ISSN: 2474-3658

Depina J, et al. J Infect Dis Epidemiol 2026, 12:336
DOI: 10.23937/2474-3658/1510336
Volume 12 | Issue 1
Open Access

Journal of Infectious Diseases and Epidemiology

REVIEW ARTICLE

Dermatologic Manifestations and Immune Responses of *Chlamydia trachomatis* Co-Infections: Addressing Racial Disparities in Diagnosis and Management

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Abstract

Chlamydia trachomatis (CT) is an intracellular pathogen that can infect the epithelial cells of the genital tract and lead to significant reproductive issues such as pelvic inflammatory disease (PID) and infertility in women. CT infections are one of the most prevalent sexually transmitted infections (STIs) worldwide and are commonly coinfected with Neisseria gonorrhoeae (NG). Early detection of CT and NG infections can be challenging due to many individuals presenting asymptomatically. Due to the high risk of PID and infertility associated with CT infections, it is imperative for early detection and to understand how these infections present across different ethnic groups. Current evidence indicates strong disparities in CT prevalence amongst ethnic groups. Understanding these differences is necessary to reduce the substantial lifetime risks of CT infections across different ethnic groups.

Introduction

Chlamydia trachomatis (CT) is an obligate intracellular bacterium that can infect the epithelial cells of the genital tract [1]. Globally, CT is the most common sexually transmitted infection (STI), and in the United States, CT is the most frequently reported bacterial infection, with rates continuing to rise despite public health interventions [2]. Clinicians must be well-trained to identify CT manifestations, as the infection often remains asymptomatic, particularly in women, leading to delayed diagnosis, prolonged transmission windows, and untreated chronic inflammation [3]. CT's insidious presence allows the pathogen to persist undetected within the population, creating an ever-growing reservoir that facilitates ongoing transmission and reinfection cycles [4]. Although antibiotic treatment has a 95% effectiveness for first-time therapy, untreated or recurrent CT infections can result in severe reproductive sequelae such as pelvic inflammatory disease (PID), ectopic pregnancy, and tubal infertility, imposing a significant economic and psychological burden on patients [3].

CT often co-infects human hosts with a variety of other viral and bacterial STIs, creating a synergistic environment that can alter the natural history of each pathogen. Neisseria gonorrhoeae (NG) is the second most prevalent STI globally and, similarly to CT, is a facultative intracellular bacterium that frequently presents asymptomatically [5]. Although difficult to estimate, the incidence of CT co-infection with NG is estimated at nearly 50-70% [6]. Viral co-infections are also common with CT, and include human immunodeficiency virus (HIV), human papillomavirus (HPV), and herpes simplex virus (HSV) [7]. Critically,

Citation: Depina J, Iska N, Shelestak J, et al. (2026) Dermatologic Manifestations and Immune Responses of *Chlamydia trachomatis* Co-Infections: Addressing Racial Disparities in Diagnosis and Management. J Infect Dis Epidemiol 12:336. doi.org/10.23937/2474-3658/1510336

Accepted: April 02, 2026 : **Published:** April 04, 2026

the presence of CT in genital mucosa has been shown to increase the risk of HIV acquisition and transmission by recruiting susceptible immune cells to the region. Beyond cellular recruitment, CT infection disrupts the physical integrity of the mucosal barrier itself, further lowering the threshold for viral entry and systemic dissemination [8]. Advancing dermatologists' understanding of the interactions between CT and other STIs is critical to inform preventative strategies and improve clinical management in the efforts of mitigating the disease burden of *chlamydia* co-infections on global public health.

Historically, dermatologists have been intimately tied to the study of STIs, known as venereology. CT's dermatologic manifestations primarily occur through presentations of reactive arthritis (Reiter syndrome), presenting as mucocutaneous lesions as part of the classic triad of urethritis, conjunctivitis, and arthritis [9]. Additionally, specific serovars can cause lymphogranuloma venereum, which presents with characteristic skin ulcers that can progress to chronic lymphedema and genital elephantiasis [10]. Both lymphogranuloma venereum and reactive arthritis are difficult to treat due to diagnostic challenges and limited awareness. Cutaneous manifestations, including keratoderma blennorrhagica, can often mimic more common conditions like psoriasis, leading to misdiagnosis in the absence of a thorough sexual history [11]. These difficulties are often compounded in communities of color, where variations in erythema presentation and lesion morphology, on Fitzpatrick types IV-VI, are commonly under-represented in medical literature, education, and training [12]. Limited awareness and clinical understanding highlight a significant area of need and further education for both dermatologists and other health care providers.

