Joint position statement from the National Psoriasis Foundation Medical Board and the International Psoriasis Council on routine testing for latent tuberculosis infection prior to and during treatment of psoriasis patients with interleukin 17 or interleukin 23 inhibitors

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Background: Although testing for latent tuberculosis (TB) infection has been standard practice for psoriasis patients being treated with interleukin (IL) 17 or IL-23 inhibitors, evidence for this practice is weak.

Objectives: To review evidence on safety of IL-17 and IL-23 inhibitors in the setting of latent TB infection and to provide a new Joint Position Statement on this topic.

Methods: Experts from the National Psoriasis Foundation and the International Psoriasis Council reviewed evidence regarding progression of latent TB infection to active disease in psoriasis patients receiving IL-17 or IL-23 blockers. A Joint Position Statement was formulated and approved to provide updated guidance to clinicians.

Results: 87.5% of the members from the National Psoriasis Foundation Medical Board and International Psoriasis Council approved a new Joint Position Statement regarding psoriasis patients being treated with IL-17 or IL-23 inhibitors, stating that testing for latent TB infection is not required.

Limitations: This position statement allows for exceptions where continued testing for late nt TB infection could be considered, including for patients on concomitant immunosuppressive therapy and for those living in TB endemic areas.

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Conclusion: Psoriasis experts reached consensus that routine testing for latent TB infection is not required in psoriasis patients being treated with IL-17 or IL-23 inhibitors. (J Am Acad Dermatol 2026;94:802-9.)

Key words: biologics; IL-17 inhibitors; IL-23 inhibitors; psoriasis; safety; tuberculosis.

INTRODUCTION

Testing for latent tuberculosis (TB) infection has been standard clinical practice prior to initiation of biologic therapy for patients with psoriasis.[1] Historically, this practice was largely driven by experience with tumor necrosis factor (TNF) inhibitors. TNF, a prototypic proinflammatory cytokine with broad roles in both health and disease, is involved in granuloma formation and function, which helps contain and control *Mycobacterium tuberculosis* infection within tissue.[2,3] Treatment of psoriasis and other immune-mediated inflammatory diseases (IMIDs) with TNF inhibitors can inhibit granuloma formation or function and can promote progression of latent TB infection to active disease.[4-7] Thus, testing for latent TB infection prior to and during treatment with TNF inhibitors has been an integral and important part of clinical trial designs and clinical practice for decades.

Testing for latent TB infection has also been an integral part of clinical trials and clinical practice prior to and during treatment with other drug classes that can potentially lead to progression of latent TB infection to active disease, including ustekinumab and tyrosine kinase 2 inhibitors, drugs that inhibit both interleukin (IL) 12 and IL-23 actions, and Janus kinase inhibitors, which have broad immunomodulatory effects on a number of cytokines, including interferon-γ. IL-12 and interferon-γ orchestrate Th1 immune responses, which are important for controlling TB infection.[2,3] Two other classes of biologics used to treat psoriasis, IL-17 inhibitors and IL-23 inhibitors, also have requirements for latent TB infection testing. These testing recommendations were primarily based on early mouse studies[8-10] and on historic precedent with older biologic therapies rather than on direct evidence of risk. Currently, both the biologic basis for this testing and the evidence for progression of latent TB infection to active disease in clinical settings, in both trials and in real-world practice, are limited and weak for both IL-17 and IL-23 inhibitors.[11]

CAPSULE SUMMARY

- Clinical data suggest that progression of latent tuberculosis infection to active disease during psoriasis treatment with interleukin-17 or interleukin-23 inhibitors is rare.
- Experts formulated a new Joint Position Statement, highlighting that testing for latent tuberculosis infection is not required in psoriasis patients being treated with interleukin-17 or interleukin-23 inhibitors.

