

Clinical Review of Moxifloxacin for Drug-Sensitive Pulmonary Tuberculosis

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Abstract

Moxifloxacin is a newer-generation fluoroquinolone antibiotic with strong activity against *Mycobacterium tuberculosis*. It is widely used in the management of multidrug-resistant tuberculosis (MDR-TB) and is being evaluated in shorter TB treatment regimens. The drug acts by inhibiting bacterial DNA gyrase, thereby preventing DNA replication and causing bacterial cell death. Moxifloxacin possesses enhanced antimycobacterial activity due to structural modifications such as the methoxy group at the C-8 position. It has excellent pharmacokinetic properties including rapid oral absorption, high bioavailability, long half-life, and good penetration into lung tissues and macrophages, which are important sites of TB infection. Clinical studies have demonstrated significant bactericidal activity against tuberculosis bacteria, and ongoing trials are assessing its role in treatment-shortening strategies. Moxifloxacin is generally well tolerated with minimal serious adverse effects. It is mainly metabolized through glucuronide and sulphate conjugation pathways and is commonly administered orally at a dose of 400 mg once daily in tuberculosis therapy. Overall, moxifloxacin is considered a promising drug in modern anti-tubercular treatment regimens aimed at improving patient outcomes and reducing treatment duration.

I. Introduction

Moxifloxacin is a fourth-generation, 8-methoxy fluoroquinolone antimicrobial agent belonging to the quinolone class of antibiotics. It was developed and marketed by under the brand name *Avelox*. The drug received regulatory approval for the treatment of a variety of bacterial infections, including acute bacterial sinusitis, acute exacerbations of chronic bronchitis, community-

acquired pneumonia, and uncomplicated skin and soft tissue infections¹.

Moxifloxacin possesses broad-spectrum antibacterial activity against both Gram-positive and Gram-negative organisms, as well as atypical pathogens. Owing to its potent antimycobacterial activity, it has also emerged as an important second-line therapeutic option in the management of tuberculosis (TB), particularly multidrug-resistant tuberculosis (MDR-TB). Although widely incorporated into MDR-TB treatment regimens and investigated extensively in clinical trials, moxifloxacin has not received specific regulatory approval for the treatment of drug-susceptible TB or MDR-TB in many countries^{2,3}.

The antimicrobial activity of moxifloxacin is mediated through inhibition of bacterial DNA gyrase and topoisomerase IV, enzymes that are essential for DNA replication, transcription, repair, and bacterial cell division. Inhibition of these enzymes leads to disruption of bacterial DNA synthesis and ultimately results in irreversible bacterial cell death. The presence of a methoxy group at the C-8 position of the quinolone nucleus enhances its antimycobacterial potency and reduces the likelihood of resistance development compared with earlier fluoroquinolones.

Moxifloxacin demonstrates favorable pharmacokinetic characteristics, including excellent oral bioavailability, extensive tissue penetration, prolonged elimination half-life, and high intracellular concentrations within macrophages and pulmonary tissues, which are important sites of *Mycobacterium tuberculosis* infection. These properties support its utility in pulmonary tuberculosis treatment regimens^{4,5}.

An important pharmacological advantage of moxifloxacin is its minimal interaction with the cytochrome P450 enzyme system. Since many antiretroviral drugs (ARVs) used in the management

of HIV/AIDS are metabolized through cytochrome P450 pathways, moxifloxacin exhibits relatively low potential for clinically significant drug–drug interactions with ARVs. This characteristic makes it a favorable and “antiretroviral-friendly” option for TB treatment in patients co-infected with HIV.

Several preclinical studies, animal models, and phase II and phase III clinical trials have evaluated moxifloxacin-containing regimens as potential treatment-shortening strategies for tuberculosis. Although these studies demonstrated rapid bactericidal activity and faster sputum culture conversion, current evidence has not conclusively established the effectiveness of shortening standard tuberculosis therapy to four months using moxifloxacin-based regimens alone. Nevertheless, the drug continues to play a significant role in modern MDR-TB management and in ongoing research aimed at improving tuberculosis treatment outcomes^{6,7}.

Dosage of Moxifloxacin

Moxifloxacin is available in multiple pharmaceutical formulations for systemic and topical administration:

1. Tablet

Available strength: 400 mg

This is the most commonly prescribed oral dosage form.