Furthermore, CT exhibits a critically disparate prevalence dependent on race. CT infections present at a higher rate amongst black adolescent and young adult females (5.8%) relative to their white counterparts [13]. These disparities likely reflect a complex interplay between biological, socioeconomic, and structural factors, including the "weathering" effect of chronic systemic stressors and historical medical mistrust. Furthermore, the presence of a "dermatologic desert", geographic regions with limited access to board-certified dermatologists, often correlates with areas of high STI prevalence, potentially alluding to a compounded and nuanced barrier for timely diagnosis [14]. Hispanic women and non-Hispanic black women are historically underrepresented in dermatology education and clinical case presentations, which could contribute to higher rates of misdiagnosis and delayed care. Underrepresentation in these populations can impact how STIs, like CT, are recognized and managed across different ethnic groups. Structural barriers add to the difficulty in the clinical challenges faced in communities of color. Limited access to diagnostic testing, such as LGV genotyping, disproportionately affects populations with reduced healthcare access [15]. The absence of race-stratified data on dermatologic complications due to CT in communities of color represents a critical research gap, suggesting underdiagnosis, undertreatment, and an area of care that remains unmeasured and unaddressed [15].

This review aims to evaluate the racial disparities in the diagnosis and management of dermatologic manifestations associated with CT co-infections, specifically looking at how variability in immune responses may influence clinical presentation. Furthermore, it explores how the intersection of genetic polymorphisms and social determinants of health shapes the disease trajectory in minority communities. By identifying specific barriers to timely and accurate diagnosis, this review seeks to provide a structural foundation for fostering more culturally competent and clinically adept dermatologic care [16]. We synthesize current evidence on pathophysiology, cutaneous manifestations, impacts of race, diagnostic challenges, management implications, and key gaps to guide future research and the implementation of long-overdue equitable healthcare standards that ensure optimal outcomes for all patient populations.

Pathophysiology of *C. trachomatis* in Dermatologic Conditions

Chlamydia continues to be one of the most reported bacterial STIs worldwide in both men and women. It is often seen in co-infections with other viral and bacterial STIs such as human immunodeficiency virus (HIV), human papillomavirus (HPV), and herpes simplex virus (HSV), to name a few [7]. Infections with these pathogens are well-known and studied to have long-lasting complications that can affect patients' reproductive and sexual health as well as dermatologic health; however, there is still room for expansion on how these infections can manifest differently across different ethnic groups. Importantly, the dermatologic manifestations associated with *C. trachomatis* are largely immune-mediated rather than due to direct bacterial invasion of the skin, highlighting the role of host immune response in disease expression. Due to CT being an obligate intracellular microbe, its removal and protection rely on cellular immunity, particularly involving CD4+ Th1 responses [17]. Delayed clearance and persistent intracellular infection can result in prolonged antigenic stimulation, promoting immune dysregulation and cross-reactivity that contributes to cutaneous and mucocutaneous manifestations. The effectiveness of cell-mediated immune responses can vary among different ethnic groups due to genetic and environmental factors. Variations in immune responses also have the potential to influence the variations in serovar distribution and prevalence, and severity of

different co-infections. Geisler. reported that chlamydial serovars and race can influence the transmission and effects of CT, particularly those in the black community [18]. Specific seroprevalence of *C. trachomatis* PgP3 IgG and serovar Ia was found to be the most prevalent in those of non-Hispanic blacks, Mexican Americans, and other Hispanics when compared to non-Hispanic whites [18,19]. Higher seroprevalence in these populations may reflect repeated exposure to prolonged immune activation, resulting in more robust inflammatory responses and increased tissue damage. The higher seroprevalences may be associated with ethnic and environmental differences, which can result in a more severe inflammatory response, resulting in more tissue damage and more severe clinical manifestations.

