]	21.3 [-]	18.8 [-]	22.6 [-]	21.1 [-]
AFTER	S	YES	9.8 [-]	9.9 [-]	9.8 [-]	9.8 [-]	9.8 [-]	21.9 [B]	23.3 [-]	23.0 [-]	22.4 [-]	22.5 [-]
	M	NO	9.3 [-]	9.6 [-]	9.4 [-]	9.5 [-]	9.5 [-]	20.6 [B]	23.0 [-]	20.4 [-]	22.1 [-]	21.7 [-]
	N	DK	9.8 [-]	9.7 [-]	9.7 [-]	9.8 [-]	9.7 [-]	24.7 [A]	22.7 [-]	20.8 [-]	24.0 [-]	22.7 [-]
IMPROVEM'T		YES	0.3 [A]	0.1 [-]	-0.3 [-]	0.2 [-]	0.2 [-]	0.5 [B]	0.7 [-]	3.8 [-]	0.6 [B]	1.0 [-]
	x	NO	-0.5 [B]	0.2 [-]	0.7 [-]	-0.1 [-]	0.1 [-]	-0.3 [C]	0.2 [-]	2.9 [-]	0 [C]	0.7 [-]
	H	DK	0.3 [A]	0.3 [-]	0.7 [-]	0.3 [-]	0.5 [-]	1.5 [A]	1.3 [-]	2.0 [-]	1.4 [A]	1.7 [-]
%INCREASE	F	YES	5 (31%)	1 (9%)	0	6 (22%)	6 (19%)	10 (63%)	5 (45%)	4 (100%)	15 (56%)	19 (61%)
		NO	1 (9%)	3 (16%)	4 (40%)	4 (13%)	8 (20%)	2 (18%)	8 (42%)	6 (50%)	10 (33%)	16 (40%)
		DK	2 (33%)	1 (33%)	2 (33%)	3 (33%)	5 (33%)	6(100%)	3 (100%)	4 (67%)	9 (100%)	13 (87%)
P-VALUE			-	-	-	-	-	0.038	-	-	0.006	0.02

Table 8o - Knowledge Scores as a Function of # Visits.

GROUPS			SCORE1 MELANOM'	SCORE1 NEVUS	SCORE1 PLC	SCORE2 MELANOM'	SCORE2 NEVUS	SCORE2 PLC
BEFORE	S	NEW	5.6 [-]	7.3 [-]	6.5 [-]	5.4 [-]	5.7 [-]	5.5 [-]
	T	RETURN	7.1 [-]	7.9 [-]	7.5 [-]	5.1 [-]	5.2 [-]	5.1 [-]
AFTER	I	NEW	6.0 [-]	7.2 [-]	6.6 [-]	5.6 [-]	6.3 [-]	6.0 [-]
	S	RETURN	6.9 [-]	7.9 [-]	7.4 [-]	5.6 [-]	5.4 [-]	5.5 [-]
IMPROVEM'T	I	NEW	0.4 [-]	-0.2 [-]	0.1 [-]	0.2 [-]	0.7 [-]	0.5 [-]
	V	RETURN	-0.2 [-]	0 [-]	-0.1 [-]	0.5 [-]	0.2 [-]	0.4 [-]
%INCREASE		NEW	3 (60%)	1 (17%)	4 (36%)	1 (20%)	4 (67%)	5 (45%)
	#	RETURN	8 (29%)	5 (19%)	13 (24%)	14 (50%)	8 (30%)	22 (40%)
P-VALUE			-	-	-	-	-	-

GROUPS			SCORE3 MELANOM'	SCORE3 NEVUS	SCORE3 PLC	SCORE4 MELANOM'	SCORE4 NEVUS	SCORE4 PLC
BEFORE	S	NEW	9.4 [-]	9.7 [-]	9.5 [-]	20.4 [-]	22.7 [-]	21.6 [-]
	T	RETURN	9.6 [-]	9.5 [-]	9.5 [-]	21.8 [-]	22.6 [-]	22.2 [-]
AFTER	I	NEW	10.0 [-]	9.8 [-]	9.9 [-]	21.6 [-]	23.3 [-]	22.5 [-]
	S	RETURN	9.5 [-]	9.7 [-]	9.6 [-]	22.0 [-]	23.0 [-]	22.5 [-]
IMPROVEM'T	I	NEW	0.6 [-]	0.2 [-]	0.4 [-]	1.2 [-]	0.7 [-]	0.9 [-]
	V	RETURN	0 [-]	0.1 [-]	0.1 [-]	0.3 [-]	0.4 [-]	0.3 [-]
%INCREASE		NEW	2 (40%)	1 (17%)	3 (27%)	4 (80%)	4 (67%)	8 (73%)
	#	RETURN	6 (21%)	4 (15%)	10 (18%)	14 (50%)	12 (44%)	26 (47%)
P-VALUE			-	-	-	-	-	-

Table 9a - Skin Self-Examination Practices as a Function of Subject Groups.

GROUPS		MELANOMA	SSE NEVUS	CONTROL	p-value	PLC	SSE CONTROL	p-value
BEFORE		49.6 [B]	18.0 [C]	77.2 [A]		33.7 [-]	77.2 [-]	
AFTER		52.3 [B]	15.2 [C]	76.8 [A]		33.7 [-]	76.8 [-]	
IMPROVEMENT		2.7 [-]	-2.8 [-]	-0.5 [-]		0 [-]	-0.5 [-]	
%INCREAS	GENERAL	12 (36%)	13 (39%)	6 (30%)	-	25 (38%)	6 (30%)	-
% NO SSE								
BEFORE		4 (12%)	3 (9%)	6 (30%)	0.054	7 (11%)	6 (30%)	0.035
AFTER		4 (12%)	1 (3%)	5 (25%)	0.017	5 (8%)	5 (25%)	0.034
% OPTIMAL[1]								
BEFORE		9 (27%)	12 (36%)	3 (15%)	-	21 (32%)	3 (15%)	-
AFTER		16 (48%)	18 (55%)	6 (30%)	-	34 (52%)	6 (30%)	-
INCR OPTIMA		9 (27%)	5 (25%)	11 (33%)	-	20 (30%)	11 (33%)	-

[1] Optimal SSE defined to be from once per month to once every four months.

