

Opposing Views of 2 Academies About the Nature of Solar Keratosis

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These are facts: (1) Clinically, it is not possible to determine where a so-called solar keratosis (actinic keratosis [AK]) ends and a so-called invasive squamous cell carcinoma (SCC) begins (Figure 1). (2) Histopathologically, it is not possible to determine where a so-called solar keratosis ends and a so-called invasive SCC begins (Figure 2). (3) Biologically, what begins as a so-called solar keratosis may eventuate in metastatic SCC.

How are these facts best explained? By recognizing that solar keratosis, clinically, histopathologically, and biologically, is an SCC of one type at a superficial (early, incipient) stage of that malignant neoplastic process. Other types of cutaneous SCC are Bowen's, bowenoid papulosis, keratoacanthomatous (follicular and non-follicular),¹ and verrucous. Each of those types of SCC fulfills criteria for malignancy, namely, the capability (if left to its own devices) to kill either by destruction of tissue locally or by metastasis widely.

Clinically, a superficial SCC of the solar keratotic type (solar keratosis) is a scaly or keratotic papule or plaque whose base often is red. Histopathologically, such a lesion involves the lower part of the surface epidermis and the outer part of the infundibular epidermis. The keratocytes that comprise it have nuclei that are crowded, large, pleomorphic, and heterochromatic, as well as being in mitosis episodically, and cytoplasm that is eosinophilic, a sign of aberrant cornification. Other signs of abnormal cornification in solar keratosis are dyskeratotic cells and parakeratosis. These findings,



Figure 1. Clinically, it is not possible to determine where “solar keratosis” ends and “squamous cell carcinoma” begins.

in toto, by inspection grossly and by examination microscopically, are diagnostic of SCC.

Since the Ackerman Academy of Dermatopathology was begun in 1999, no small number of publications about solar keratosis have emanated from it²⁻¹⁰—all of them unwaveringly consistent in propounding the principle that solar keratosis **is** SCC, **not** a precancer or a precursor of cancer. The American Academy of Dermatology (AAD) was founded in 1938, and during the years that followed, never has it stated, straightforwardly, unequivocally, and unambiguously, that solar keratosis **is** SCC. On the contrary, it has averred, repeatedly, that solar keratosis is a precancer, a precursor of cancer, or a condition *en route* to cancer. In the past year, the AAD published separate brochures for “Actinic Keratoses” and “Squamous Cell Carcinoma.”^{11,12} What follows are quotations

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from publications of the AAD between 1999 and 2003 about the subject of solar keratosis.

1999

The following quotations are from conference proceedings printed in 1999 in *Dermatology World*,¹³ an official publication of the AAD.

Moulds—“Millions of these lesions [AKs] are seen in this country each year, and a substantial number will progress to squamous cell carcinoma unless appropriately treated.”¹³

Moy—“The exact rate at which actinic keratoses progress to squamous cell carcinoma that involves the dermis can be argued. . . it is impossible to predict the exact outcome of an individual actinic keratosis, or to always be able to distinguish the difference between an AK and SCC. . . Actinic keratosis is the initial lesion in a disease continuum that progresses to squamous cell carcinoma. . . The thickened lesions can progress to squamous cell carcinoma and be indistinguishable from squamous cell carcinoma.”¹³

Glogau—“Clinically actinic keratoses observed over some interval of time have three evolutionary possibilities: 1) spontaneous resolution, 2) persistence, or, 3) evolution into invasive skin cancer, usually squamous cell carcinoma. . . You know the overall risk is probably low, but how do you pick which one will become malignant?”¹³

Salasche—“There are many more actinic keratoses than squamous cell carcinomas. The order of magnitude is exponential. Therefore, it is clear that all AKs do not progress to invasive SCC. . . Although any one individual actinic keratosis has a low probability to progress to an invasive squamous cell carcinoma, their importance lies in that they identify individuals who not only have a unique genetic propensity but also sufficient solar radiation exposure and consequent damage to produce these incipient malignancies.”¹³

