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(REVIEW ARTICLE)

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# Abrocitinib: A panacea for atopic dermatitis

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## Abstract

Atopic Dermatitis [AD] is a chronic relapsing inflammatory dermatological disease characterized by itch, dry skin, localized or disseminated eczematous lesions and hyperpigmentation of skin. There are several exogeneous and endogenous risk factors for development of AD. The endogenous factors include genetic factors, atopy, skin hyperreactivity and exogeneous factors such as food, dust, pollen, epidermal, fungal, bacterial, and vaccine-associated factors. The management of AD includes topical treatments such as topical corticosteroids, topical capsaicin, topical calcineurin inhibitors and phosphodiesterase inhibitors. Systemic therapies such as corticosteroids, immunosuppressants, biologicals, gabapentinoids and antidepressants are provided to patients with inadequate systemic therapies have wide range of side effects limiting their use in many patients. Therefore, there is a need for innovative treatment options. This led to development of Janus kinase inhibitors. First generation JAK inhibitors includes Tofacitinib, Baricitinib, Ruxolitinib, Oclacitinib. Second generation drugs such as Upadacitinib and Abrocitinib are more specific towards the JAK isoforms. This review addresses the importance of Abrocitinb for the treatment of Atopic Dermatitis. Abrocitinib an oral once daily treatment for moderate to severe atopic dermatitis developed by Pfizer. Studies shows that Abrocitinib is better alternative in non- responders to Dupilumab therapy or patients with contraindications to the Dupilumab. The patient's adherence to medication will also be better with oral route rather than injections. Abrocitinib will be widely accepted because of its clinical benefits such as reduction in itching and more convenient oral route of administration.

**Keywords:** Abrocitnib; Atopic dermatitis; Janus kinase inhibitors; Calcineurin inhibitors

## 1. Introduction

Atopic Dermatitis [AD] is a chronic relapsing inflammatory dermatological disease characterized by itch, dry skin, localized or disseminated eczematous lesions and hyperpigmentation of skin[1]. The usual onset of the disease is from childhood and both males and females are equally affected[2]. About 2-8 % of adults are affected by AD and in 25% of them the disease started in adulthood. The term Atopic Dermatitis was first used by Fred Wise and Marion Sulzberger in 1933[3]. AD is a heterogeneous disorder that is classified into various subtypes based on phenotypes and genotypes. The various subtypes includes the IgE- high, extrinsic subtype and the IgE-normal, intrinsic subtype. European, American and Asian AD subtypes have been also proposed. This phenotypic classification could help to provide personalised medicine to the individual patients for better prognosis of disease[4].

There are several exogeneous and endogenous risk factors for development of AD[5]. The endogeneous factors includes genetic factors, atopy, skin hyperreactivity and exogeneous factors such as food, dust, pollen, epidermal, fungal,

