Lasers and Light Devices for Psoriasis, Part 1: Excimer Laser and Phototherapy

Marilyn Zabielinski, BS; Katherine Ferris, BA; Yasser Al-Qubaisy, MD; Michael McLeod, MS; Sonal Choudhary, MD; Keyvan Nouri, MD

Psoriasis is a common inflammatory skin condition with high morbidity. Unfortunately, the etiology of the disease currently is unknown, although genetic and environmental factors appear to play important roles in its pathogenesis. Because lasers are used extensively in dermatology to treat various conditions with proven efficacy and safety, they have been investigated as alternatives for treating psoriasis due to side effects that can occur with long-term use of potent topical corticosteroids. Lasers also have the potential to target specific plaques. Modalities that have been investigated for treatment of various forms of psoriasis include the 308-nm excimer laser, psoralen plus UVA (PUVA), narrowband UVB (NB-UVB), the pulsed dye laser, the 1064-nm Nd:YAG laser, the CO_2 laser, and photodynamic therapy. In part 1 of this series, we review the etiology and clinical features of psoriasis and evaluate the efficacy and safety of the 308-nm excimer laser, PUVA, and NB-UVB in the treatment of this common disease.

Cosmet Dermatol. 2012;25:34-41.

Psoriasis is an inflammatory skin disease characterized by erythematous, sharply demarcated, scaly plaques of varying shapes and sizes. Psoriasis is considered a T-cell–mediated autoimmune disease resulting from persistent stimulation of T cells by immunogens of epidermal origin. Genetic factors also have been shown to contribute to the pathogenesis of psoriasis.[1] The psoriasis area and severity index (PASI) is commonly used to assess overall severity and coverage of psoriasis in affected patients.[2] The PASI is used to evaluate the degree of erythema, thickness, and scaling of psoriatic plaques and estimate the extent of involvement of these features in 4 anatomical areas: the head, trunk, and upper and lower extremities. A patient's PASI can range from 0 to 72, is more quantitative than descriptive, and relies on estimates of the involved body surface area.[2] As such, a patient with a PASI score of 72 would have most of his/her body covered with severe, erythematous plaques, while a patient with a PASI score of 0 has no visible evidence of psoriasis. Common histologic features observed in psoriasis patients include epidermal hyperplasia,

From the Department of Dermatology and Cutaneous Surgery, University of Miami, Miller School of Medicine, Florida.
The authors report no conflicts of interest in relation to this article.
This article is the first of a 2-part series. The second part on additional treatment options will appear next month.
Correspondence: Sonal Choudhary, MD, Department of Dermatology and Cutaneous Surgery, University of Miami, Miller School of Medicine, 1475 NW 12th Ave, Miami, FL 33136 (drsonalchoudhary@gmail.com).

dilated and prominent blood vessels in the dermis, and an inflammatory infiltrate of leukocytes that are primarily located in the dermis.[3]

Psoriasis has no validated diagnostic criteria; therefore, estimates of the prevalence of psoriasis in various parts of the world range from 0.6% to 4.8%.[4] Two population-based studies of adults in the United States have found the prevalence among Americans to be approximately 2.2%[5] to 2.6%.[1] More recently, a cross-sectional study using data from the National Health and Nutrition Examination Survey 2003-2004 showed the prevalence of diagnosed psoriasis in adults aged 20 to 59 years to be approximately 3.15%.[3]

Prevalence rates also vary greatly among patients from different ethnic backgrounds.[3] For instance, psoriasis is more common among white individuals and affects only approximately 0.3% of the general population of China.[6] Interestingly, latitude also appears to affect prevalence, possibly because of the beneficial effects of sunlight on psoriatic lesions.[7] The mean age of onset for psoriasis vulgaris has been estimated to be approximately 33 years, with 75% of cases occurring before the age of 46 years.[8] The onset of psoriasis has been observed to be bimodal, with peaks at 16 to 22 years of age and 57 to 60 years of age.[9] The age of onset seems to be slightly younger in women than men, but psoriasis tends to have approximately similar prevalence in both genders.[10] There are different subtypes of psoriasis and patients may exhibit more than 1 type. The most common type, plaque psoriasis, affects 80% to 90% of patients with psoriasis. On the other hand, guttate psoriasis occurs in less than 2% of patients with psoriasis.[11]

