with modulation by a granulocyte-macrophage colony-stimulation factor-based antitumor vaccine. *J Clin Oncol* 25:2546–53

Frey AB (2006) Myeloid suppressor cells regulate the adaptive immune response to cancer. *J Clin Invest* 116:2587–90

Gattinoni L, Ranganathan A, Surman DR *et al.* (2006) CTLA-4 dysregulation of self/tumor-reactive CD8+ T-cell function is CD4+ T-cell dependent. *Blood* 108:3818–23

Hamid O, Robert C, Daud A *et al.* (2013) Safety and tumor responses with lambrolizumab (anti-PD-1) in melanoma. *N Engl J Med* 369:134–44

Hodi FS, O'Day SJ, McDermott DF *et al.* (2010) Improved survival with ipilimumab in patients with metastatic melanoma. *N Engl J Med* 363:711–23

Ilkovitch D, Lopez DM (2008) Immune modulation by melanoma-derived factors. *Exp Dermatol* 17:977–85

Nishimura H, Nose M, Hiai H *et al.* (1999) Development of lupus-like autoimmune diseases by disruption of the PD-1 gene encoding an ITIM motif-carrying immunoreceptor. *Immunity* 11:141–51

Tivol EA, Borriello F, Schweitzer AN *et al.* (1995) Loss of CTLA-4 leads to massive lymphoproliferation and fatal multiorgan tissue destruction, revealing a critical negative regulatory role of CTLA-4. *Immunity* 3:541–7

Ethnicity and Hidradenitis Suppurativa

Journal of Investigative Dermatology (2014) **134,** 2842–2843; doi:10.1038/jid.2014.220; published online 5 June 2014

TO THE EDITOR

There is a lack of definitive epidemiologic information on patients who suffer from hidradenitis suppurativa (HS), as well as poor understanding of the etiology of this process. It is essential to gather more accurate information regarding HS as understanding of disease associations may lead to improved comprehension of the pathology. In their recent report, Vazques *et al.* (2013) have put forth an excellent population-based study on this disease process, which helps to provide valuable information on such associations. They found that 90.3% of the patients with HS in Olmsted County, Minnesota, were white, but specifically note that this racial breakdown may be a reflection of the racial demographics of the county itself, which is predominantly white. The classical teaching regarding HS has been that this process occurs more frequently in those of African descent (McMichael *et al.*, 2008). However, many of the published epidemiologic studies are similar to this publication in reporting a predominance of whites among those affected. It is important to consider that many of these studies are drawn from the northern or western European populations. This may again be a reflection of the demographics of these regions, but this message dominates the available literature.

Over the last several years, we have increasingly recognized that there is a large population of patients in our area who suffer from follicular disorders, particularly HS. As such, we have performed our own retrospective review of all HS patients seen in our clinic during an 18-month time period from 1 January 2011 to 31 May 2012.

All encounters were searched, and there were 366 patients identified with the clinical diagnosis of HS seen in the Dermatology Clinic at the Henry Ford Medical Center during this time period. Given that overall there were 8,480 patients seen in the clinic during this time, HS patients comprised 4.3% of the dermatology clinic patient population. For 6,664 of these patients, including all 366 of the patients with the diagnosis of HS, there were data available regarding race. In addition, the US Census Bureau data for Wayne County, Michigan, in 2010 (http://quickfacts.census.gov/qfd/states/26/26163lk.html, accessed 21 May 2013) indicate that the population of Wayne County was 54.2% white and 41.8% black. For the HS patients, 54.4% (199) were black, 25.7% (94) were white, and the remaining 19.9% (73) were classified as ''other.'' Looking at the overall population of patients seen in the Henry Ford Dermatology Clinic for all diagnoses, 47.0% (3,131) were black, 35.8% (2,389) were white, and 17.2% (1,144) fell into the category of ''other.'' We found that 6.4% of the black patients as compared with 3.9% of the white patients seen in the clinic were seen for the diagnosis of HS (*P*-value of <0.001), using a chi-square test, SAS9.2 (SAS, Cary, NC) (Table 1). We feel our data support the traditional teaching that HS is more common in those of African descent, which is not currently substantiated in the medical literature.

CONFLICT OF INTEREST

Dr Hamzavi has served and/or currently serves as an investigator in clinical trial activities for the following companies: Microdermis Corporation, Galderma, ViroXis, Basilea, Abbvie, La Roche – Posay, Dow, Centocor, Amgen, Clinuvel, and

Table 1. Ethnicity of HS patients versus non-HS patients for the Henry Ford Dermatology Clinic

	White	Black	Other	*P*-value
All non-HS patients	2,295 (96.1%)	2,932 (93.6%)	1,071 (93.6%)	<0.001
HS patients	94 (3.9%)	199 (6.4%)	73 (6.4%)	

Abbreviation: HS, hidradenitis suppurativa.

Abbreviation: HS, hidradenitis suppurativa

Accepted article preview online 12 May 2014; published online 5 June 2014

Pfizer. Dr Reeder and Ms Mahan state no conflict of interest.