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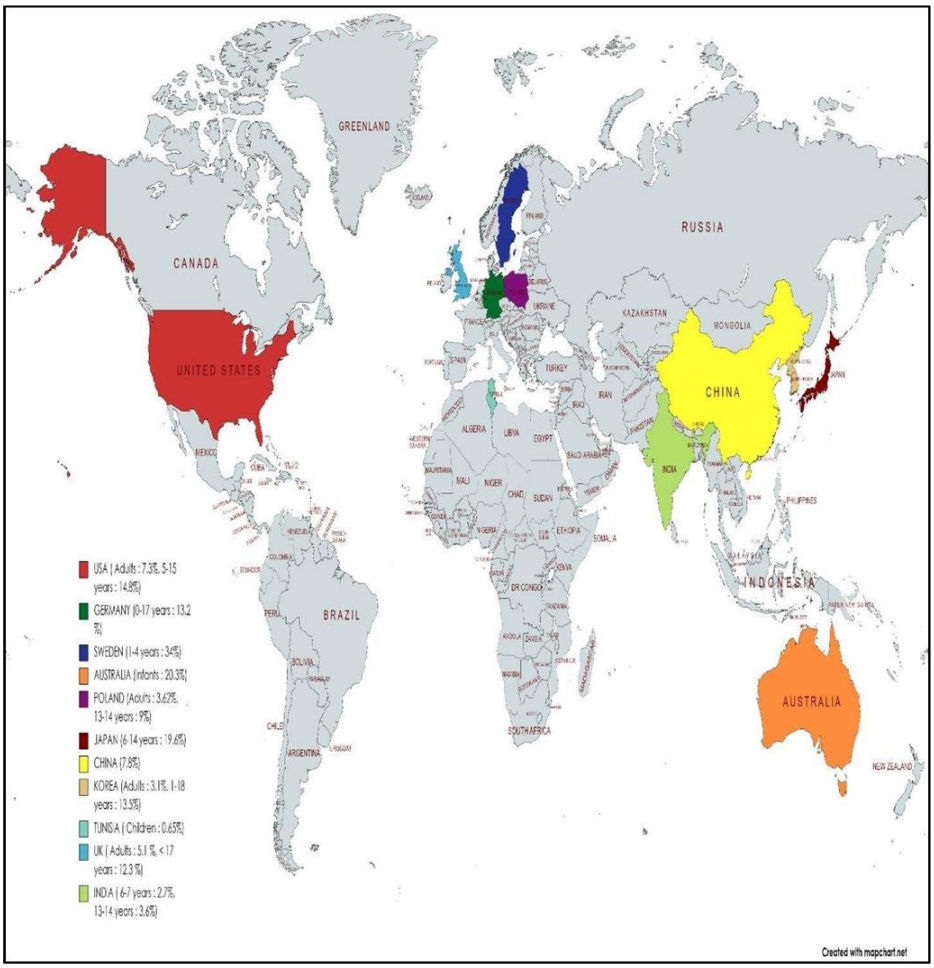
bacterial, and vaccine-associated factors, as well as tobacco smoke, pollutants, xenobiotics, climate, geographical factors, malnutrition, non-compliance with personal hygiene practices, viral infections, and psychological stress[6]. It is a multifactorial disease that affects the person's quality of life by decreasing productivity due to work disruptions, higher direct medical costs, reduced daily activity, fatigue, and daytime sleepiness due to sleep disturbances.

Treatment should be provided to shorten the clinical course of the disease in these patients for better quality of life[7]. The management of AD includes topical treatments such as topical corticosteroids, topical capsaicin, topical calcineurin inhibitors and phosphodiesterase inhibitors. Systemic therapies such as corticosteroids, immunosuppressants, biologicals, gabapentinoids and antidepressants are provided to patients with in adequate response[8].

These systemic therapies have wide range of side effects limiting their use in many patients. New drug such as the Dupilumab is beneficial to patients, but its subcutaneous administration resulted in poor patient acceptance. There is a need for innovative treatment options that led to development of Janus kinase inhibitors. Ruxolitinib was first approved topical Janus kinase inhibitor by FDA for noncontinuous chronic treatment of mild to moderate AD in patients of above 12 years[9]. First generation JAK includes Tofacitinib (oral), Baricitinib (oral), Ruxolitinib (topical cream), Oclacitinib (veterinary use). Second generation drugs such as Upadacitinib and Brocitinib are more specific towards the JAK isoforms[10]. This review addresses the importance of Abrocitinib for the treatment for the treatment of Atopic Dermatitis.