ETIOLOGY

Genetic, immunologic, and environmental factors are believed to play a role in triggering the onset of psoriasis. Nine chromosomal loci (*PSOR1–PSOR9*) have a linkage to psoriasis.[12] *PSOR1* has been shown to account for at least 35% to 50% of psoriasis heritability, which is located on the major histocompatibility complex on chromosome arm 6p.[13] Recently, a deletion of the 32.2-kb region coding for *late cornified envelope 3B* and *3C* on 1q21 was found to be more common in patients with psoriasis (68% [1926/2852]) than in control patients (59% [1648/2812]) and has been shown to be a risk factor for the development of psoriasis.[14] Other genes with variants that appear to be associated with psoriasis include *IL-23R, IL-12B, cyclin-dependent kinase 5 regulatory subunit–associated protein 1–like 1*, and *zinc finger protein 313* (also called *ring finger protein 114*).[14-17]

There also is strong evidence that the innate and adaptive immune systems play a role in initiating as well as maintaining psoriatic plaques. The majority of the cellular infiltrate consists of $CD4^+$ and $CD8^+$ T cells, which are known to precede the epidermal keratinocyte proliferation associated with psoriasis. Natural killer cells, myeloid dermal dendritic cells, and neutrophils also are involved in the inflammatory reaction.[18-20] Some adhesion molecules, such as intercellular adhesion molecule-1 on epidermal keratinocytes and E-selectin on dermal capillaries, are highly expressed in the affected areas and promote leukocyte adherence.[21] Although psoriasis is believed to be an autoimmune disease, no known autoantigen has been identified.[6]

Other factors associated with psoriasis include obesity, smoking, alcohol consumption, a prior episode of an infectious disease or upper respiratory tract infection, and prior infection with *Streptococcus pyogenes*. Certain drugs (ie, antibiotics, beta-blockers, lithium, and antimalarial drugs) also are associated with psoriatic exacerbations. Diabetes mellitus, cardiovascular disease, and an increased history of traumatic experiences in childhood or adulthood (eg, emotional abuse, neglect) also may be risk factors.[22-27]

SKIN AND NAIL FINDINGS

Psoriatic lesions typically demonstrate well-demarcated, raised, red plaques with overlying, silvery white scales. Lesions may begin as small papules that coalesce into larger plaques covering a substantial amount of the body surface area. Common sites include the elbows, knees, scalp, nails, and intertriginous regions.[28] Attempts to slough the white scales can elicit bleeding, a phenomenon known as the Auspitz sign.[29] Lesions also may be accompanied by pruritus, pain, superimposed infection, and arthritis. They are more likely to present symmetrically and on extensor surfaces, with sparing of the palms, plantar surfaces, and face, though localized palmoplantar variants do exist.

Nail involvement tends to be underappreciated but is estimated to affect 80% of psoriatic patients at some point in their lives.[30] Nail lesions may manifest as scattered pits or indentations and often herald the presence of concomitant arthritis. Other nail findings include onycholysis and subungual brown macules.[28]

Histologically, early psoriatic lesions typically demonstrate edema and mononuclear cell infiltration of the upper dermis, which arrive via dilated venules. The epidermis proliferates, demonstrating parakeratosis and elongation of local capillaries, while the dermis becomes flooded by other immune cells, including T cells and dendritic cells. Mature lesions are characterized by elongated rete pegs, loss of the epidermal granular layer, and transition from the normal basket weave appearance of the stratum corneum to a compact appearance.

The histopathology of nail findings depends on the site of involvement. Nail pitting reflects matrix disease and demonstrates parakeratotic islands of variable size, which may progress to thick but friable plates.[31] Onycholysis demonstrates spongiform pustulation. Microvascular changes as well as extravasation from capillaries and the trapping of neutrophils under the nail bed also may be apparent in the clinical setting of oil spotting and splinter hemorrhages. Finally, more proximal nail involvement often appears histologically similar to findings of skin sites.

MODALITIES USED FOR MANAGING PSORIASIS

The therapeutic options that are available for the management of psoriasis include topical agents (eg, calcipotriene, steroids, anthralin, tazarotene, tar); systemic agents, such as methotrexate, acitretin, and cyclosporine; anti–T-cell agents (eg, efalizumab, alefacept); anti–tumor necrosis factor agents (eg, infliximab, adalimumab, etanercept)[13,32]; and anti-interleukin agents (eg, ustekinumab).