Table 9b - Skin Self-Examination as a Function of Sex.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		FEMALE	36.1 [-]	36.1 [-]	82.1 [-]	25.5 [-]	38.9 [-]
		MALE	70.3 [-]	70.3 [-]	65.8 [-]	51.5 [-]	54.7 [-]
AFTER		FEMALE	46.4 [-]	11.9 [B]	78.0 [-]	27.2 [-]	39.3 [-]
		MALE	61.4 [-]	25.4 [A]	73.8 [-]	47.7 [-]	53.5 [-]
IMPROVEM'T		FEMALE	10.3 [-]	-5.1 [-]	-4.1 [-]	1.7 [-]	0.4 [-]
		MALE	-8.9 [-]	4.5 [-]	8.0 [-]	-3.8 [-]	-1.2 [-]
%INCREAS		FEMALE	8 (40%)	9 (36%)	4 (29%)	17 (38%)	21 (36%)
		MALE	4 (31%)	4 (50%)	2 (10%)	8 (38%)	10 (37%)
P-VALUE			-	-	-	-	-
% NO SSE							
BEFORE	SEX	FEMALE	1 (5%)	3 (12%)	5 (36%)	4 (9%)	9 (15%)
		MALE	3 (23%)	0	1 (17%)	3 (14%)	4 (15%)
P-VALUE			-	-	-	-	-
AFTER		FEMALE	1 (5%)	1 (4%)	3 (21%)	2 (4%)	5 (8%)
		MALE	3 (23%)	0	2 (33%)	3 (14%)	5 (19%)
P-VALUE			-	-	-	-	-
% OPTIMAL							
BEFORE		FEMALE	7 (35%)	9 (36%)	0	16 (36%)	16 (27%)
		MALE	2 (22%)	3 (38%)	3 (50%)	5 (24%)	8 (30%)
P-VALUE			-	-	0.005	-	-
AFTER		FEMALE	11 (55%)	13 (52%)	5 (36%)	24 (53%)	11 (41%)
		MALE	5 (38%)	5 (63%)	1 (17%)	10 (48%)	14 (52%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		FEMALE	3 (23%)	8 (32%)	5 (36%)	14 (31%)	19 (32%)
		MALE	3 (23%)	3 (38%)	0	6 (29%)	6 (22%)
P-VALUE			-	-	-	-	-

Table 9c - Skin Self-Examination as a Function of Age.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		18-35	10.8 [-]	24.3 [-]	156.0 [-]	20.5 [-]	37.4 [-]
		36-50	52.2 [-]	12.7 [-]	98.8 [-]	32.4 [-]	41.3 [-]
		51-85	63.8 [-]	12.4 [-]	52.4 [-]	50.3 [-]	51.1 [-]
AFTER		18-35	22.0 [-]	13.4 [-]	125.3 [-]	15.9 [-]	29.5 [-]
		36-50	46.3 [-]	19.2 [-]	107.0 [-]	32.7 [-]	42.6 [-]
		51-85	70.9 [-]	10.0 [-]	56.2 [-]	54.8 [-]	55.4 [-]
IMPROVEM'T		18-35	11.2 [-]	-10.9 [-]	-30.7 [-]	-4.6 [-]	-7.9 [-]
		36-50	-5.9 [-]	6.5 [-]	8.3 [-]	0.3 [-]	1.4 [-]
		51-85	7.1 [-]	-2.4 [-]	3.9 [-]	4.6 [-]	4.3 [-]
%INCREAS		18-35	4 (67%)	5 (33%)	0	9 (43%)	9 (38%)
		36-50	5 (38%)	7 (54%)	1 (25%)	12 (46%)	13 (43%)
		51-85	3 (21%)	1 (20%)	5 (38%)	4 (21%)	9 (28%)
P-VALUE			-	-	-	-	-
% NO SSE							
BEFORE	AGE	18-35	1 (17%)	2 (13%)	1 (33%)	3 (14%)	4 (17%)
		36-50	1 (8%)	0	0	1 (4%)	1 (3%)
		51-85	2 (14%)	1 (20%)	5 (38%)	3 (16%)	8 (25%)
P-VALUE			-	-	-	-	-
AFTER		18-35	1 (17%)	1 (7%)	1 (33%)	2 (10%)	3 (13%)
		36-50	1 (8%)	0	1 (25%)	1 (4%)	2 (7%)
		51-85	2 (14%)	0	3 (23%)	2 (10%)	5 (16%)
P-VALUE			-	-	-	-	-
% OPTIMAL							
BEFORE		18-35	2 (33%)	6 (40%)	0	8 (38%)	8 (33%)
		36-50	2 (15%)	5 (38%)	1 (25%)	7 (27%)	8 (27%)
		51-85	5 (36%)	1 (20%)	2 (15%)	6 (32%)	8 (25%)
P-VALUE			-	-	-	-	-
AFTER		18-35	3 (50%)	8 (53%)	1 (33%)	11 (52%)	12 (50%)
		36-50	6 (46%)	6 (46%)	1 (25%)	12 (46%)	13 (43%)
		51-85	7 (50%)	4 (80%)	4 (31%)	11 (48%)	15 (47%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		18-35	2 (33%)	5 (33%)	1 (33%)	7 (33%)	8 (33%)
		36-50	4 (31%)	3 (23%)	1 (25%)	7 (27%)	8 (27%)
		51-85	3 (21%)	3 (60%)	3 (23%)	6 (32%)	9 (28%)
P-VALUE			-	-	-	-	-