Recently, international experts have extensively reviewed the roles of IL-17 and IL-23 in TB pathogenesis as well as current clinical evidence for the progression of latent TB infection to active disease during treatment with either IL-17 or IL-23 inhibitors; these authorities further suggested several modifications to existing treatment guidelines for these 2 classes of biologics.[11,12] Inspired by these efforts, here, the National Psoriasis Foundation (NPF) Medical Board and the International Psoriasis Council (IPC) review clinical evidence supporting the safety of IL-17 and IL-23 inhibitors regarding the progression of latent TB infection to active disease. A consensus proposal is presented that urges modifications to existing clinical and regulatory guidelines for the routine testing requirement for latent TB infection prior to and during treatment of psoriasis patients with these 2 newer classes of biologics. Implementing this evidence-based change in future clinical trial designs and clinical practice would avoid unnecessary medical costs, reduce avoidable regulatory burden, alleviate concerns for false positive results, and expedite treatment of patients with psoriasis.

METHODS

An evidence-based review was performed examining scientific literature on progression of latent TB to active disease in patients with psoriasis being treated with IL-17 or IL-23 inhibitors. Literature from relevant preclinical studies, clinical trials, and real-world studies were examined and summarized.

To further investigate the possible role of IL-17 and IL-23 inhibitors in the progression of latent TB infection to active disease, a structured search of the US Food and Drug Administration Adverse Event Reporting System (FAERS) was conducted as a signal detection exercise

Abbreviations used:

FAERS:	Food and Drug Administration Adverse Event Reporting System
IL:	interleukin
IMID:	immune-mediated inflammatory disease
IPC:	International Psoriasis Council
M tuberculosis:	*Mycobacterium tuberculosis*
NPF:	National Psoriasis Foundation
TB:	tuberculosis
TNF:	tumor necrosis factor

(Table I). The FAERS database was queried from January 2004 (when TNF inhibitors were first approved for psoriasis in the United States) through May 31, 2025 and was restricted to reports submitted in the United States. Searches used MedDRA Preferred Terms, including tuberculosis, disseminated tuberculosis, miliary tuberculosis, tuberculous meningitis, tuberculous peritonitis, tuberculous hepatitis, tuberculous nephritis, and tuberculous osteomyelitis. Product names included IL-17 inhibitors (secukinumab, ixekizumab, brodalumab, and bimekizumab), IL-23 inhibitors (guselkumab, tildrakizumab, and risankizumab), and, for comparison, TNF inhibitors (adalimumab, infliximab, etanercept, and certolizumab). The analysis focused on extra-pulmonary, internal-organ TB as a specific proxy for immunosuppression-associated disease, since TB involvement of internal organs is more likely to reflect impaired host immunity.

A Joint Position Statement was formulated by authors from the NPF Medical Board and the IPC. This consensus statement was then presented to all members of the NPF Medical Board and to all councilors of the IPC for voting, asking each person if they agreed or not to the Joint Position Statement with a simple "yes" or "no" answer.

RESULTS

IL-17 inhibitors

IL-17A and IL-17F are critical cytokines involved in the pathogenesis of psoriasis.[13] There are now 4 approved therapies for psoriasis that inhibit function of IL-17 family members: secukinumab and ixekizumab (which both target IL-17A), brodalumab (which targets IL-17RA, a component of the IL-17 receptor), and bimekizumab (which targets IL-17A and IL-17F). Although the exact role for IL-17 family members in the immunopathogenesis of *Mycobacterium tuberculosis* (*M tuberculosis*) infection is debated,[14,15] in mice, selective inhibition of IL-17A, IL-17F, IL-17RA, or IL-22 does not impair host control of *M tuberculosis*, while TNF-deficient mice rapidly succumb to infection.[16,17] Furthermore, using a human microgranuloma model *in vitro*, secukinumab-induced inhibition of IL-17A did not reverse *M tuberculosis* dormancy, whereas ustekinumab-induced inhibition of IL-12/IL-23 and adalimumab-induced inhibition of TNF promoted *M tuberculosis* reactivation.[18,19]

Table I. FAERS reports of extra-pulmonary internal organ TB in patients with psoriasis treated with IL-17, IL-23, or TNF inhibitors in the United States

Exposure	Total no. of adverse event reports (any cause)	No. of extra-pulmonary internal organ TB reports
IL-17 inhibitors		
Secukinumab	43,725	0
Ixekizumab	12,078	0
Brodalumab	1913	0
Bimekizumab	286	0
IL-23 inhibitors		
Guselkumab	7630	0
Tildrakizumab	513	0
Risankizumab	30,206	0
TNF inhibitors		
Etanercept	67,566	7
Infliximab	1765	4
Adalimumab	66,359	15
Certolizumab	4135	0

FAERS, Food and Drug Administration Adverse Event Reporting System; *IL*, interleukin; *TB*, tuberculosis; *TNF*, tumor necrosis factor.

