



JAK INHIBITORS IN PSORIATIC ARTHRITIS: A NETWORK META-ANALYSIS OF EFFICACY AND SAFETY

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ABSTRACT

Psoriatic Arthritis (PsA) is an inflammatory systemic condition that includes different forms of musculoskeletal and cutaneous symptoms such as peripheral arthritis, axial involvement, enthesitis, dactylitis, nail involvement, and psoriasis. There has been a marked advancement in the treatment of patients with active PsA due to the emergence of targeted synthetic disease-modifying antirheumatic agents. JAK inhibitors play a crucial role in this context since they target the signalling of cytokines at intracellular level and affect multiple inflammatory processes at the same time. Tofacitinib, upadacitinib, and filgotinib have been tested in randomized trials, whereas other targeted kinase inhibitors have further advanced the field. Nevertheless, comparisons are necessary due to the diversity of molecular selectivity, dosing, efficacy in various disease domains, and safety issues. The network meta-analysis is the ideal approach for assessing treatment comparison when there is insufficient direct evidence since it involves both direct and indirect data using common comparators. Available studies show that JAK inhibitors have proved to be superior to placebo regarding peripheral arthritis by increasing ACR20, ACR50, and ACR70 responses. These drugs improve psoriasis, enthesitis, dactylitis, and physical function. In one published Bayesian network meta-analysis study, which included five randomized controlled trials and 2,539 participants, filgotinib 200 mg demonstrated the highest probability of ACR20 response, while upadacitinib 30 mg was the best for ACR50 and ACR70 responses; tofacitinib 10 mg showed the highest probability of PASI75 response. Further evidence that has been provided by studies carried out later shows that most JAK inhibitors perform better than placebo in the context of PsA. It is important to note that the statistically significant differences in favor of the JAK inhibitors involve upadacitinib. Evaluation of safety is quite challenging since such issues as infections, herpes zoster, cardiovascular disorders, thromboembolic disorder, malignancy, and discontinuation of treatment need to be addressed. The network meta-analysis carried out in 2025 demonstrated that there were differences between some of the JAK treatments in terms of adverse-event outcomes; particularly, upadacitinib at a dose of 30 mg performed differently from others. However, the study suggested that 15 mg of upadacitinib had efficacy and safety similar to other regimens.

Keywords: Psoriatic arthritis, JAK inhibitors, Janus kinase, network meta-analysis, tofacitinib, upadacitinib, filgotinib, ACR20, ACR50, PASI75, efficacy, safety.

INTRODUCTION

Psoriatic arthritis (PsA) is a chronic immune-mediated inflammatory condition that occurs along with psoriasis and features a high level of heterogeneity. This condition may involve peripheral joints, the axial skeleton, entheses, digits, nails, and the skin, and each patient experiences various combinations

of these aspects of PsA. Chronic inflammation may lead to pain, stiffness, fatigue, impairment of physical functioning, inability to perform job duties, decreased quality of life, and progression of joint destruction. Given the complicated nature of the PsA, treatment can be not limited by the suppression of peripheral joint inflammation. An efficient therapy should target predominant disease domains and take into account other diseases like psoriasis, inflammatory bowel disease, uveitis, cardiovascular disease, and metabolic risk factors. The modern treatment strategy includes conventional synthetic disease-modifying antirheumatic drugs (DMARDs), biologic DMARDs, and targeted synthetic drugs. JAK inhibitors have become one of the important groups of targeted synthetic drugs because of the oral administration of JAK inhibitors and their impact on intracellular signaling pathways participating in various inflammatory cytokines.

The emergence of JAK inhibitors is a revolutionary shift in the pharmacotherapy of inflammatory arthritis. Unlike biological treatments, which mainly affect extracellular cytokines or their receptors, JAK inhibitors have an intracellular mechanism of action. Cytokines interact with the corresponding receptors localized on the cell surface, resulting in the activation of JAK enzymes and the phosphorylation of signal transducer and activator of transcription proteins. The latter move into the nucleus and affect the transcription of genes associated with the immune response and inflammation. Thus, the blockade of particular JAKs leads to the interference with signaling from multiple cytokines. Tofacitinib has a relatively high affinity to JAK1 and JAK3, as well as JAK2, whereas upadacitinib and filgotinib selectively inhibit JAK1. The difference in the selectivity profile may affect the pharmacological properties of drugs, but biochemical selectivity does not fully define its effectiveness and safety.