Table 9d - Skin Self-Examination as a Function of Hair Color.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		BLOND/RED	60.3 [-]	25.8 [-]	48.3 [-]	46.5 [-]	46.8 [-]
		L. BROWN	45.4 [-]	8.8 [-]	168.8 [-]	35.6 [-]	68.9 [-]
		BRWN/BLCK	41.4 [-]	16.7 [-]	46.1 [-]	24.7 [-]	30.3 [-]
AFTER		BLOND/RED	75.0 [-]	15.3 [-]	42.0 [-]	51.1 [-]	49.6 [-]
		L. BROWN	34.2 [-]	7.5 [-]	152.8 [-]	27.1 [-]	58.5 [-]
		BRWN/BLCK	45.0 [-]	16.6 [-]	54.8 [-]	25.7 [-]	33.4 [-]
IMPROVEM'T		BLOND/RED	14.8 [-]	-10.5 [-]	-6.3 [-]	4.7 [-]	2.8 [-]
		L. BROWN	-11.2 [-]	-1.3 [-]	-16.0 [-]	-8.5 [-]	-10.4 [-]
		BRWN/BLCK	3.6 [-]	-0.1 [-]	8.7 [-]	1.1 [-]	3.1 [-]
%INCREAS		BLOND/RED	5 (42%)	3 (38%)	1 (25%)	8 (40%)	9 (38%)
		L. BROWN	3 (27%)	1 (25%)	1 (20%)	4 (27%)	5 (25%)
		BRWN/BLCK	4 (40%)	9 (43%)	4 (36%)	13 (42%)	17 (40%)
P-VALUE	HAIR COLOR		-	-	-	-	-
% NO SSE							
BEFORE		BLOND/RED	0	2 (25%)	2 (50%)	2 (10%)	4 (17%)
		L. BROWN	1 (9%)	0	1 (20%)	1 (7%)	2 (10%)
		BRWN/BLCK	3 (30%)	1 (5%)	3 (27%)	4 (13%)	7 (17%)
P-VALUE			0.036	-	-	-	-
AFTER		BLOND/RED	0	0	2 (50%)	0	2 (8%)
		L. BROWN	1 (9%)	1 (25%)	1 (20%)	2 (13%)	3 (15%)
		BRWN/BLCK	3 (30%)	0	2 (18%)	3 (10%)	5 (12%)
P-VALUE			0.036	-	-	-	-
% OPTIMAL							
BEFORE		BLOND/RED	3 (25%)	2 (25%)	0	5 (25%)	5 (21%)
		L. BROWN	3 (27%)	4 (100%)	1 (20%)	7 (47%)	8 (40%)
		BRWN/BLCK	3 (30%)	6 (29%)	2 (18%)	9 (29%)	11 (26%)
P-VALUE			-	-	-	-	-
AFTER		BLOND/RED	6 (50%)	7 (88%)	1 (25%)	13 (65%)	15 (48%)
		L. BROWN	6 (55%)	3 (75%)	1 (20%)	9 (60%)	10 (50%)
		BRWN/BLCK	4 (40%)	8 (38%)	4 (36%)	12 (39%)	16 (38%)
P-VALUE			-	0.014	-	0.059	-
INCR OPTIMA		BLOND/RED	5 (42%)	5 (63%)	1 (25%)	10 (50%)	11 (46%)
		L. BROWN	3 (27%)	0	1 (20%)	3 (20%)	4 (20%)
		BRWN/BLCK	1 (10%)	6 (29%)	3 (27%)	7 (23%)	10 (24%)
P-VALUE			-	-	-	0.051	-

Table 9e - Skin Self-Examination as a Function of Eye Color.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		BLUE/GREEN	50.1 [-]	15.7 [-]	89.1 [-]	35.8 [-]	45.5 [-]
		HAZEL/GREY	81.7 [-]	10.8 [-]	12.3 [-]	49.5 [-]	41.5 [-]
		L/D BROWN	15.5 [-]	23.3 [-]	99.3 [-]	20.8 [-]	44.1 [-]
AFTER		BLUE/GREEN	56.1 [-]	15.3 [-]	75.0 [-]	39.1 [-]	45.6 [-]
		HAZEL/GREY	76.0 [-]	16.8 [-]	18.3 [-]	49.1 [-]	42.5 [-]
		L/D BROWN	15.3 [-]	14.3 [-]	110.0 [-]	14.6 [-]	42.9 [-]
IMPROVEM'T		BLUE/GREEN	6.0 [-]	-0.3 [-]	-14.1 [-]	3.3 [-]	0.2 [-]
		HAZEL/GREY	-5.7 [-]	6.0 [-]	6.0 [-]	-0.4 [-]	1.0 [-]
		L/D BROWN	-0.2 [-]	-9.0 [-]	10.8 [-]	-6.2 [-]	-1.2 [-]
%INCREAS		BLUE/GREEN	8 (38%)	5 (33%)	2 (25%)	13 (36%)	15 (34%)
	COLOR	HAZEL/GREY	2 (33%)	2 (40%)	1 (33%)	4 (36%)	5 (36%)
		L/D BROWN	2 (33%)	6 (46%)	3 (38%)	8 (42%)	11 (41%)
P-VALUE			-	-	-	-	-
% NO SSE							
BEFORE		BLUE/GREEN	3 (14%)	2 (13%)	3 (38%)	5 (14%)	8 (18%)
		HAZEL/GREY	0	1 (20%)	0	1 (9%)	1 (7%)
	EYE	L/D BROWN	1 (17%)	0	2 (25%)	1 (5%)	3 (11%)
P-VALUE			-	-	-	-	-
AFTER		BLUE/GREEN	3 (14%)	1 (7%)	2 (25%)	4 (11%)	6 (14%)
		HAZEL/GREY	0	0	1 (33%)	0	1 (7%)
		L/D BROWN	1 (17%)	0	1 (13%)	1 (5%)	2 (7%)
P-VALUE			-	-	-	-	-
% OPTIMAL							
BEFORE		BLUE/GREEN	4 (19%)	5 (33%)	0	9 (25%)	9 (20%)
		HAZEL/GREY	1 (17%)	3 (60%)	1 (33%)	4 (36%)	5 (36%)
		L/D BROWN	4 (67%)	4 (31%)	2 (25%)	8 (42%)	10 (37%)
P-VALUE			0.044	-	-	-	-
AFTER		BLUE/GREEN	8 (38%)	9 (60%)	3 (38%)	17 (47%)	20 (45%)
		HAZEL/GREY	4 (67%)	3 (60%)	1 (33%)	7 (64%)	8 (57%)
		L/D BROWN	4 (67%)	6 (46%)	2 (25%)	10 (53%)	12 (44%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		BLUE/GREEN	5 (24%)	5 (33%)	3 (38%)	10 (28%)	13 (30%)
		HAZEL/GREY	3 (50%)	2 (40%)	0	5 (45%)	5 (36%)
		L/D BROWN	1 (17%)	4 (31%)	2 (25%)	5 (26%)	7 (26%)
P-VALUE			-	-	-	-	-

