A Review of the *Journal of Investigative Dermatology*'s Most Cited Publications over the Past 25 Years and the Use of Developing Bibliometric Methodologies to Assess Journal Quality

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The *JID* is a major resource for publishing dermatologic research. Here we document bibliometric systems that permit detailed analysis of *JID*'s relative scientific quality. We provide an overview of metrics employed by ISI Thomson Reuters Web of Knowledge and Elsevier's open-access Scopus to measure *JID*'s comparative performance. We list *JID*'s 50 most cited articles between 1986 and 2010 and summarize the six most cited papers published during this period. We conclude by showing how selected cited papers have influenced research in the *JID* subcategories of immunology/infection and photobiology during this period. *JID* has thrived as the strength of its editorial leadership and the quality of dermatologic science have grown apace.

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Anyone old enough to recall the weekly arrival of *Current Contents* and the painfully slow process of combing through each issue searching for relevant articles of interest, manually addressing postcards requesting reprints, or trudging to the library to copy a paper of interest will remember the name Eugene Garfield. He was responsible for *Current Contents*, a true pioneer in addressing the explosion of scientific information before we all had laptops and desktops. The introduction of PubMed in 1996 and Google Scholar in 2004 provided rapid online access to the literature. Dr. Garfield was also the founder of the Institute for Scientific Information (ISI). In 2004, the ISI was acquired by the science division of the Thomson Reuters Company. In this review we attempt to provide a glimpse of the newly developing bibliometric tools that have become available for assessing journal quality and to compare the position of *JID* among other leading dermatology journals using some of these tools. We then offer perspectives on *JID*'s growth as an influential source of knowledge in the field of cutaneous biology over the past 25 years.

ISI Thomson Reuters Web of Knowledge

The ISI Thomson Reuters Web of Knowledge provides quick, powerful access to the world's leading citation databases. It covers more than 10,000 of the highest-impact journals worldwide. In addition to *Current Contents*, Garfield created numerous innovative bibliographic products. Together with Irwin Sher, he first proposed the concept of impact factor by re-sorting the author citation index into the Journal Citation Index and, with the support of the National Institutes of Health, was thereafter able to create the Science Citation Index (SCI) (Garfield, 2006). This led to the recognition that there was a core group of highly cited journals that would form the core of the SCI.

Using the Web of Knowledge, we identified the 50 most cited articles published in *JID* between 1986 and 2010, listed the number of times each was cited during that time, and subcategorized the articles using the subcategories utilized by *JID* since 2002. These data are shown in Table 1 (see also Supplementary Table S1 online).

Journal impact factor

The impact factor is defined as the ratio of the number of citations in the current year (numerator) to all articles and reviews published in the previous 2 years (denominator). Example of the calculation of the 2010 *JID* impact factor:

> Total citations in 2010 to articles published in 2008 (1,705) and 2009 (1,844) = 3,549; number of articles published in 2008–2009 = 566
>
> *JID* impact factor = 3,549/566 = 6.270

There has been gradual improvement in *JID*'s impact factor over the years, and between 2006 and 2010 it rose steadily from 4.535 to 6.270 (Figure 1). Its impact factor places the *Journal* first in a list of the top 20 dermatology journals ranked by the Web of Knowledge (Supplementary Table S2 online).

The use of the 2-year window to calculate the impact factor has been criticized as being too short in that it does not represent a typical value to account for changes that could occur over a longer time span. This has led to the use of the 5-year impact factor calculated identically to the original 2-year impact factor but over 5 years.

Five-year journal impact factor

The 5-year journal impact factor is defined as the ratio of the number of citations in the current year (numerator) to all articles and reviews published in the previous 5 years (denominator).

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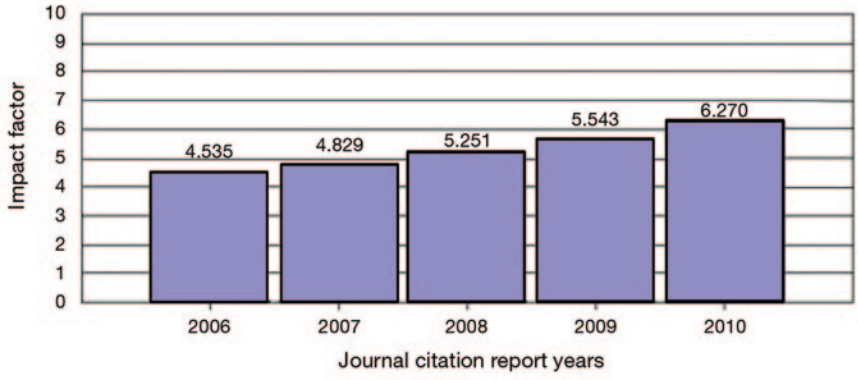

Figure 1. Five-year trend of Institute for Scientific Information (ISI) Thomson impact factor for the *Journal of Investigative Dermatology*.

Example of the calculation of the 2010 *JID* 5-year impact factor:

Total citations between 2005 and 2009 = 8,435; number of articles published between 2005 and 2009 = 1,465

JID 5-year impact factor = 8,435/1,465 = 5.758

The utility of the impact factor has been questioned as a tool for assessing the quality of scientific journals. For example, it has been pointed out that the SCI database includes only normal articles, notes, and reviews in the denominator as citable items but records citations to all types of documents (including editorials, letters, and meeting abstracts) in the numerator (Favaloro, 2008; Elsaie and Kammer, 2009). As a result, journals that include meeting reports, editorials, and extensive correspondence sections could inflate that journal's impact factor relative to those that do not. Review articles may also help to increase the impact factor because of increased citations. Despite these limitations, it is generally agreed that in each specialty the best journals are those in which it is most difficult to have an article accepted, and these are the journals (most of which predated the concept of impact factor) that tend to have higher impact factors (Hoeffel, 1998).

Wolthoff *et al*. (2011) have attempted to address some of the limitations of impact factor rankings as they pertain to dermatology journals by proposing the use of the comprehensive citation factor (CCF) (Supplementary Table S3 online). The CCF is based on data obtained in 2007 and includes in the denominator all citable articles, specifically editorials and letters. Their intent is to discourage the high proportion of editorials and letters to the editor that can artificially inflate a journal's impact factor. They address another potential shortcoming of impact factor, namely, the fact that the classification of journal articles by the Web of Knowledge is performed manually by multiple individuals, thereby raising questions about the accuracy and consistency of these designations. Rossner *et al*. (2008) have also expressed concerns regarding the arbitrary manner in which the Web of Knowledge computes impact factors and interprets their databases.