Chlamydia infections can often manifest as reactive arthritis, which has common dermatologic manifestations such as keratoderma blenorrhagicum, circinate balanitis, ulcerative vulvitis, nail and oral lesions [20]. These dermatologic manifestations are often influenced by the different serovar distributions, co-infection, and variability in immune responses across patient populations. Characteristic skin changes caused by keratoderma blenorrhagica are believed to be due to pathogenic antigens that cause a plasmocellular immune response that results in cross-reactive autoantibodies that contribute to manifestations of dermatitis, arthritis, conjunctivitis, and tendonitis [9]. This process is thought to represent molecular mimicry, in which chalmydial antigens trigger autoimmune-like inflammatory pathways affecting the skin and mucosa. The various dermatologic manifestations are often attributed to the presence of co-infections as well as the patients' immune system responsiveness. Njobvu found that co-infection of CT with HIV was noted to have an accelerated course of reactive arthritis, with manifestations often presenting earlier and becoming chronic [21]. Immunosuppression in HIV-positive patients may impair effective clearance of CT, amplifying inflammatory sequelae and worsening cutaneous disease. This could imply the need for more aggressive recognition and treatment measures for patients who are known to have a positive HIV diagnosis. Ensuring HIV positive patients are adherent to their medications can prevent the more severe and persistent symptoms of CT infections. Proper management of immunosuppressed patients can potentially help mediate some of the rarer and potentially difficult-to-diagnose dermatologic manifestations due to CT infections.

Cutaneous manifestations of *chlamydia* (CT) in ethnically diverse patient populations can present with variability in their presentation due to co-infections affecting presentation. CT conjunctivitis, although rare in adults, can present with concurrent anogenital infections of CT but is often mistaken for viral conjunctivitis due to its similar presentation [22]. Due to CT often presenting asymptomatically, its clinical presentation can often delay treatment due to it being mistaken for other disorders. Other challenges dermatologists face when trying to manage the skin manifestations of CT and other STI co-infections are partly due to knowledge gaps in the clinical presentation of dermatologic disease in skin of color at all levels of medical training [12]. Limited representation of skin of color in educational resources may further obscure early inflammatory or subtle mucocutaneous findings associated with CT infection. With the population of the US becoming increasingly diverse, addressing the knowledge gaps of STI infections is essential in understanding immune responses and early treatment interventions in patients from different ethnic groups [23]. The lack of diverse imaging of dermatologic lesions in learning materials can lead to delayed and often misdiagnoses due to a lack of awareness of overlapping signs and symptoms, as well as mimickers due to CT infections.

Immune Response to *C. trachomatis* and Co-infections

Understanding the role of the innate immune response to *chlamydia* (CT) infections is critical in patients of skin of color and who are co-infected with other pathogens. Innate immune signaling not only determines initial infection control but also influences downstream inflammatory pathways that contribute to dermatologic sequelae. CT is an intracellular pathogen that often relies on the functionality of the innate immune response, particularly the body's ability to produce a robust CD4+ Th1 response [19]. The innate immune system is the body's first line of defense against any pathogens and can be compromised in patients who are dealing with multiple infections, are on immunosuppressants, or are dealing with immunosuppressive infections like HIV. Impairment of early innate responses can permit prolonged intracellular persistence of CT, increasing the likelihood of immune-mediated cutaneous manifestations. Activation of toll-like receptor 2 (TLR2) is essential in the process of CT recognition and recruitment of other immune cells, such as neutrophils and monocytes [24]. Activation of TLRs plays a crucial role in controlling the primary infection and can often be compromised in co-infections, which can result in exacerbation of CT infections. Lehr reported that the presence of a particular cryptic plasmid has been identified in a variety of *chlamydia* servovars (A, B, D, E, L1, L2) and *C. muridarum* species and plays a role in the pathogenicity of CT infections [25]. This plasmid has been implicated in enhanced immune activation and inflammatory signaling, which may influence disease severity and tissue specific manifestations. The presence of this specific plasmid may play a role in how robustly the innate immune response is activated and how this might affect its connection to the adaptive immune response.