Considerable clinical trial data and postmarketing real-world evidence support the concept that IL-17 inhibitors do not increase the risk of progression of latent TB infection to active disease in humans with psoriasis and other IMIDs.[20-32] For clinical trials, patients with latent TB infection were typically eligible for enrollment into IL-17 inhibitor studies, provided that they received preventive TB therapy according to local guidelines before or at the beginning of the trials. No cases of active TB were observed in trials among such individuals, which numbered in the hundreds.[11] Although IL-17 inhibitors are safe in patients with properly treated latent TB infection, administration of prior TB preventive therapy introduces a confounding factor in assessing the specific safety of IL-17 inhibitors in the setting of TB infection.

Some particular patient scenarios are notable from the clinical trial literature on IL-17 inhibitors and TB infection. Six patients receiving ixekizumab tested negative for latent TB infection before entering clinical studies, but later tested positive during the trials. These patients did not receive treatment for latent TB infection, continued receiving ixekizumab, and did not develop active TB.[26]

A considerable body of real-world evidence also exists on the safety of IL-17 inhibitors in patients with latent TB infection, even in the absence of TB

preventive therapy.[11,32-44] Details from these reports reveal hundreds of patients with IMIDs who were treated with IL-17 inhibitors and did not receive TB preventive therapy for a variety of reasons, including concomitant liver disease, contraindications due to existing comorbidities or potential drug-drug interactions, and patient preference. Only 1 patient in these reports exhibited signs of progression of latent TB infection to active disease,[11] which falls well within the 5% to 10% lifetime risk for progression of latent TB infection to active disease.[1] Although TNF inhibitor-associated progression of latent TB infection to active disease is typically observed within 12 months of starting treatment,[45] atypical extrapulmonary TB was observed in this patient 14 months after starting an IL-17 inhibitor. The progression to active TB infection may have been independent of biologic treatment.[11] No progression of latent TB infection to active disease was reported in all other cases, underscoring the relative safety of IL-17 inhibitor treatment for psoriasis and other IMIDs in the setting of latent TB infection.

IL-23 inhibitors

IL-23 plays a critical role in the pathogenesis of psoriasis.[46] IL-23 consists of 2 protein subunits, p40 and p19; the former subunit is shared with IL-12, whereas p19 is specific for IL-23. IL-23 inhibitors that block p19 are important treatment options for psoriasis patients; they include guselkumab, tildrakizumab, and risankizumab. Similar to IL-17A, IL-23 has been implicated in protective responses against *M tuberculosis* infection. However, IL-12–mediated Th1 responses are dominant in controlling TB infection, with IL-23/Th17 responses playing a more protective role in the setting where Th1 responses are compromised.[47] Individuals genetically deficient in *IL-12p40* or *IL-23R* are susceptible to TB infection,[48] although current clinical data do not indicate an increased risk of progression of latent TB infection to active disease with therapeutic inhibition of IL-23. This discrepancy likely reflects differences between complete genetic ablation and the partial, reversible, and context-dependent inhibition achieved with pharmacologic agents.

Many reviews have highlighted the safety of IL-23 inhibitors in treating psoriasis and other IMIDs, including in the setting of latent TB infection.[21,49,50] Extensive data from clinical trials have been pooled and analyzed on this issue.[51-53] Analyses that included 4399 patients treated with guselkumab with an exposure of 10,787 patient-years,[51] 1413 patients treated with tildrakizumab,[52] and 2072 patients treated with risankizumab with an exposure of 7927 patient-years[53] revealed no cases of progression of latent TB infection to active disease in clinical trials using IL-23 inhibitors. Similarly, no cases of progression of latent TB infection to active disease have been reported in clinical trials using IL-23 inhibitors to treat other IMIDs, such as psoriatic arthritis, ulcerative colitis, and Crohn's disease.[51,54-58] As discussed above with IL-17 inhibitor trials, patients with latent TB infection were typically included in these IL-23 inhibitor studies only if they had previously received TB preventive therapy, which introduces confounding bias when fully evaluating the safety of these drugs in the setting of latent TB infection.