Inflammatory signaling that is affected by JAK inhibitors plays a role in a number of PsA presentations. Cytokines that affect T-cell activation, innate immunity, and inflammation cell recruitment cause synovitis, enthesitis, dactylitis, and psoriasis. Thus, modification of JAK-dependent pathways might help to achieve the effect in several clinical areas. This is especially important for PsA patients who might have active arthritis along with significant psoriasis or enthesitis. Based on the data from randomized trials, JAK inhibitors might be helpful in treating these conditions; therefore, they might be considered as an alternative to traditional treatment or biologicals in cases where these drugs cannot be used. According to the pooled data from randomized trials, JAK inhibitors provide better ACR20, ACR50, ACR70, PASI75, enthesitis, and dactylitis results than placebo.

While having proven their effectiveness, the comparative effectiveness of each JAK inhibitor is a key issue. The vast majority of clinical studies have examined only one effective drug versus a placebo or another background therapy without a direct comparison of all JAK inhibitors against each other. As a result, pairwise meta-analysis fails to give a full hierarchy. Network meta-analysis can solve the issue by using both direct and indirect comparisons in the framework of the treatment network. For example, if treatments A and B have both been tested against a placebo, it is possible to evaluate the comparative effectiveness of A versus B with the help of a common placebo comparator. The method will be especially helpful for PsA due to the limited number of direct head-to-head studies among the JAK inhibitors.

The objective of this paper is to review the comparative effectiveness and safety of JAK inhibitors in PsA using data obtained from randomized controlled trials, systematic reviews, and network meta-analyses. The efficacy endpoints evaluated are ACR20, ACR50, ACR70, PASI75, resolution of enthesitis, resolution of dactylitis, and functional improvement. The safety endpoints are total adverse events, serious adverse events, infections, herpes zoster, major adverse cardiovascular events, thromboembolism, malignancy, and discontinuation of therapy. Special attention is paid to the comparison of tofacitinib, upadacitinib, and filgotinib and the interpretation of their treatment rankings taking into account uncertainty and individual risk.

METHODOLOGY

The research in this paper utilizes an evidence synthesis method based on published randomized controlled trials and network meta-analysis that examines JAK inhibitors used for the treatment of active PsA in adults. The target population consists of adults with active PsA in which joints, entheses, digits, skin or any other domains of the disease have been affected. Interventions under study include tofacitinib, upadacitinib, and filgotinib at doses tested in randomized trials. Placebo and active comparator drugs are included in this analysis only in case they have been included in the particular treatment networks. The outcome measures of efficacy include ACR20, ACR50, ACR70, PASI75, resolution of enthesitis, resolution of dactylitis and changes in functional scales including HAQ-DI. The safety outcome measures include adverse events, serious adverse events, infections and other clinically important events. The basis of this method is the randomized controlled trial, because randomization allows one to determine treatment efficacy best. There is one prior systematic review for PsA that contained five randomized controlled trials with 3,293 patients and showed a significant difference in ACR20 between JAK inhibitors and placebo. There is one network meta-analysis which consisted of five randomized controlled trials with 2,539 patients and evaluated various doses of upadacitinib, tofacitinib, and filgotinib. There have been several more recent pieces of network evidence that expanded on the efficacy and safety endpoints and confirmed the efficacy of JAK inhibitors.

The network meta-analysis approach uses both direct and indirect evidence based on the assumptions of transitivity and consistency. The transitivity assumption entails comparability of important clinical and methodological aspects of the trials. Transitivity is especially relevant in PsA since different trials can vary in terms of disease duration, disease activity at baseline, previous exposure to biological treatments, concurrent disease-modifying drugs, and psoriasis severity. In other words, the interpretation of indirect comparisons should take into consideration possible discrepancies between the study populations. Treatment ranking is also part of network meta-analysis. Indicators, such as surface under the cumulative ranking curve, may assess the probability that the treatment in question belongs to the group of the most effective and safest treatments. Nevertheless, ranking cannot be seen as proof of clinical superiority, since the treatment may be the first due to its small advantage, which involves statistical uncertainty.

Mechanism of Action of JAK Inhibitors

JAK-STAT signaling pathway is one of the major intracellular signaling pathways that enable cytokines to convey their biological information. There are four main types of JAK proteins that have been recognized – JAK1, JAK2, JAK3, and TYK2. The JAK proteins bind to the cytokine receptors and

become active after the cytokine-binding process. Active JAK proteins phosphorylate the STAT molecules that regulate the gene expression processes. This signaling pathway enables immune cell differentiation, proliferation, activation and inflammation. In PsA, there are several cytokine signaling pathways that partially rely on the JAK signaling. Tofacitinib is an inhibitor of JAK1 and JAK3 and, in addition, a weak JAK2 inhibitor. Upadacitinib displays more selectivity towards JAK1, whereas filgotinib is JAK1 selective as well. Selectivity differences offer a basis for comparison of each agent from a pharmacological perspective, although many other factors than enzyme selectivity are responsible for the clinical effects. One major benefit of the JAK inhibitors is that they can be given orally. Patients may find oral therapy preferable to biologic injectables. JAK inhibitors are fast-acting drugs compared with some conventional treatment methods. Nevertheless, oral therapy does not exclude the need for monitoring, because of the involvement of JAK signaling in immune system processes.

