Review Article

# Pyogenic Brain Abscess: A Comprehensive Review of Epidemiology, Pathogenesis, Diagnosis, and Management

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## Abstract

**Background:** Pyogenic brain abscess is a severe neurological infection associated with significant morbidity and mortality. Despite advances in diagnostic techniques, neurosurgical procedures, and antimicrobial therapy, managing pyogenic brain abscesses remains challenging. This article reviews the current understanding of the epidemiology, pathogenesis, diagnosis, and treatment of pyogenic brain abscesses, highlighting the importance of a multidisciplinary approach to improve patient outcomes.

**Materials and Methods:** A comprehensive literature review was conducted using PubMed, Scopus, and Google Scholar databases. The search terms included "pyogenic brain abscess," "epidemiology," "pathogenesis," "diagnosis," "treatment," "antibiotic therapy," and "prognosis." Relevant articles published in English between 2010 and 2023 were selected, focusing on the most recent advances and evidence-based recommendations for managing pyogenic brain abscesses.

**Results:** Pyogenic brain abscess is a life-threatening condition that requires prompt diagnosis and treatment. A multidisciplinary approach involving neurosurgeons, infectious disease specialists, and radiologists is necessary to manage pyogenic brain abscesses successfully.

**Conclusion:** Early recognition, appropriate antibiotic therapy, and timely neurosurgical intervention are essential for improving patient outcomes and minimizing neurological sequelae. Antibiotic therapy and surgical approach should be tailored to the individual patient, considering factors such as the suspected pathogens, immune status, and the primary source of infection. Long-term follow-up is crucial, as recurrence and neurological sequelae are common among survivors. Continued research is needed to improve our understanding of this complex condition and develop more effective treatment strategies.

**Keywords:** Pyogenic brain abscess, Intracranial infection, Neuroimaging, Antibiotic therapy, Neurosurgical intervention

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# Introduction

A brain abscess is a severe intracranial infection characterized by a localized collection of purulent material within the brain parenchyma. The clinical presentation of brain abscesses can be highly variable, making diagnosis and management challenging. In recent years, significant advancements have been made in imaging technologies, laboratory diagnostics, minimally invasive neurosurgical procedures, and antimicrobial therapy. These developments have greatly impacted the diagnosis and treatment of brain abscesses. However, despite these advances, managing brain abscesses can still be complex and demanding in certain cases. This review comprehensively examines literature using PubMed, Scopus, and Google Scholar databases. The search terms included "pyogenic brain abscess," "epidemiology," "pathogenesis," "diagnosis," "treatment," "antibiotic therapy," and "prognosis." Relevant original articles, case reports, and reviews published in English between 2010 and 2023 were selected, and a total of 132 articles were selected. The current understanding of the epidemiology, diagnostic approaches, and treatment strategies for brain abscesses sheds light on recent advances and ongoing challenges in this field.

**Epidemiology:** Brain abscesses can manifest at any age, though most cases occur during the third to fifth decades of life [1]. Contemporary research has revealed substantial variability in the mean age of presentation, spanning 24 to 57 years, with developed nations generally reporting higher average ages than developing countries[2,3]. A consistent finding across the literature is a significant male predominance, regardless of geographical location[3,4]. In the United States, brain abscesses occur in approximately 1500–2500 cases per year[5,6]. Studies have consistently shown a male predominance, with male-to-female ratios ranging from 1.3:1 to 3.0:1[4,7,8].

**Predisposing factors:** In the majority of cases, brain abscesses develop as a result of predisposing factors, such as underlying diseases (e.g., human immunodeficiency virus HIV), the use of immunosuppressive drugs, disruption of the natural protective barriers surrounding the brain (e.g., due to trauma, mastoiditis, sinusitis, or dental infection), or a systemic source of infection (e.g., endocarditis or bacteremia)[9]. Studies showed that Diabetes mellitus was the most common comorbidity [4]. Bacterial species frequently implicated in cases of contiguous spread include *Staphylococcus aureus* and both anaerobic and aerobic *Streptococcus* strains[4,9]. Nevertheless, reports have also identified other microorganisms, such as *Enterobacteriaceae* (including *Klebsiella* and *Proteus*)[10] and *Salmonella* species[11], as causative agents in some instances. Hematogenous bacterial dissemination can occur in patients with endocarditis[12], congenital heart defects, pulmonary infections, or dental infections, accounting for approximately one-third of brain abscess cases[13]. In instances related to endocarditis, the typical causative pathogens are *Staphylococcus aureus* and *Streptococcus* species[12,13]. However, brain abscesses arising from underlying dental or sinus infections are frequently polymicrobial, potentially involving microorganisms such as *Fusobacterium*, *Actinomyces*, *Bacteroides*, and *Haemophilus* species [14]. Hereditary hemorrhagic telangiectasia (Osler–Weber–Rendu disease) is also associated with the development of brain abscesses [15]. Such increased susceptibility is likely attributable to pulmonary arteriovenous malformations, which enable septic microemboli to circumvent the pulmonary filtration system and access the cerebral vasculature. HIV infection is associated with brain abscesses caused by *Toxoplasma gondii* and *Mycobacterium tuberculosis* infection[16,17]. HIV testing should be recommended for all patients presenting with brain abscesses, as cerebral toxoplasmosis may represent the initial opportunistic infection in previously undiagnosed HIV cases[18]. Solid organ transplantation constitutes a significant risk factor for brain abscesses caused by *Nocardia* species and fungal abscesses caused by *Aspergillus* or *Candida* species[19]. Notably, up to 90% of cerebral abscesses among solid organ transplant recipients are attributable to fungal infections[19]. Brain abscesses can also develop following neurosurgical procedures[20].

