Psoriasis is a systemic, chronic, autoimmune disease that affects approximately three percent of the US adult population. Plaque psoriasis, also referred to as psoriasis vulgaris, is the most common type of psoriasis and affects 85 to 90 percent of individuals with the disease. Even though psoriasis is an immune-mediated condition that affects the skin, it is associated with a variety of major medical comorbidities (e.g., cardiovascular, pulmonary, and gastrointestinal disorders; diabetes; and renal disease), and the burden of these comorbid diseases increases with the severity of psoriasis.

The red, itchy, scaly, and painful skin lesions that are characteristic of psoriasis can negatively impact a patient’s quality of life. Evidence indicates that total skin clearance, which is becoming increasingly achievable with newer, more effective therapies, results in reduced symptom severity and improved health-related quality of life. Therefore, the goal of psoriasis treatment is to achieve the highest possible level of clearance. Maintaining clearance, however, is also of paramount importance given the frustration and psychological stress that patients experience anticipating recurrence. We, as healthcare providers (HCPs), have a responsibility not only to address the patient’s desire for clear skin but to also partner with the patient to maintain that level of clearance.

Evidence indicates that psoriasis is underdiagnosed and undertreated, and overall patient satisfaction with treatment is low. One factor potentially impacting patient satisfaction is unrealistic treatment expectations. HCPs and their staff should work with patients to establish realistic expectations in terms of the time to visual symptom improvement, which is not instantaneous, and chronology of symptom improvement (e.g., itching may improve more quickly than redness). While treatment success may be defined using a threshold (e.g., ≥75 percent improvement in Psoriasis Area and Severity Index [PASI 75] score) in clinical trials, success should be defined in a more patient-centric manner in clinical practice, being derived from the burden of the disease and how it is perceived by the patient. Severity of disease is generally defined by the extent of body surface area (BSA) involved (mild, <5 percent; moderate, ≥5 percent to <10 percent; and severe, ≥10 percent), as well as location of involvement.

While psoriasis on the hands, feet, face, or genital regions may cover less BSA, it may substantially impact self-regard and significantly interfere with activities of daily life. Unfortunately, this nuance is not captured using the current clinical scoring systems. Additionally, patients with moderate-to-severe psoriasis are more likely to experience greater frequency of flare-ups, and more intense symptomology, such as skin pain, itching, and stinging than patients with mild disease.

Herein, we provide a brief overview of available therapeutic options for psoriasis, summarize the immune processes underlying this disease, and discuss efficacy and safety considerations for secukinumab, an interleukin (IL)-17A antagonist approved to treat moderate-to-severe plaque psoriasis.

UNDERSTANDING IMMUNE SYSTEM ABERRATIONS IN PSORIASIS

Multiple theories exist regarding triggers for psoriasis (e.g., infectious episode, traumatic insult, stressful life event); however, no obvious trigger can be identified in many patients. Additionally, it is not possible for HCPs to tell a patient exactly why he or she develops psoriasis. Regardless, once triggered, T cells are recruited to the dermis and epidermis, resulting in the characteristic psoriatic plaques. Though the pathogenesis of psoriasis is not completely understood, genetic and immune-mediated components have been identified, which establish the involvement of inflammatory pathways that serve as targets for therapeutic agents. Both the innate and adaptive immune systems are involved in the uncontrolled proliferation of keratinocytes, activation of dendritic cells, release of pro-inflammatory cytokines, and recruitment of T cells to the skin (see Table 1 for common...
immunology terms). Several inflammatory mediators, including tumor necrosis factor alpha (TNF-α), IL-12, IL-23, and IL-17, have been implicated in this complex, multifactorial process (Figure 1). Upon activation by TNF-α, dendritic cells will secrete cytokines including IL-12 and IL-23 that act upon subsets of T helper cells to further upregulate the inflammatory cascade observed in psoriasis. IL-12 induces T helper 1 (Th1) cells to produce TNF-α and interferon-γ. IL-23 induces Th17 cells to produce IL-17, IL-22, and TNF-α. These cytokines then promote formation and exacerbation of psoriatic plaques. For instance, IL-17A stimulates keratinocytes and the resultant secretion of chemokines, as well as proinflammatory mediators, ultimately leading to further recruitment of other types of inflammatory cells, such as neutrophils and additional Th17 cells. This process results in further positive feedback to the inflammatory cascade and maintenance or development of psoriatic plaques. Through selective and specific inhibition of IL-17A, secukinumab (a human monoclonal antibody) restores the normal function of keratinocytes, which decreases the production of neutrophil chemoattractants from keratinocytes. Subsequently, there is clearance of cutaneous neutrophils and resolution of psoriatic plaques.

