Table I. Clinical contexts at time samples were obtained for bacterial cultures

Clinical context	N = 429, n (%)
Patient prescribed antibiotics before sample collection	63 (14.7)
Patient prescribed oral antibiotics at the time of sample collection	225 (52.4)
Incision and drainage performed at the time of sample collection	116 (27.0)
Incision and drainage performed and antibiotics prescribed at the time of sample collection	61 (14.2)
Infection or lesion type	
Abscess, carbuncle, furuncle, cyst	156 (36.4)
Folliculitis, acne, pustule	86 (20.0)
Cellulitis	14 (3.3)
Surgical wound infection	43 (10.0)
Other wound infection (friction ulcer, traumatic ulcer, leg ulcer, penile ulcer, erosions)	50 (11.7)
Impetigo, superinfected dermatitis	39 (9.1)
Hidradenitis suppurativa	9 (2.1)
Bullae and vesicles	20 (4.7)
Paronychia	12 (2.8)

cultured organisms and their sensitivities in only 7.7% of cases. Categorized by clinical context, changes in management occurred in 8.3% of abscess cases, of which 76.9% were concordant with sensitivities. For cultures performed at the same time as incision, drainage, and empiric antibiotic treatment, change in management occurred in only 4.5% of cases. Changes in management occurred in 20.5% of impetigo and superinfected dermatitis cases, 75% of which were concordant with sensitivities. No changes in management occurred among cases of hidradenitis suppurativa, bullae, and vesicles. In 4.2% of bacterial cultures, organisms were resistant to empiric therapy, but there were no subsequent changes in therapy.

Although this study involved a small sample size from a single clinic, it appears as though bacterial culturing infrequently leads to changes in management. It also appears that culture utility might vary depending on clinical context, with cultures of hidradenitis suppurativa, bullae, and vesicles being least likely to result in therapeutic changes. Furthermore, some cultures were ordered after patients had started antibiotics and over half of the bacterial skin cultures grew organisms that are not typical cutaneous pathogens, making these results difficult to interpret and utilize clinically.

This pilot study highlights the need to further investigate bacterial cultures for skin and soft tissue infections to determine the best practices and improve patient care while decreasing unnecessary costs. In an effort to cut wasteful tests in medicine, the optimal use of bacterial cultures in outpatient dermatology settings should be further studied.

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Sunscreen may prevent the development of basal cell carcinoma in individuals with basal cell carcinoma nevus syndrome: A retrospective survey study

To the Editor: A recent survey study of individuals with basal cell carcinoma nevus syndrome (BCCNS) suggested that sun exposure is responsible for basal cell carcinoma (BCC) development in individuals with BCCNS.[1] On the basis of this demonstration that individuals with and without BCCNS share a trigger for BCC development, BCCNS provides an excellent model for studying BCC prevention strategies, as individuals with BCCNS have an elevated risk of BCC development (lifetime average of 257 BCCs), which facilitates powering studies.[1] The proper use of sunscreen is frequently recommended as a method to prevent BCCs despite the failure of several prospective studies to confirm the efficacy of this strategy.[2] A 33-question survey was utilized to assess

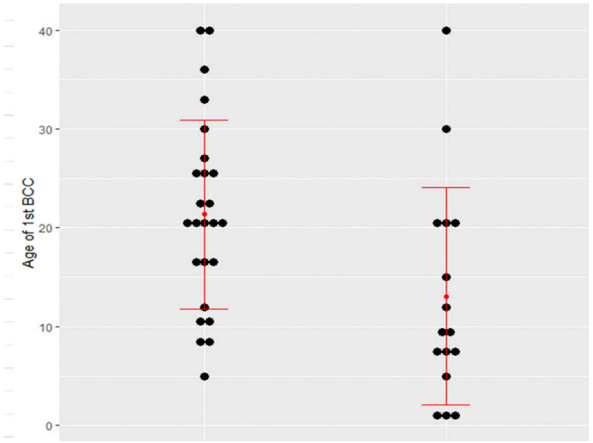

Fig 1. Comparison of childhood sunscreen use with age of first basal cell carcinoma (BCC).

sunscreen use and the development of BCCs in individuals with BCCNS.

Participants were contacted through the BCCNS Alliance List Serv. There were 47 respondents. The exact number of surveys distributed to individuals meeting study inclusion criteria is unknown. Data were analyzed by using linear regression analysis of log (total BCCs + 1) versus age, with data grouped binomially on the basis of predetermined predictors of interest (eg, childhood sunscreen use). The study revealed several key findings: (1) 76.6% of individuals with BCCNS currently apply sunscreen at least half of the time during which they are outdoors; (2) children with BCCNS who apply sunscreen at least half of the time during which they are outdoors develop BCCs at a younger age than those who do not (Fig 1) (this finding may be due to the fact that children who develop BCCs at a young age subsequently apply sunscreen consistently in an attempt to prevent additional BCCs); and (3) neither childhood sunscreen use nor current sunscreen use was associated with a statistically significant decrease in the number of BCCs developed by individuals with BCCNS, although both of these outcomes trended toward statistical significance and linear regression analysis of the data for both outcomes demonstrated clear trends supporting the efficacy of sunscreen in BCC prevention (Fig 2). This failure to achieve statistical significance may suggest that (1) sunscreen does not in fact prevent BCC development in this population, (2) stronger adherence to sunscreen use regimens (eg, >90% sunscreen use) may be required to confer a preventative benefit, (3) our study was underpowered owing to small sample size, and/or (4) individuals with BCCNS who use sunscreen