## 2. Epidemiology




Source: Created with Map chart.net

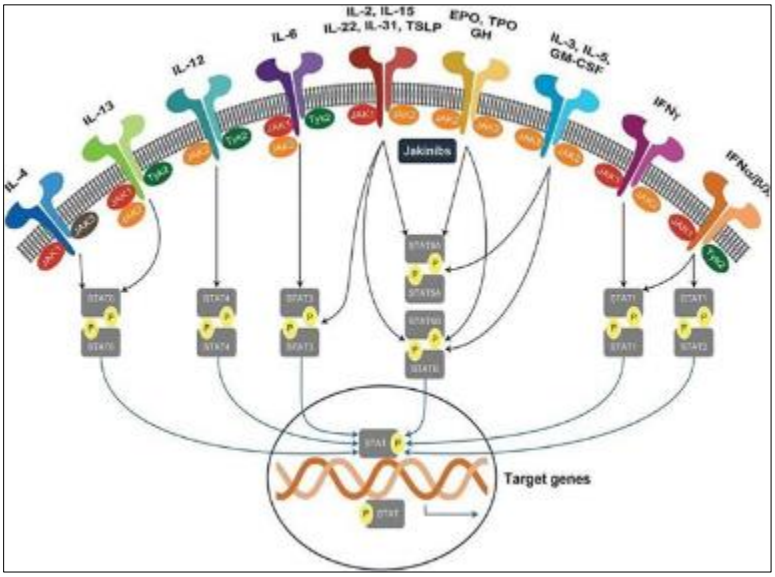
**Figure 1** Epidemiology of Atopic Dermatitis

The prevalence of the AD was higher among the pediatric population when compared to the adults. The highest prevalence was among Swedish children (34%) and lowest prevalence among Tunisian children (0.65%)[11]. The prevalence of AD in India is rising but low when compared to developed countries. As per the ISAAC phase 3 study

conducted worldwide across many countries the prevalence of AD is increasing worldwide. This rise in prevalence is caused due to urbanization[12].

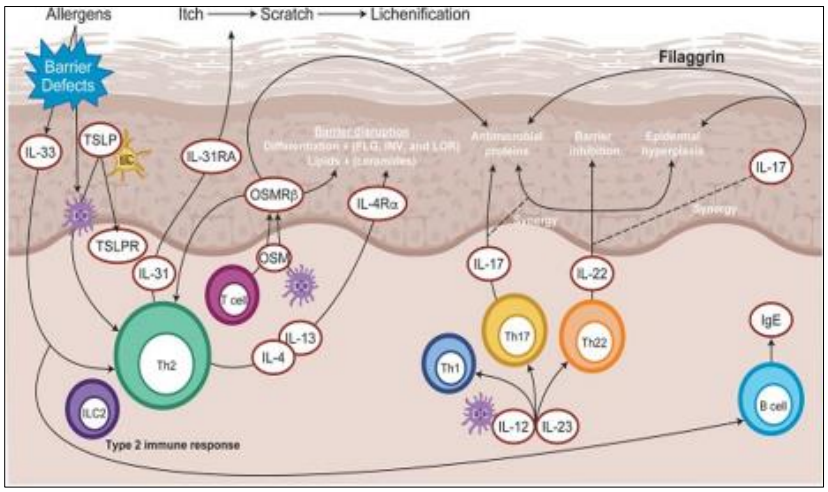
## 3. Pathophysiology and clinical features

The etiopathogenesis of AD is complex, but it is mainly due to increased Th2 immunity driven by JAKSTAT signaling pathway. The cellular infiltrate of AD lesions mainly consist of CD4+ T cells, which are considered keydrivers of inflammation. Lesional skin is characterized by an over expression of inflammatory Th2-cytokines (IL-4, IL-13, IL-31), and Th22- cytokines (IL-22). The symptoms of Atopic dermatitis can appear anywhere on the body and vary from person Toperson. They may include: Dry, Cracked skin, Pruritis, Rash on swollen skin that varies in colour depending onthe skin colour, Thickened skin, Darkening of the skin around the eyes, oozing and crusting.[14]




Source: Nakashima C, Yanagihara S, Otsuka A. Innovation in the treatment of atopic dermatitis: Emerging topical and oral Janus kinase inhibitors. Allergol Int. 2022