Table 9f - Skin Self-Examination as a Function of Tendency to Sunburn.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		SUNBURN	57.3 [-]	8.5 [-]	73.6 [-]	27.4 [-]	37.3 [-]
		MILD-MOD TAN	43.1 [-]	25.8 [-]	57.6 [-]	40.1 [-]	43.9 [-]
AFTER		SUNBURN	53.8 [-]	9.6 [-]	66.2 [-]	33.7 [-]	40.7 [-]
		MILD-MOD TAN	50.5 [-]	19.8 [-]	63.9 [-]	33.8 [-]	40.2 [-]
IMPROV		SUNBURN	10.7 [-]	1.1 [-]	-7.3 [-]	6.3 [-]	3.4 [-]
		MILD-MOD TAN	-6.8 [-]	-6.0 [-]	6.3 [-]	-6.4 [-]	-3.6 [-]
%INCREAS		SUNBURN	7 (39%)	6 (40%)	2 (22%)	13 (39%)	15 (36%)
		MILD-MOD TAN	5 (33%)	7 (39%)	4 (44%)	12 (36%)	16 (38%)
P-VALUE			-	-	-	-	-
% NO SSE							
BEFORE		SUNBURN	2 (11%)	2 (13%)	4 (44%)	4 (12%)	8 (19%)
		MILD-MOD TAN	2 (13%)	1 (6%)	1 (11%)	3 (9%)	4 (10%)
P-VALUE			-	-	-	-	-
AFTER		SUNBURN	2 (11%)	0	4 (44%)	2 (6%)	6 (14%)
		MILD-MOD TAN	2 (13%)	1 (6%)	0	3 (9%)	3 (7%)
P-VALUE			-	-	-	-	-
% OPTIMAL							
BEFORE		SUNBURN	6 (33%)	5 (33%)	0	11 (33%)	11 (26%)
		MILD-MOD TAN	3 (20%)	7 (39%)	3 (33%)	10 (30%)	13 (31%)
P-VALUE			-	-	-	-	-
AFTER		SUNBURN	10 (56%)	8 (53%)	2 (22%)	18 (55%)	20 (48%)
		MILD-MOD TAN	6 (40%)	10 (56%)	4 (44%)	16 (48%)	20 (48%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		SUNBURN	6 (33%)	5 (33%)	2 (22%)	11 (33%)	13 (31%)
		MILD-MOD TAN	3 (20%)	6 (33%)	3 (33%)	9 (27%)	12 (29%)
P-VALUE			-	-	-	-	-

TENDENCY TO BURN

Table 9g - Skin Self-Examination as a Funtion of Inability to Tan.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		FRCKL/PEEL+TAN	61.7 [-]	19.5 [-]	121.8 [-]	42.9 [-]	60.1 [-]
		MOD-DEEP TAN	44.3 [-]	17.8 [-]	43.9 [-]	30.8 [-]	33.6 [-]
AFTER		FRCKL/PEEL+TAN	84.2 [-]	18.4 [-]	85.6 [-]	54.9 [-]	61.6 [-]
		MOD-DEEP TAN	38.4 [-]	14.5 [-]	57.2 [-]	26.2 [-]	32.9 [-]
IMPROVEM'T		FRCKL/PEEL+TAN	22.5 [-]	-1.1 [-]	-36.2 [-]	12.0 [-]	1.5 [-]
		MOD-DEEP TAN	-5.9 [-]	-3.4 [-]	13.2 [-]	-4.6 [-]	-0.7 [-]
%INCREAS	INABILITY TO TAN	FRCKL/PEEL+TAN	3 (30%)	3 (38%)	0	6 (33%)	6 (26%)
		MOD-DEEP TAN	9 (39%)	10 (42%)	6 (46%)	19 (40%)	25 (42%)
P-VALUE			-	-	-	-	-
% NO SSE							
BEFORE		FRCKL/PEEL+TAN	0	1 (13%)	1 (20%)	1 (5%)	2 (9%)
		MOD-DEEP TAN	4 (17%)	2 (8%)	4 (31%)	6 (13%)	10 (17%)
P-VALUE			-	-	-	-	-
AFTER		FRCKL/PEEL+TAN	0	0	2 (40%)	0	2 (9%)
		MOD-DEEP TAN	4 (17%)	1 (4%)	2 (15%)	5 (11%)	7 (12%)
			-	-	-	-	-
% OPTIMAL							
BEFORE		FRCKL/PEEL+TAN	3 (30%)	2 (25%)	0	5 (28%)	5 (22%)
		MOD-DEEP TAN	6 (26%)	9 (38%)	3 (23%)	15 (32%)	18 (30%)
P-VALUE			-	-	-	-	-
AFTER		FRCKL/PEEL+TAN	5 (50%)	5 (63%)	1 (20%)	10 (56%)	11 (48%)
		MOD-DEEP TAN	11 (48%)	12 (50%)	5 (38%)	23 (49%)	28 (47%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		FRCKL/PEEL+TAN	3 (30%)	3 (38%)	1 (20%)	6 (33%)	7 (30%)
		MOD-DEEP TAN	6 (26%)	8 (33%)	4 (31%)	14 (30%)	18 (30%)
P-VALUE			-	-	-	-	-