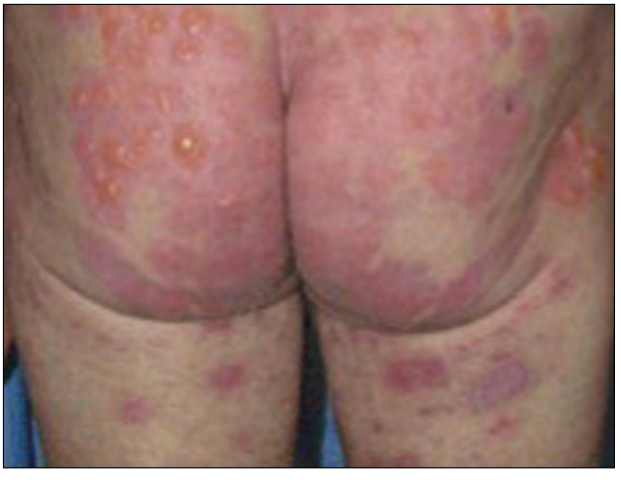
Lasers have been investigated as an alternative for treating psoriasis due to the numerous side effects that can occur with long-term use of potent topical corticosteroids. Most notably, corticosteroids can induce cutaneous atrophy, striae, and telangiectasia. Rare side effects include hypothalamic-pituitary-adrenal axis suppression, hepatotoxicity, and nephrotoxicity.[32] In addition, the data for the long-term safety of biologics used for the treatment of psoriasis, such as etanercept, adalimumab, infliximab, ustekinumab, efalizumab, and alefacept, are relatively limited.[33] Lasers are currently extensively used for various dermatologic conditions with proven efficacy and safety. They have the potential to target specific plaques in the treatment of psoriasis. Specifically, UV radiation with wavelengths in the range of 311 to 313 nm has been shown to be highly effective in the treatment of psoriasis while minimizing the risk for burns and erythema.[34-36] This review will focus on the various lasers and light sources that can be utilized for the treatment of psoriasis. Part 1 discusses the excimer laser as well as narrowband UVB (NB-UVB) and psoralen plus UVA (PUVA) therapy; part 2 will discuss other lasers and light devices such as the pulsed dye laser, Nd:YAG laser, CO_2 laser, and photodynamic therapy.

Excimer Laser (308 nm)

The 308-nm excimer laser, approved by the US Food and Drug Administration in 2000 for the treatment of psoriasis, generates a single wavelength of UVB radiation with a spot size of 2×2 cm and a pulse repetition rate of 200 Hz or less. The pulse width is 30 nanoseconds and the average power delivered is 2 to 3 W. Potential adverse effects include pain, erythema, burning, blistering, and discoloration at the treatment site (Figure). The excimer laser appears to exert its therapeutic effect by direct T-cell cytotoxicity. It induces T-cell apoptosis, inhibits cytokine secretion, and impairs antigen presentation by Langerhans cells.[38]

Overall, it appears that substantial improvement has been observed in psoriatic plaques after treatment with the 308-nm excimer laser (Table). Most studies reported the following promising findings: no pain or minimal pain during the treatment, treatment sessions were completed in several minutes at maximum, and side effects were temporary. However, most of the studies in the literature evaluated a small number of participants, which limits the power of the conclusions. The studies by Hadi et al[40] and Feldman et al[53] included larger study groups (98 and 92 participants, respectively), and both demonstrated clearance of psoriatic plaques with use of the 308-nm excimer laser. More studies should be conducted prospectively with larger sample populations.

In addition to the excimer laser, another option is monochromatic excimer light (MEL), which utilizes a lamp that emits noncoherent monochromatic 308-nm light; unlike PUVA, MEL does not require a photosensitizing agent and requires less frequent treatment sessions than NB-UVB.[55] Another advantage of MEL is that it targets affected sites only, avoiding irradiation of normal skin and thereby preventing unnecessary carcinogenic



Potential adverse effects of treatment with the 308-nm excimer laser include erythema, blisters, and sunburn sensation. Reprinted with permission from Gerber et al.[37]