**Figure 2** JAK-STAT signalling pathways



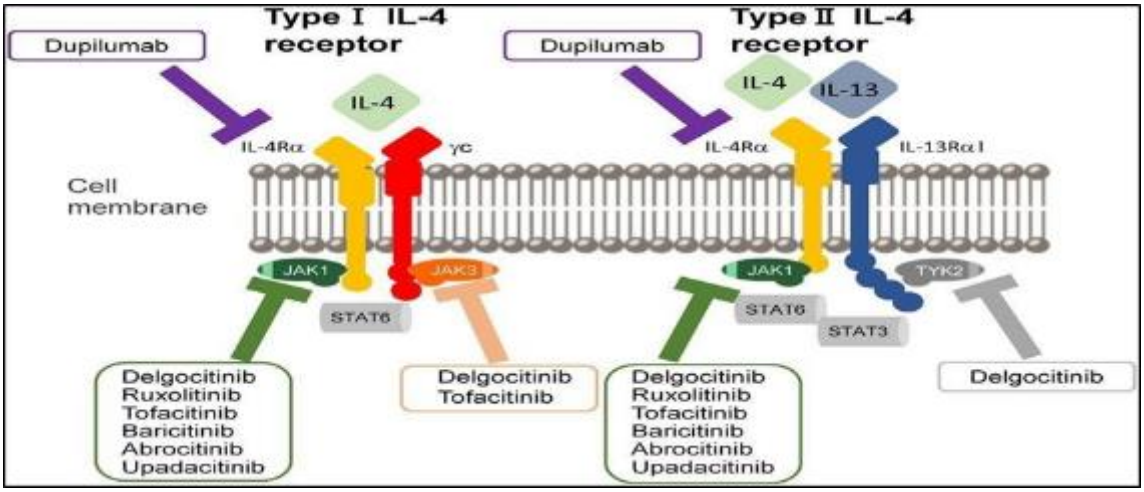

Source: Howell MD, Kuo FI, Smith PA. Targeting the Janus ; Kinase Family inAutoimmune Skin Diseases. Front Immunol.

**Figure 3** Immunopathogenesis of Atopic Dermatitis

## 4. Role of janus kinase inhibitors in atopic dermaitis

The word Janus kinase is based on the name of Roman God "Janus", the God of beginning and end. JAKsindicates a family of receptor associated kinases. JAKs functions are firmly integrated to the cytoplasmic STAT (signal transducer and activator of transcription) transcription factors, of which seven are currently expressed (STAT1, STAT2, STAT3, STAT4, STAT5A/5B, and STAT6). [15] AD is illustrated by multi-cytokine polarization, and a corrective attitude capable of inhibiting more than one cytokine cascade. JAKs bind to intracellular chains of the cytokine receptor and generates functional signaling complexes and regulate the inflammatory processby activating the signal transducer and activator of transcription STAT molecules. STAT proteins dimerize upon activation, translocate into the nucleus, and either positively or negatively regulate downstream target gene expression, namely, inflammatory mediators.[16] Thus, inhibiting JAK activitymay be more productive than targeting a single cytokine.

Further, JAK repression due to the disruptionof cytokine signaling reduces a chronic itch propelled by type-2 cytokine and receptor interplay in sensory neurons.[17] JAK repression also improves skin barrier function by regulating the expression offilaggrin, a skin barrier protein. [18] These findings hinted that JAK is a versatile target for AD treatment, along with its role in regulating epithelial barrier function and immune response and its neuronal mechanisms of action.




Source: Nakashima C, Yanagihara S, Otsuka A. Innovation in the treatment of atopicdermatitis: Emerging topical and oral Janus kinase inhibitors. Allergol Int. 2022 Jan;71(1):40-46.