Cockerell—“The exact number of AKs that progress to involve the dermis is unknown, and estimates vary depending on the number of risk factors present. The likelihood of a fully developed SCC evolving from a given AK has been estimated to occur at a rate of 0.075-0.096% per lesion per year. Thus, for a person with 7.7 AKs, the average number present on the skin of an affected individual, SCC would develop at a rate of 10.2% over 10 years.”¹³

2000

Callen—“Although it is usually possible to diagnose actinic keratosis on the basis of the clinical appearance of a lesion, it may on occasion be

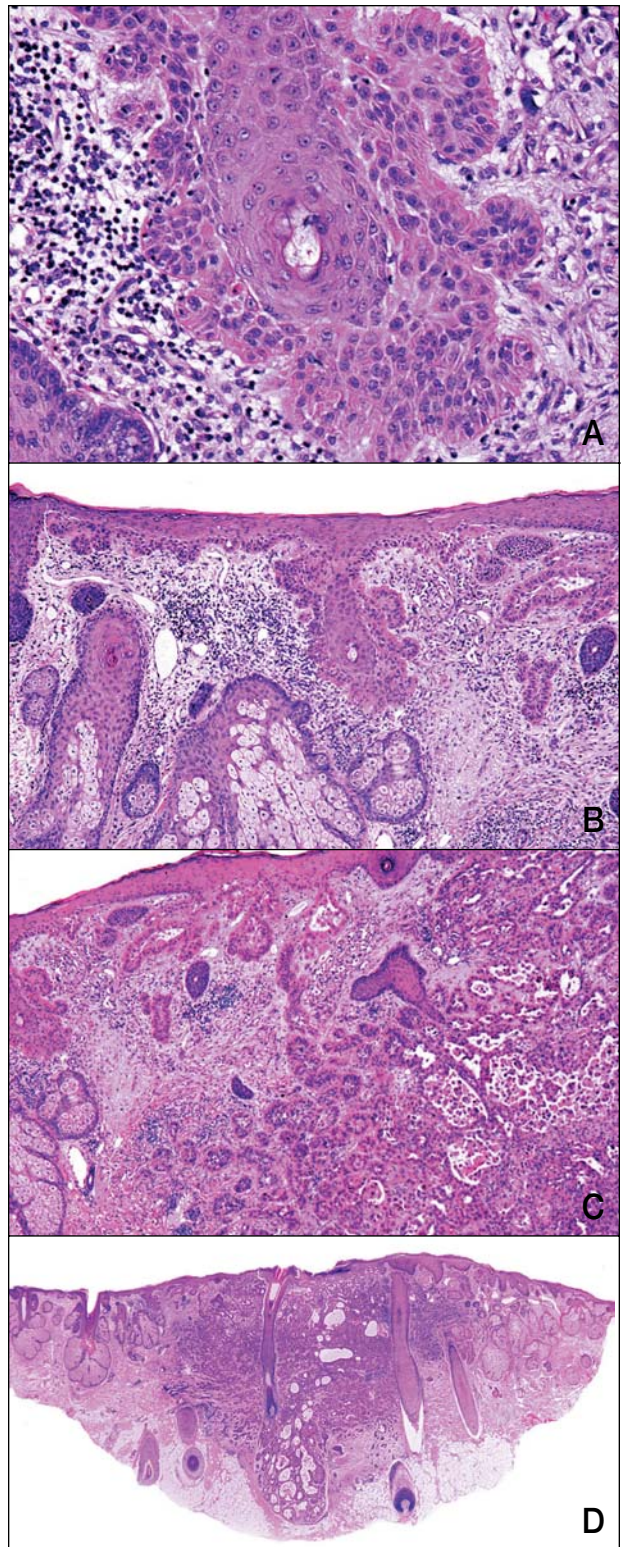


Figure 2. In this section of tissue, it is not possible to determine histopathologically where “solar keratosis” ends and “squamous cell carcinoma” begins (A through D). Note that the cytopathologic attributes of the so-called solar keratosis shown at high magnification (A) are the very same as those of the so-called squamous cell carcinoma depicted at progressively lower magnifications (H&E).