2. Intravenous Injection/Infusion

Available strength: 400 mg/250 mL

Administered as a slow intravenous infusion over approximately 60 minutes.

Standard Adult Dosage

1. Tuberculosis (including Multidrug-Resistant Tuberculosis [MDR-TB])

400 mg once daily, administered orally or intravenously.

Used as part of combination anti-tubercular therapy.

2. Respiratory Tract Infections

400 mg once daily for 5–10 days

3. Skin and Soft Tissue Infections

400 mg once daily for 7–14 days

4. Intra-abdominal Infections

400 mg once daily for 5–14 days

Administration Guidelines :

Moxifloxacin may be administered with or without food. Tablets should be swallowed whole with an adequate amount of water. Concurrent administration with antacids or supplements containing calcium, magnesium, iron, or zinc should be avoided, as these agents may significantly reduce drug absorption and therapeutic efficacy

Table.1 Comparison Between Ethambutol and Moxifloxacin

Parameter	Ethambutol	Moxifloxacin
Drug Class	First-line antitubercular agent	Fluoroquinolone antibacterial agent
Primary Clinical Use	Management of drug-sensitive Tuberculosis	Management of multidrug-resistant tuberculosis (MDR-TB) and various bacterial infections
Mechanism of Action	Inhibits arabinosyl transferase enzymes, thereby impairing mycobacterial cell wall synthesis	Inhibits bacterial DNA gyrase and topoisomerase IV, resulting in inhibition of DNA replication and transcription
Pharmacological Activity	Primarily bacteriostatic	Bactericidal
Activity Against Mycobacterium tuberculosis	Effective against actively replicating tubercle bacilli	Demonstrates potent activity against susceptible and certain drug-resistant strains of Mycobacterium tuberculosis.
Role in Tuberculosis Regimen	Included as a standard first-line agent in the HRZE regimen.	Commonly used as a second-line or reserve drug in MDR-TB and special treatment regimens
Route of Administration	Oral	Oral and intravenous (IV)
Major Adverse Effects	Optic neuritis, decreased visual acuity, red-green color discrimination defects	Gastrointestinal disturbances, dizziness, QT interval prolongation, tendinopathy
Monitoring Requirements	Baseline and periodic ophthalmologic evaluation	ECG monitoring in high-risk patients; assessment for tendon-related adverse effects

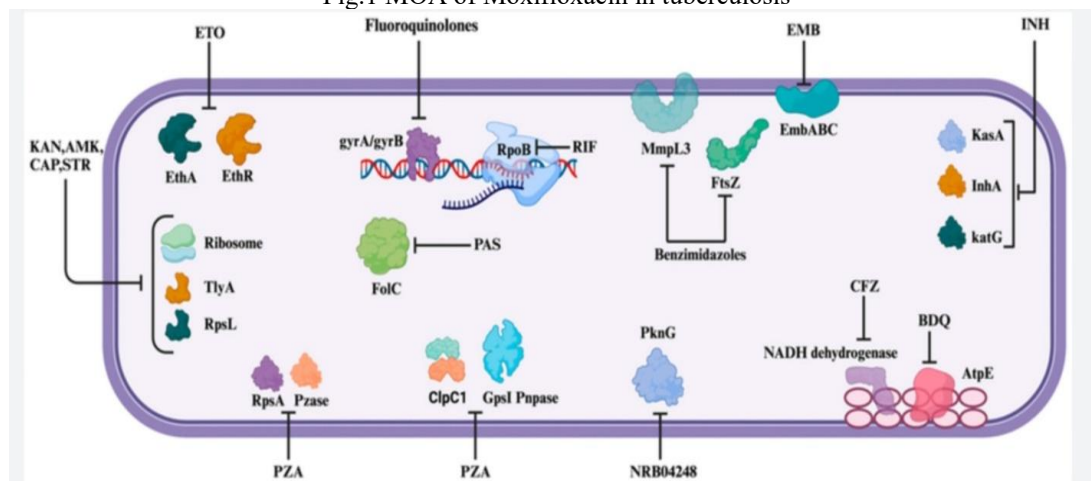
Dose Considerations	Adjustment	Dose adjustment required in renal impairment	Caution advised in hepatic dysfunction and patients with cardiac risk factor
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1.1 Mechanism of Action and Activity of Moxifloxacin Against Mycobacterium tuberculosis

Moxifloxacin, an 8-methoxy fluoroquinolone, exhibits potent bactericidal activity against a broad spectrum of Gram-positive and Gram-negative organisms, including

Mycobacterium tuberculosis. Its antimicrobial effect is mediated through inhibition of bacterial topoisomerase II (DNA gyrase), an essential enzyme involved in DNA replication, transcription, and repair. By binding to this enzyme, moxifloxacin interferes with critical bacterial DNA processes, ultimately leading to cell death.