Comparative Efficacy

The most compelling rationale for the use of JAK inhibitors in PsA is the benefit in peripheral arthritis. ACR20 is commonly employed as one of the primary measures of therapeutic response and requires improvements in tender and swollen joint counts as well as other disease-specific measures from the perspective of the patient and physician. ACR50 and ACR70 are even better measures of clinical response. A systematic review of five randomized controlled trials with 3,293 participants demonstrated that the administration of JAK inhibitors increased the odds of attaining an ACR20 response by 3.78.) This constitutes strong evidence of clinically relevant efficacy of JAK inhibition for PsA. Network meta-analysis has been performed to compare the effectiveness of JAK inhibitors. A previous Bayesian network model analysis included upadacitinib 30 mg, upadacitinib 15 mg, tofacitinib 10 mg, tofacitinib 5 mg, and filgotinib 200 mg. Out of all the analyzed treatment regimens, filgotinib 200 mg demonstrated the highest probabilities for ACR20 response, followed by upadacitinib 30 mg, upadacitinib 15 mg, tofacitinib 10 mg, and tofacitinib 5 mg. In the case of ACR50 and ACR70 responses, the highest probabilities were shown for upadacitinib 30 mg, upadacitinib 15 mg, and filgotinib 200 mg, respectively. This may indicate some variations between each JAK inhibitor and its dosage, but

Further evidence has continued to confirm the efficiency of JAK inhibitors. A network meta-analysis conducted in 2025 found five trials in which there was an improvement in the ACR20 and PASI75 responses for JAK inhibitors in a total of 2,757 patients. Filgotinib 200 mg and upadacitinib 30 mg had better ACR20 responses compared to tofacitinib 5 mg twice daily in network analysis. Recent systematic review and network meta-analysis found that most of the JAK inhibitors were more efficient than placebo for ACR20, ACR50, ACR70, PASI75, enthesitis, dactylitis, and functional responses, with significant differences between some drugs, including upadacitinib. The significance of the ACR50 and ACR70 findings is that they show that there is a greater level of improvement in disease status. While ACR20 is known as a gold standard, it would make sense to test whether higher response rates would prove the greater effectiveness of treatments. The very good rank of the upadacitinib 30 mg treatment in the previous network analysis in terms of ACR50 and ACR70 speaks for its high efficiency. Still, the high dose should be viewed in combination with the safety of the treatment.

Another important therapeutic area in PsA is psoriasis. PASI75 is frequently utilized to measure skin disease improvement. A meta-analysis on JAK inhibitors in psoriasis and PsA consisted of 17 clinical

trials with 6,802 patients. Higher ACR20 and PASI75 responses were observed in JAK inhibitors vs. placebo. The pooled ACR20 risk ratio was 2.09, while the pooled PASI75 risk ratio was 4.03. In subgroup analysis, Tofacitinib 10 mg twice a day had a higher PASI75 response rate than 5 mg in subgroup analysis. These results show the ability of JAK inhibition to offer clinically meaningful skin improvement along with the joint improvement. Another important aspect is enthesitis and dactylitis since they can cause considerable pain and disability. A meta-analysis of randomized placebo-controlled trials showed the effectiveness of JAK inhibitors in the treatment of enthesitis and dactylitis resolution in comparison with placebo. The pooled risk ratio for enthesitis resolution was 1.82, while the pooled risk ratio for dactylitis resolution was 1.85. These results confirm the multitherapeutic area of JAK inhibitors' action and prove that their effects are not limited only to peripheral synovitis. Functional improvement is another important aspect. Prolonged PsA could impair physical activity levels and lower quality of life despite low levels of objective joint inflammation. The benefits of JAK inhibitors on the HAQ-DI and other measures of physical function have been shown. In the recent network analysis, HAQ-DI was also included, and JAK inhibitors were shown to be better than placebo with respect to functional outcome. It is important to note that an effective treatment will not just improve inflammatory scores but improve daily function as well.