Studies of the 308-nm Excimer Laser for the Treatment of Psoriasis

Reference (Year)	Study Objective	Study Group	Results
Goldberg et al[39] (2011)	Examine efficacy of 308-nm excimer laser in treatment of palmoplantar psoriasis	10 participants presenting with mild to severe psoriasis involving palms and soles	All participants showed 50%–100% improvement in PASI score with no relapse reported at 3 mo posttreatment
Hadi et al[40] (2010)	Investigate effectiveness of 308-nm excimer laser for treatment of various forms of localized stable psoriasis	Retrospective chart review of 98 patients (41 males, 57 females)	60.2% (59/98) of patients demonstrated significant improvement (≥70% clearance)
Gattu et al[41] (2009)	Review of recent trials on efficacy of 308-nm excimer laser in treating psoriasis	18 trials from comprehensive review of medical literature in PubMed database using search terms *psoriasis* and *308-nm excimer laser*	Useful and effective for psoriasis and may be used as a compliment to topical medications and NB-UVB
Trott et al[42] (2008)	Compare therapeutic response of PUVA plus up to 4 UVB 308-nm radiations vs PUVA monotherapy in patients with moderate to severe plaque-type psoriasis	256 participants with moderate to severe plaque-type psoriasis completed the study; 272 participants were enrolled (149 received PUVA plus excimer combination therapy; 123 received PUVA monotherapy)	No statistically significant difference between PUVA and PUVA plus excimer in clearance rate; on average, participants treated with combination therapy went into remission in half the treatment time
He et al[38] (2007)	Evaluate efficacy and safety of 308-nm excimer laser for treatment of psoriasis vulgaris	40 participants presenting with macular-type (26/40) or chronic plaque-type (14/40) psoriasis vulgaris who completed 15 laser treatments	PASI scores improved by 90.19% for macular-type and 77.34% for chronic plaque-type psoriasis
Morison et al[43] (2006)	Retrospective analysis of 308-nm excimer laser for treatment of scalp psoriasis	35 participants (10 males, 25 females) ranging in age from 8–81 y	49% (17/35) of participants demonstrated clearance of >95%; 45% (16/35) demonstrated clearance of 50%–95% (mean number of treatments, 21)

continued on page 38

(CONTINUED)

Reference (Year)	Study Objective	Study Group	Results
Nisticò et al[44] (2006)	Efficacy analysis of 308-nm excimer laser in treatment of palmoplantar psoriasis	54 participants (29 males, 25 females)	57% (31/54) of participants demonstrated complete remission; 24% (13/54) partial remission; 19% (10/54) moderate improvement
Taibjee et al[45] (2005)	Controlled prospective trial comparing efficacy of 308-nm excimer laser to PDL	15 of 22 participants presenting with localized plaque psoriasis completed study	PASI improvement was significantly greater in excimer group (4.7) vs PDL group (2.7)(P=.003)
Pahlajani et al[46] (2005)	Comparison of efficacy and safety of 308-nm excimer laser for treatment of localized mild to moderate plaque-type psoriasis in children vs adults	4/7 children and 12/18 adults completed the study	Children group had a greater reduction from mean baseline PASI (P=.008)
Köllner et al[47] (2005)	Compare 308-nm excimer laser and 308-nm excimer lamp with 311-nm NB-UVB in treatment of psoriasis	15 participants presenting with 3 plaques	PASI scores did not demonstrate a statistically significant difference
Taylor and Racette[48] (2004)	Pilot study to investigate effectiveness of 308-nm excimer laser in combination with a hair blower for treatment of scalp psoriasis	13 participants (7 males, 6 females) completed the study (mean age, 44 y)	Mean MED, 311 mJ/cm^2; mean treatment duration, <5 min; mean number of treatments, 29; difference in mean modified PASI scores between the control and treated sites, 4.0 (P<.0001); 12/13 participants improved
Gerber et al[37] (2003)	Develop new treatment parameters and determine whether effectiveness of excimer laser could be improved; also to determine whether cumulative dose and adverse effects could be minimized	120 standard protocol participants with chronic plaque psoriasis; 43 participants received an experimental supra-erythemogenic treatment	Standard protocol group: 65.8% (67/102) demonstrated ≥90% clearance after maximum of 10 treatment sessions; experimental group: demonstrated a similar clearance rate (83.7%) in 34 of 40 participants
Taneja et al[49] (2003)	Evaluate response of psoriatic plaques to 308-nm excimer laser	14 participants presenting with recalcitrant plaque psoriasis completed the study (44 total plaques)	Treatment group showed significant improvement compared to control (P<.001)