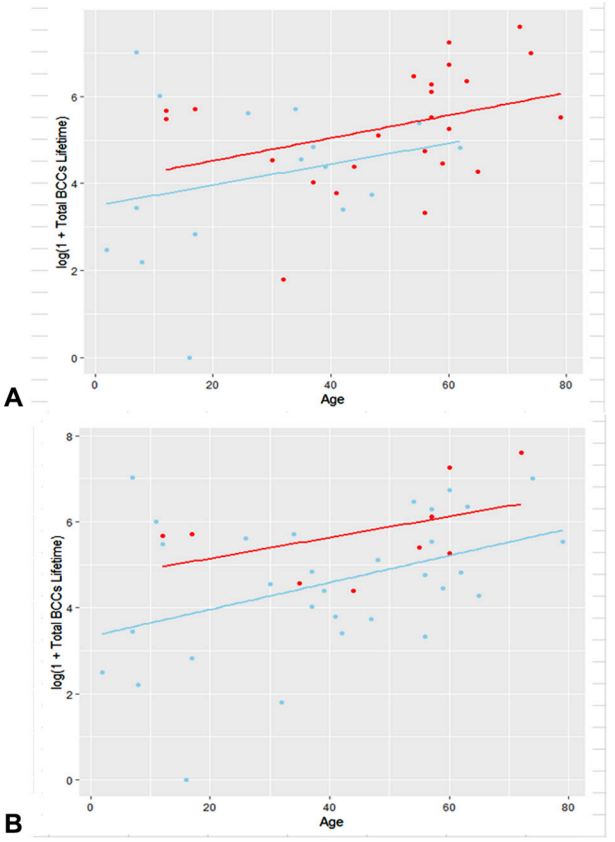

Fig 2. Effect of childhood (**A**) and current (**B**) sunscreen use on the development of basal cell carcinomas (BCCs) in individuals with BCC nevus syndrome. Red lines indicate sunscreen use less than 50% of the time; blue lines indicate sunscreen use at least 50% of the time.

frequently are more phenotypically severe and therefore not comparable to individuals who do not use sunscreen.

This study was meant to highlight the potential for BCCNS to serve as an appropriate model for studying prevention of BCC development and also to highlight the fact that BCC prevention strategies that are successfully utilized in individuals without BCCNS may be useful for individuals with BCCNS. Additionally, our study trended toward supporting existing evidence that sunscreen is effective for the prevention of BCC, although the robustness of these findings was limited by the study's small sample size and its retrospective nature. Limitations of this study include small sample size, use of self-reported data, and high variability of rate of BCC development of individuals with BCCNS.

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Counting keratinocyte carcinomas as a step to preventing them

To the Editor: Keratinocyte carcinomas (KCs) (ie, basal cell carcinoma and squamous cell carcinoma) account for three-fourths of cancers in the United States.[1] Twenty-five years ago, KC was known as nonmelanoma skin cancer. During their training, many of the physicians practicing at the time had learned to call basal cell carcinoma "epithelioma," suggesting that it was a benign entity. KC frequency was poorly documented, it was not included in most cancer registries, and its morbidity was largely unmeasured. Consequently, the impact of KCs was underestimated.

Naming and enumerating these cancers facilitates their appreciation and ultimately reduces their numbers and public health impact. For KCs to truly have an impact on public policy, they need to be counted. Quantification is a stimulus to action. The goal of our report in the *Journal of the American Academy of Dermatology* was to sharpen the focus on skin cancers.[2]

Progress has occurred over the past quarter-century. KCs were formerly known (and are still often known) as nonmelanoma skin cancers, which is an ambiguous and otherwise (for multiple reasons) suboptimal term.[1] At that time, an estimated 480,000 Americans developed KC annually, an estimate that was then 16 years old and doubled with the publication of our report.[2]

Since then, estimation of KC incidence has improved, and the incidence has been increasing—a trend that has been amplified by increasing iatrogenic immunosuppression.[3] The improvements in the estimation of KC are due to better access to and utilization of big databases.

It is now time to cut KC incidence and burden. Many efforts have been focused on prevention with sun protection, dating back to Slip! Slop! Slap! (slip on a shirt, slop on the sunscreen, slap on a hat), which was publicized initially by the Anti-Cancer Council of Victoria, Australia, nearly 40 years ago.[4] Since then, we have learned much about the difficulty of changing the sun protection habits of the population, the long delay between changes in those habits, and the limited progress in moderation of the skin cancer incidence rate despite extensive population-wide efforts. Additional measures are needed.

Particularly promising are efforts to reduce KC risk despite existing skin damage from prior ultraviolet radiation exposure. The sole topical agent demonstrated to be efficacious in preventing KC for a prolonged period after its use is 5-fluorouracil. It lowers risk of squamous cell carcinoma by three-fourths during the year after application of a standard 2- to 4-week course to the face and ears; it is cost-effective, and indeed, cost-saving.[5] The usefulness of other topical agents for chemoprevention of KC is unproved. Skin cancer risk may not be substantially reduced by actinic keratosis destruction alone. We need randomized trials to guide practice.

Oral nicotinamide has modest efficacy for reducing KC risk, but its limitations include lack of effect after ingestion ends and the potential for increased risk of more aggressive KCs. Oral retinoids decrease KC risk during use but not afterward, and they have potential concerning adverse effects. Neither of these treatments are officially indicated for skin cancer chemoprevention.

The way forward is to produce convincing evidence of a practical, safe strategy with the ultimate goal of reducing incidence (and associated morbidity and costs) in the general population.

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