The adaptive immune response plays a key role in controlling and clearing out pathogens. Efficient and proper clearance of *chlamydia* (CT) infections involves upregulation of MHC II expression on epithelial cells infected with CT to mediate their clearance via CD4+ Th1 cells that express granzyme B [26]. Failure to mount an effective adaptive response can lead to persistent antigen exposure, driving chronic inflammation. Stimulation of T cells to produce granzymes can contribute to the cytotoxic abilities of these cells and aid in the clearance of CT infections. Helble reported that the specific memory cells that are first in line to respond to reinfection are tissue resident memory T cells (Trms) - these cells can respond more rapidly than other memory T cells and can help efficiently clear out reinfection [27]. Other key adaptive immune responses include the production of antibodies to assist with earlier and faster recognition of infection. Geisler reported that the predominant immunoglobulins (Ig) produced in response to CT infections were subtypes IgG, specifically IgG1 and IgG3 [28]. Production of CT-specific immunoglobulins, such as IgG1 and IgG3, can play a crucial role in CT infections; however, it is commonly known that CT is often an asymptomatic infection, which can imply that there are ways for CT to evade the host immune responses.

Chlamydia's (CT) ability to evade the host immune response can complicate the management of dermatologic manifestations such as keratoderma blennorrhagicum, especially in patients with skin of color. Rajeeve noted that the asymptomatic presentation of CT can be attributed to CT's ability to impair and "paralyze" neutrophils' ability to generate extracellular traps (NETs) and oxidative bursts, which are essential for neutrophils' ability to clear bacterial infections [29]. This evasion strategy allows CT to persist without triggering overt inflammation, delaying diagnosis while sustaining immune dysregulation. The paralysis of neutrophils can significantly impact the innate immune response, which in turn affects the adaptive immune response in patients. The lack of immune response to these infections can further impact the long-term outcome of CT infections, resulting in either severe or persistent clinical manifestations. Along with evasion of immune responses, CT has also been shown to enhance infectivity and replication of other infections such as HIV-1. Dzakah reported that cells infected with CT showed increased levels of a particular chemokine, CCL3L1, as a major regulator of co-infection of CT and HIV-1 [30]. This immunologic synergy may exacerbate inflammatory skin disease and worsen outcomes in co-infected individuals. CT's impact on a variety of different immune regulators can significantly impact the host immune response and ability to maintain a satisfactory immune response, and can significantly worsen the prognostic dermatologic conditions associated with CT.

Chlamydia's (CT) effect on the host immune responses can be greatly impacted by genetic and environmental differences across ethnic groups. Specific human leukocyte antigen (HLA) variants have been identified in African American women as a higher risk marker for CT reinfection. Olson reported that African American women with HLA-DQB1*06 were found to have a higher risk of CT reinfection and should have more frequent CT screenings [31]. HLA variations such as the one identified by Olson imply the increased risk of reinfection and severe disease outcomes in patients with specific variants. Variations that result in impaired immune responses further exacerbate future manifestations of CT and potential co-infections that can arise. Along with HLA differences, the human microbiota also plays an important role in immune surveillance and protection against pathogens. The microbiome functions as a critical modulator of mucosal immunity and inflammatory tone. When vaginal microbiota is disrupted, the pH of the vaginal environment increases, resulting in an increased risk of STIs such as CT [32]. Such disruptions of the vaginal microbiota can be due to bacterial vaginosis infections, resulting in elevated pH, which creates a more hospitable environment for bacteria to grow and disrupt natural host defenses. Microbiome dysbiosis may therefore indirectly contribute to downstream dermatologic complications through sustained immune activation. Disruptions in human microbiota can be influenced by socioeconomic and behavioral factors, but should not be considered the only factors leading to higher CT infection rates. Hulstein reported that socioeconomic and behavioral factors among different ethnic groups in the Netherlands could not fully explain the differences in CT infections due to socioeconomic status, sexual risk behavior, and sexual health-care seeking behaviors [33]. Despite the higher prevalence of CT infections among African Surinamese and Ghanaian residents of Amsterdam, Hulsltein's findings show that ethnic differences should not be the main indicator of CT risk. These findings emphasize the importance of biologic and immunologic contributors beyond social determinants alone.