Some patient cohorts in IL-23 inhibitor clinical trials are noteworthy. For example, a subset of 31 patients in the IMMhance risankizumab trial for psoriasis tested positive for latent TB infection during screening, but did not receive TB preventive therapy prior to study initiation; these individuals were exposed to risankizumab for 55 weeks and none reported progression of latent TB infection to active disease.[59] In a pooled analysis from 2 guselkumab studies, a subset of 7 patients began TB preventive therapy concurrently with the initiation of guselkumab therapy and 5 additional patients began TB preventive therapy after starting guselkumab; none of these 12 patients experienced progression of latent TB infection to active disease.[60]

Real-world data on psoriasis patients being treated with IL-23 inhibitors provide additional support for the safety of these drugs in the setting of latent TB infection. Twenty-five patients who tested positive for latent TB infection, yet were either untreated or inadequately treated, received guselkumab for 24 to 32 months, with no cases of progression of latent TB infection to active disease reported.[11,38,61] Similarly, 23 patients with latent TB infection who did not receive or received inadequate TB preventive therapy were treated for 24 to 32 months with tildrakizumab, again with no cases of progression of latent TB infection to active disease reported.[11,62] Finally, 68 patients with latent TB infection, but who received either no or inadequate TB preventive therapy, were treated with risankizumab for periods ranging from 9 to 32 months; no cases of progression of latent TB infection to active disease were reported in this cohort.[38,39,11,59]

FAERS database search

To further investigate the possible role of IL-17 inhibitors and IL-23 inhibitors in the progression of latent TB infection to active disease, the FAERS was searched for cases of extra-pulmonary internal organ TB, which generally occur in patients with underlying immune compromising conditions. A total of

96,351 safety reports from the United States associated with the use of IL-17 or IL-23 inhibitors for the treatment of psoriasis were revealed in the FAERS database (Table I). For IL-17 inhibitors (secukinumab, ixekizumab, brodalumab, and bimekizumab) and IL-23 inhibitors (guselkumab, tildrakizumab, and risankizumab), no cases of extra-pulmonary internal organ TB and no cases of disseminated TB associated with the use of these medications were found. As a positive control, cases of extra-pulmonary internal organ TB in US patients treated with TNF inhibitors were queried.

Among 139,825 safety reports associated with the use of TNF inhibitors (etanercept, infliximab, adalimumab, and certolizumab) for the treatment of psoriasis, 26 reports of extra-pulmonary internal organ TB were found, including 8 cases of disseminated TB (Table I). Thus, despite nearly a decade of use of biologics that target IL-17 or IL-23 for the treatment of psoriasis in the United States, no signal was detected in the FAERS database for types of TB infection associated with immunosuppression, but this signal was confirmed with TNF inhibitors.

Joint position statement

There are 36 members of the Medical Board of the NPF, a US-based nonprofit organization focused on advancing clinical care, patient advocacy, and research on psoriatic disease. Membership to the Medical Board is by invitation only and includes dermatologists, rheumatologists, nurse practitioners, and physician assistants generally recognized as national/international experts on psoriatic disease. The IPC is a global nonprofit organization focused on psoriasis. Membership into the council occurs through a comprehensive vetting process and includes approximately 200 individuals worldwide, including dermatologists, rheumatologists, other physicians, and basic research scientists who are generally recognized as international experts in psoriasis. To formalize endorsement of the joint position statement, a vote was conducted among members of the Medical Board of the NPF and Board Members/Councilors of the IPC. Overall, 87.5% of respondents (112 votes received) voted in favor of endorsement, reflecting strong support and in alignment with established consensus thresholds. In general, most dissenters practiced dermatology in TB endemic areas and were more comfortable in continuing to test all their psoriasis patients receiving biologic therapies. The following statement was formally adopted by both organizations: *Routine testing for latent tuberculosis infection is not required prior to or during treatment of psoriasis patients with IL-17 or IL-23 inhibitors.*