**OVERVIEW OF AVAILABLE OPTIONS FOR TREATING PSORIASIS**

Decision-making regarding the appropriate treatment options for plaque psoriasis is generally guided by disease severity. For mild disease (<5 percent BSA), treatment primarily...
consists of topical therapy with corticosteroids, retinoids (e.g., tazarotene), vitamin D analogs and targeted phototherapy. However, topical monotherapy is not appropriate for patients with moderate-to-severe disease (≥5 percent BSA) or those with psoriasis involving the genitals, hands, feet, and/or face, which requires treatment with phototherapy or systemic agents. Systemic options include various nonbiologics such as methotrexate, cyclosporine, second-generation retinoids (acitretin), and the small molecule phosphodiesterase 4 inhibitor apremilast; however, side effects and contraindications must be considered. Several biologics also are available, including TNF-α inhibitors (adalimumab, etanercept, and infliximab), IL-12/IL-23 inhibitors (ustekinumab), and IL-17A inhibitors (ixekizumab and secukinumab). Biologics are a welcome addition to the growing armamentarium of options for treating psoriasis given their potential for less gastrointestinal distress and less organ toxicity (renal, hepatic, and bone marrow), relative to nonbiologics, and lack of teratogenicity.

### TABLE 2. PIVOTAL, RANDOMIZED, CONTROLLED, PHASE 3 TRIALS OF SECUKINUMAB IN PSORIASIS

<table>
<thead>
<tr>
<th>Trial</th>
<th>Treatment</th>
<th>Efficacy at Week 12</th>
<th>Maintenance of response from Week 12 to Week 52</th>
</tr>
</thead>
</table>
| ERASURE<sup>26</sup> N=738 | Subcutaneous secukinumab (300 mg or 150 mg) or placebo at Baseline, Weeks 1, 2, and 3, and then every 4 weeks from Week 4 to 48 | PASI 75  
• Secukinumab 300 mg: 81.6%<sup>a</sup>  
• Secukinumab 150 mg: 71.6%<sup>a</sup>  
• Placebo: 4.5%  
PASI 90  
• Secukinumab 300 mg: 59.2%<sup>a</sup>  
• Secukinumab 150 mg: 39.1%<sup>a</sup>  
• Placebo: 1.2%  
PASI 100  
• Secukinumab 300 mg: 28.6%<sup>a</sup>  
• Secukinumab 150 mg: 12.8%<sup>a</sup>  
• Placebo: 0.8%  | Maintenance of PASI 75:  
• Secukinumab 300 mg: 80.5%  
• Secukinumab 150 mg: 72.4%  
• Placebo: NE  
Maintenance of IGA mod 2011 responses of 0 (clear) or 1 (almost clear):  
• Secukinumab 300 mg: 74.4%  
• Secukinumab 150 mg: 59.2%  
• Placebo: NE  |
| FIXTURE<sup>26</sup> N=1306 | Subcutaneous secukinumab (300 mg or 150 mg) or placebo at Baseline, Weeks 1, 2, and 3, and then every 4 weeks from Week 4 to 48. Etanercept (50 mg) twice weekly for 12 weeks, then once weekly | PASI 75  
• Secukinumab 300 mg: 77.1%<sup>a,b</sup>  
• Secukinumab 150 mg: 67.0%<sup>a,b</sup>  
• Etanercept: 44.0%  
• Placebo: 4.9%  
PASI 90  
• Secukinumab 300 mg: 54.2%<sup>a,b</sup>  
• Secukinumab 150 mg: 41.9%<sup>a,b</sup>  
• Etanercept: 20.7%  
• Placebo: 1.5%  
PASI 100  
• Secukinumab 300 mg: 24.1%<sup>b,c</sup>  
• Secukinumab 150 mg: 14.4%<sup>b,c</sup>  
• Etanercept: 4.3%  
• Placebo: 0%  | Maintenance of PASI 75:  
• Secukinumab 300 mg: 84.3%<sup>b</sup>  
• Secukinumab 150 mg: 82.2%<sup>d</sup>  
• Etanercept: 72.5%  
• Placebo: NE  
Maintenance of IGA mod 2011 responses of 0 (clear) or 1 (almost clear):  
• Secukinumab 300 mg: 79.7%<sup>b</sup>  
• Secukinumab 150 mg: 67.7%<sup>e</sup>  
• Etanercept: 56.8%  
• Placebo: NE  |