Another problem with the concept of impact factor is its use to evaluate the scholarly credentials of scientists rather than journals (Fersht, 2009). Fersht suggests that this is an inappropriate use of impact factor and that assessment of academic merit requires careful and meticulous analysis by expert scholars in the subject area—the use of a simple metric for this purpose should never be a substitute for the evaluation of research quality. Despite these reservations, impact factor remains an objective measure of quality for the best journals in a specialty.

Immediacy index

The immediacy index of a journal is calculated by dividing the number of citations to articles published in a given year by the number of articles published in that year. It is an indicator of the speed with which citations to a specific journal appear in the published literature. Example of the calculation of the 2010 *JID* immediacy index:

Citations to items published in 2010 = 412

Numbers of items published in 2010 = 250

JID immediacy index = 412/250 = 1.648

Because it is a per-article average, the immediacy index tends to discount the advantage of large journals over small ones. However, frequently issued journals may have an advantage because an article published early in the year has a better chance of being cited than one published later in the year. Many publications that publish infrequently or late in the year have low immediacy indexes. For comparing journals specializing in cutting-edge research, however, the immediacy index can provide a useful perspective.

Cited half-life

The cited half-life for a journal is the median age in years of its items cited in the current year. It is defined by the number of publication years from the current year that account for 50% of the citations received by the journal. Half of the total citations to the journal are to items published within the cited half-life.

JID cited half-life = 7.9 years

Citing half-life

The citing half-life for a journal is the median age of the items the journal cited in the current year. Half of the citations in the journal are to items published within the citing half-life.

JID citing half-life = 6.6 years

Further efforts have been made to find additional metrics for measuring the quality of scientific journals (Rousseau and Stimulate 8 Group, 2009). In the report, alternatives to the impact factor were compared to ascertain their value. These include the Eigenfactor score and the Article Influence score. It was

shown that although these indicators are calculated using different methods and databases, they strongly correlate with the Web of Knowledge impact factor and with one another.

Eigenfactor score

The Eigenfactor score of a journal is an estimate of the percentage of the time that researchers actually spend with that particular journal. The Eigenfactor algorithm corresponds to a simple model of research in which readers follow chains of citations as they move from journal to journal. Imagine a researcher in a library selecting a journal article at random. After reading the article, the researcher randomly selects a citation from the article and proceeds to the cited journal, reads a random article there, and selects a citation in another journal volume. This process is then repeated over and over. The Eigenfactor score is the sum of normalized citations received from other journals weighted by the status of the citing journals. Citations are normalized with respect to the total amount of cited references of the citing journal. The citation target period is 5 years.

JID Eigenfactor score = 0.05137

Article Influence score

The Article Influence score is a measure of the average influence per article of each of its papers over the first 5 years after its publication. Article Influence scores are normalized so that the mean article in the entire ISI Thomson Journal Citation Reports (JCR) database has an article influence of 1.00. Thus, in 2010 *JID* had an Article Influence score of 1.800. This means that the average article in *JID* has 1.8 times the influence of the mean journal in the JCR.

The data in Supplementary Table S2 online show how the 20 highest cited dermatology journals in the Web of Knowledge compare in terms of these various metrics. Given that *JID* has a reputation for publishing research articles and reviews focused on basic research and increasingly on translational application of that research, it is perhaps not surprising that it is ranked highest in virtually all of these bibliometric categories.

Franceschet (2010) compared 2-year impact factor, 5-year impact factor, Eigenfactor score, and Article Influence score as measures of journal quality. Article Influence and the 2-year impact factor were close to the 5-year impact factor as tools in this regard. Article influence was shown to be the most stable indicator across different scientific disciplines.

Rizkallah and Sin (2010) also used a combined approach to assess journal quality by comparing impact factor, Eigenfactor, and Article Influence scores in a series of highly cited journals between 2001 and 2008. Their analysis of impact factor and Eigenfactor score yielded a similar rank order of medical journals, although some discrepancies were apparent. For example, journals that publish large numbers of papers have higher Eigenfactor scores than would be expected for their impact factor, whereas the reverse is true for journals that publish fewer papers.

h-Index

The h-index was first proposed by Jorge Hirsch, a physicist at the University of California, San Diego (Hirsch, 2005). It is defined as the highest number of published papers by a scientist receiving at least that number of citations. For example, someone with an h-index of 50 has written 50 papers, each of which has been cited at least 50 times. Hirsch believes that this is more objective than measures based on numbers of publications because a large number of mediocre publications would create a false impression of superior scholarship. Since its introduction, the h-index has become a widely accepted indicator of scientific performance and is included in major bibliographic databases, including the Web of Knowledge. It is said to have several advantages, including simplicity and the fact that citation impact and publication numbers are combined in a single number (Bornmann *et al.*, 2011). Loscalzo (2011), however, questions the utility of the h-index and emphasizes that it suffers from the same limitation associated with citation indexes and is not a surrogate for scientific quality. He adds that it seems unlikely that any substitute for the impact factor will be found in the near future because it has become such an embedded measure both academically and commercially.

We initially attempted to provide h-index data for the authors of the top 50 cited papers in *JID* between 1986 and 2010 (Table 1). However, this analysis was complicated by a number of confounders, including duplicate names and initials, which in our opinion made confirmation of these scores uncertain, and we have therefore not included them here. The assignment and use of methods to more precisely identify authors should help to enhance the accuracy of author h-indexes in the future.

Elsevier's Scopus, also known as SciVerse Scopus, is another citation database containing both peer-reviewed research literature and Web sources. This is an open-access portal that also attempts to address the quantity and the quality of scientific publications. It provides comprehensive coverage of the scientific, technical, medical, and social sciences fields as well as, recently, the arts and humanities. SCImago is their portal that includes the journals and country scientific indicators developed from information in the Scopus database (Elsevier B.V.). These indicators can also be used to assess and analyze scientific domains. This platform takes its name from the SCImago Journal Rank (SJR) indicator, which in turn is derived from Google's PageRank system. This indicator ranks journals in the Scopus database. The Scopus ranking for the top 25 dermatology journals is shown in Supplementary Table S4 online. In addition to SJR rank, Scopus has identified the number of citations per article over the prior 2 years as a meaningful indicator of journal quality. Using this indicator, *JID*, with a total of 6.24 citations per article, is the top-ranking dermatology journal. The Scopus and the Web of Knowledge rankings are quite similar (Supplementary Table S2). The Scopus ranking of selected dermatology journals relative to more than 18,750 other covered scientific journals is shown in Supplementary Table S5. *JID* ranks 309th of the more than 18,750 journals currently in the Scopus database.