DISCUSSION

Although this new joint position statement from the NPF and the IPC represents a change from current drug labeling and current sponsor and regulatory practices, which assume screening for latent TB infection is obligated, it is based on biologic, clinical trial, and real-world evidence that support the safety of IL-17 and IL-23 inhibitors in the setting of latent TB infection. This recommendation is applicable to both current clinical practice and future clinical trials. Regarding the latter, emerging oral medications for psoriasis that selectively target IL-17 or IL-23 have not shown signals that increase the risk of progression of latent TB infection to active disease,[63] and thus should also not have routine testing performed for latent TB infection before or during treatment.

This new joint position statement allows for exceptions, which may occur for specific patients and in certain clinical conditions where continued testing for latent TB infection may be prudent or warranted, for example, in areas of the world where TB is endemic or when patients are receiving concomitant medications, such as prednisone or other immunosuppressants, that may increase the risk of progression of latent TB infection to active disease. Leading experts who use IL-17 and IL-23 inhibitors to treat patients with IMIDs other than psoriasis, such as psoriatic arthritis, ankylosing spondylitis, hidradenitis suppurativa, and inflammatory bowel disease, are also encouraged to make similar position statements. In summary, avoiding routine TB testing that is scientifically and medically unwarranted would alleviate regulatory burden, save healthcare costs, alleviate concerns for false positive tests, and expedite treatment of psoriasis patients with an IL-17 or IL-23 inhibitor.

Conflicts of interest

Dr Blauvelt is a speaker (received honoraria) for Almirall, Eli Lilly, Sanofi, and UCB; is a scientific adviser (received honoraria) for AbbVie, Almirall, Alumis, Amgen, Anaptysbio, Apogee, Arcutis, Astria, Boehringer Ingelheim, Bristol Myers Squibb, Celltrion, Corvus, Dermavant, Eli Lilly, Galderma, GlaxoSmithKline, Immunovant, Incyte, IQVIA, Janssen, Leo, Lipidio, Merck, Novartis, Oruka, Paragon, Pfizer, Rani Therapeutics, Regeneron, Sanofi, Spherix Global Insights, Sun Pharma, Syncona, Takeda, UCB, Union, and Zai Lab; is a clinical study investigator (institution has received clinical study funds) for AbbVie, Acelyrin, Almirall, Alumis, Amgen, Arcutis, Boehringer Ingelheim, Bristol-Myers Squibb, Dermavant, Eli Lilly, Galderma, Incyte, Janssen, Leo, Merck, Novartis, Pfizer, Regeneron, Sanofi, Sun Pharma, Takeda, and UCB; and owns stocks in Lipidio and Oruka. Dr Strober is a consultant (honoraria) for AbbVie, Alumis, Almirall, Amgen, Apogee, Arcutis, Boehringer Ingelheim, Bristol Myers Squibb, Capital One,