Fig.1 MOA of Moxifloxacin in tuberculosis



The bactericidal activity of fluoroquinolones, including moxifloxacin, is believed to occur through a two-step mechanism involving the formation of quinolone–gyrase–DNA complexes followed by irreversible chromosomal fragmentation. A characteristic feature of Tuberculosis is the ability of *Mycobacterium tuberculosis* to persist in a dormant or non-replicating state, during which the organism demonstrates reduced susceptibility to many conventional antimicrobial agents. Moxifloxacin has demonstrated a unique capacity to exert antimycobacterial activity even in the absence of active protein synthesis and against dormant bacilli. This property is considered particularly important for achieving complete eradication of infection and reducing the risk of disease relapse⁸.

Moxifloxacin is a fluoroquinolone characterized structurally by the presence of a methoxy group at the C-8 position and a diazabicyclononyl ring at the C-7 position of the quinolone nucleus. The methoxy substitution at the C-8 position significantly broadens the antibacterial spectrum, particularly enhancing activity against

Gram-positive organisms and mycobacteria. In addition, compounds containing a 2,4-difluorophenyl group at the N-1 position of the quinolone nucleus demonstrate enhanced bactericidal activity, including improved efficacy against *Streptococcus pneumoniae*. Structural modifications at these positions are considered important determinants of the antimicrobial potency and spectrum of activity of fluoroquinolones.

II. Pharmacokinetics

2.1 Absorption

Moxifloxacin is rapidly and efficiently absorbed following oral administration, with an oral bioavailability exceeding 90%. This high bioavailability supports effective systemic drug exposure after oral dosing.

2.2 Drug Distribution

Suboptimal long-term treatment outcomes observed in some patients, despite negative sputum cultures indicating clearance of *Mycobacterium tuberculosis*

(MTB), suggest that persistent bacilli may remain within lung cavities, granulomas, lesions, abscesses, and other pulmonary or extra pulmonary tissue sites that are relatively inaccessible to anti-tubercular therapy. Adequate penetration of anti-tuberculosis agents into these pathological lesions is therefore considered essential for complete eradication of infection and prevention of relapse in Tuberculosis.

2.3 Metabolism and Drug Transport

Moxifloxacin undergoes hepatic metabolism primarily through glucuronide and sulphate conjugation mediated by the cytosolic enzymes glucuronosyltransferase and sulphotransferase. The principal uridine diphosphate (UDP)-glucuronosyltransferase (UGT) isoenzymes involved in the formation of the M2 metabolite are UGT1A1, UGT1A3, and UGT1A9, with UGT1A1 serving as the predominant isoform. Moxifloxacin is also a substrate of P-glycoprotein, a drug transporter protein that significantly influences its absorption, distribution, and elimination^{10,11}.

Unlike many antimicrobial agents, the cytochrome P450 (CYP450) enzyme system is neither involved in the metabolism of moxifloxacin nor affected by its administration. Additionally, neither moxifloxacin nor its metabolites demonstrate inhibitory effects on CYP450 enzymes or major human UDP-glucuronosyltransferases, thereby minimizing the potential for clinically significant metabolic drug interactions.

The sulphate conjugate metabolite (M1) accounts for approximately 38% of the administered oral dose and is predominantly excreted via the feces, whereas nearly 14% of the dose is converted to the glucuronide conjugate metabolite (M2) and eliminated through the urine. Peak plasma concentrations of metabolites M1 and M2 are reported to be less than 10% and approximately 40% of the parent drug concentration, respectively. Overall, nearly 45% of an orally administered dose is excreted unchanged, while approximately 51% is eliminated as the biologically inactive metabolites M1 and M2^{12,13,15}.