Reference (Year)	Study Objective	Study Group	Results
Rodewald et al[50] (2002)	Assess patient impressions and satisfaction with 308-nm laser for treatment of mild to moderate psoriasis	124 participants with mild to moderate psoriasis	55% (68/124) of patients reported overall satisfaction with treatment; 63% (78/124) thought they needed additional treatments; 25% (31/124) reported laser treatments were better than any prior treatments they had tried
Trehan et al[51] (2002)	Evaluate role of high-dose 308-nm excimer laser treatments for treatment of stable plaque-type psoriasis	16 participants completed the study; average age, 40.7 y	At baseline, mean modified PASI score was 6.31; 4 weeks after a single treatment, the mean PASI score was 3.56 ($P<.001$); 11/16 (69%) demonstrated substantial improvement after 4 weeks; 5/16 (31%) demonstrated minimal improvement after 4 weeks
Trehan et al[52] (2002)	Evaluate efficacy of multiple medium-dose 308-nm excimer laser treatments for psoriasis	15 participants completed study (8 males, 7 females); mean age, 44.9 y	Mean cumulative UV radiation dose was 6.1 J/cm^2; mean remission time was 3.5 mo; mean number of treatments to achieve >95% clearance was 10.6
Feldman et al[53] (2002)	Demonstrate the efficacy of the 308-nm excimer laser for treatment of psoriasis	92 participants with stable mild to moderate plaque-type psoriasis	Initial dose based on multiples of predetermined MED; subsequent doses based on response to treatment; 72% (66/92) of participants achieved at least 75% clearance (mean number of treatments, 6.2)
Asawanonda et al[54] (2000)	Determine the dose-response relationship of excimer laser–generated 308-nm UVB for treating plaque psoriasis	13 patients with at least 4 large stable plaques (mean age, 36.9 y; baseline PASI, 6)	Treatment with high fluences produced significantly better results than with medium and low fluences at weeks 4, 6, 8, and 10 ($P<.05$); mean cumulative dose, 203.03 mJ/cm^2

Abbreviations: PASI, psoriasis area and severity index; NB-UVB, narrowband UVB; PUVA, psoralen plus UVA; PDL, pulsed dye laser; MED, minimal erythema dose.

exposure. Monochromatic excimer light and the 308-nm excimer laser appear to have similar efficacy and evade some of the problems than can arise when utilizing other light therapies.

Phototherapy

Phototherapy using UVA or UVB irradiation is another important treatment option for psoriasis. Psoralen plus UVA employs the use of topical psoralen, which is absorbed preferentially by rapidly proliferating cells, followed by the application of 320- to 400-nm wavelength UVA light. The UVA light is strongly absorbed by the psoralen, having a mutagenic effect and inducing apoptosis. In a review of the literature, Dogra and De[56] determined that PUVA is comparable to NB-UVB in efficacy but may induce longer remission times; in one retrospective study, Brazzelli et al[57] estimated a remission time of approximately 386 days for PUVA. Unfortunately, PUVA simultaneously irradiates both affected and normal skin, thereby increasing carcinogenicity. Narrowband UVB therapy applies light at a wavelength of 311 nm and, similar to other phototherapies, affects DNA integrity, promotes cell-cycle arrest and apoptosis,[56] and also exerts some anti-inflammatory effects. Narrowband UVB has demonstrated efficacy in clearing moderate to severe chronic psoriasis; however, similar to PUVA, NB-UVB poses a carcinogenic risk to exposed skin.[58] Both PUVA and NB-UVB therapies serve as important modalities for chronic psoriasis, especially in patients with disease that is refractory to topical treatment, but these therapies also pose a risk for future cutaneous malignancy due to an accumulation of UV irradiation. The magnitude for this risk is still being elucidated.

CONCLUSION

Psoriasis has a complex pathophysiology in which both environmental and genetic factors play a role. Unfortunately, there is no known specific etiology for this relatively common chronic disease. Many therapeutic options are available for treating psoriasis, such as topical agents, systemic agents, NB-UVB and PUVA light modalities, and various lasers. Each of these modalities targets a contributing factor in the pathophysiology of psoriasis and contains specific side effects. Lasers have been investigated as alternatives for treating psoriasis due to the side effects of long-term use of potent topical corticosteroids, such as atrophy, striae, and telangiectasia. Overall, the 308-nm excimer laser is an effective treatment option for psoriasis. Most of the studies are small, though some larger ones also exist to support the positive results of the smaller studies. There are few side effects and the actual treatment time is only a few minutes. Phototherapy using UVA (320–400-nm light) or NB-UVB (311-nm light) is also effective in treating psoriasis, except, unlike the excimer laser, it may pose a higher risk for skin cancer due to the greater amount of healthy skin exposed to the carcinogenic light.