**Pathogenesis:** Brain abscesses begin with a localized area of cerebritis and subsequently evolve into suppurative lesions surrounded by vascularized fibrotic capsules. The stages of abscess formation can be visualized using CT scans and MRI[21]. The stages are

**Table 1.** Stages of Brain Abscess Formation[21].

| Stages | Characteristics |
|---|---|
| **Early stage or early cerebritis (Days 1-3)** | This stage is characterized by the accumulation of neutrophils, edema, and tissue necrosis. Additionally, the activation of microglia and astrocytes is evident during this stage. |
| **Intermediate or late cerebritis (Days 4-9)** | The events of this stage are predominantly linked to the infiltration of macrophages and lymphocytes. |
| **Early capsule formation (Days 10-13)** | This stage is associated with the formation of a well-vascularized abscess wall that isolates the infection. The capsule tends to be thinner on the medial or ventricular side of the abscess and is prone to rupture in this direction. |
| **Late capsule formation (After Day 14)** | During this stage, the capsule develops gliotic, collagenous, and granulation layers. |

**Table 2.** Organisms Associated with Specific Etiologies[34].

| Etiology of the abscess | Organisms |
|---|---|
| **Otogenic abscesses** | *Proteus, Streptococcus milleri* group microorganisms, and *Streptococcus pneumoniae* (9,26,31) |
| **Paranasal sinusitis** | *Streptococcus spp, Staphylococcus spp,* and less frequently, *Enterobacteriaceae* (9,33) |
| **Traumatic brain injury** | *Staphylococcus aureus, Staphylococcus epidermidis,* and Enterobacteriaceae (32) |
| **Neurosurgical procedures** | *S. aureus, S. epidermidis, P. aeruginosa,* and *Propionibacterium acnes* (7,32) |

shown in table 1. Although the immune system plays a crucial role in limiting the extent of infection, it can also cause damage to the surrounding normal brain tissue[22]. It has been proposed that the persistent production of pro-inflammatory mediators following *S. aureus* infection effectively increases damage to the surrounding normal brain parenchyma[23]. This collateral damage highlights the delicate balance between the immune response's protective and potentially harmful effects in the context of brain abscesses[24].

The location of brain abscesses often correlates with the preceding infection. Abscesses associated with otitis media and mastoiditis tend to develop in the inferior temporal lobe and cerebellum[25,26]. On the other hand, abscesses associated with sinusitis primarily occur in the frontal or temporal lobes[27,28]. The frontal lobe is also commonly affected following mandibular dental infections[29].

**Microbiology:** According to a meta-analysis, aerobic bacteria are found more frequently than anaerobes, with *Streptococci* being the most common among aerobes[11]. *Peptostreptococcus spp* and *Bacteroides fragilis* are the most often found anaerobic pathogens [30]. *Streptococci* were the most abundant species among cultured microorganisms (33% - 35%), with the viridans group (*S. mutans, S. constellatus, S. sanguinis, S. mitis, and S. salivarius*) being the most frequently isolated[31]. *Streptococcus pneumoniae* was the causative agent in only 2.4% of patients[32]. *Staphylococcus spp.* was the second most common group, causing 18% of cases, with *S. aureus* accounting for 84% and *S. epidermidis* for 16% of the identified *Staphylococcus* species[33]. Gram-negative enteric bacteria (*Klebsiella spp, Proteus spp, Enterobacteriaceae, and Escherichia coli*) accounted for 15% of cases and were more frequently found in polymicrobial brain abscesses[34]. There was no significant difference in the bacteria cultured from adults and children[35,36]. The causative organisms, according to the etiology of the abscess, are shown in Table 2.