III. Pharmacodynamic Parameters

The pharmacodynamic data describe the steady-state concentrations of Moxifloxacin and evaluate how the drug behaves when administered alone or together with rifamycins such as Rifampicin or rifapentine. The studies mainly assess drug exposure using the area under the concentration–

time curve (AUC), which reflects the total amount of drug available in the body over time^{11,12}.

Key findings indicate that co-administration of rifampicin or rifapentine reduces moxifloxacin plasma concentrations due to drug interactions, potentially affecting therapeutic efficacy in Tuberculosis treatment. Different dosing schedules, including once-weekly, twice-weekly, and thrice-weekly regimens, were also evaluated to understand their impact on drug exposure and pharmacodynamic outcomes^{17,18}.

Adverse Effects and Safety Warnings of Moxifloxacin

Moxifloxacin may produce mild to moderate adverse effects during therapy. Commonly reported side effects include nausea, vomiting, abdominal pain, diarrhea, constipation, and heartburn. These reactions are usually transient; however, persistent or severe symptoms require medical evaluation. These include severe antibiotic-associated diarrhea with watery or bloody stools, which may develop during or even several weeks after treatment. Hypersensitivity reactions such as rash, urticarial, pruritus, skin peeling, blistering, facial or throat swelling, hoarseness, and difficulty in breathing or swallowing have also been reported¹⁹.

Moxifloxacin may cause hepatotoxicity, characterized by jaundice, dark urine, pale stools, or liver dysfunction. Alterations in blood glucose levels, including hypoglycemia and hyperglycemia, may occur, especially in diabetic patients, presenting with symptoms such as tremors, sweating, palpitations, blurred vision, excessive thirst, or anxiety.

Additional serious reactions include syncope, decreased urine output, abnormal bleeding or bruising, and sudden severe pain in the chest, abdomen, or back, which may indicate vascular complications. Fluoroquinolones, including moxifloxacin, have also been associated with musculoskeletal toxicity involving bones, joints, and periarticular tissues. Therefore, the drug is generally not recommended in children and adolescents below 18 years of age unless the potential benefits clearly outweigh the risks²⁰.

Drug Interactions of Moxifloxacin

1. Antacids and Mineral Supplements

Concomitant administration of antacids or supplements containing magnesium, aluminum, calcium, iron, or zinc may significantly reduce the gastrointestinal absorption of Moxifloxacin, thereby

decreasing its therapeutic efficacy. Administration should be separated by at least 2–4 hours.

2. QT Interval–Prolonging Agents

Co-administration with medications known to prolong the QT interval, including antiarrhythmic agents, macrolide antibiotics, antipsychotics, and tricyclic antidepressants, may increase the risk of ventricular arrhythmias and torsades de pointes.

3. Corticosteroids

Concurrent use with corticosteroids may potentiate the risk of fluoroquinolone-associated tendonitis and tendon rupture, particularly in elderly patients.

4. Antidiabetic Medications

Moxifloxacin may interfere with glucose homeostasis when administered with insulin or oral hypoglycemic agents, potentially resulting in hypoglycemia or hyperglycemia.

5. Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

Simultaneous use with NSAIDs may enhance central nervous system stimulation and increase the risk of seizures in susceptible individuals.

6. Warfarin and Other Anticoagulants

Moxifloxacin may potentiate the anticoagulant effect of warfarin and related agents, increasing the risk of bleeding. Regular monitoring of the international normalized ratio (INR) is recommended.

7. Theophylline

Although the interaction is less pronounced than with earlier fluoroquinolones, concomitant administration with theophylline should be undertaken cautiously because of the potential for central nervous system adverse effects.

8. Drugs Causing Electrolyte Disturbances

Medications such as diuretics that induce hypokalemia or hypomagnesemia may increase the likelihood of QT interval prolongation when administered with moxifloxacin.

9. Rifampicin

Rifampicin may reduce plasma concentrations of moxifloxacin through induction of metabolic pathways, potentially decreasing its therapeutic effectiveness in tuberculosis treatment²¹.

Clinical Study on Moxifloxacin

The study was a randomized, double-blind, placebo-controlled Phase III clinical trial designed to evaluate whether Moxifloxacin-containing regimens could successfully shorten the standard tuberculosis treatment duration from 6 months to 4 months in patients with uncomplicated, smear-positive pulmonary tuberculosis.

