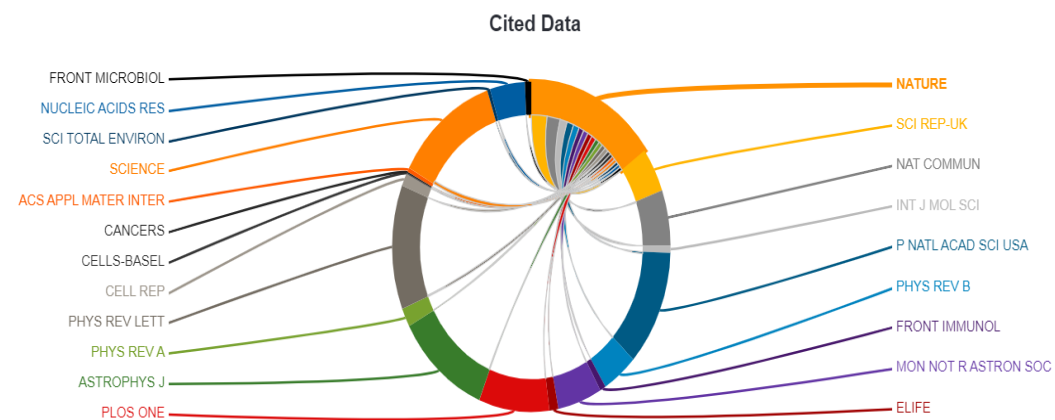
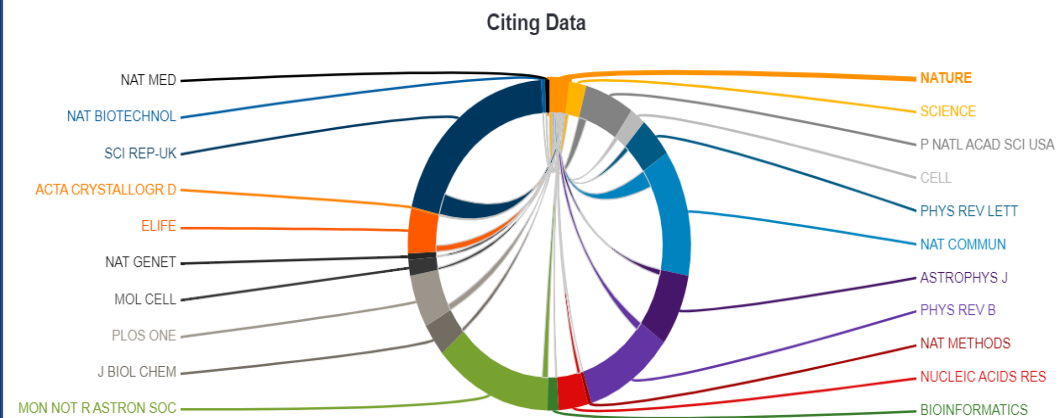


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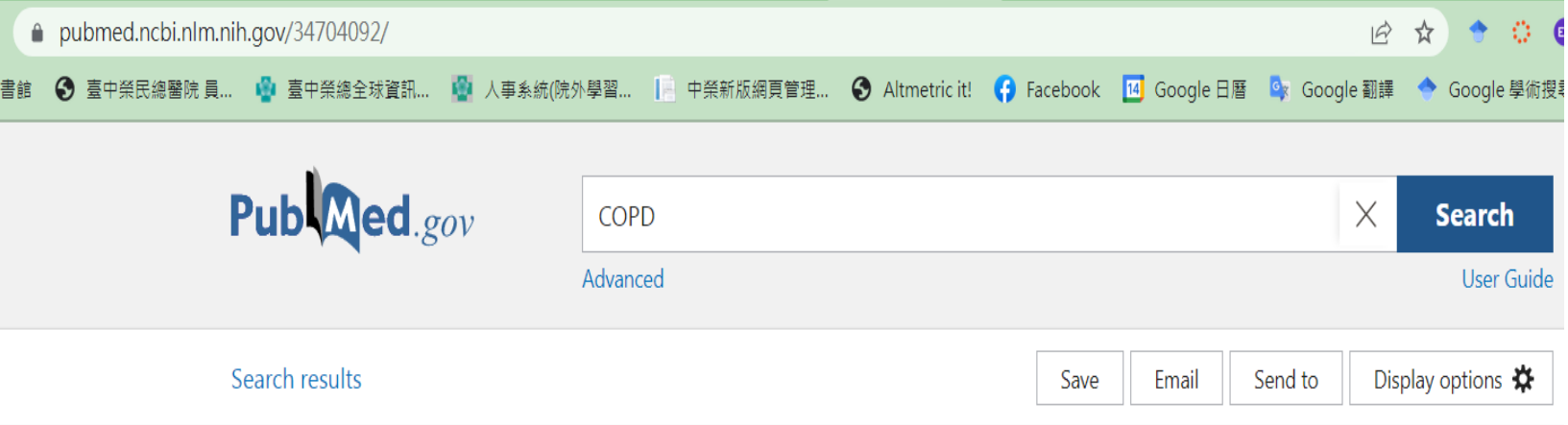
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Modeling and Readmission: Visual Analytical Approach

Bhavnani S.K., Zhang W., Visweswaran S., Raji M., Kuo Y.-F. View author addresses