Table 9h - Skin Self-Examination as a Function of the Objective, Physician-Determined Nevus Count.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		0-33	46.9 [B]	16.0 [-]	71.2	35.3 [-]	48.7 [-]
		34-66	27.4 [C]	17.3 [-]	-	21.9 [-]	21.9 [-]
		67-100 & 100+	141.3 [A]	21.3 [-]	-	51.3 [-]	51.3 [-]
AFTER		0-33	57.5 [-]	13.0 [-]	131.6	40.8 [-]	55.5 [-]
		34-66	18.8 [-]	10.9 [-]	-	14.5 [-]	14.5 [-]
		67-100 & 100+	129.33 [-]	23.7 [-]	-	50.1 [-]	50.1 [-]
IMPROVEM'T	COUNT	0-33	10.6 [-]	-3.0 [-]	9.0	5.5 [-]	6.8 [-]
		34-66	-8.6 [-]	-6.4 [-]	-	-7.4 [-]	-7.4 [-]
		67-100 & 100+	-12.0 [-]	2.3 [-]	-	-1.3 [-]	-1.3 [-]
%INCREAS		0-33	8 (40%)	6 (50%)	6 (32%)	14 (44%)	20 (39%)
		34-66	4 (40%)	3 (25%)	-	7 (32%)	7 (32%)
		67-100 & 100+	0	4 (44%)	-	4 (33%)	4 (33%)
P-VALUE			-	-	-	-	-
% NO SSE					-		
BEFORE		0-33	4 (20%)	3 (25%)	6 (32%)	7 (22%)	13 (25%)
		34-66	0	0	-	0	0
		67-100 & 100+	0	0	-	0	0
P-VALUE			-	0.04	-	0.011	0.004
AFTER		0-33	4 (20%)	0	5 (26%)	4 (13%)	9 (18%)
		34-66	0	0	-	0	0
		67-100 & 100+	0	1 (11%)	-	1 (8%)	1 (8%)
P-VALUE			-	-	-	-	-
% OPTIMAL							
BEFORE		0-33	5 (25%)	5 (42%)	3 (16%)	10 (31%)	13 (25%)
		34-66	4 (40%)	4 (33%)	-	8 (36%)	8 (36%)
		67-100 & 100+	0	3 (33%)	-	3 (25%)	3 (25%)
P-VALUE			-	-	-	-	-
AFTER		0-33	7 (35%)	8 (67%)	5 (26%)	15 (47%)	20 (39%)
		34-66	7 (70%)	6 (50%)	-	13 (59%)	13 (59%)
		67-100 & 100+	2 (67%)	4 (44%)	-	6 (50%)	6 (50%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		0-33	4 (20%)	6 (50%)	4 (21%)	10 (31%)	14 (27%)
		34-66	3 (30%)	3 (25%)	-	6 (27%)	6 (27%)
		67-100 & 100+	2 (67%)	2 (22%)	-	4 (33%)	4 (33%)
P-VALUE			-	-	-	-	-

NEVUS OBJECTIVE

Table 9i - Skin Self-Examination as a Function of the Subjective, Self-Determined Nevus Count.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		0-33	20.0 [-]	14.9 [-]	79.5 [-]	17.9 [-]	39.2 [-]
		34-66	94.1 [-]	12.5 [-]	0 [-]	64.5 [-]	59.1 [-]
		67-100 & 100^{+}	85.4 [-]	21.8 [-]	96.5 [-]	41.2 [-]	45.6 [-]
AFTER		0-33	15.3 [C]	11.9 [-]	89.4 [-]	13.9 [B]	40.1 [-]
		34-66	131.4 [A]	17.0 [-]	4.0 [-]	89.8 [A]	82.7 [-]
		67-100 & 100^{+}	73.7 [B]	17.3 [-]	6.0 [-]	34.5 [B]	32.2 [-]
IMPROVEM'T		0-33	-4.7 [-]	-2.9 [-]	9.9 [-]	-4.0 [-]	0.8 [B]
		34-66	37.3 [-]	4.5 [-]	4.0 [-]	25.4 [-]	23.6 [A]
		67-100 & 100^{+}	-11.7 [-]	-4.5 [-]	-90.5 [-]	-6.7 [-]	-13.4 [C]
%INCREAS		0-33	7 (37%)	8 (62%)	5 (29%)	15 (67%)	20 (41%)
		34-66	3 (43%)	1 (25%)	1 (100%)	4 (36%)	5 (42%)
		67-100 & 100^{+}	2 (29%)	4 (25%)	0	6 (26%)	6 (24%)
P-VALUE			-	0.051	-	-	-
% NO SSE							
BEFORE		0-33	3 (16%)	3 (23%)	5 (29%)	6 (19%)	11 (22%)
		34-66	0	0	1 (100%)	0	1 (8%)
		67-100 & 100^{+}	1 (14%)	0	0	1 (4%)	1 (4%)
P-VALUE			-	0.036	-	-	0.031
AFTER		0-33	3 (16%)	0	4 (24%)	3 (9%)	7 (14%)
		34-66	0	0	0	0	0
		67-100 & 100^{+}	1 (14%)	1 (6%)	1 (50%)	2 (9%)	3 (12%)
P-VALUE			-	-	-	-	-
% OPTIMAL							
BEFORE		0-33	6 (32%)	4 (38%)	3 (18%)	10 (31%)	13 (27%)
		34-66	3 (43%)	3 (75%)	0	6 (55%)	6 (50%)
		67-100 & 100^{+}	0	5 (31%)	0	5 (22%)	5 (20%)
P-VALUE			-	-	-	-	-
AFTER		0-33	11 (58%)	8 (62%)	4 (24%)	19 (59%)	23 (47%)
		34-66	3 (43%)	2 (50%)	1 (100%)	5 (45%)	6 (50%)
		67-100 & 100^{+}	2 (29%)	8 (50%)	1 (50%)	10 (43%)	11 (44%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		0-33	7 (37%)	7 (54%)	3 18%)	14 (44%)	17 (35%)
		34-66	0	0	1 (100%)	0	1 (8%)
		67-100 & 100^{+}	2 (29%)	4 (25%)	1 (50%)	6 (26%)	7 (28%)
P-VALUE			-	-	-	-	-