Racial Disparities in Diagnosis and Management

Historically, racial diversity in medical research and education has been underemphasized despite being a crucial aspect of understanding a growing and diverse population. This lack of representation has direct implications for recognizing inflammatory and infectious dermatologic conditions across diverse skin tones. The epidemiologic prevalence of sexually transmitted infections (STIs) and particularly *chlamydia* (CT), across different racial and ethnic groups, exemplifies this gap. Notably, non-Hispanic black individuals account for 32.4% of all cases of CT, gonorrhea, and primary and secondary syphilis despite only making up approximately 13% of the population [34]. Chambers reported that for

women born between 1980 and 1984, the lifetime risk of CT diagnosis was 64.9% for non-Hispanic black women, compared to just 14% for non-Hispanic white women [35]. Higher baseline prevalence increases cumulative inflammatory exposure, which may contribute to more frequent or severe dermatologic immune path. These disparities reflect how high community prevalence can increase the likelihood of being exposed to an affected individual, leading to a cascade effect [34]. Multiple factors contribute to this phenomenon, including differential access to healthcare, variations in sexual behavioral patterns, and individual desire to seek care. Cultural and systemic barriers in dermatology remain a significant obstacle for many patients, especially as only 3% of US dermatologists identify as black [36]. The underrepresentation of ethnically diverse dermatologists and the persistent gaps in dermatologic education that inadequately address skin conditions in patients with skin of color result in diagnostic delays and a disproportionate burden to patients who, in turn, seek numerous consultations for an accurate diagnosis [36,37]. The combination of these factors presents real and significant burdens that are faced by the black community to seek care and to receive medical advice from dermatologists who have experience with their specific needs.

Beyond the challenges of seeking timely and adequate dermatologic care, as well as proper diagnoses, there are additional challenges in the management of dermatologic conditions due to *chlamydia* (CT) infections. Management disparities may also influence treatment adherence, antibiotic access, and timely monitoring of cutaneous disease. Patients with lower income and educational levels, particularly among Hispanic and black patients, were less likely to visit dermatology clinics overall [38]. Other barriers contributing to lower attainment of dermatologic care by Hispanic and black patients were often due to insurance-related barriers. Receiving insurance coverage and approval for a dermatologic visit was decreased among those with public insurance, more commonly held by black and Hispanic patients, compared to private insurance holders [39,40]. Reduced access to specialty care may result in under-recognition of atypical or subtle dermatologic presentations associated with CT infections. Regardless of patients' intent to seek medical attention for dermatology-related issues, factors such as insurance denials and an inability to continue with follow-up care can result in improper management and suboptimal care for patients who are either Hispanic or black. Failure to obtain proper and timely dermatologic care in black and other marginalized communities is an area of active research, as the full scope of the repercussions is not fully understood. These systemic barriers may contribute to delayed treatment initiation, recurrent infection cycle, and more persistent dermatologic disease. Future research should explore treatment gaps, long-term management, and downstream effects of delayed or improper diagnosis of infections, such as *chlamydia* and its effect on co-infections in minoritized population groups. Efforts in creating more equitable access to care are essential in addressing the complex interplay of racial disparities and how they can affect access to care, socioeconomic status, and provider education to improve clinical outcomes for those disproportionately affected by preventable dermatologic infectious diseases. Improving provider education, expanding representation in dermatologic imagery, and increase equitable access to STI screening and dermatologic evaluation are essential steps towards reducing disparities in CT-associated skin disease outcomes.

Future Directions

Addressing disparities in the clinical presentation and management of *chlamydia* (CT) and its dermatologic manifestations requires a multifaceted approach focusing on expanding access to care, advancing diagnostic approaches, and enhancing medical education to ensure more equitable outcomes across all populations.

Efforts to expand healthcare access are essential in closing the gap in sexually transmitted infection (STI) outcomes between different racial and ethnic groups. Increasing healthcare availability in underserved areas through mobile clinics, community-based screening initiatives, and federally supported health services can help reduce delays in diagnosis and treatment. The growing role of telemedicine, particularly in dermatology, presents an opportunity to access patients who may experience geographical or logistical barriers to specialized care, such as dermatology. Virtual consultations can facilitate earlier identification of dermatologic symptoms and improve continuity of care. Allowing patients the opportunity to meet with their providers virtually can help eliminate some of the potential causes that result in patients being lost to follow-up appointments, such as unreliable transportation and financial barriers. Healthcare policy changes that support insurance equity, remove administrative hurdles to specialist care, and fund public health initiatives aimed at high-risk populations can further reduce structural barriers that limit access. Investing in programs that work to provide equitable access to care and education is instrumental in mitigating the high-risk sequelae of common infections, such as CT, that are known to have positive outcomes with early detection and treatment.