Table 1. The 50 most cited *JID* articles in the Institute for Scientific Information Thomson Reuters Web of Knowledge over the past 25 years and their subcategories

Rank	Total times cited	Reference	Category
1	676	Ades EW, Candal FJ, Swerlick RA *et al*. (1992) HMEC-1: establishment of an immortalized human microvascular endothelial cell line. *J Invest Dermatol* 99:683–90	Vascular biology
2	431	Frazier K, Williams S, Kothapalli D *et al*. (1996) Stimulation of fibroblast cell growth, matrix production, and granulation tissue formation by connective tissue growth factor. *J Invest Dermatol* 107:404–11	Connective tissue
3	391	Rajadhyaksha M, Grossman M, Esterowitz D *et al*. (1995) *In vivo* confocal scanning laser microscopy of human skin: melanin provides strong contrast. *J Invest Dermatol* 104:946–52	Clinical research
4	381	Tschachler E, Groh V, Popovic M *et al*. (1987) Epidermal Langerhans cells—a target for HTLV-III/LAV infection. *J Invest Dermatol* 88:233–7	Immunology/infection
5	378	Imokawa G, Abe A, Jin K *et al*. (1991) Decreased level of ceramides in stratum corneum of atopic dermatitis: an etiologic factor in atopic dry skin? *J Invest Dermatol* 96:523–6	Clinical research
6	361	Smith EL, Walworth NC, Holick MF (1986) Effect of 1 α,25-dihydroxyvitamin D3 on the morphologic and biochemical differentiation of cultured human epidermal keratinocytes grown in serum-free conditions. *J Invest Dermatol* 86:709–14	Cell biology
7	351	Romani N, Lenz A, Glassel H *et al*. (1989) Cultured human Langerhans cells resemble lymphoid dendritic cells in phenotype and function. *J Invest Dermatol* 93:600–9	Immunology/infection
8	339	Giudice GJ, Emery DJ, Diaz LA (1992) Cloning and primary structural analysis of the bullous pemphigoid autoantigen BP180. *J Invest Dermatol* 99:243–50	Immunology/infection
9	323	van der Heijden FL, Wierenga EA, Bos JD *et al*. (1991) High frequency of IL-4-producing $CD4^+$ allergen-specific T lymphocytes in atopic dermatitis lesional skin. *J Invest Dermatol* 97:389–94	Immunology/infection
10	304	Yoshikawa T, Rae V, Bruins-Slot W *et al*. (1990) Susceptibility to effects of UVB radiation on induction of contact hypersensitivity as a risk factor for skin cancer in humans. *J Invest Dermatol* 95:530–6	Photobiology
11	303	Wysocki AB, Staiano-Coico L, Grinnell F (1993) Wound fluid from chronic leg ulcers contains elevated levels of metalloproteinases MMP-2 and MMP-9. *J Invest Dermatol* 101:64–8	Vascular biology
12	299	Mansbridge JN, Knapp AM (1987) Changes in keratinocyte maturation during wound healing. *J Invest Dermatol* 89:253–63	Wound healing
13	284	Uyemura K, Yamamura M, Fivenson DF *et al*. (1993) The cytokine network in lesional and lesion-free psoriatic skin is characterized by a T-helper type 1 cell-mediated response. *J Invest Dermatol* 101:701–5	Immunology/infection
14	276	Borradori L, Sonnenberg A (1999) Structure and function of hemidesmosomes: more than simple adhesion complexes. *J Invest Dermatol* 112:411–8	Connective tissue
15	274	Detmar M, Brown LF, Schön MP *et al*. (1998) Increased microvascular density and enhanced leukocyte rolling and adhesion in the skin of VEGF transgenic mice. *J Invest Dermatol* 111:1–6	Vascular biology
16	273	Rajadhyaksha M, González S, Zavislan JM *et al*. (1999) *In vivo* confocal scanning laser microscopy of human skin II: advances in instrumentation and comparison with histology. *J Invest Dermatol* 113:293–303	Clinical research
17	265	Igarashi A, Nashiro K, Kikuchi K *et al*. (1996) Connective tissue growth factor gene expression in tissue sections from localized scleroderma, keloid, and other fibrotic skin disorders. *J Invest Dermatol* 106:729–33	Connective tissue
18	247	Schlaak JF, Buslau M, Jochum W *et al*. (1994) T cells involved in psoriasis vulgaris belong to the Th1 subset. *J Invest Dermatol* 102:145–9	Immunology/infection
19	246	Stern RS, Lange R (1988) Non-melanoma skin cancer occurring in patients treated with PUVA five to ten years after first treatment. *J Invest Dermatol* 91:120–4	Photobiology
20	246	Müller-Röver S, Handjiski B, van der Veen C *et al*. (2001) A comprehensive guide for the accurate classification of murine hair follicles in distinct hair cycle stages. *J Invest Dermatol* 117:3–15	Appendages
21	245	Wood GS, Tung RM, Haeffner AC *et al*. (1994) Detection of clonal T-cell receptor gamma gene rearrangements in early mycosis fungoides/Sezary syndrome by polymerase chain reaction and denaturing gradient gel electrophoresis (PCR/DGGE). *J Invest Dermatol* 103:34–41	Clinical research
22	240	Parsa R, Yang A, McKeon F *et al*. (1999) Association of p63 with proliferative potential in normal and neoplastic human keratinocytes. *J Invest Dermatol* 113:1099–105	Cell biology
23	236	Haake AR, Polakowska RR (1993) Cell death by apoptosis in epidermal biology. *J Invest Dermatol* 101:107–12	Keratinocytes/epidermis
24	230	Cooper KD (1994) Atopic dermatitis: recent trends in pathogenesis and therapy. *J Invest Dermatol* 102:128–37	Immunology/infection
25	226	Millar SE (2002) Molecular mechanisms regulating hair follicle development. *J Invest Dermatol* 118:216–25	Appendages