**Figure 4** Action of Janus kinase Inhibitors on Various Receptors

## 5. Management of atopic dermatitis

**Table 1** Available JAK inhibitors and approved indications

| JAK inhibitors | Indications |
|---|---|
| Abrocitinib | Atopic dermatitis |
| Tofacitinib | Rheumatoid arthritis<br>Psoriatic arthritis Ulcerative colitis |
| Ruxolitinib | Myelofibrosis Plaque psoriasis |
| Baricitinib | Rheumatoid arthritis Atopic dermatitis SLE |
| Upadacitinib | Atopic<br>dermatitis<br>SLE |
| Delgocitinib | Atopic dermatitis Chronic hand dermatitis |
| Oclacitinib | Atopic dermatitis |

Topical agents are mainly used for the treatment of Atopic dermatitis. In severe cases systemic or phototherapy are used. Moisturizers, Wet wrap therapy etc. are some non-pharmacological interventions for the management of AD[16]. Topical corticosteroids are used for the management of AD in both adults and children and these agents are used as anti-inflammatory agents. Topical calcineurin inhibitors are also used as second-class anti-inflammatory agents for the management of AD. Other agents used are topical antimicrobials, antihistamines etc. Artificial UV radiation is widely used as a second line treatment for moderate to severe AD. Systemic therapies are available and it is recommended in severe, chronic and resistant forms of AD. Cyclosporine is the first choice for systemic therapy of moderate to severe AD. Others are Azathioprine, Methotrexate, Mycophenolic mofetil etc[17].

### 5.1. Abrocitinib approval status

Abrocitinib (CIBINQO) an oral once daily treatment for moderate to severe atopic dermatitis developed by Pfizer. The FDA announced its approval in January 2022 [18].

### 5.2. Dose of Abrocitinib

Abrocitinib has a recommended dose of 100mg/day. Dose may be increased to maximum of 200mg/day, if inadequate response is obtained after 12 weeks[18].

### 5.3. Pharmacokinetics of Abrocitinib

Abrocitinib was rapidly absorbed within 1 hour. The steady state concentrations were achieved 48 hours after oral administration. Active metabolites M1 and M2 binds predominantly to the albumin. Metabolism is mediated by CYP2C19 and CYP2C9. The mean elimination half life of M1 and M2 is 3- 5 hours[19].

### 5.4. Pharmacodynamics of Abrocitinib

Abrocitinib caused dose dependent reductions from baseline serum inflammatory markers including CRP, interferon gamma induced protein-10 and reduction in disease biomarkers IL-31 and thymus and activation regulated chemokine[19].

## 6. Safety and efficacy of abrocitinib in clinical trials

The phase 1 study was conducted among the 79 healthy individuals which included healthy males and females (postmenopausal or non-child-bearing age) of 18-55 years. They received a single dose of placebo or 3mg, 10mg, 30mg, 100mg, 200mg, 400mg, or 800 mg of Abrocitinib and placebo or 30mg OD, 100mg OD, 200mg OD, 400mg OD, 100mg BD, 200mg BD of Abrocitinib for 10 consecutive days. Severe adverse events and deaths were not observed during the study. The adverse events observed were headache (13 subjects), diarrhea (11 subjects), nausea (11 subjects) and mild or moderate infection (17 subjects). Most of these adverse events were mild and occurred at higher frequencies in both 400 mg BD and 400mg OD and 200mg BD. The phase 1 study shows that Abrocitinib is safe and well tolerated in healthy subjects[20].

Phase 2 randomised controlled trials were conducted to evaluate the safety and efficacy of Abrocitinib for patients with AD. This study included 267 participants with clinically diagnosed AD for 1 year or more and inadequate response or contraindications to topical medications for 4 weeks or more within 12 months. The subjects received Abrocitinib 200mg, 100mg, 30mg or 10mg or a placebo OD for 12 weeks. At 12th week there was improvement in investigators global assessment scale by 2 grades or more in patients receiving Abrocitinib. There was reductions in eczema area and severity index which was 82.6% in 200mg Abrocitnib group and 59% for 100mg Abrocitinib. Most common adverse events were upper respiratory tract infections, headache, nausea, diarrhea. Dose dependent decrease in platelet count were observed which increased later towards baseline levels after 4 weeks[21].