CorEvitas, Dermavant, Immunovant, Janssen, Leo, Eli Lilly, Maruho, Oruka, Meiji Seika Pharma, Protagonist, Takeda, Novartis, Pfizer, UCB Pharma, Rapt, Regeneron, Sanofi-Genzyme, and Union Therapeutics; owns stock options in Connect Biopharma and Mindera Health; is a speaker for AbbVie, Arcutis, Dermavant, Eli Lilly, Incyte, Janssen, Regeneron, and Sanofi-Genzyme; is the Scientific Co-Director (consulting fee) of CorEvitas Psoriasis Registry; is an investigator for CorEvitas Psoriasis Registry; and is the Editor-in-Chief (honorarium) for Journal of Psoriasis and Psoriatic Arthritis. Dr Kerkhof received consultancy service or lecturerships from Almirall, Eli Lilly, Novartis, Janssen Pharmaceutica, Bristol Myers Squibb, UCB, Boehringer Ingelheim Centrion, and Sandoz. Dr Puig received consultancy/speaker's honoraria from and/or participated in clinical trials sponsored by AbbVie, Almirall, Amgen, Biogen, Boehringer Ingelheim, Bristol Myers Squibb, Fresenius-Kabi, Horizon (DSMB), J&J Innovative Medicine, Leo-Pharma, Lilly, Novartis, Pfizer, Samsung-Bioepis, STADA, Sun-Pharma, and UCB. Dr Lebwohl is an employee of Mount Sinai which receives research funds from AbbVie, Arcutis, Avotres, Boehringer Ingelheim, Cara therapeutics, Clexio, Dermavant Sciences, Eli Lilly, Incyte, Inozyme, Johnson & Johnson, Pfizer, Sanofi-Regeneron, and UCB and is a consultant for Aikium, Almirall, AltruBio Inc, Amgen, Apogee, Arcutis, Inc, AstraZeneca, Atomwise, Avotres Therapeutics, Boehringer-Ingelheim, Bristol-Myers Squibb, Castle Biosciences, Celltrion, CorEvitas, Dermavant Sciences, Dermsquared, Evommune, Inc, Facilitation of International Dermatology Education, Forte biosciences, Galderma, Genentech, Incyte, LEO Pharma, Goodrx-Mayne, Meiji Seika Pharma, Mindera, Mirium Pharmaceuticals, Oruka, Pfizer, Sanofi-Regeneron, Revolo, Seanergy, Strata, Sun Pharma, Takeda, Trevi, and Verrica. Dr Romiti is a research investigator, scientific advisor, or speaker to AbbVie, Boehringer Ingelheim, Bristol Myers Squibb, Leo, Johnson & Johnson, Lilly, Novartis, Pfizer, Sanofi, Sun Pharma, Takeda, and UCB. Dr Armstrong is a research investigator, scientific advisor, or speaker to AbbVie, Arcutis, Boehringer Ingelheim, Bristol Myers Squibb, Leo, UCB, Janssen, Lilly, Novartis, Ortho, Sun, Dermavant, Sanofi, Takeda, Regeneron, and Pfizer. Dr Choon is an advisor, speaker, or consultant for AbbVie, Almirall, Boehringer Ingelheim, Johnson and Johnson, Novartis, and Sanofi. Dr Gelfand is a consultant for AbbVie, Artax (DSMB), Bristol Myers Squibb, Boehringer Ingelheim, Celldex (DSMB), FIDE (which is sponsored by multiple pharmaceutical companies) GSK, Inmagene (DSMB), Lilly, Leo, Moonlake (DSMB), Janssen Biologics, Novartis Corp, UCB (DSMB), Neuroderm (DSMB), Oruka, Inc, Teva (DSMB); receives research grants (to the Trustees of the University of Pennsylvania) from Amgen, Bristol Myers Squibb, and Pfizer Inc; and received payment for continuing medical education work related to psoriasis that was supported indirectly pharmaceutical sponsors. Dr Gelfand is a Deputy Editor for the Journal of Investigative Dermatology receiving honoraria from the Society for Investigative Dermatology, is Chief Medical Editor for Healio Dermatology (receiving honoraria), and is a member of the Board of Directors for the International Psoriasis Council and the Medical Dermatology Society, receiving no honoraria. Dr Merola is a consultant and/or investigator for Amgen, Astra-Zeneca, Biogen, Boehringer Ingelheim, Bristol Myers Squibb, AbbVie, Dermavant, Eli Lilly, Moonlake, Novartis, Janssen, Oruka, UCB, Sanofi, Regeneron, Sun Pharma, Galderma, Biogen, and Pfizer. Dr Winthrop received research grant funds and/or consulting honoraria from Bristol Myers Squibb, UCB, AbbVie, Galapagos, Sanofi, Regeneron, Janssen, Takeda, and Eli Lilly and Company. Dr Torres is a speaker, scientific adviser, and clinical study investigator for AbbVie, Almirall, Amgen, Apogee, Arena Pharmaceuticals, Biocad, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Johnson & Johnson, LEO Pharma, Eli Lilly, MSD, Novartis, Pfizer, Samsung-Bioepis, Sanofi-Genzyme, Sandoz, STADA, and UCB. Dr Eakin, Dr Howard, Christy Langan, and Dr Ramessur have no conflicts of interest to declare.

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