SUBJECTIVE NEVUS COUNT

Table 9j - Skin Self-Examination as a Function of the Belief in Having More-than-Average Nevus Counts.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		YES	82.5 [-]	16.6 [-]	139.3 [-]	39.2 [-]	49.4 [-]
		NO	31.6 [-]	24.9 [-]	70.1 [-]	29.4 [-]	44.4 [-]
		DK	28.0 [-]	6.0 [-]	3.0 [-]	21.7 [-]	17.6 [-]
AFTER		YES	105.7 [-]	15.3 [-]	95.0 [-]	46.3 [-]	51.3 [-]
		NO	21.9 [-]	14.9 [-]	78.8 [-]	19.5 [-]	41.4 [-]
		DK	21.6 [-]	15.0 [-]	26.0 [-]	19.7 [-]	21.1 [-]
IMPROVEM'T		YES	23.2 [-]	-1.3 [-]	-44.3 [-]	7.1 [-]	1.8 [-]
		NO	-9.8 [-]	-10.0 [-]	8.7 [-]	-9.8 [-]	-3.0 [-]
		DK	-6.4 [-]	9.0 [-]	23.0 [-]	-2.0 [-]	3.6 [-]
%INCREAS	MORE NEVI ?	YES	5 (42%)	7 (30%)	1 (25%)	12 (34%)	13 (33%)
		NO	6 (38%)	4 (40%)	4 (29%)	10 (42%)	14 (37%)
		DK	1 (20%)	2 (100%)	1 (50%)	3 (43%)	4 (44%)
P-VALUE			-	0.051	-	-	-
% NO SSE							
BEFORE		YES	0	1 (4%)	1 (25%)	1 (3%)	2 (5%)
		NO	2 (13%)	1 (13%)	4 (29%)	3 (13%)	7 (18%)
		DK	2 (40%)	1 (50%)	1 (50%)	3 (43%)	4 (44%)
P-VALUE			0.029	0.032	-	0.002	0.004
AFTER		YES	0	1 (4%)	1 (25%)	1 (3%)	2 (5%)
		NO	2 (13%)	0	3 (21%)	2 (8%)	5 (13%)
		DK	2 (40%)	0	1 (50%)	2 (29%)	3 (33%)
P-VALUE			0.029	-	-	0.021	0.021
% OPTIMAL							
BEFORE		YES	3 (25%)	8 (35%)	0	11 (31%)	11 (28%)
		NO	5 (31%)	3 (38%)	2 (14%)	8 (33%)	10 (26%)
		DK	1 (20%)	1 (50%)	1 (50%)	2 (29%)	3 (33%)
P-VALUE			-	-	-	-	-
AFTER		YES	6 (50%)	11 (48%)	2 (50%)	17 (49%)	19 (49%)
		NO	9 (56%)	6 (75%)	4 (29%)	15 (63%)	19 (50%)
		DK	1 (20%)	1 (50%)	0	2 (29%)	2 (22%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		YES	3 (25%)	5 22%)	0	8 (23%)	10 (26%)
		NO	6 (38%)	5 (63%)	5 (26%)	11 (46%)	14 (37%)
		DK	0	1 (50%)	0	1 (14%)	1 (11%)
P-VALUE			-	-	-	-	-

Table 9k - Skin Self-Examination as a Function of Personal Hx of Atypical Nevi.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		YES	56.6 [-]	17.9	364.0 [A]	33.4 [-]	39.3 [-]
		NO	35.6 [-]	-	62.1 [B]	35.6 [-]	52.4 [-]
AFTER		YES	66.2 [-]	15.9	364.0 [A]	35.6 [-]	41.4 [-]
		NO	24.6 [-]	-	61.6 [B]	24.6 [-]	48.0 [-]
IMPROVEM'T		YES	9.6 [-]	-2.8	0.0 [-]	2.2 [-]	2.1 [-]
		NO	-11.1 [-]	-	-0.5 [-]	-11.1 [-]	-4.4 [-]
%INCREAS		YES	9 (41%)	13 (39%)	0	22 (40%)	22 (39%)
		NO	3 (27%)	-	6 (32%)	3 (27%)	9 (30%)
P-VALUE			-	-	-	-	-
% NO SSE							
BEFORE		YES	1 (5%)	3 (9%)	0	4 (7%)	4 (7%)
		NO	3 (27%)	-	6 (32%)	3 (27%)	9 (30%)
P-VALUE			-	-	-	0.051	0.005
AFTER		YES	1 (5%)	1 (3%)	0	2 (4%)	2 (4%)
		NO	3 (27%)	-	5 (26%)	3 (27%)	8 (27%)
P-VALUE			-	-	-	0.007	0.002
% OPTIMAL							
BEFORE		YES	7 (32%)	12 (36%)	0	19 (35%)	19 (34%)
		NO	2 (18%)	-	3 (16%)	2 (18%)	5 (17%)
P-VALUE			-	-	-	-	-
AFTER		YES	11 (50%)	18 (55%)	0	29 (53%)	29 (52%)
		NO	5 (45%)	-	6 (32%)	5 (45%)	11 (37%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		YES	6 (27%)	11 (33%)	0	17 (31%)	17 (30%)
		NO	3 (27%)	-	3 (16%)	3 (27%)	8 (27%)
P-VALUE			-	-	-	-	-