Comparative Safety

Safety is among the most critical considerations in comparing the efficacy of different JAK inhibitors. While most randomized trials typically show satisfactory safety in the short term, adverse events are still fairly frequent. A meta-analysis of PsA randomized trials showed that JAK inhibitors had a greater likelihood of experiencing any adverse event compared to placebo, with a risk ratio of about 1.14. The same study showed that the incidence of infection was elevated by approximately 1.23. This highlights the necessity of monitoring patients with infections. Another systematic review comprising 15 randomized trials with 6,757 subjects showed no statistically significant differences in the occurrence of serious adverse events compared to placebo. In addition, an earlier meta-analysis in patients with PsA failed to show any increased occurrence of serious adverse events compared to placebo. However, serious adverse events are relatively rare, and many PsA randomized trials are often not big enough and long enough to establish differences in rare events.

One area of special consideration with JAK inhibitors is herpes zoster. The effect of alteration of immune signal transduction pathways in cells could potentially impair antiviral defense mechanisms and increase susceptibility to reactivation of the disease. The issue is something to take into account while assessing patients with a history of recurring infections or taking other immunosuppressive medications. Vaccination and careful monitoring could play a crucial role in the safety of the treatment. The comparative safety of various JAK treatment strategies has been addressed in recent network analyses. A 2025 study conducted a network meta-analysis which showed that a 30 mg dose of upadacitinib was linked to a higher probability of adverse events than placebo and showed poorer safety profile than filgotinib 200 mg regarding severe adverse events. Upadacitinib 15 mg, the standard dose of the drug, had efficacy and safety profiles similar to other studied strategies.

Cardiovascular and Thromboembolic Safety

The cardiovascular safety of patients is an especially important aspect when considering JAK inhibitors. The regulatory concern arose based on data from a safety trial of tofacitinib in patients with rheumatoid arthritis and risk factors for cardiovascular disease. This led to updated warnings for the risk of major adverse cardiovascular events, malignancy, thrombosis, and death for certain JAK inhibitors. It should be noted that although these data were not collected for patients with PsA, they affect clinical evaluation of the whole class of drugs. PsA increases cardiometabolic risk due to the presence of both chronic inflammation and the risk factors for cardiovascular diseases such as obesity, hypertension, diabetes, and others.

PsA specific data are becoming more abundant. For example, a network meta-analysis for MACE and thromboembolic events carried out in 2025 included 11 studies. From the combined randomized and long-term extension trials data, there were 23 MACE and 26 thromboembolic events identified, with respective incidence rates being 0.25 and 0.29 per 100 person years. From the randomized data, 12 MACE and 8 thromboembolic events were identified. The conclusion made by the authors was that tofacitinib and upadacitinib had equivalent outcomes related to cardiovascular and thromboembolic events compared to other comparators, but due to the relatively small number of events, one cannot be absolutely sure about this. This is an example of why absolute risks are as important as relative risks. The importance of the potential increase in cardiovascular risk is very dependent on baseline patient characteristics.

Dose-Response Relationship

The dose level is an important determinant in both efficacy and safety. The higher the dose, the greater the likelihood of achieving a response in terms of ACR50 and ACR70, but there will be increased risks of adverse events. The previous network meta-analysis found that upadacitinib 30 mg was the best choice for ACR50 and ACR70. But the latest data show that upadacitinib 30 mg poses a greater risk of adverse events. This is an important principle of the clinical approach to treatment: the best regimen from the point of view of efficacy does not have to be the most appropriate for the treatment course.

DISCUSSION

The evidence presented in this article shows that JAK inhibitors offer substantial benefits to PsA patients with active disease. These agents show efficacy across many aspects such as peripheral arthritis, psoriasis, enthesitis, dactylitis, and physical function. The consistency of superiority over placebo in the context of multiple meta-analyses supports the use of JAK inhibitors as a valid approach to PsA treatment. Comparative evidence, on the other hand, is slightly more complicated. Early network analysis showed that filgotinib 200 mg was the top-ranking agent with respect to ACR20, whereas upadacitinib 30 mg was the top-ranking drug with regard to ACR50 and ACR70. Recent network evidence also highlighted the fact that filgotinib 200 mg and upadacitinib 30 mg were among the most effective treatments compared to tofacitinib 5 mg with respect to ACR20. This evidence should not be considered conclusive since it is based on limited indirect comparison of small samples.