Virginia J. Reeder[1], Meredith G. Mahan[2] and Iltefat H. Hamzavi[1]

[1]Department of Dermatology, Henry Ford Health System, Detroit, Michigan, USA and [2]Department of Public Health Science, Henry Ford Health System, Detroit, Michigan, USA
E-mail: Ihamzav1@hfhs.org

REFERENCES

McMichael A, Guzman-Sanchez D, Kelly P (2008) Folliculitis and other follicular disorders. In: Bolognia JL, Jorizzo JL, Rapini RP, (eds) *Dermatology*. Mosby Elsevier: Spain, 528

Vazquez BG, Alikhan A, Weaver AL *et al.* (2013) Incidence of hidradenitis suppurativa and associated factors: a population-based study of Olmsted County, Minnesota. *J Invest Dermatol* 133:97–103

Abnormal Epidermal Barrier Recovery in Uninvolved Skin Supports the Notion of an Epidermal Pathogenesis of Psoriasis

Journal of Investigative Dermatology (2014) **134,** 2843–2846; doi:10.1038/jid.2014.205; published online 5 June 2014

TO THE EDITOR

Psoriasis is generally considered to be an immunologically initiated disorder, which shares certain common susceptibility loci with autoimmune diseases (Zhang, 2012). Yet, both clinical experience (Gottlieb *et al.*,1990; Griffiths *et al.*, 1995; Volden *et al.*, 2001) and recent molecular studies (Mischke *et al.*, 1996; Kim *et al.*, 2011; Vermeij *et al.*, 2011; Bergboer *et al.*, 2012) support an emerging concept that psoriasis could be ''driven'' by a primary defect in epidermal permeability barrier function. Clinicians know well that psoriasis predictably flares during winter months (Park and Youn, 1998; Kwon *et al.*, 2012) when the barrier is under additional stress owing to low stratum corneum (SC) hydration, which accelerates transepidermal water loss (TEWL) rates (Lin, 2009; Muizzuddin *et al.*, 2013). They also appreciate that sites vulnerable to epidermal trauma, such as the extensors of the extremities and the scalp, are preferentially involved in psoriasis. The Koebner phenomenon offers an additional, eloquent example of how psoriasis can be provoked by external perturbations. Finally, improvement of epidermal permeability barrier function by occlusion alone often alleviates psoriasis (e.g., Friedman, 1987).

Among psoriasis susceptibility genes, PSORS4 is located on chromosome 1q21, within the epidermal differentiation complex, which encodes numerous proteins required for epidermal differentiation and formation of the cornified envelope (Mischke *et al.*, 1996), a structure that is critical for the permeability barrier (Vermeij *et al.*, 2011). In addition, deletion of differentiation-related proteins, such as keratin1, whose levels are reduced in psoriasis (Thewes *et al.*, 1991; Bata-Csörgö and Szell, 2012), not only compromises the permeability barrier but also leads to upregulation of inflammatory genes and altered cytokine production in a pattern that resembles psoriasis (Roth *et al.*, 2012). Finally, in experimental models, disruption of the epidermal permeability barrier in otherwise normal skin stimulates epidermal hyperproliferation (Proksch *et al.*, 1991; Man *et al.*, 2008), cytokine production (Denda *et al.*, 1996; Wood *et al.*, 1996), downstream inflammatory cell infiltration (Proksch *et al.*, 1996; Lin *et al.*, 2013), and epidermal vascular endothelial growth factor production, leading to dermal capillary proliferation (Elias *et al.*, 2008), all of which are prominent features of psoriasis.

Although prior studies have demonstrated phenotype-dependent abnormalities in basal permeability barrier function in psoriatic lesions (Ghadially *et al.*, 1996), the uninvolved skin in psoriasis reportedly displays normal TEWL levels (Takahashi *et al.*, 2014). Yet, expression levels of filaggrin, a protein of known importance for the barrier (Scharschmidt *et al.*, 2009; Irvine *et al.*, 2011), and loricrin are reportedly lower than normal in uninvolved skin sites of psoriasis (Kim *et al.*, 2011). Because alterations in the epidermal differentiation should predict an abnormality in barrier function, we hypothesized that if abnormal epidermal function has a role in the pathogenesis of psoriasis then epidermal function should be abnormal in uninvolved psoriatic skin. Hence, we assessed changes in epidermal function in the uninvolved and involved skin of a large cohort of Chinese patients, with either stable or progressive psoriasis.

In all, 44 patients with psoriasis vulgaris and 68 normal controls were enrolled in the study (Table 1). All participants were provided written informed consent. This study was carried out according to the Helsinki Declaration Principles and the protocol approved by the Human Research Subcommittee, Dalian Skin Disease Hospital. Stable psoriasis was defined as no recent development of new lesions, as well as no recent expansion of pre-existing lesions, whereas progressive psoriasis was defined as recent or ongoing development of new lesions and prominent inflammation, often accompanied by

Abbreviations: SC, stratum corneum; TEWL, transepidermal water loss

Accepted article preview online 29 April 2014; published online 5 June 2014