REFERENCES

1. Bos JD, Hulsebosch HJ, Krieg SR, et al. Immunocompetent cells in psoriasis. in situ immunophenotyping by monoclonal antibodies. *Arch Dermatol Res.* 1983;275:181-189.
2. Gottlieb AB, Chaudhari U, Baker DG, et al. The National Psoriasis Foundation Psoriasis Score (NPF-PS) system versus the Psoriasis Area Severity Index (PASI) and Physician's Global Assessment (PGA): a comparison. *J Drugs Dermatol.* 2003;2:260-266.
3. Kurd SK, Gelfand JM. The prevalence of previously diagnosed and undiagnosed psoriasis in US adults: results from NHANES 2003-2004 [published online ahead of print November 20, 2008] [published correction appears in *J Am Acad Dermatol.* 2009;61:507]. *J Am Acad Dermatol.* 2009;60:218-224.
4. Naldi L. Epidemiology of psoriasis. *Curr Drug Targets Inflamm Allergy.* 2004;3:121-128.
5. Stern RS, Nijsten T, Feldman SR, et al. Psoriasis is common, carries a substantial burden even when not extensive, and is associated with widespread treatment dissatisfaction. *J Investig Dermatol Symp Proc.* 2004;9:136-139.
6. Griffiths CE, Barker JN. Pathogenesis and clinical features of psoriasis. *Lancet.* 2007;370:263-271.
7. Braathen LR, Botten G, Bjerkedal T. Prevalence of psoriasis in Norway. *Acta Derm Venereol Suppl (Stockh).* 1989;142:5-8.
8. Nevitt GJ, Hutchinson PE. Psoriasis in the community: prevalence, severity and patients' beliefs and attitudes towards the disease. *Br J Dermatol.* 1996;135:533-537.
9. Henseler T, Christophers E. Psoriasis of early and late onset: characterization of two types of psoriasis vulgaris. *J Am Acad Dermatol.* 1985;13:450-456.
10. Plunkett A, Marks R. A review of the epidemiology of psoriasis vulgaris in the community. *Australas J Dermatol.* 1998;39:225-232.
11. Menter A, Gottlieb A, Feldman SR, et al. Guidelines of care for the management of psoriasis and psoriatic arthritis: section 1. overview of psoriasis and guidelines of care for the treatment of psoriasis with biologics. *J Am Acad Dermatol.* 2008;58:826-850.
12. Bowcock AM, Krueger JG. Getting under the skin: the immunogenetics of psoriasis [published correction appears in *Nat Rev Immunol.* 2005;5:826]. *Nat Rev Immunol.* 2005;5:699-711.
13. Nestle FO, Kaplan DH, Barker J. Psoriasis. *N Engl J Med.* 2009;361:496-509.
14. de Cid R, Riveira-Munoz E, Zeeuwen PL, et al. Deletion of the late cornified envelope LCE3B and LCE3C genes as a susceptibility factor for psoriasis [published online ahead of print January 25, 2009]. *Nat Genet.* 2009;41:211-215.
15. Cargill M, Schrodi SJ, Chang M, et al. A large-scale genetic association study confirms IL12B and leads to the identification of IL23R as psoriasis-risk genes [published online ahead of print December 21, 2006]. *Am J Hum Genet.* 2007;80:273-290.
16. Capon F, Di Meglio P, Szaub J, et al. Sequence variants in the genes for the interleukin-23 receptor (IL23R) and its ligand (IL12B) confer protection against psoriasis [published online ahead of print June 22, 2007]. *Hum Genet.* 2007;122:201-206.
17. Wolf N, Quaranta M, Prescott NJ, et al. Psoriasis is associated with pleiotropic susceptibility loci identified in type II diabetes