ATYPICAL NEVI Hx

Table 9I - Skin Self-Examination as a Function of PHx of Non-Mel. Skin Cancer

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		YES	42.7 [-]	7.2 [-]	202.7 [-]	24.9 [-]	60.5 [-]
		NO	51.1 [-]	20.3 [-]	57.8 [-]	35.7 [-]	40.8 [-]
AFTER		YES	75.3 [-]	10.5 [-]	142.7 [-]	42.9 [-]	62.9 [-]
		NO	47.2 [-]	16.2 [-]	67.7 [-]	31.7 [-]	39.9 [-]
IMPROVEM'T		YES	32.7 [-]	3.3 [-]	-60.0 [-]	18.0 [-]	2.4 [-]
		NO	-3.9 [-]	-4.1 [-]	9.9 [-]	-4.0 [-]	-0.8 [-]
%INCREAS		YES	1 (17%)	2 (33%)	0	3 (25%)	3 (20%)
		NO	11 (41%)	11 (41%)	5 (31%)	22 (41%)	27 (39%)
P-VALUE			-	-	-	-	-
% NO SSE							
BEFORE		YES	1 (17%)	1 (17%)	0	2 (17%)	2 (13%)
		NO	3 (11%)	2 (7%)	6 (38%)	5 (9%)	11 (16%)
P-VALUE			-	-	-	-	-
AFTER		YES	1 (17%)	0	0	1 (9%)	1 (7%)
		NO	3 (11%)	1 (4%)	5 (31%)	4 (7%)	9 (13%)
P-VALUE			-	-	-	-	-
% OPTIMAL							
BEFORE		YES	1 (17%)	4 (67%)	0	5 (42%)	5 (33%)
		NO	8 (30%)	8 (30%)	2 (13%)	16 (30%)	18 (26%)
P-VALUE			-	-	0.015	-	-
AFTER		YES	3 (50%)	3 (50%)	1 (33%)	6 (50%)	7 (47%)
		NO	13 (81%)	15 (56%)	5 (31%)	28 (52%)	33 (47%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		YES	2 (33%)	1 (17%)	1 (33%)	3 (25%)	4 (27%)
		NO	7 (26%)	10 (37%)	4 (25%)	17 (31%)	21 (30%)
P-VALUE			-	-	-	-	-

NON-MEL SKIN CA PHx

Table 9m - Skin Self-Examination as a Function of Family Hx of Melanoma.

GROUPS			MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		YES	17.0 [-]	25.1 [-]	52.0 [-]	22.0 [-]	24.4 [-]
		NO	60.3 [-]	14.5 [-]	82.4 [-]	41.7 [-]	53.5 [-]
		DK	78.0 [-]	5.8 [-]	68.0 [-]	29.8 [-]	42.6 [-]
AFTER		YES	19.1 [-]	20.5 [-]	32.0 [-]	20.0 [-]	20.9 [-]
		NO	67.7 [-]	12.8 [-]	95.7 [-]	45.5 [-]	59.9 [-]
		DK	32.0 [-]	5.3 [-]	12.0 [-]	14.2 [-]	13.4 [-]
IMPROVEM'T		YES	2.1 [-]	-4.6 [-]	-20.0 [-]	-2.0 [-]	-3.4 [B]
		NO	7.4 [-]	-1.7 [-]	13.3 [-]	3.7 [-]	6.5 [A]
		DK	-46.0 [-]	-0.5 [-]	-56.0 [-]	-15.7 [-]	-29.1 [C]
%INCREAS		YES	4 (44%)	8 (57%)	1 (50%)	12 (52%)	13 (52%)
		NO	8 (36%)	5 (33%)	4 (27%)	13 (35%)	17 (33%)
	M	DK	0	0	1 (33%)	0	1 (11%)
P-VALUE	M		-	-	-	0.026	0.054
% NO SSE							
BEFORE	x	YES	0	2 (14%)	1 (50%)	2 (9%)	3 (12%)
	H	NO	4 (18%)	1 (7%)	4 (27%)	5 (14%)	9 (17%)
	F	DK	0	0	1 (33%)	0	1 (11%)
P-VALUE			-	-	-	-	-
AFTER		YES	0	0	0	0	0
		NO	4 (18%)	1 (7%)	4 (27%)	5 (14%)	9 (17%)
		DK	0	0	1 (33%)	0	1 (11%)
P-VALUE			-	-	-	-	-
% OPTIMAL							
BEFORE		YES	3 (33%)	4 (29%)	0	7 (30%)	7 (28%)
		NO	6 (27%)	6 (40%)	2 (13%)	12 (32%)	14 (27%)
		DK	0	2 (50%)	1 (33%)	2 (33%)	3 (33%)
P-VALUE			-	-	-	-	-
AFTER		YES	7 (78%)	7 (50%)	1 (50%)	14 (61%)	15 (60%)
		NO	8 (30%)	9 (60%)	4 (27%)	17 (46%)	21 (40%)
		DK	1 (50%)	2 (50%)	1 (33%)	3 (50%)	4 (44%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		YES	4 (44%)	4 (29%)	1 (50%)	8 (35%)	9 (36%)
		NO	4 (18%)	7 (47%)	3 (20%)	11 (30%)	14 (27%)
		DK	1 (50%)	0	1 (33%)	1 (17%)	2 (22%)
			-	-	-	-	-

Table 9n - Skin Self-Examination as a Function of FHx of Non-Melanoma Skin Cancer