Rank	Total times cited	Reference	Category
26	225	Norris P, Poston RN, Thomas DS *et al*. (1991) The expression of endothelial leukocyte adhesion molecule-1 (ELAM-1), intercellular adhesion molecule-1 (ICAM-1), and vascular cell adhesion molecule-1 (VCAM-1) in experimental cutaneous inflammation: a comparison of ultraviolet B erythema and delayed hypersensitivity. *J Invest Dermatol* 96:763–70	Photobiology
27	225	Grossman D, McNiff JM, Li F *et al*. (1999) Expression and targeting of the apoptosis inhibitor, survivin, in human melanoma. *J Invest Dermatol* 113:1076–81	Melanocytes/melanoma
28	224	Taylor RS, Ramirez RD, Ogoshi M *et al*. (1996) Detection of telomerase activity in malignant and nonmalignant skin conditions. *J Invest Dermatol* 106:759–65	Photobiology
29	222	Nanney LB, Stoscheck CM, Magid M *et al*. (1986) Altered [^{125}I]epidermal growth factor binding and receptor distribution in psoriasis. *J Invest Dermatol* 86:260–5	Clinical research
30	222	Stasiak PC, Purkis PE, Leigh IM *et al*. (1989) Keratin 19: predicted amino acid sequence and broad tissue distribution suggest it evolved from keratinocyte keratins. *J Invest Dermatol* 92:707–16	Keratinocytes/epidermis
31	219	Hashimoto T, Ogawa MM, Konohana A *et al*. (1990) Detection of pemphigus vulgaris and pemphigus foliaceus antigens by immunoblot analysis using different antigen sources. *J Invest Dermatol* 94:327–31	Immunology/infection
32	218	Austin LM, Ozawa M, Kikuchi T *et al*. (1999) The majority of epidermal T cells in psoriasis vulgaris lesions can produce type 1 cytokines, interferon-γ, interleukin-2, and tumor necrosis factor-α, defining TC1 (cytotoxic T lymphocyte) and TH1 effector populations: a type 1 differentiation bias is also measured in circulating blood T cells in psoriatic patients. *J Invest Dermatol* 113:752–9	Immunology/infection
33	213	Darr D, Fridovich I (1994) Free radicals in cutaneous biology. *J Invest Dermatol* 102:671–5	Photobiology
34	210	Dorschner RA, Pestonjamasp VK, Tamakuwala S *et al*. (2001) Cutaneous injury induces the release of cathelicidin anti-microbial peptides active against group A Streptococcus. *J Invest Dermatol* 117:91–7	Immunology/infection
35	206	Mischke D, Korge BP, Marenholz I *et al*. (1996) Genes encoding structural proteins of epidermal cornification and S100 calcium-binding proteins form a gene complex ("epidermal differentiation complex") on human chromosome 1q21. *J Invest Dermatol* 106:989–92	Keratinocytes/epidermis
36	204	Teunissen MB, Koomen CW, de Waal Malefyt R *et al*. (1998) Interleukin-17 and interferon-γ synergize in the enhancement of proinflammatory cytokine production by human keratinocytes. *J Invest Dermatol* 111:645–9	Immunology/infection
37	200	Madison KC, Swartzendruber DC, Wertz PW *et al*. (1987) Presence of intact intercellular lipid lamellae in the upper layers of the stratum corneum. *J Invest Dermatol* 88:714–8	Keratinocytes/epidermis
38	200	McCall CA, Cohen JJ (1991) Programmed cell death in terminally differentiating keratinocytes: role of endogenous endonuclease. *J Invest Dermatol* 97:111–4	Cell biology
39	198	Hou SY, Mitra AK, White SH *et al*. (1991) Membrane structures in normal and essential fatty acid-deficient stratum corneum: characterization by ruthenium tetroxide staining and x-ray diffraction. *J Invest Dermatol* 96:215–23	Keratinocytes/epidermis
40	196	Paus R, Müller-Röver S, Van Der Veen C *et al*. (1999) A comprehensive guide for the recognition and classification of distinct stages of hair follicle morphogenesis. *J Invest Dermatol* 113:523–32	Appendages
41	195	Schröder JM, Christophers E (1986) Identification of C5A des arg and an anionic neutrophil-activating peptide (ANAP) in psoriatic scales. *J Invest Dermatol* 87:53–8	Immunology/infection
42	195	Bos JD, Zonneveld I, Das PK *et al*. (1987) The skin immune system (SIS): distribution and immunophenotype of lymphocyte subpopulations in normal human skin. *J Invest Dermatol* 88:569–73	Immunology/infection
43	194	Igarashi A, Nashiro K, Kikuchi K *et al*. (1995) Significant correlation between connective tissue growth factor gene expression and skin sclerosis in tissue sections from patients with systemic sclerosis. *J Invest Dermatol* 105:280–4	Connective tissue
44	192	Madsen P, Rasmussen HH, Leffers H *et al*. (1991) Molecular cloning, occurrence, and expression of a novel partially secreted protein "psoriasin" that is highly up-regulated in psoriatic skin. *J Invest Dermatol* 97:701–12	Immunology/infection
45	191	Bertaux B, Hornebeck W, Eisen AZ *et al*. (1991) Growth stimulation of human keratinocytes by tissue inhibitor of metalloproteinases. *J Invest Dermatol* 97:679–85	Keratinocytes/epidermis
46	190	Wollenberg A, Wagner M, Günther S *et al*. (2002) Plasmacytoid dendritic cells: a new cutaneous dendritic cell subset with distinct role in inflammatory skin diseases. *J Invest Dermatol* 119:1096–102	Immunology/infection
47	188	Grøndahl-Hansen J, Lund LR *et al*. (1988) Urokinase- and tissue-type plasminogen activators in keratinocytes during wound reepithelialization *in vivo*. *J Invest Dermatol* 90:790–5	Vascular biology
48	187	Teunissen MB, Wormmeester J, Krieg SR *et al*. (1990) Human epidermal Langerhans cells undergo profound morphologic and phenotypical changes during *in vitro* culture. *J Invest Dermatol* 94:166–73	Immunology/infection
49	187	Shindo Y, Witt E, Packer L (1993) Antioxidant defense mechanisms in murine epidermis and dermis and their responses to ultraviolet light. *J Invest Dermatol* 100:260–5	Photobiology
50	187	Schwarz A, Bhardwaj R, Aragane Y *et al*. (1995) Ultraviolet-B-induced apoptosis of keratinocytes: evidence for partial involvement of tumor necrosis factor-α in the formation of sunburn cells. *J Invest Dermatol* 104:922–7	Photobiology

The vast majority of rankings of journals and rankings of scientists have been developed independently. Buoyssoua and Marchant (2010) argue that a consistent approach to both rankings would be preferable because there is striking interdependence between the quality of a journal and the quality of the work done by the scientists who publish in that journal. They used the impact factor to assess journal quality and combined this with two rankings for scientists using either the total number of citations or the total number of citations weighted by the inverse of the number of coauthors. They concluded that these metrics provide a consistent assessment of journal and scientist quality.