difficult (or impossible) to distinguish one from a squamous cell carcinoma of the skin. . . . Actinic keratoses must be evaluated because of their role as a precursor of invasive squamous cell carcinoma. Early treatment of these lesions may avoid potential invasion and more extensive treatment of subsequent malignancy."¹⁴

Salasche—"There are relatively more AKs on the dorsum of the hand and forearms than SCCs in these locations when compared with the AK/SCC ratio on the face."¹⁵

Moy—"Actinic keratosis is a skin lesion that can progress to squamous cell carcinoma but cannot always be clinically distinguished from a squamous cell carcinoma."¹⁶

Cockerell—"Because AK represents a proliferation of keratinocytes confined to the epidermis, one term we propose is *keratinocytic intraepidermal neoplasia* or *keratinocytic intraepidermal malignant neoplasia* (KIN) for this process."¹⁷

Leffell—"The laboratory techniques of molecular analysis have made it possible, when combined with our expanding knowledge of the human genome, to evaluate the genetic behavior of the AK cell and compare it with that of SCC and normal skin."¹⁸

Glogau—"Yet it is evident that the more severely photodamaged the skin, the higher the risk of progression from AK to SCC."¹⁹

Dinehart—"Because it is not possible to tell which AK will progress to SCC, treatment of all lesions is generally recommended."²⁰

2001

The following quotation is from *Derm Coding Consult*, an official publication of the AAD.

"Actinic keratoses (AKs), also known as solar keratoses, are common, sun-induced lesions that are confined to the epidermis and have the potential to become cancer. . . . An alternative approach to treating AKs is to observe the lesions over time and remove them only if they exhibit specific clinical features suggesting possible transformation to invasive squamous cell carcinoma (SCC)."²¹

2002

Dermatology World—"The Academy's [AAD] comprehensive educational campaign to heighten public awareness of AKs included a series of events to educate and screen the public for AKs and skin cancer."²²

AAD—"Further changes in cell growth can turn AKs into squamous cell carcinoma, a type of skin cancer. . . . Sun damage to unprotected skin begins in childhood and puts the child at risk for actinic keratoses and skin cancer later in life."¹¹

The Ackerman Academy of Dermatopathology has set forth, in what is meant to be logical, incisive, and compelling fashion, reasons why solar keratosis is SCC. In contrast, the AAD has not provided any evidence on behalf of its theses (the plural being apropos, ever changing as those theses are, from solar keratosis being a precancer/precursor of SCC to being an early stage in the evolution of SCC) and has not uttered a syllable of refutation of the assertion that solar keratosis is SCC.

It is curious that dermatologists and general pathologists everywhere are able to comprehend readily that Bowen disease is a superficial expression of SCC of one type, but they are refractory to the idea that solar keratosis, which is made up of the very same neoplastic cells as those that constitute Bowen disease (a neoplasm which, at times, so closely simulates solar keratosis histopathologically that the latter then is designated "bowenoid solar keratosis"), is just as authentically an SCC as is Bowen disease! The reason for that seeming contradiction is that the synonym, worldwide, for Bowen disease is "squamous cell carcinoma in situ," but the synonyms for solar keratosis are simply *actinic keratosis* and *senile keratosis*, neither of which, like the term solar keratosis itself, communicates any sense for the essentially carcinomatous character of the neoplasm. Moreover, ever since Dubreuilh, more than a century ago, spawned the ill-conceived notion of "precancerosis," solar keratosis has been deemed to be the stereotypical example of it. Dermatologists and general pathologists also appreciate readily the fact that superficial basal cell carcinoma, as the title of it conveys clearly, is a true basal cell carcinoma. In fact, solar keratosis, Bowen disease, and superficial basal cell carcinoma are superficial carcinomas with potential to develop chronologically in similar fashion (ie, descending progressively further into the dermis and tissues subjacent to it)(Figure 3).