The development and implementation of comprehensive and standardized STI testing protocols are crucial for early and accurate detection, especially in patients with co-infections or atypical presentations. Incorporating multiplex testing and expanding access to point-of-care diagnostics can improve screening

efficiency. The continuous growth and diversity of the US population continue to contribute to the ever-changing immunologic gene pool, implicating health and disease susceptibility [41]. The change in the genetic landscape in the US could potentially influence the prevalence and severity of different STIs and co-infections, resulting in the continued need for continuous education and research. Expansion of diagnostic tools and clinical algorithms that reflect population-specific differences in immune response, serovar prevalence, and co-morbid conditions should be included in future steps to address the racial differences seen in STI infections. Tailored diagnostic approaches considering ethnic and environmental factors can lead to more precise treatment and better disease outcomes. Enhancing health equity, communication strategies, medical school, and residency curricula can better equip providers to deliver effective care to patients of all backgrounds.

Lastly, improving education for future dermatologists and physicians who are trained to recognize and manage dermatologic conditions in diverse populations is fundamental to reducing disparities in minoritized populations. Current gaps in dermatologic education of conditions that appear in darker skin tones can significantly contribute to delayed or missed diagnoses, leading to severe adverse outcomes [42]. The lack of formal training at all educational levels can result in physicians who are not comfortable in providing quality care to minoritized groups,m resulting in the potential to distrust medical professionals among patients. Janodia reported the increasing need for medical schools to increase patient demographics when designing their curricula to better prepare medical students for future practice [43]. Expanding the representation of skin of color in educational resources, such as textbooks, image databases, and clinical training scenarios, is necessary to improve diagnostic accuracy across patient populations. Beyond clinical recognition, education must also emphasize cultural competency. Narla expresses a critical need for dermatologists to be aware of the existing racial and ethnic disparities that are experienced by their minoritized patients [44]. The current gaps in medical education on both the clinical and cultural sides of minoritized populations hinder dermatologists' ability to provide equitable care. Medical schools and residency programs play an important role in providing more equitable and diverse learning opportunities to better bridge this gap. Incorporation of structured training in culturally responsive care, communication strategies, and awareness of systemic health inequities can better prepare physicians to deliver empathetic and effective care across diverse communities.

Conclusion

This review highlights the critical importance of recognizing the dermatologic manifestations of *chlamydia trachomatis* (CT) and its co-infections, particularly as they vary across racial and ethnic groups. Immunologic variability, atypical presentations, and systemic healthcare inequities often contribute to delays in diagnosis, misdiagnoses, and suboptimal treatment in minoritized populations. These challenges are further exacerbated by limited cultural competence amongst providers, gaps in medical education regarding manifestations across diverse skin tones, and inadequate access to affordable dermatological care, resulting in poorer outcomes for underserved communities.

A multi-step approach should be taken to improve dermatologic care and access for minoritized populations. Improvement in access to dermatologic care and STI management will require dermatologists to encourage and advocate for their patients outside of the clinic. Advocating for expansion and improvements in areas such as teledermatology and community clinics in underserved areas will increase access and promote more community engagement with patients. Medical institutions such as medical schools and residency programs should encourage policymakers to advocate for bills that promote dermatologic research advancement, which work towards overcoming diagnostic challenges commonly faced by dermatologists. Equipping dermatologists with better diagnostic tools will have the potential to reduce diagnostic delay, enhance provider confidence, and decrease STI prevalence in minoritized groups. Medical institutions also play a critical role in providing quality education to future and current dermatologists. The purposeful incorporation of more diverse patients in medical education can help to establish more competency in physicians and trust in patients. This multifaceted approach can reduce diagnostic delays, enhance provider confidence in identifying dermatologic conditions across all skin tones, and improve overall delivery of care. Prioritizing culturally competent training, expanding telemedicine in dermatology, and investing in research focused on diverse populations are critical next steps. These efforts will begin to create equity in dermatologic sexually transmitted infection outcomes and ensure that all patients, regardless of their race and ethnicity, receive gold-standard care.

Sources of Support

The authors declare that no funding was received from the National Institutes of Health (NIH), the Wellcome Trust, or any other organization for the preparation of this manuscript.

Statement of Equal Author Contributions

Each author of this manuscript contributed equally to this work and share co-authorship.

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