A total of 1,931 adult patients with newly diagnosed, drug-susceptible pulmonary tuberculosis

were randomized into three treatment groups. The control group received the standard 6-month regimen consisting of isoniazid, rifampin, pyrazinamide, and ethambutol. In the experimental groups, moxifloxacin replaced either ethambutol or isoniazid in the treatment regimen²².

The study demonstrated that moxifloxacin-containing regimens produced a more rapid initial decline in bacterial load and faster sputum culture conversion compared with the standard regimen, indicating strong bactericidal activity. However, the overall treatment success rates were lower in the 4-month moxifloxacin groups than in the standard 6-month control group. Favorable outcomes were observed in 92% of patients receiving the standard regimen compared with 85% and 80% in the two moxifloxacin-containing regimens²³.

Although moxifloxacin showed excellent antimycobacterial activity and favorable pharmacokinetic properties, the study failed to demonstrate no inferiority of the shortened 4-month regimens. Therefore, shortening tuberculosis treatment to 4 months using these regimens was not considered effective. Importantly, the incidence of severe adverse events was similar across all treatment groups, supporting the acceptable safety and tolerability profile of moxifloxacin. Overall, the trial confirmed the potent bactericidal activity of moxifloxacin but indicated that its incorporation into shortened treatment regimens alone was insufficient to replace the standard 6-month therapy for drug-susceptible pulmonary tuberculosis²⁴.

Study Procedures

Following initial screening and baseline assessment, patients were scheduled for eight consecutive weekly follow-up visits, followed by additional visits extending up to 18 months after randomization. Baseline evaluation included comprehensive clinical assessment comprising poster anterior chest radiography, pregnancy testing where applicable, collection of two sputum samples for microbiological analysis, physical examination, visual acuity testing using Ishihara and Snellen charts, and routine urinalysis.

Safety monitoring was conducted at screening and during treatment at weeks 2, 8, 12, and 17, with additional liver function assessment at week 4. Monitoring parameters included hepatic function tests such as aspartate aminotransferase (AST), alanine aminotransferase (ALT), and bilirubin levels, along with vitamin K estimation, prothrombin time, activated partial thromboplastin time, complete blood count including hemoglobin and platelet

count, renal function tests including urea and creatinine, and serum electrolyte evaluation.

For microbiological analysis, sputum specimens were decontaminated using the acetylcysteine–sodium hydroxide method, followed by microscopic examination and culture on Lowenstein–Jensen solid medium as well as in liquid culture using the Mycobacteria Growth Indicator Tube (MGIT) system²³.

Rational Use of Moxifloxacin

Rational use of Moxifloxacin refers to its appropriate, safe, and evidence-based utilization to achieve optimal therapeutic efficacy while minimizing adverse effects, antimicrobial resistance, and unnecessary healthcare costs.

Principles of Rational Use

1. Appropriate Clinical Indication

Moxifloxacin should be prescribed only for confirmed or strongly suspected bacterial infections caused by susceptible organisms. In tuberculosis management, it is primarily reserved for multidrug-resistant tuberculosis (MDR-TB) or in situations where first-line anti-tubercular agents are contraindicated or ineffective.

2. Correct Dose and Duration of Therapy

The recommended adult dosage is generally 400 mg once daily. Duration of therapy should strictly adhere to established clinical guidelines to ensure complete eradication of infection and to reduce the risk of relapse and development of resistance.

3. Use in Combination Therapy for Tuberculosis

In tuberculosis treatment, moxifloxacin should never be administered as monotherapy because isolated use may rapidly promote the emergence of resistant *Mycobacterium tuberculosis* strains. It should always be used in combination with other effective anti-tubercular agents.

4. Avoidance of Inappropriate Use

Unnecessary or irrational use, particularly for viral illnesses such as the common cold or influenza, should be avoided. Injudicious prescribing contributes significantly to antimicrobial resistance.

5. Monitoring for Adverse Effects

Patients receiving moxifloxacin should be carefully monitored for adverse reactions, including QT interval prolongation, gastrointestinal disturbances, central nervous system effects, and fluoroquinolone-associated tendon disorders. Special caution is required in elderly individuals and patients with pre-existing cardiac conditions.