The growing influence of online access to journals on citation frequency

Evans (2008) addressed the issue of the rapid development of online access to journal articles and to citation frequency. He found that articles published more recently listed fewer and more recent citations, and he expressed concern that this trend may result in less comprehensive scholarly review. He emphasized that a major weakness of print library research is poor indexing of titles and authors in core journals, which resulted in the integration of science and scholarship. This has been disputed, and some believe that online access is actually having the opposite effect and encouraging more citations.

PageRank

PageRank is a link analysis algorithm named after one of the founders of Google, Larry Page. This proprietary system assigns a numerical weighting to each element of a hyperlinked set of documents, such as the World Wide Web, with the purpose of "measuring" its relative importance within the set. The goal as stated by Google is to permit more rapid searching of more sites more quickly, thereby providing more relevant results by applying a hierarchy of importance to results, allowing users to spend less time with irrelevant retrievals. Based on PageRank but distinct from it, Dellavalle *et al*. (2007) have developed a weighted algorithm for dermatology journals (Figure 2). This algorithm assigns greater weight to citations originating in more frequently cited journals. A high impact factor generally corresponds with a high PageRank weight (PRw). In some ways this resembles the Eigenfactor method described above in that it is a measure of the time spent reading a citation.

The explosion in online access to journals has led to a decline in print subscriptions, along with a rise in electronic subscriptions. Lo and Fisher (2011) made this point in the journal *Stroke*. Their analysis showed that in 2010 *Stroke* had an 11.9% decline in combined individual and institutional print subscriptions compared with 2009, whereas electronic subscriptions increased. Electronic access to *Stroke* articles increased by 21.4% in 2010, and the number of articles read/downloaded on mobile devices such as cell phones and portable electronic devices increased dramatically as well. These trends strongly suggest that online access will be the dominant gateway to scientific articles in the future. At present, *JID* is different in that no electronic-only subscriptions are offered and, aside from institutions (libraries) and industry, almost all *JID* print subscriptions originate from society memberships (either the SID or the European Society for Dermatological Research).

Google Scholar

Google Scholar is a freely accessible, Web-based search engine that indexes the full text of scholarly literature across an array of publishing formats and disciplines. It includes most peer-reviewed online journals of European and American publishers. It is similar in function to other freely available citation tools, including Scirus from Elsevier, CiteSeerX, and getCITED (Beel and Gipp, 2009). Google Scholar's statistical model is based on author names, bibliographic data, and article content to group articles probably written by the same author. Three metrics are available: the h-index; the i-10 index, which is the number of articles with at least 10 citations; and the total number of citations to articles. It is possible to enable automatic addition of newly published articles to one's profile. This would instruct the Google Scholar indexing system to update the author's profile as it discovers new articles. Authors can manually update profiles by adding missing articles, fixing bibliographic errors, and merging duplicate entries. Some have criticized the quality control of Google Scholar, and it is generally seen to be a browsing tool as opposed to more rigorous bibliometric tools such as the Web of Knowledge and Scopus. On the other hand, Google Scholar covers journals

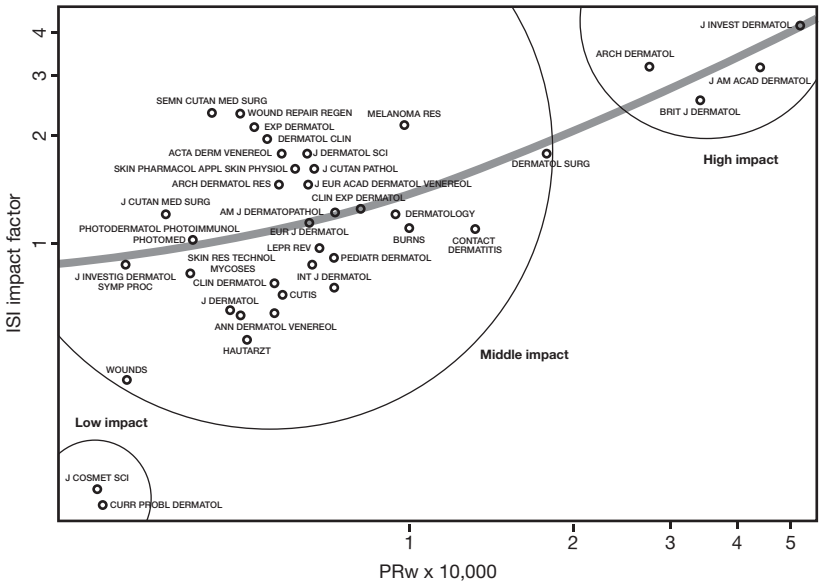

Figure 2. Weighted algorithm for dermatology journals. ISI, Institute for Scientific Information; JCR, Journal Citation Reports; PRw; PageRank weight.

not included in the JCR, such as the *Malaysian Journal of Medicine*, whose contents achieved international recognition based on the citations and impact score it received in Google Scholar (Sanni and Zainab, 2010).

In July 2011, Google began the launch of Google Scholar Citations, designed to provide a simple way for authors to compute citation metrics and track them over time. This feature is described at http://scholar.google.com/intl/en/scholar/citations.html. The service is currently limited to a small number of users, but interested individuals are directed to a page where they can register to be notified when the availability of Google Scholar Citations is expanded.

Six citation classics: then (1989) and now (2011)

In 1989 a special *JID* supplement was published to celebrate the 50th anniversary of the founding of the SID. David Norris, the *JID* editor at that time, chose for focused discussion six highly cited papers (he designated them "citation classics") that had been published in the *Journal*. The most highly cited paper was one by the late Albert Kligman (1966) in which he described an *in vivo* testing procedure that proved to be very useful in defining the risk of contact sensitization to chemicals in human populations. As pointed out by Norris, this highly predictive and reliable assay continues to be essential for the pharmaceutical and cosmetic industries.