<sup>a</sup> P<0.001 versus placebo. <sup>b</sup> P<0.001 versus etanercept. <sup>c</sup> No comparison with placebo was performed because there were no patients with a response in the placebo group. <sup>d</sup> P=0.009 versus etanercept. <sup>e</sup> P=0.002 versus etanercept.
**SECUKINUMAB: EFFICACY AND SAFETY**

Understanding the role of IL-17 in the pathogenesis of psoriasis led to the development of novel, targeted treatment options. Secukinumab is a recombinant, high-affinity, fully human immunoglobulin (Ig)G1 monoclonal antibody that selectively binds IL-17A and impedes interaction with the IL-17 receptor, thereby inhibiting release of proinflammatory cytokines and chemokines. In 2015, secukinumab (Cosentyx; Novartis Pharmaceuticals Corporation) became the first FDA-approved agent for the treatment of plaque psoriasis targeting IL-17A. In addition to the treatment of moderate-to-severe plaque psoriasis in adults who are candidates for systemic therapy or phototherapy, secukinumab is also approved for use in adults with active psoriatic arthritis or active ankylosing spondylitis. For plaque psoriasis, secukinumab is self-administered as a subcutaneous injection at Weeks 0, 1, 2, 3, and 4 (5-week loading dosage) and then once every 4 weeks. The recommended dosage of secukinumab is 300mg, and it is available in a prefilled syringe and an autoinjector.

Approval for the psoriasis indication was based on results from two multicenter, randomized, double-blind, placebo-controlled trials. In these trials, secukinumab was evaluated at a dose of 300mg or 150mg. Only the 300mg dose of secukinumab will be discussed here because 300mg is the recommended dosage of secukinumab (Results for both doses are presented in Table 2). Across both trials, the response to secukinumab was rapid with high efficacy observed at early time points and a favorable safety profile. In the two largest phase 3 trials (ERASURE and FIXTURE), significantly more patients achieved PASI 75, PASI 90, and PASI 100 response rates at Week 12 with secukinumab 300mg than with placebo. It is important to note the high levels of almost-complete (PASI 90) or complete (PASI 100) skin clearance with secukinumab 300mg because these rates of response are associated with greater improvement in quality of life than PASI 75 responses. At Week 12, PASI 90 and PASI 100 responses were achieved by 54.2 percent to 59.2 percent and 24.1 percent to 28.6 percent of patients receiving secukinumab 300mg, respectively, and these rates of clearance were well-maintained to Week 52. Maintenance of clear or almost-clear skin from Week 12 to Week 52 was observed in 74.4 percent to 79.7 percent of patients receiving secukinumab 300mg, indicating that many patients experience sustained initial rates of skin clearance. Improvements were also rapid with secukinumab 300mg and the median time to a 50 percent reduction in baseline PASI scores was 3 weeks with secukinumab 300mg. Further, reductions in PASI scores were sustained over 2 years of treatment with secukinumab.

Secukinumab treatment was also superior to the biologic agents etanercept (a TNF-α inhibitor) and ustekinumab (an IL-12/23 inhibitor). In the FIXTURE trial, secukinumab 300mg treatment provided significantly greater efficacy than etanercept at Week 12 with respect to PASI 75, PASI 90, and PASI 100 response rates. In addition, response to treatment was less rapid with etanercept as a 50 percent reduction in baseline PASI score was achieved after seven weeks, 4 weeks longer than with secukinumab 300mg. Similar efficacy findings were reported for the comparison of secukinumab to ustekinumab in the CLEAR trial. Significantly greater responses with secukinumab 300mg in comparison to ustekinumab were observed as early as Week 1 for PASI 75, Week 3 for PASI 90, and Week 4 for PASI 100. The statistically greater efficacy of secukinumab 300 mg was maintained to the last time point reported (Week 16) for each measure. Treatment with secukinumab 300 mg was also accompanied by significantly greater improvement in dermatology-related quality of life than ustekinumab as early as four weeks after initiation of therapy.