6. Consideration of Drug Interactions

Potential drug interactions should be evaluated before initiation of therapy. Concurrent administration with antacids, iron, calcium, or other QT-prolonging medications should be avoided or appropriately managed to prevent reduced efficacy and increased toxicity.

7. Promotion of Patient Adherence

Patients should be advised to complete the full prescribed course of therapy even if clinical improvement occurs early. Adequate adherence enhances therapeutic success, prevents relapse, and minimizes the development of antimicrobial resistance²⁴.

Importance

Rational use of moxifloxacin is essential to optimize clinical outcomes, reduce adverse drug reactions, limit the emergence of resistant microorganisms, and preserve the long-term effectiveness of this important fluoroquinolone antimicrobial agent²⁵.

Safety Considerations of Moxifloxacin

Moxifloxacin should be administered with appropriate clinical caution to ensure therapeutic effectiveness while minimizing the risk of adverse effects and complications.

Important Safety Considerations

1. QT Interval Prolongation

Moxifloxacin has the potential to prolong the QT interval, thereby increasing the risk of serious ventricular arrhythmias, including torsades de pointes. Cautious use is recommended in patients with pre-existing cardiac disease, electrolyte abnormalities, or concurrent use of other QT-prolonging medications.

2. Tendonitis and Tendon Rupture

Fluoroquinolone therapy has been associated with tendon inflammation and tendon rupture, particularly involving the Achilles tendon. The risk is higher in elderly patients and in those receiving concomitant corticosteroid therapy.

3. Central Nervous System Effects

Moxifloxacin may cause neurological adverse effects such as dizziness, headache, confusion, insomnia, and, rarely, seizures. Patients with epilepsy or other neurological disorders should be monitored carefully during treatment.

4. Hypersensitivity Reactions

Serious hypersensitivity reactions, including rash, pruritus, urticaria, angioedema, and anaphylaxis, may occur. Immediate discontinuation of therapy is warranted if severe allergic reactions develop.

5. Gastrointestinal Effects

Common gastrointestinal adverse effects include nausea, vomiting, diarrhea, and abdominal discomfort. Rarely, severe antibiotic-associated colitis, including *Clostridioides difficile*-associated diarrhea, may occur.

6. Hepatic Toxicity

Elevation of hepatic enzymes and rare cases of severe hepatotoxicity have been reported. Liver function monitoring is recommended during prolonged therapy or in patients with pre-existing hepatic impairment.

7. Blood Glucose Disturbances

Dysglycemic events, including hypoglycemia and hyperglycemia, may occur, particularly in diabetic patients receiving insulin or oral hypoglycemic agents.

8. Use in Pregnancy and Lactation

Moxifloxacin is generally avoided during pregnancy and breastfeeding unless the anticipated clinical benefit outweighs the potential risk, owing to concerns regarding effects on fetal and neonatal cartilage development.

9. Pediatric Use

Routine use in children and adolescents is not recommended because of the potential risk of cartilage and joint toxicity associated with fluoroquinolone therapy.

10. Drug Interactions

Antacids and preparations containing calcium, iron, magnesium, or zinc may significantly reduce the absorption of moxifloxacin. Concurrent administration with other QT-prolonging agents may further increase the risk of cardiac adverse effects.

11. Clinical Monitoring

Patients receiving long-term therapy should undergo appropriate monitoring, including electrocardiography (ECG), liver function tests, blood glucose evaluation, and assessment for signs of tendon injury or musculoskeletal complications²⁶.

IV. Conclusion

Ethambutol and Moxifloxacin are important antimicrobial agents used in the management of Tuberculosis, but they differ significantly in their pharmacological class, mechanism of action, therapeutic role, and adverse effect profile. Ethambutol is a first-line antitubercular drug primarily used in standard drug-sensitive TB regimens, whereas moxifloxacin is mainly reserved for multidrug-resistant TB or special clinical situations. Ethambutol is particularly associated with optic neuritis requiring visual monitoring, while moxifloxacin carries risks such as QT interval prolongation and tendon-related toxicity. Appropriate

drug selection should be based on the type of tuberculosis, drug susceptibility pattern, patient comorbidities, and safety considerations to ensure effective and safe treatment outcomes.

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