The continuing importance of the original Harvard-based cooperative clinical trial for psoralen UVA (PUVA) photochemotherapy is aptly demonstrated by the second citation classic of 1989 (Stern and Lange, 1988), which is also one of the most highly cited papers between 1986 and 2010 (number 19; Table 1). This is also a clear example of the important work in clinical research that has been published in *JID* throughout its history (see "Photobiology" below).

The third citation classic of 1989 was a paper by Stanley Cohen describing the identification of an extract from murine submaxillary glands that could stimulate epidermal keratinization; the extract later became known as epidermal growth factor (Cohen and Elliott, 1963). Cohen and Rita-Levi Montalcini shared the Nobel Prize in 1986 for this seminal work, which paved the way to our current understanding of the importance of growth factors in cutaneous biology.

Norris (1989) cited the article by Birbeck *et al*. (1961) that described the characteristic cytoplasmic granules in epidermal Langerhans cells (LCs) now known as Birbeck granules. This paper, along with numerous others, presaged the growing recognition of the importance of these cells in cutaneous immunobiology.

The fifth citation classic identified in 1989 was that by Karasek (1966), in which the collagen gel method for culturing human keratinocytes was described. This was one of many publications that contributed enormously to the development of epidermal cell biology.

Finally, the sixth citation classic of 1989 focused on numerous review articles and their importance for *JID*. In particular, the paper by Beutner *et al*. (1968) was selected because of its in-depth discussion of the development of immunofluorescence techniques that revolutionized the clinical management of patients with autoimmune blistering diseases. As stated by Norris, "This is clearly one of the best examples of basic research changing clinical practice." Indeed, this paper is a classic forerunner of current recognition of the importance of translational research.

Borrowing David Norris's idea, we have selected six of the most highly cited papers published in *JID* over the past 25 years for brief discussion (see Table 1 for full citations for the 50 most cited articles). By far the most highly cited paper is that by Ades *et al*. (1992; 676 citations), which described the creation of an immortalized human microvascular endothelial cell line. In fact, this paper has 50% more citations than does the next most cited paper. This is due in part to the extraordinary utility of this cell line for studies addressing the pathogenesis of cutaneous inflammation. Studies conducted with these cells have advanced our understanding of the mechanisms of synthesis and secretion of proinflammatory cytokines, as well as surface adhesion molecules that facilitate binding of circulating leukocytes to bind to endothelial cells and also enhance binding of endothelial cells to matrix proteins.

Frazier *et al*. (1996; number 2, with 431 citations) described the identification of connective-tissue growth factor (CTGF) and showed it to be an important downstream mediator of transforming growth factor-β (TGF-β) effects on connective tissue cells by enhancing fibroblast proliferation and the synthesis of connective tissue matrix. The identification of CTGF, now also known as CCN2, has enhanced research into wound healing and fibrotic disorders, although as yet there are no effective treatments for tissue fibrosis. Because it seems clear that CCN2/CTGF plays an important role in the production and maintenance of tissue fibrosis, the development of therapeutic agents that inhibit this factor may prove effective in fibrosing skin disorders such as scleroderma.

Two papers by Rajadhyaksha *et al*. describing the development and refinement of *in vivo* confocal scanning laser microscopy of human skin are among the 50 most cited *JID* articles in this time period (1995; number 3, with 391 citations; and 1999; number 16, with 273 citations). These papers described the application of developing noninvasive imaging techniques to the diagnosis of skin disorders. These imaging tools may permit the assessment of skin cancer morphology in real time and ultimately compete with histopathological methods, particularly for pigmented lesions. More recent studies designed to evaluate the diagnostic accuracy of this modality for pigmented lesions suggest that it may be superior to dermoscopy. In the future, confocal laser microscopy could provide an additional tool for the more accurate noninvasive diagnosis of melanocytic and other skin lesions.

Tschachler *et al*. (1987; number 4, with 381 citations) showed that LCs are infected by HTLV-III/LAV and that infection of LCs with this retrovirus may have deleterious consequences for the immunologic functions of this cell system, which may thus contribute to both the acquisition of immunodeficiency and the infectious and neoplastic complications of AIDS (see below).

Imokawa *et al*. (1991; number 5, with 378 citations) showed that the stratum corneum of patients with atopic dermatitis manifests a deficiency in ceramides and drew attention to the fact that compromised skin barrier function may contribute to the pathogenesis of skin diseases. The skin barrier is important for minimizing transepidermal water loss, as well as for minimizing physical and chemical insults from the environment. This consists of protein-enriched corneocytes and lipid-enriched intercellular domains. During epidermal differentiation, lipids are synthesized in keratinocytes and then help to form the cornified envelope. Ceramides are then covalently bound to the cornified envelope that cross-links with filaggrin. Indeed, several studies have revealed that approximately 25–50% of patients with atopic dermatitis manifest filaggrin mutations as a predisposing factor.

Smith *et al*. (1986; number 6, with 361 citations) showed that 1α,25-dihydroxy D_3 is a potent inhibitor of cultured human epidermal keratinocyte proliferation and enhances terminal differentiation of these cells.

These studies helped pave the way for the development of topical derivatives of vitamin D for the treatment of psoriasis. These agents act to inhibit keratinocyte proliferation and promote normalization of epidermal differentiation, thereby addressing two of the cardinal manifestations of the skin disease psoriasis.

In the sections below, an effort is made to provide a perspective regarding the manner in which highly cited articles in the journal have influenced the fields of immunology/infection and photobiology.

Immunology/infection

Approximately one-third of the top 50 articles published in *JID* between 1986 and 2010 are in the "immunology/infection" subcategory. In the past 25 years, a major advance in immunology has been the realization that there are two distinct components to the immune system: the innate and the adaptive immune response. The innate immune response is genetically preprogrammed and facilitates the rapid detection of biochemical types of ligands that characterize classes of microbial pathogens as well as the host. In this manner, the innate immune response provides the first line of defense against danger. The adaptive immune response involves T and B cells, with rearranged receptors; it involves memory and is therefore the target of vaccines.