In 1938, the year of the birth of the AAD, Richard L. Sutton, Jr., wrote these perceptive, poignant, and largely correct lines about solar keratosis²³:

"They [senile keratoses] are called 'precancerous' lesions; they are, in fact, cancerous already—early, superficial, and requiring time to manifest those characteristics of cancer with which all clinicians are familiar, but cancerous, nevertheless.

Either a lesion does become cancer or it does not. It is not eligible to statistical analysis. If it does become cancer, it must have been cancer; and if it is cancer, no one can say when it was

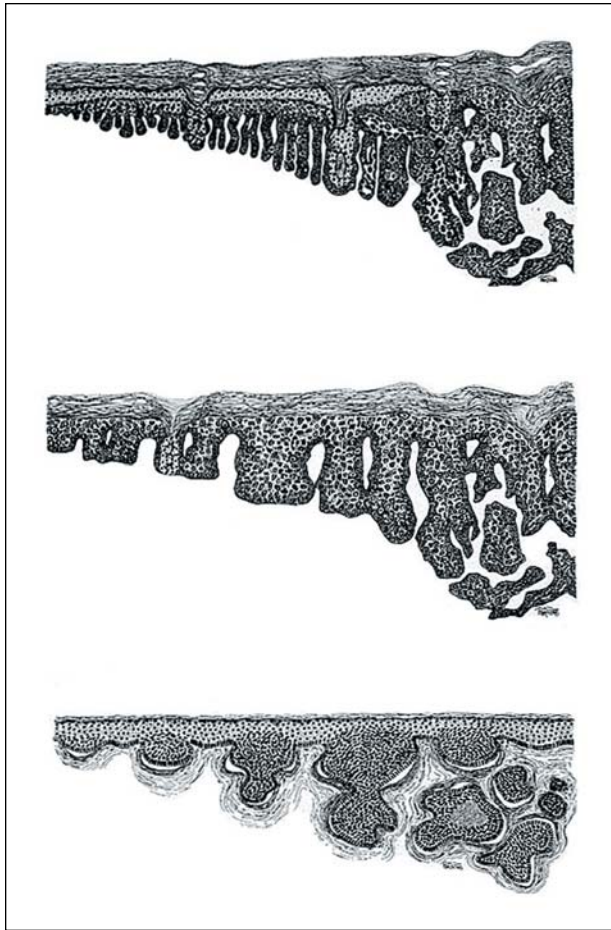


Figure 3. Solar keratosis, Bowen disease, and superficial basal cell carcinoma, pictured from top to bottom, are superficial carcinomas whose neoplastic cells may, in the manner of a phalanx, descend progressively further into the skin and tissues beneath it.

not cancer. I believe that a lesion is the same thing throughout its existence and that it cannot properly change its name merely because it changes its size. The acorn is *Quercus* just as is the oak tree; the fertilized ovum in the human uterus is *Homo sapiens*; the 1-mm circular, scaling intra-epidermal macule is cancer as appropriately as the stinking ulcer that destroys the malar bone and orbit.

The scaling macule, the ‘precancerosis’ and the cancer evolved from it, are the same thing. The lesion was, is and will continue to be the same cancer, and its degree of malignancy has not altered in the process.

. . . In summary, ‘precancerosis’ is a hypothesis, not a lesion. ‘Precancerosis’ is anything the user of the term may define it to be. . . . ‘Precancerosis’ is anything in which neoplastic

alteration may take place; any mammal as a whole is a ‘Precancerosis.’

It [precancerosis] is as unscientific a term as ‘rheumatism’ and covers as many different things. It is both useless and confusing for biologic comprehension.

This theory does away completely with the concept of ‘precancerosis;’ there is no such thing. Small lesions are neoplastic or not neoplastic; if neoplastic they may grow slowly and be benign or grow fast and be malignant. They should all be destroyed.”

There is every reason for hope that by 2038, the AAD will catch up with Sutton.

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