A double blind randomised phase 3 clinical trial was conducted in adults and adolescents with moderate to severe atopic dermatitis. Out of 387 patients, 156 patients were given Abrocitinib 100mg, 154 were given Abrocitinib 200mg and 77 were assigned to placebo for a study period of 12 weeks.

This study shows that Abrocitinib was effective for atopic dermatitis with moderate to severe intensity. The serious adverse effects considered to be treatment related include chronic inflammatory bowel disease in a patient who received 200mg Abrocitinib and acute pancreatitis in a patient who took 100mg Abrocitinib[22].

In another randomised phase 3 study of Abrocitinib in adults with moderate to severe atopic dermatitis the patients were randomised to receive 16 weeks of oral 200mg or 100mg OD, Dupilumab 300mg SC every 2 weeks or placebo with background topical therapy. Patient reported outcomes such as night time itch, patient oriented eczema measure, scoring Atopic dermatitis, hospital anxiety and depression scale, dermatology life quality index, pruritis and symptoms assessments for atopic dermatitis and Patient global assessment were analysed, all of which showed improvement in the scores within 16 week period. Abrocitinib 200mg provided greater effects when compared to placebo Dupilumab in patients[23]. A recent phase 3 study were patients with moderate to severe atopic dermatitis after Dupilumab in a JADE COMPARE. This study was conducted to study efficacy and safety of Abrocitinib in patients who received prior Dupilumab.

The JADE EXTEND was designed to study the long term safety and efficacy of 200mg or 100mg OD Abrocitinib in moderate to severe atopic dermatitis patients. In JADE EXTEND patients who responded to Dupilumab in JADE COMPARE maintained the clinical benefits and a significant proportion of Dupilumab non responders in JADE COMPARE were able to achieve clinical benefits with Abrocitinib for 12 weeks. The most common adverse events observed were nausea, diarrhoea, headache, upper respiratory tract infection. The adverse events were less in JADE EXTEND than Dupilumab treatment of JADE COMPARE. The study shows that Abrocitinib is good oral treatment option for moderate to severe atopic dermatitis, regardless of prior Dupilumab response. It is also suitable for patients who prefer oral route to be convenient[24].

**Table 2** Clinical trials on abrocitinib

| **Name of study** | **Study type** | **Study status** | **Identifier number** | **Study Location** |
|---|---|---|---|---|
| A study to learn about abrocitinib tablets in people with atopic dermatitis in india. | Intervention-al phase 3 clinical trial | Recruiting | NCT05375929 | India |
| Study of abrocitibib compared with dupilumab in adults with atopic dermatitis on background topical Therapy | Intervention-al phase 3 clinical trial (JADE DARE) | Completed | NCT04345367 | Multicentered |
| Study To Evaluate Efficacy And Safety of Pf-04965842 With Or Without Topical Medications in Subjects Aged 12 Years Or Older With Moderate To SevereAtopic Dermatitis | Intervention-al phase 3 clinical trial (JADE EXTEND) | Active, not recruiting | NCT03422822 | Multicentered |
| Study evaluating the mechanism of action of Pf-04965842 monotherapy for moderate to severe atopic dermatitis | Intervention-al phase 2 clinical trial (JADE MOA) | Completed | NCT03915496 | Multicentered |
| Study to investigate efficacy and safety of Pf- 04965842 in subjects aged 12years and over with moderate to severe Atopic Dermatitis with the Optionof Rescue treatment in flaring Subjects | Intervention-al phase 3 clinical trial (JADE REGIMEN) | Completed | NCT03627767 | Multicentered |
| Jak 1 Inhibitor with medicated topical therapying adolescents with atopic dermatitis | Interventional phase 3 clinical trial (JADE TEEN) | Completed | NCT03796676 | Multicentered |
| A study to learn about the study medicine (CIBINQO) in people with atopic dermatitis | Observationalstudy | Recruiting | NCT05387980 | Pfizer local country office Tokyo, Japan |