The epidermis is not only a physical barrier to the outside environment but also active immunologically, with keratinocytes secreting proteins that are immunomodulatory and/or antimicrobial. Schröder and Christophers (1986; number 41, with 195 citations) identified two peptides in psoriatic scales—C5a des-Arg and anionic neutrophil-activating peptide (also known as IL-8)—which stimulated neutrophil functional activities including chemotaxis and generation of superoxide free radicals. Dorschner *et al*. (2001; number 34, with 210 citations) from the Gallo laboratory showed that the antimicrobial peptide cathelicidin is upregulated and released by keratinocytes in response to injury. Furthermore, they found that cathelicidin was processed into an active C-terminal form with antimicrobial activity against group A Streptococcus. Madsen *et al*. (1991; number 44 with 192 citations) cloned psoriasin, an S100 family member known as S100A7. Psoriasin is upregulated in psoriatic epidermis and has antimicrobial and inflammatory properties. Together, these studies have helped establish the concept that keratinocytes are part of the innate immune system, contributing to host defense. These studies were complemented by the report by Norris *et al*. (1991; number 26, with 225 citations), which analyzed the expression of adhesion molecules on human dermal vasculature induced by intradermal injection of an immunogenic stimulus (purified protein derivative) and provided insights into pathways by which inflammatory cells enter skin.

In the past quarter century, dendritic cells (DCs) have been recognized as a key cell component of the innate immune system, with the capacity to instruct and modulate the adaptive T-cell response. Three highly cited papers deal with Langerhans cells (LCs), which are DCs that are resident in the epidermis. As previously stated, Tschachler *et al*. (1987; number 4, with 381 citations) demonstrated that LCs are a target for HTLV-III infection. LCs were the only target for HTLV-III infection in the epidermis, and infection resulted in a disruption of cell morphology. Romani *et al*. (1989; number 7, with 351 citations) found that human LCs could be differentiated *in vitro*, resembling lymphoid DCs and characterized by more potent T-cell stimulatory activity. They concluded that human LCs represent immature precursors of lymphoid DCs in skin-draining lymph nodes. The changes involved in the differentiation of LCs were further detailed in elegant studies by Teunissen *et al*. (1990; number 48 with 187 citations). Another DC subtype, the plasmacytoid DC, has a major role in the induction of type I interferons. Wollenberg *et al*. (2002; number 46, with 190 citations) surveyed the distribution of plasmacytoid DCs in skin. They found increased numbers of plasmacytoid DCs in the dermis of psoriasis and lupus erythematosus lesions, as compared with atopic dermatitis and normal skin. These studies, taken together, provide insight into the role of DC populations in skin, in both health and disease.

Two important advances in T-cell biology—first the definition of CD4 and CD8 T-cell subsets using monoclonal antibodies and then the identification of functional T-cell subsets based on the pattern of secreted cytokines—have led to advances in immunodermatology. Bos *et al*. (1987; number 42, with 195 citations) identified the $CD4^+$ and $CD8^+$ resident T-cell subpopulations in normal skin. van der Heijden *et al*. (1991; number 9, with 323 citations) first showed the high frequency of IL-4-producing T cells in lesional skin of patients with atopic dermatitis. Uyemura *et al*. (1993; number 13, with 284 citations) described the presence of a Th1-like cytokine pattern in skin lesions of patients with psoriasis, typified by the presence of IL-2, IFN-γ, and tumor necrosis factor-α (TNF-α). These findings were extended by Schlaak *et al*. (1994; number 18, with 247 citations) and Austin *et al*. (1999; number 32, with 218 citations). Teunissen *et al*.

(1998; number 36, with 204 citations) identified a synergistic role for IL-17 and IFN-γ in enhancing proinflammatory cytokine production by human keratinocytes. Cooper (1994; number 24, with 230 citations) reviewed the pathogenesis of atopic dermatitis and provided clear examples of how advances in understanding the immunology of skin diseases have led to the development of novel immunotherapeutic strategies.

Identification of the antigens recognized by the adaptive T-cell and B-cell response is pivotal to the understanding of the pathogenesis of many skin diseases, but in particular has proven to be key to understanding the immunobiology of the autoimmune vesiculobullous diseases. Hashimoto *et al*. (1990; number 31, with 219 citations) reported the relationship between the antigens involved in pemphigus vulgaris and pemphigus foliaceus using sera from patients and immunoblot analysis. Giudice *et al*. (1992; number 8, with 339 citations) reported the cloning of the bullous pemphigoid antigen BP180. These studies provided insight into the important structural role of these antigens in skin biology and how, serving as targets of autoantibody responses, they contribute to the pathogenesis of autoimmune blistering diseases.

Photobiology

Approximately 12% of articles published in *JID* over the past 25 years are found in the subcategory of photobiology. Harnessing the photosensitizing properties of the naturally occurring psoralens was a major accomplishment and led to the development of PUVA photochemotherapy for the treatment of psoriasis and other dermatologic diseases in the 1970s. This breakthrough revolutionized the outpatient management of patients with psoriasis and cutaneous T-cell lymphoma. On the other hand, because of the knowledge that PUVA causes DNA damage, from the outset there has been concern regarding the potential skin carcinogenicity of this modality. Indeed, the careful design of the original cooperative clinical trials of PUVA therapy has provided a unique resource permitting close follow-up of these 1,380 patients and monitoring them for treatment-related toxic effects. The publication by Stern and Lange (1988; number 19, with 246 citations) showed that patients treated more than 260 times had an 11-fold increased risk of developing squamous cell carcinoma compared with patents receiving 160 or fewer treatments. There was also a smaller but significantly increased risk of developing basal cell carcinoma.

Recognition of the immunosuppressive effects of solar UVB radiation has provided important insights into the pathogenesis of nonmelanoma skin cancer. The induction of allergic contact dermatitis to potent skin allergens such as dinitrochlorobenzene (DNCB) is diminished/abrogated by prior exposure of the application site to low doses of UVB, and antigen-specific tolerance also ensues. Intradermal injection of subinflammatory doses of cytokines such as TNF-α evokes the same response. UVB induces *cis*-urocanic acid that also augments the release of TNF-α and attenuates upregulation of the proinflammatory cytokines IL-6 and IL-8. UVB immunosuppression was initially attributed to the induction of hapten-specific suppressor T cells, now known as regulatory T cells. Regulatory T cells reside in draining lymph nodes of UVB-irradiated skin, where they induce expression of the immunosuppressive cytokine IL-10.