Similar efficacy and safety for secukinumab were observed in the smaller phase 3 FEATURE and JUNCTURE trials, which evaluated administration of secukinumab by prefilled syringe and autoinjector, respectively. In these trials, patients reported high levels of usability and satisfaction when secukinumab was administered by self-injection with either device. Additionally, in patients with a PASI 75 response after 12 weeks of secukinumab therapy in the SCULPTURE trial, fixed interval dosing (every 4 weeks) of secukinumab showed benefit over secukinumab retreatment-as-needed (retreatment after loss of 20 percent or more of maximum PASI score improvement versus baseline, plus loss of PASI 75 response) for maintenance of efficacy. From Week 12 to Week 52 in this trial, PASI 75 responses with secukinumab 300mg were maintained in 78.2 percent of patients receiving fixed interval dosing and by 67.7 percent of patients being retreated-as-needed.

The specific and selective targeting of IL-17A by secukinumab allows for downstream targeting in the inflammatory cascade responsible for the development of psoriasis. Thus, unlike other less specifically targeted biologic agents that inhibit upstream elements or components with broad functions such as TNF-α, specific inhibition of IL-17A is not expected have as widespread effects. Specific and selective targeting of IL-17A may allow for a broader spectrum of patients to be considered for secukinumab therapy. This more-targeted approach lowers the risk of side effects, such as susceptibility to tuberculosis. Accordingly, in our opinion, the use of secukinumab may be considered with care in cases of latent tuberculosis positivity, as well as in multiple sclerosis, HIV infection, an immunosuppressed state, and previous history of cancer, in collaboration with necessary specialists.
SAFETY CONSIDERATIONS

Patients with tuberculosis infection or Crohn’s disease should be identified during initial screening, and completion of age-appropriate vaccinations before drug initiation should be considered. Live vaccines should be avoided during treatment, and nonlive vaccines may produce a blunted immune response that is not sufficient to prevent disease. Caution should be used in patients with Crohn’s disease because new exacerbation of existing disease, have been reported in clinical trials of secukinumab. In addition, new onset of inflammatory bowel disease has been reported in patients receiving secukinumab. Also, monitoring for infection-related safety signals (particularly mucosal/superficial candidiasis and staphylococcal infections) is necessary. However, the approved product labeling for secukinumab does not include extensive warnings such as risk of demyelinating diseases, lymphoma, congestive heart failure, and pancytopenia or anaplastic anemia, as are listed in the labels of TNF-α inhibitors (etanercept, adalimumab, and infliximab). Secukinumab-treated patients should be advised about the risks of hypersensitivity reactions, including latex allergies to the natural-rubber–based cap of the delivery devices (pen or syringe). Latex-sensitive patients may consider having a caregiver administer the secukinumab dose to avoid any contact with the cap.

In a pooled analysis of 10 phase II and phase III trials of secukinumab in patients with moderate-to-severe psoriasis, the most common adverse events over 52 weeks with any dose of secukinumab were nasopharyngitis (29.3 cases per 100 patient-years), headache (11.0 cases per 100 patient-years), and upper respiratory tract infection (8.8 cases per 100 patient-years). These were also the most common adverse events observed during the 12-week induction period. Overall rates of adverse events were similar between secukinumab, etanercept, and ustekinumab. Of note, more candidiasis was observed over 52 weeks with secukinumab 300mg (0.43 cases per 100 patient-years) and secukinumab 150mg (0.35 cases per 100 patient-years) than with etanercept (0 cases per 100 patient-years). However, this finding is expected due to the role of IL-17A in host-defense mechanisms against Candida species. Importantly, candida infections in patients receiving secukinumab did not require discontinuation of treatment. Further, these infections were generally mild, nonserious, localized, and responsive to standard treatment.

A safety concern inherent to administration of monoclonal antibodies is the potential for development of neutralizing antibodies. Low rates of antisecukinumab antibodies were observed in FIXTURE (0.4 percent) and ERASURE (0.3 percent) trials, and only a single isolated case of neutralizing antibodies was reported. No instances of reduced efficacy in the setting of antibody development, neutralizing or otherwise, has been reported.