Abstract

Background A primary goal of precision medicine is to identify patient subgroups and infer their underlying disease processes, with the aim of designing targeted interventions. However, few methods automatically identify both patient subgroups and their co-occurring characteristics simultaneously, measure their significance, and visualize the results. Such methods could enhance the interpretability of patient subgroups, and inform the design of classification and predictive models. Objectives To analyze patient subgroups in hospital readmitted patients using a three-step modeling approach. (1) Visual analytical modeling to automatically identify patient subgroups and their co-occurring comorbidities, and determine their statistical significance and clinical interpretability. (2) Classification modeling to classify patients into subgroups and measure its accuracy. (3) Prediction modeling to

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Summary

There is evidence that Janus kinase (JAK)–signal transducer and activator of transcription (STAT) signaling plays a role in the pathogenesis of sarcoidosis. We treated a patient with cutaneous sarcoidosis with the JAK inhibitor tofacitinib; the patient had not previously had a response to medications and had not received systemic glucocorticoids. This treatment resulted in clinical and histologic remission of her skin disease. Sequencing of RNA and immunohistochemical examination of skin-lesion samples obtained from the patient before and during therapy and immunohistochemical testing of lesion samples obtained from other patients with cutaneous sarcoidosis support a role for JAK-STAT signaling in cutaneous sarcoidosis. (Funded by the Ranjini and Ajay Poddar Resource Fund for Dermatologic Diseases Research and others.)

Sarcoidosis is an inflammatory disease that is associated with the formation of noncaseating granulomas in one or multiple organ systems. Skin involvement is seen in approximately 25% of patients with sarcoidosis.¹ Systemic glucocorticoids are the initial treatment for sarcoidosis with systemic involvement and may be used for the treatment of cutaneous sarcoidosis.^{2,3} Granulomas in patients with sarcoidosis are composed primarily of macrophages and T cells.⁴ The activation of macrophages in granulomas is considered to be dependent on helper T cells and mediated in part by interferon- γ .^{5,6} Interferon- γ activates the Janus kinase (JAK)–signal transducer and activator of transcription (STAT) signaling pathway, resulting in the up-regulation of STAT1 transcriptional targets. Several studies have shown that JAK-STAT pathway activation signatures, especially STAT1-dependent transcripts, are characteristic of the transcriptome in both peripheral-blood mononuclear cells and other tissues in patients with sarcoidosis.^{7–10}

We treated a patient who had refractory cutaneous sarcoidosis with the oral JAK inhibitor tofacitinib, which resulted in clinical and histologic remission of skin lesions. We also performed molecular characterization of the response using global gene-expression profiling

From the Departments of Dermatology (W.D., D.T., A.G., B.K.), Immunobiology (W.D.), and Pathology (A.G.) and the Department of Internal Medicine, Section of Pulmonary, Critical Care, and Sleep Medicine (N.E.), Yale School of Medicine, New Haven, CT. Address reprint requests to Dr. King at the Department of Dermatology, Yale School of Medicine, 333 Cedar St., LCI 501, P.O. Box 208059, New Haven, CT 06520, or at brett.king@yale.edu.

N Engl J Med 2018;379:2540–6.
DOI: 10.1056/NEJMoa1805958

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BRIEF REPORT

Tofacitinib Treatment and Molecular Analysis of Cutaneous Sarcoidosis

William Damsky, M.D., Ph.D., Durga Thakral, M.Phil., M.S., Nkiruka Emeagwali, M.D., Ph.D., Anjela Galan, M.D., and Brett King, M.D., Ph.D.

SUMMARY

There is evidence that Janus kinase (JAK)–signal transducer and activator of transcription (STAT) signaling plays a role in the pathogenesis of sarcoidosis. We treated a patient with cutaneous sarcoidosis with the JAK inhibitor tofacitinib; the patient had not previously had a response to medications and had not received systemic glucocorticoids. This treatment resulted in clinical and histologic remission of her skin disease. Sequencing of RNA and immunohistochemical examination of skin-lesion samples obtained from the patient before and during therapy and immunohistochemical testing of lesion samples obtained from other patients with cutaneous sarcoidosis support a role for JAK-STAT signaling in cutaneous sarcoidosis. (Funded by the Ranjini and Ajay Poddar Resource Fund for Dermatologic Diseases Research and others.)

SARCOIDOSIS IS AN INFLAMMATORY DISEASE THAT IS ASSOCIATED WITH the formation of noncaseating granulomas in one or multiple organ systems. Skin involvement is seen in approximately 25% of patients with sarcoidosis.¹ Systemic glucocorticoids are the initial treatment for sarcoidosis with systemic involvement and may be used for the treatment of cutaneous sarcoidosis.^{2,3} Granulomas in patients with sarcoidosis are composed primarily of macrophages and T cells.⁴ The activation of macrophages in granulomas is considered to be dependent on helper T cells and mediated in part by interferon- γ .^{5,6} Interferon- γ activates the Janus kinase (JAK)–signal transducer and activator of transcription (STAT) signaling pathway, resulting in the up-regulation of STAT1 transcriptional targets. Several studies have shown that JAK-STAT pathway activation signatures, especially STAT1-dependent transcripts, are characteristic of the transcriptome in both peripheral-blood mononuclear cells and other tissues in patients with sarcoidosis.^{7–10}

We treated a patient who had refractory cutaneous sarcoidosis with the oral JAK inhibitor tofacitinib, which resulted in clinical and histologic remission of skin lesions. We also performed molecular characterization of the response using global gene-expression profiling of skin-lesion samples obtained from this patient, and we analyzed a series of biopsy samples obtained from other patients with cutaneous sarcoidosis.

METHODS

CLINICAL DATA AND SPECIMEN COLLECTION

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2540

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