and Crohn disease [published online ahead of print November 9, 2007]. *J Med Genet.* 2008;45:114-116.
18. Valdimarsson H, Baker BS, Jónsdóttir I, et al. Psoriasis: a T-cell-mediated autoimmune disease induced by streptococcal superantigens? *Immunol Today.* 1995;16:145-149.
19. Gaspari AA. Innate and adaptive immunity and the pathophysiology of psoriasis. *J Am Acad Dermatol.* 2006;54(3, suppl 2):S67-S80.
20. Nickoloff BJ. Skin innate immune system in psoriasis: friend or foe? *J Clin Invest.* 1999;104:1161-1164.
21. Robert C, Kupper TS. Inflammatory skin diseases, T cells, and immune surveillance. *N Engl J Med.* 1999;341:1817-1828.
22. Huerta C, Rivero E, Rodríguez LA. Incidence and risk factors for psoriasis in the general population. *Arch Dermatol.* 2007;143:1559-1565.
23. Neimann AL, Shin DB, Wang X, et al. Prevalence of cardiovascular risk factors in patients with psoriasis [published online ahead of print September 25, 2006]. *J Am Acad Dermatol.* 2006;55:829-835.
24. Henseler T, Christophers E. Disease concomitance in psoriasis. *J Am Acad Dermatol.* 1995;32:982-986.
25. Telfer NR, Chalmers RJ, Whale K, et al. The role of streptococcal infection in the initiation of guttate psoriasis. *Arch Dermatol.* 1992;128:39-42.
26. Sommer DM, Jenisch S, Suchan M, et al. Increased prevalence of the metabolic syndrome in patients with moderate to severe psoriasis [published online ahead of print September 22, 2006]. *Arch Dermatol Res.* 2006;298:321-328.
27. Simonić E, Kaštelan M, Peternel S, et al. Childhood and adulthood traumatic experiences in patients with psoriasis. *J Dermatol.* 2010;37:793-800.
28. Menter M, Stoff B. Clinical manifestations of psoriasis. In: Menter M, Stoff B. *Psoriasis.* London, England: Manson Publishing Ltd; 2010:25-46.
29. Gudjonsson JE, Elder JT. Psoriasis. In: Wolff K, Goldsmith LE, Katz S, et al, eds. *Fitzpatrick's Dermatology in General Medicine.* 7th ed. New York, NY: McGraw-Hill; 2008.
30. Fernández-Guarino M, Harto A, Sánchez-Ronco M, et al. Pulsed dye laser vs. photodynamic therapy in the treatment of refractory nail psoriasis: a comparative pilot study [published online ahead of print March 11, 2009]. *J Eur Acad Dermatol Venereol.* 2009;23:891-895.
31. González-Serva A. Disorders of the nail apparatus. In: Barnhill RL, Crowson AN, Magro CM, et al. *Dermatopathology.* 3rd ed. New York, NY: McGraw-Hill; 2010:987-998.
32. Tournas JA, Lowe NJ, Yamauchi PS. Laser and novel light source treatments for psoriasis. *Lasers Surg Med.* 2004;35:165-173.
33. van Lümig P, Driessen R, Berends M, et al. Safety of treatment with biologics for psoriasis in daily practice: 5-year data [published online ahead of print]. *J Eur Acad Dermatol Venereol.* doi: 10.1111/j.1468-3083.2011.04044.x.
34. Green C, Ferguson J, Lakshmipathi T, et al. 311 nm UVB phototherapy—an effective treatment for psoriasis. *Br J Dermatol.* 1988;119:691-696.
35. Coven TR, Burack LH, Gilleaudead R. Narrowband UV-B produces superior clinical and histopathological resolution of moderate-to-severe psoriasis in patients compared with broadband UV-B. *Arch Dermatol.* 1997;133:1514-1522.
36. Walters IB, Burack LH, Coven TR, et al. Suberythemogenic narrow-band UVB is markedly more effective than conventional UVB in treatment of psoriasis vulgaris. *J Am Acad Dermatol.* 1999;40 (6, pt 1):893-900.
37. Gerber W, Arheilger B, Ha TA, et al. Ultraviolet B 308-nm excimer laser treatment of psoriasis: a new phototherapeutic approach. *Br J Dermatol.* 2003;149:1250-1258.
38. He YL, Zhang XY, Dong J, et al. Clinical efficacy of a 308 nm excimer laser for treatment of psoriasis vulgaris. *Photodermatol Photoimmunol Photomed.* 2007;23:238-241.