PRACTICAL ASPECTS OF CARE DURING SECUKINUMAB THERAPY

After initiating secukinumab therapy, patients should be evaluated at each appointment for psoriatic lesions and asked specific questions such as, “Have you noticed any itching?” (a potential sign of recurrence; Table 3). Visits also should include a skin evaluation for candidiasis. Patients with psoriasis should be screened for psoriasis-associated comorbidities, including ulcerative colitis, Crohn’s disease, and other gastrointestinal diseases, before starting any treatment. Surveillance for, and management of, these and other comorbidities, including those associated with systemic inflammation (e.g., hypertriglyceridemia, hypertension, diabetes, obesity, or metabolic syndrome) and joint involvement (psoriatic arthritis or other rheumatic diseases), should continue throughout treatment and patients should be appropriately referred if necessary, for example, to an endocrinologist for diabetes, a cardiologist for hypertension and hypertriglyceridemia, or a rheumatologist for joint disease. A clinical consensus report on psoriasis comorbidities, issued by the National Psoriasis Foundation, provides additional guidance on screening and managing comorbidities and recommendations from this report are summarized in Table 4.

Psoriatic arthritis affects 30 percent of patients with psoriasis and these conditions share common disease mechanisms, including involvement of IL-17. As such, therapies for psoriasis, such as secukinumab, also have efficacy for treating psoriatic arthritis. Special considerations for the care of patients with both skin and joint involvement are needed. For example, if a patient has severe skin disease and joint disease, treatment should be initiated for the skin disease, with referral to a rheumatologist for evaluation of the joint disease. If a patient presents with limited psoriasis but more-severe joint disease, the patient should be promptly referred to a rheumatologist for evaluation. Under these conditions, it is possible that another immune-mediated disorder, such as ankylosing
<table>
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<tr>
<th>Comorbidity</th>
<th>Recommendation</th>
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<tbody>
<tr>
<td>General cardiovascular risk</td>
<td>Screening for patients without a known risk factor*&lt;br&gt;Risk factor screening as early as age 20 years&lt;br&gt;Risk factor screening at age 40 years for:&lt;br&gt;• Blood pressure: at least every 2 years; target &lt;120/80 mmHg&lt;br&gt;• BMI: at least every 2 years; target &lt;25 kg/m²&lt;br&gt;• Waist circumference: at least every 2 years; target &lt;35 inches for women and &lt;40 inches for men&lt;br&gt;• Pulse: at least every 2 years&lt;br&gt;• Fasting serum lipoprotein or total and HDL cholesterol level: at least every 5 years, or every 2 years if risk factors† are present:&lt;br&gt;  ◦ Total cholesterol level &lt;200 mg/dL&lt;br&gt;  ◦ HDL level ≥50 mg/dL&lt;br&gt;  ◦ LDL level&lt;br&gt;    – Optimal &lt;100 mg/dL&lt;br&gt;    – Near/above optimal 100–129 mg/dL&lt;br&gt;    – Bordeline high 130–159 mg/dL&lt;br&gt;    – High 160–189 mg/dL&lt;br&gt;    – Very high ≥190 mg/dL&lt;br&gt;• Fasting blood glucose level: at least every 5 years, or every 2 years if risk factors† are present.&lt;br&gt;  Target &lt;100 mg/dL&lt;br&gt;Management&lt;br&gt;Smoking cessation, moderate alcohol intake, and exercise 3 times a week for 30 minutes or more</td>
</tr>
<tr>
<td>Metabolic syndrome</td>
<td>Screening&lt;br&gt;Metabolic syndrome can be identified by 3 or more of the following:&lt;br&gt;• Increased waist circumference:&lt;br&gt;  ◦ Men ≥40 inches&lt;br&gt;  ◦ Women ≥35 inches&lt;br&gt;• Elevated triglyceride level:&lt;br&gt;  ≥150 mg/dL&lt;br&gt;• Reduced HDL cholesterol level:&lt;br&gt;  ◦ Men &lt;40 mg/dL&lt;br&gt;  ◦ Women &lt;50 mg/dL&lt;br&gt;• Elevated blood pressure:&lt;br&gt;  ≥130/85 mmHg&lt;br&gt;• Elevated fasting glucose level:&lt;br&gt;  ≥100 mg/dL&lt;br&gt;Management&lt;br&gt;Weight loss to achieve a BMI &lt;25 kg/m², increased physical activity with 30 minutes of moderate-intensity activity most days, and healthy eating habits</td>
</tr>
<tr>
<td>Obesity</td>
<td>Screening&lt;br&gt;BMI ≥30 is considered obese and BMI 25–29.9 is considered overweight&lt;br&gt;Management&lt;br&gt;Alter lifestyle to achieve an ideal BMI and exercise 3 times a week for 30 minutes or more</td>
</tr>
<tr>
<td>Depression</td>
<td>Screening&lt;br&gt;Ask patients about depressed mood and anhedonia&lt;br&gt;Management&lt;br&gt;Treatment of psoriasis has been shown to improve depression&lt;br&gt;Referral to a psychiatrist or psychologist</td>
</tr>
</tbody>
</table>
spondylitis or rheumatoid arthritis, is involved.