An important factor contributing to variation in network rankings is the heterogeneity of PsA. PsA can present predominantly with peripheral arthritis in one patient, severe psoriasis in another, or axial/enthesal disease in yet another. Furthermore, the treatment response may vary depending on the

prior biological therapy experience. An overall treatment ranking may overlook some important domain-specific differences in this case. Finally, the safety profile adds an additional layer of complexity. According to the 2025 network analysis, although upadacitinib 30 mg demonstrated excellent efficacy, it was associated with a higher rate of adverse events. Hence, this example corroborates the idea of the need to consider a treatment on the basis of the balance between benefits and risks rather than an efficacy ranking. It is especially important to evaluate the link between JAK inhibitors and cardiovascular/thromboembolic outcomes. The regulatory warnings were based on evidence in high-risk rheumatoid arthritis populations, whereas the recent evidence specific to PsA population showed relatively low event rates.

One more crucial problem is that of long-term safety. Randomized trials usually evaluate the efficiency for relatively short time periods and may lack statistical power to reveal rare effects. Thus, the use of long-term extension studies and registry data is necessary. The information from such analyses may show us rare adverse events that may not appear during the course of randomized treatment. Oral administration of JAK inhibitors is one more significant benefit. Patients might find it easier to take drugs by mouth since it does not require injections. But we should not forget about the safety issues while discussing the convenience of oral treatment. Choice of therapy must be done in the process of shared decision making when patients are aware of efficacy, side effects, required monitoring, and other treatment modalities. Also, JAK inhibitors must be chosen considering other advanced therapies for PsA. Therapies against TNF, IL-17, IL-12/23, and IL-23 pathways still play an important role in the treatment of PsA. Network meta-analysis of all available therapies for PsA shows us that different biological approaches are efficient. Thus, JAK inhibitors must be selected based on patient's specific conditions.

Limitations

There are several factors that influence the understanding of the current evidence. First, there is a lack of direct comparison between JAK inhibitors. The major part of comparative evidence is based on indirect comparison through placebo. Second, the population characteristics, background therapy, disease severity, previous use of biologics, and follow-up periods for different trials are not similar. All this can influence the effectiveness of treatments and even violate the assumptions of network meta-analysis. Third, rare safety events like malignancy, MACE, thromboembolism, and mortality can be assessed only by a huge sample size and long follow-up period that is not provided in most PsA randomized trials. Fourth, the treatment rankings can amplify small differences between treatments. Fifth, regulatory approval and dosages may differ in different countries; therefore, results of the network analysis can not be extrapolated to other healthcare systems. Rapid changes in treatment modalities mean that network meta-analysis can be outdated in a short period of time due to the introduction of new drugs and evidence on long-term treatment.

Future Research

Future research should aim to compare JAK inhibitors head to head with other novel therapies. Pragmatic randomized trials would yield better evidence than small indirect comparisons. Patient-individual data network meta-analyses would further help in making choices about which treatment to use by determining whether age, gender, prior biological therapy, phenotypes, disease severity or cardiovascular

risk impacts the efficacy of these treatments. Future safety research should be focused on myocardial infarction, death, venous thromboembolism, malignancy, infections, and mortality. Since these outcomes are quite rare, it would be particularly important to establish registries and collect long-term follow-up data internationally. In addition, future research should look into patient outcomes, persistence of therapy, quality of life, work productivity and medication adherence. Even a highly effective treatment from an ACR response point of view might not show good performance if there is low patient adherence.

CONCLUSION

JAK inhibitors have proven to be very useful therapeutic approaches in the management of active psoriatic arthritis. Studies done in randomized controlled trials and network meta-analyses indicate that the medications offer great improvements in peripheral arthritis, and also help in improving psoriasis, enthesitis, dactylitis, and physical function. Early studies indicated that filgotinib 200 mg had a higher likelihood of achieving an ACR20, whereas upadacitinib 30 mg exhibited good results regarding ACR50 and ACR70. Recent evidence has continued to validate the efficacy of JAK inhibitors despite the fact that there are some differences between certain regimens.

Safety issues are just as vital. The overall adverse events and infections could be increased in JAK inhibition therapy, but the serious adverse events occur rarely in randomized PsA trials. Long-term cardiovascular, thromboembolic, and malignancy risk assessments have to be done individually depending on patients who have preexisting risks. The current evidence with regard to cardiovascular risk in PsA is relatively positive, but still limited because of few numbers of rare events.

The collective body of evidence thus indicates that JAK inhibitors can serve as efficacious options for oral therapy for PsA, although there is no JAK inhibitor that can be deemed the ideal one for all patients at present. The ideal treatment for a patient should be decided upon on the basis of the predominant disease domains, prior medication exposure, efficacy and safety profile of the specific dose, infection risk, cardiovascular and thrombotic risks, concomitant disorders, preferences of the patient, and relevant regulatory guidelines.

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