GROUPS			SSE MELANOMA	SSE NEVUS	CONTROL	PLC	ALL
BEFORE		YES	56.7 [-]	22.1 [-]	78.5 [-]	42.6 [-]	47.2 [-]
		NO	44.1 [-]	17.1 [-]	41.4 [-]	27.0 [-]	30.6 [-]
		DK	40.7 [-]	8.3 [-]	136.0 [-]	29.9 [-]	72.3 [-]
AFTER		YES	73.9 [-]	18.1 [-]	19.8 [-]	51.2 [-]	47.1 [-]
		NO	38.9 [-]	13.9 [-]	64.0 [-]	23.1 [-]	33.3 [-]
		DK	19.3 [-]	12.3 [-]	136.0 [-]	17.0 [-]	64.6 [-]
IMPROVEM'T		YES	17.2 [-]	-4.0 [-]	-58.8 [C]	8.6 [-]	-0.1 [-]
		NO	-5.2 [-]	-3.2 [-]	22.6 [A]	-3.9 [-]	2.7 [-]
		DK	-21.3 [-]	4.0 [-]	0.0 [B]	-12.9 [-]	-7.7 [-]
%INCREAS		YES	8 (50%)	3 (27%)	1 (25%)	11 (41%)	12 (39%)
		NO	4 (36%)	9 (47%)	4 (40%)	13 (43%)	17 (43%)
		DK	0	1 (33%)	1 (17%)	1 (11%)	2 (13%)
P-VALUE			0.036	-	-	-	0.054
% NO SSE							
BEFORE		YES	1 (6%)	0	0	1 (4%)	1 (3%)
		NO	3 (27%)	3 (16%)	5 (50%)	6 (20%)	11 (28%)
		DK	0	0	1 (17%)	0	1 (7%)
P-VALUE			-	-	-	-	-
AFTER		YES	1 (6%)	0	0	1 (4%)	1 (3%)
		NO	3 (27%)	1 (5%)	4 (40%)	4 (13%)	8 (20%)
		DK	0	0	1 (17%)	0	1 (7%)
P-VALUE			-	-	-	-	-
% OPTIMAL							
BEFORE		YES	3 (19%)	3 (27%)	2 (50%)	6 (22%)	8 (26%)
		NO	3 (27%)	7 (37%)	0	10 (33%)	10 (25%)
		DK	3 (50%)	2 (67%)	1 (17%)	5 (56%)	6 (40%)
P-VALUE			-	-	-	-	-
AFTER		YES	6 (38%)	5 (45%)	3 (75%)	11 (41%)	14 (45%)
		NO	6 (55%)	12 (63%)	2 (20%)	18 (60%)	20 (50%)
		DK	4 (67%)	1 (33%)	1 (17%)	5 (56%)	6 (40%)
P-VALUE			-	-	-	-	-
INCR OPTIMA		YES	5 (31%)	2 (18%)	2 (50%)	7 (26%)	9 (29%)
		NO	3 (27%)	9 (47%)	2 (20%)	12 (40%)	14 (35%)
		DK	1 (17%)	0	1 (17%)	1 (11%)	2 (13%)
P-VALUE			-	-	-	-	-

FHx NON-MELSKINCA

Table 9o - Skin Self-Examination as a Function of the # of Clinic Visits.

GROUPS			MELANOMA	SSE NEVUS	PLC
BEFORE		NEW	24.0 [-]	49.7 [A]	38.0 [-]
		RETURN	54.1 [-]	10.9 [B]	32.9 [-]
AFTER		NEW	12.8 [-]	22.7 [-]	18.2 [-]
		RETURN	59.4 [-]	13.5 [-]	36.8 [-]
IMPROVEMENT		NEW	-11.2 [-]	-27.0 [B]	-19.8 [-]
		RETURN	5.2 [-]	2.6 [A]	3.9 [-]
%INCREAS	NEW	NEW	3 (60%)	1 (17%)	4 (36%)
		RETURN	9 (32%)	12 (44%)	21 (38%)
P-VALUE			-	-	-
% NO SSE					
BEFORE	VS.	NEW	1 (20%)	0	1 (9%)
		RETURN	3 (11%)	3 (11%)	6 (11%)
P-VALUE			-	-	-
AFTER		NEW	1 (20%)	1 (17%)	2 (18%)
		RETURN	3 (11%)	0	3 (5%)
P-VALUE			-	0.034	-
% OPTIMAL					
BEFORE		NEW	1 (20%)	1 (17%)	2 (18%)
		RETURN	8 (29%)	11 (41%)	19 (34%)
P-VALUE			-	-	-
AFTER		NEW	2 (40%)	2 (35%)	4 (36%)
		RETURN	14 (50%)	16 (59%)	30 (55%)
P-VALUE			-	-	-
INCR OPTIMA		NEW	2 (40%)	2 (33%)	4 (36%)
		RETURN	7 (25%)	9 (33%)	16 (29%)
P-VALUE			-	-	-

Table 10 - Skin Self-Examination of Different Body Zones.

	PLC	CONTROL	p-value
Exam w/ "Buddy"[1]			
Optimal?	26 (39%)	6 (30%)	-
MEAN	19 [-]	28 [-]	
Examine Front[1]			
Optimal?	30 (45%)	4 (20%)	0.043
MEAN	68 [-]	95 [-]	
Examine Sides[1]			
Optimal?	32 (48%)	3 (15%)	0.008
MEAN	48 [B]	127 [A]	
Examine Back[1]			
Optimal?	30 (45%)	6 (30%)	0.043
MEAN	31 [-]	71 [-]	
Examine Other (Scalp/Genitals)[2]			
Optimal?	23 (35%)	3 (15%)	-
MEAN	15 [B]	84 [A]	

[1] Defined to be from three times per year to once a month.

[2] Defined to be from once per year to once a month.

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