Patients with psoriasis have elevated cardiovascular disease risk, and disease severity is associated with additional cardiovascular risk when traditional risk factors are controlled for, suggesting that inflammation from psoriatic plaques can act on remote sites. Interestingly, data from clinical trials in patients with psoriatic arthritis or rheumatoid arthritis indicate that secukinumab treatment leads to a reduction in C-reactive protein levels, an important inflammatory marker for cardiovascular risk. Further, IL-17A is involved in the underlying pathogenesis of obesity and vascular dysfunction. These observations raise the question whether inhibition of IL-17A may provide benefits to reducing risks from cardiovascular and metabolic disorders but additional studies are needed to fully elucidate the impact of IL-17A inhibition on these conditions.

**CONCLUSIONS**

Psoriasis is a chronic immune-mediated disease affecting the skin but also associated with a number of comorbidities of various organ systems, as well as with substantial risk for quality of life impairment. Fortunately, for patients and HCPs, various options, including topical and systemic approaches, are available for tailoring treatment. Biologic options target various underlying aberrations of the immune system that collectively contribute to the pathogenesis of psoriasis. Secukinumab, which is directed against the pro-inflammatory and keratinocyte-specific cytokine IL-17A, is the first of a new class of biologic agents to become available in the US. Therapy with secukinumab produces high rates of response that are both rapid in onset and durable for clearance of psoriatic lesions. The importance of maintaining responses over time cannot be overstated, given the frustration that is
enthused and dedicated in pursuing new research, new drugs, and alternative therapies in order to enhance the quality of life for those afflicted with psoriatic disease.

Scott Freeman earned his Bachelor of Science and Master in Clinical Medical Science degrees from the University of Florida (Gainesville) in 1996 and Barry University (Miami Shores, FL) in 1999, respectively. As a nationally certified physician assistant, Scott has over ten years of experience in the clinical field, and he has served as a supervising physician assistant at the Spencer Dermatology and Skin Surgery Center in St. Petersburg, FL since 2007. Though already having possessed a broad knowledge base of dermatology, with a keen interest in the treatment and management of acne and acne scarring, Scott continues to further his knowledge and understanding of skin and skin pathology under the guidance of his colleagues and through his clinical work and experiences. He maintains active membership with state and national organizations related to the profession of physician assistant as well as the field of dermatology.

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Joe Gorelick earned his Bachelor of Science degree in Physiology from the University of California at Davis and his Master of Science degree from Vanderbilt University in Nashville, TN. He has over 15 years experience as a nurse practitioner in all aspects of medical, surgical, and cosmetic dermatology, and is an adjunct faculty member of the Nurse Practitioner Program at Holy Names College in Oakland, CA, where he lectures on topics including surgical techniques and dermatological conditions. Joe is the Chairman and Founder of the Dermatology Essential Resource Meeting (DERM) CME meeting for nurse practitioners and physician assistants.

Wendy Cantrell earned her Bachelor of Science and Master of Science degrees in Nursing from the University of Alabama at Birmingham in 1996 and 2000, and in 2011 she earned the title Doctor of Nursing Practice. Currently, she is an assistant professor of Dermatology in Birmingham, AL, and she is the manager of the clinical research division for the department. A prolific scholar, Dr. Cantrell has published many peer-reviewed articles on a number of dermatological issues during her 15 years of practice. With a family history of psoriasis, Dr. Cantrell has expressed her enthusiasm and dedication in pursuing new research, new drugs, and alternative therapies in order to enhance the quality of life for those afflicted with psoriatic disease.

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