| | | | | |
|---|---|---|---|---|
| JAK Inhibtion in foodallergy | Interventional randomized phase 1 clinical trial | Recruiting | NCT05069831 | Icahn School of Medicine at Mount Sinai, New York, US |
| Study evaluating efficacyand safety of pf- 04965842 in subjects aged 12 years and older with moderate to Severe atopic dermatitis | Interventionalphase 3 clinical trial(JADE MONO-2) | Completed | NCT03575871 | Multicentered |
| Abrocitinib expanded access protocol in adolescents and adults with moderate to severeatopic dermatitis | Expanded Access protocol | Available | NCT04564755 | Multicentered |
| Efficacy of abrocitinib for reducing pruritis inadults with prurigo Nodularis and chronic pruritis of unknown origin | Non randomized interventional phase 2 clinical Trial | Completed | NCT05038982 | United States, Maryland |
| Study to evaluate efficacy and safety of pf-04965842 in subjects aged 12 years and olderwith moderate to Severe atopic dermatitis | Interventional phase 3 clinical trial (JADE MONO-1) | Completed | NCT03349060 | Multicentered |
| Study evaluating efficacy and safety of pf-04965842 and Dupilumab in adult subjects with moderateto severe atopic dermatitis on background topical therapy | Interventional phase 3 clinical trial (JADE COMPARE) | Completed | NCT03720470 | Multicentered |
| A Korea post marketing surveillance to learn about the study medicine abrocitinib (CIBINQO tablets) in people with atopic dermatitis | Post marketing surveillance (non interventional observational) | Not yet recruiting | NCT05391061 | Korea |
| Patient characteristics, usage and effectiveness of abrocitinib in adult patients with moderate to severe atopic Dermatitis | Observational (patient registry) | Recruiting | NCT05250115 | Germany |
| A study to estimate the effect of multiple dose of abrocitinib on caffeine, efavirenz and omeprazolein healthy Participants | Phase 1, open label, multiple dose,single fixed sequence, 2- period study | Completed | NCT05067439 | United states, Florida |
| Pre-approval single patient expanded accessfor abrocitinib | Expandedaccess | Available (New) | NCT05466578 | - |
| Effects of abrocitinib treatment on skin barrierfunction | Open label, randomised observational study (AbroSkib) | Not yet recruiting | NCT05140239 | UKSH, Campus Kiel, Schleswig-Holstein, Germany |
| A study evaluating relative bioavailabilty ofan oral suspension of abrocitinib and effect of an acid reducing agent on the bioavailabilty of abrocitinib and assessing the taste of abrocitinib oral formulations | Interventional phase 1 clinical trial | Completed | NCT04903093 | United States, Connecticut |

## 7. Conclusion

A plethora of treatment options is available for treatment of atopic dermatitis. JAK inhibitors arethe emerging newer treatment options for moderate to severe Atopic dermatitis. The topical JAK inhibitors include Deglocitinib, Ruxolitinib, Tofacitinib, Cerdulatinib. The systemic JAK inhibitors such as Batricitnib, Dupilumab (subcutaneous). The second-generation JAK inhibitors such as Upadacitinib and Abrocitinib were recently approved by FDA for atopic dermatitis. The Abrocitinib produced clinical benefits when switched from Dupilumab to Abrocitinib in JADE EXTEND study. This proves that Abrocitinib is better alternative in non-responders to Dupilumab therapy or patients with contraindications to the Dupilumab. The patient's adherence to medication will also be better with oralroute rather than injections. Abrocitinib administration produced thrombocytopenia, but it improved after 4 weeks which should be the area of focus for future studies to ensure further safety of the medicine. Abrocitinib will be widely accepted because of its clinical benefits such as reduction in itching and more convenient oral route of administration.

## Compliance with ethical standards

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*Disclosure of conflict of interest*

There is no conflict of interest for the concept.

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