39. Goldberg DJ, Chwalek J, Hussain M. 308-nm excimer laser treatment of palmoplantar psoriasis. *J Cosmet Laser Ther.* 2011;13:47-49.
40. Hadi SM, Al-Quran H, de Sá Earp AP, et al. The use of the 308-nm excimer laser for the treatment of psoriasis [published online ahead of print October 9, 2010]. *Photomed Laser Surg.* 2010;28:693-695.
41. Gattu S, Rashid RM, Wu JJ. 308-nm excimer laser in psoriasis vulgaris, scalp psoriasis, and palmoplantar psoriasis [published online ahead of print August 19, 2008]. *J Eur Acad Dermatol Venereol.* 2009;23:36-41.
42. Trott J, Gerber W, Hammes S, et al. The effectiveness of PUVA treatment in severe psoriasis is significantly increased by additional UV 308-nm excimer laser sessions [published online ahead of print December 18, 2007]. *Eur J Dermatol.* 2008;18:55-60.
43. Morison WL, Atkinson DF, Werthman L. Effective treatment of scalp psoriasis using the excimer (308 nm) laser. *Photodermatol Photoimmunol Photomed.* 2006;22:181-183.
44. Nisticò SP, Saraceno R, Stefanescu S, et al. A 308-nm monochromatic excimer light in the treatment of palmoplantar psoriasis. *J Eur Acad of Dermatol Venereol.* 2006;20:523-526.
45. Taibjee SM, Cheung ST, Laube S, et al. Controlled study of excimer and pulsed dye lasers in the treatment of psoriasis. *Br J Dermatol.* 2005;153:960-966.
46. Pahlajani N, Katz BJ, Lozano AM, et al. Comparison of the efficacy and safety of the 308 nm excimer laser for the treatment of localized psoriasis in adults and in children: a pilot study. *Pediatr Dermatol.* 2005;22:161-165.
47. Köllner K, Wimmershoff MB, Hintz C, et al. Comparison of the 308-nm excimer laser and a 308-nm excimer lamp with 311-nm narrowband ultraviolet B in the treatment of psoriasis. *Br J Dermatol.* 2005;152:750-754.
48. Taylor CR, Racette AL. A 308-nm excimer laser for the treatment of scalp psoriasis. *Lasers Surg Med.* 2004;34:136-140.
49. Taneja A, Trehan M, Taylor CR. 308-nm excimer laser for the treatment of psoriasis: induration-based dosimetry. *Arch Dermatol.* 2003;139:759-764.
50. Rodewald EJ, Housman TS, Mellen BG, et al. Follow-up survey of 308-nm laser treatment of psoriasis. *Lasers Surg Med.* 2002;31:202-206.
51. Trehan M, Taylor CR. High-dose 308-nm excimer laser for the treatment of psoriasis. *J Am Acad Dermatol.* 2002;46:732-737.
52. Trehan M, Taylor CR. Medium-dose 308-nm excimer laser for the treatment of psoriasis. *J Am Acad Dermatol.* 2002;47:701-708.
53. Feldman SR, Mellen BG, Housman TS, et al. Efficacy of the 308-nm excimer laser for treatment of psoriasis: results of a multicenter study. *J Am Acad Dermatol.* 2002;46:900-906.
54. Asawanonda P, Anderson RR, Chang Y, et al. 308-nm excimer laser for the treatment of psoriasis: a dose-response study. *Arch Dermatol.* 2000;136:619-624.
55. Mavilia L, Mori M, Rossi R, et al. 308 nm monochromatic excimer light in dermatology: personal experience and review of the literature. *G Ital Dermatol Venereol.* 2008;143:329-337.
56. Dogra S, De D. Narrowband ultraviolet B in the treatment of psoriasis: the journey so far! *Indian J Dermatol Venerol Leprol.* 2010;76:652-661.
57. Brazzelli V, Barbagallo T, Trevisan V, et al. The duration of clinical remission of photochemotherapy and narrow-band UV-B phototherapy in the treatment of psoriasis: a retrospective study. *Int J Immunopathol Pharmacol.* 2008;21:481-484.
58. Grundmann-Kollmann M, Ludwig R, Zollner TM, et al. Narrowband UVB and cream psoralen-UVA combination therapy for plaque-type psoriasis. *J Am Acad Dermatol.* 2004;50:734-739. ■