The paper by Yoshikawa *et al*. (1990; number 10, with 304 citations) was among the first to show that human subjects exhibit heterogeneous susceptibility to the induction of contact hypersensitivity to DNCB applied to UVB-irradiated skin. Approximately 60% of normal human volunteers could be sensitized by application of the allergen to skin sites previously exposed to low-dose UVB, whereas approximately 40% failed to do so. Similar studies conducted in patients with skin cancer showed that more than 90% of these individuals could not be sensitized. It was then shown that a second application of DNCB to non-UVB-irradiated skin of the normal volunteers resulted in the induction of contact hypersensitivity in 100% of these individuals but in only half of the skin cancer patients, suggesting that they had been rendered immunologically tolerant to DNCB. Hapten specificity was further verified by showing that tolerant individuals responded to the unrelated hapten diphencyprone. It was concluded that exposure of human skin to acute, low-dose UVB induces specific immune tolerance and that immunogenetic factors responsible for these effects may influence the risk of sun-induced skin cancer. Subsequent studies by others showed that the immunosuppressive effects of UVB relate to the administered dose, the induction of $CD1a^{-}$ DR^{+} epidermal macrophages, and the depletion of LCs. The importance of UVB-induced DNA damage in mediating immunosuppression has been confirmed by studies showing that topical application of the DNA repair enzyme photolyase abrogates the damage.

Epidermal cells possess a variety of defense mechanisms to ameliorate and repair UV-induced DNA damage. One such defense mechanism is the induction of apoptotic keratinocytes, also known as sunburn cells. Elimination of these cells may diminish the risk of skin cancer. Another defense mechanism is the tumor suppressor p53. UVB-induced p53 mutations can abrogate its repair function and induce upregulation of antiapoptotic pathways as well as the downregulation of proapoptotic pathways, thereby increasing the risk of skin cancer. Schwarz *et al*. (1995; number 50, with 187 citations) showed that UVB irradiation augments apoptosis of human keratinocytes and that this is associated with augmented release of TNF-α. Inhibition of the function of TNF-α using a polyclonal antibody directed against human TNF-α rescued cells from apoptosis—but only partially. Similar studies in mice showed the same result. Thus, TNF-α is important for the apoptotic sunburn-cell response, but other factors must be involved. Subsequent studies have shown the importance of the TNF-α-related apoptosis-inducing ligand (TRAIL) in tumor surveillance because animals deficient in this ligand have heightened susceptibility to skin carcinogenesis.

When human skin is exposed to erythema doses of UVB, there is upregulation of immunosuppressive IL-10. The source of this cytokine is believed to be $CD11b^{+}$ $HLA\text{-}DR^{+}$ macrophages, and

IL-10 inhibits cell-mediated immune responses. Also, $CD11b^+$ $HLA\text{-}DR^+$ neutrophils infiltrate UVB-irradiated skin, elaborate IL-10, and contribute to the immunosuppressive microenvironment created by UVB skin exposure.

Norris *et al*. (1991; number 26, with 225 citations) studied mediators of cutaneous inflammation induced by two minimal erythema doses of UVB administered to human volunteers, and immunohistochemical studies were then performed on sequential skin biopsies obtained 1, 6, 24, 72, and 168 hours thereafter. UVB-irradiated skin showed upregulation of endothelial leukocyte adhesion molecule-1 on vascular endothelium and infiltration of polymorphonuclear leukocytes within 6 hours that peaked at 24 hours. These studies showed that UVB enhances leukocyte adhesion to endothelial cells, which could explain the infiltration of neutrophils seen in UVB-irradiated human skin.

Reactive oxygen species (ROS) are thought to be involved in carcinogenesis, aging, and various inflammatory disorders of the skin. Because reliable direct measurement of ROS in skin remains challenging, the effect of pro-oxidant stress on antioxidant systems is used as a surrogate for such measurements. Shindo *et al*. (1993; number 49, with 187 citations) showed that murine skin possesses both nonenzymatic and enzymatic antioxidants. Nonenzymatic agents include lipophilic α-tocopherol, ubiquinol-9 and ubiquinone 9, hydrophilic ascorbic acid, dehydroascorbic acid, and glutathione. Enzymatic antioxidants include superoxide dismutase, catalase, glutathione peroxidase, and glutathione reductase. In general, with the exception of superoxide dismutase, all of these were shown to have higher expression in the epidermis than in the dermis of these animals. The mice were then exposed to erythema doses of solar-simulating radiation, and virtually all of these antioxidants were decreased, with the epidermis much more affected than the dermis. In recent years, methodologies such as magnetic resonance imaging, electron paramagnetic resonance, laser Doppler flowmetry, and time domain reflectometry have been developed that will permit more accurate direct measurement of ROS in skin.

The importance of ROS in cutaneous biology was reviewed by Darr and Fridovich (1994; number 33, with 213 citations). Oxygen in its ground state contains two unpaired electrons, and this favors its reduction along a univalent pathway. Reactive intermediates along this pathway include superoxide radicals, hydrogen peroxide, and the hydroxyl radical. The skin is uniquely susceptible to oxidant injury because of its double-barreled exposure to both environmental oxygen and oxygen perfused into the skin from the bloodstream. In addition, photons in solar radiation can be absorbed by skin constituents and generate excited-state molecules that can transfer their absorbed energy to oxygen, thereby generating reactive singlets and triplets that can cause injury. Each of the reactive species can be attenuated by multiple enzymatic antioxidants. Superoxide is minimized by superoxide dismutases and hydrogen peroxide by catalase and glutathione peroxidases. Nonenzymatic α-tocopherol can protect lipid-rich membranes against chain-propagating oxidant injury, and carotenoids can neutralize singlet oxygen. A continuing challenge to the present day is the availability of effective antioxidants that can consistently and safely attenuate the tissue-damaging effects of ROS.

Concluding remarks

JID has been a powerful contributor to the growth of the scientific base of cutaneous biology and dermatology over the past 25 years. Developing bibliometric systems document clearly the *Journal*'s increasing impact on the field and its recognition as an excellent resource by high-quality publications across many other disciplines in the biomedical sciences. Although several factors can be invoked to explain these impressive accomplishments, the role of the outstanding editors of the *Journal* is perhaps the most important of all (Supplementary Table S6 online). *JID* has benefited from the remarkable contributions of these leaders and is positioned well to build on these successes over the next 25 years.

CONFLICT OF INTEREST

The authors state no conflict of interest.

SUPPLEMENTARY MATERIAL

Supplementary material is linked to the online version of the paper at http://www.nature.com/jid

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