## Diagnosis

***Symptoms*:** Pyogenic brain abscess presents with diverse clinical manifestations that can vary based on the abscess's location, size, and underlying etiology.

**Table 3.** Most common clinical presentations of pyogenic brain abscess[33,34].

| Symptoms | Prevalence (% of cases) |
|---|---|
| **Headache** | 49% to 93 |
| **Fever** | 14% to 88 |
| **Altered mental status** | 33% to 70 |
| **Focal neurologic symptoms** | 29% to 71 |
| **Nausea and vomiting** | 26% to 71 |
| **Seizures** | 2% to 49 |
| **status epilepticus** | 4% to 23 |

**Table 4.** typical characteristics of imaging studies at different stages of abscess formation[39-41].

| Imaging method | Typical characteristics of imaging |
|---|---|
| **Computed Tomography (CT)** | Early cerebritis phase: show localized hypoattenuation, and contrast enhancement, if present, may exhibit a nodular or ring-like pattern that remains unchanged or progresses on delayed images.<br>Late cerebritis phase: show an area of hypoattenuation, and contrasted images demonstrate a thick ring-like or nodular enhancement.<br>Capsule formation stage: A round or ovoid area of hypoattenuation with ring enhancement that dissipates on delayed scans |
| **Magnetic Resonance Imaging (MRI)** | Early cerebritis phase: Poorly defined hyperintensity on T2-weighted sequences and hypo intensity on T1-weighted sequences.<br>Late cerebritis phase: Localized fluid collections appear hyperintense relative to CSF and hypointense relative to the surrounding white matter on T1-weighted images.<br>Capsule formation stage: A smooth contrast-enhancing capsule, isointense to hyperintense relative to white matter on T1 images and isointense to hypointense on T2 images, is evident. |
| **Diffusion-Weighted Imaging (DWI)** | The central non-enhancing portion of an abscess has diffusion restriction, appearing hyperintense on diffusion-weighted sequences and hypointense on apparent diffusion coefficient maps. |

The most common clinical presentations of pyogenic brain abscesses are summarized in Table 3.

***Laboratory findings*:** Laboratory data have limited utility in diagnosing brain abscesses. While leukocytosis and elevation in erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are common, the absence of these laboratory abnormalities does not exclude the diagnosis[33,34]. Blood cultures (40-60 mL) should be collected before initiating antibacterial treatment, as they were found to be positive in 28% of patients with brain abscesses in a recent meta-analysis[37]. The potential value of identifying the organism is substantial in circumstances where collection of abscess material cannot be performed promptly or is not advisable due to associated risks[38]. Cerebrospinal fluid (CSF) analysis may reveal pleocytosis, elevated protein, and decreased glucose. However, it will be normal in a significant proportion of individuals, and CSF culture is infrequently positive, ranging from 0% to 43%[37].

***Brain Imaging*:** Brain imaging is critical for diagnosing and managing brain abscesses and is crucial in improving patient outcomes. Table 4 and Figure 1 illustrate the typical imaging study characteristics at different stages of abscess formation[39-41].

**Treatment**

In patients with an intact mental state, without any signs of increased intracranial pressure (ICP), and with abscesses smaller than 2.5 cm, purely medical management could be attempted[42]. However, this approach should not be considered in high-risk patients, such as those with hydrocephalus or significant mass effect attributable to infratentorial abscesses[43]. Currently, most authors recommend adding vancomycin to a third-generation cephalosporin and metronidazole[37,42,44]. In patients with risk factors for *pseudomonas* infection, an appropriate cephalosporin, like ceftazidime or cefepime, or a carbapenem such as meropenem, is recommended[11,37]. Due to the wide range of potential pathogens involved in brain abscesses, antimicrobial therapy should cover both gram-negative and gram-positive bacteria, therefore, it is recommended that all patients with brain abscesses receive an extended-spectrum cephalosporin (such as cefotaxime or ceftriaxone) in combination with metronidazole as empiric treatment[37]. This regimen was found to be used in the majority (53%) of patients in the studies analyzed. 16% of patients received only antibiotic treatment without surgical intervention[20,45]. The decision to use corticosteroids should be made on a case-by-case basis, considering the potential risks and benefits. However, some retrospective case series show a relationship between steroid administration and poor outcomes[46].

***Treatment Considerations for Immunocompromised Patients:*** For patients with HIV, the standard treatment regimen for *Toxoplasma* coverage typically involves the use of pyrimethamine and sulfadiazine. Alternatively, clindamycin may be used[16]. In neutropenic patients, some clinicians suggest adding amphotericin to the treatment regimen due to the increased risk of fungal infections in this population. Other studies propose supplementing with voriconazole, trimethoprim-sulfamethoxazole, or sulfadiazine to provide coverage against fungi, yeasts,

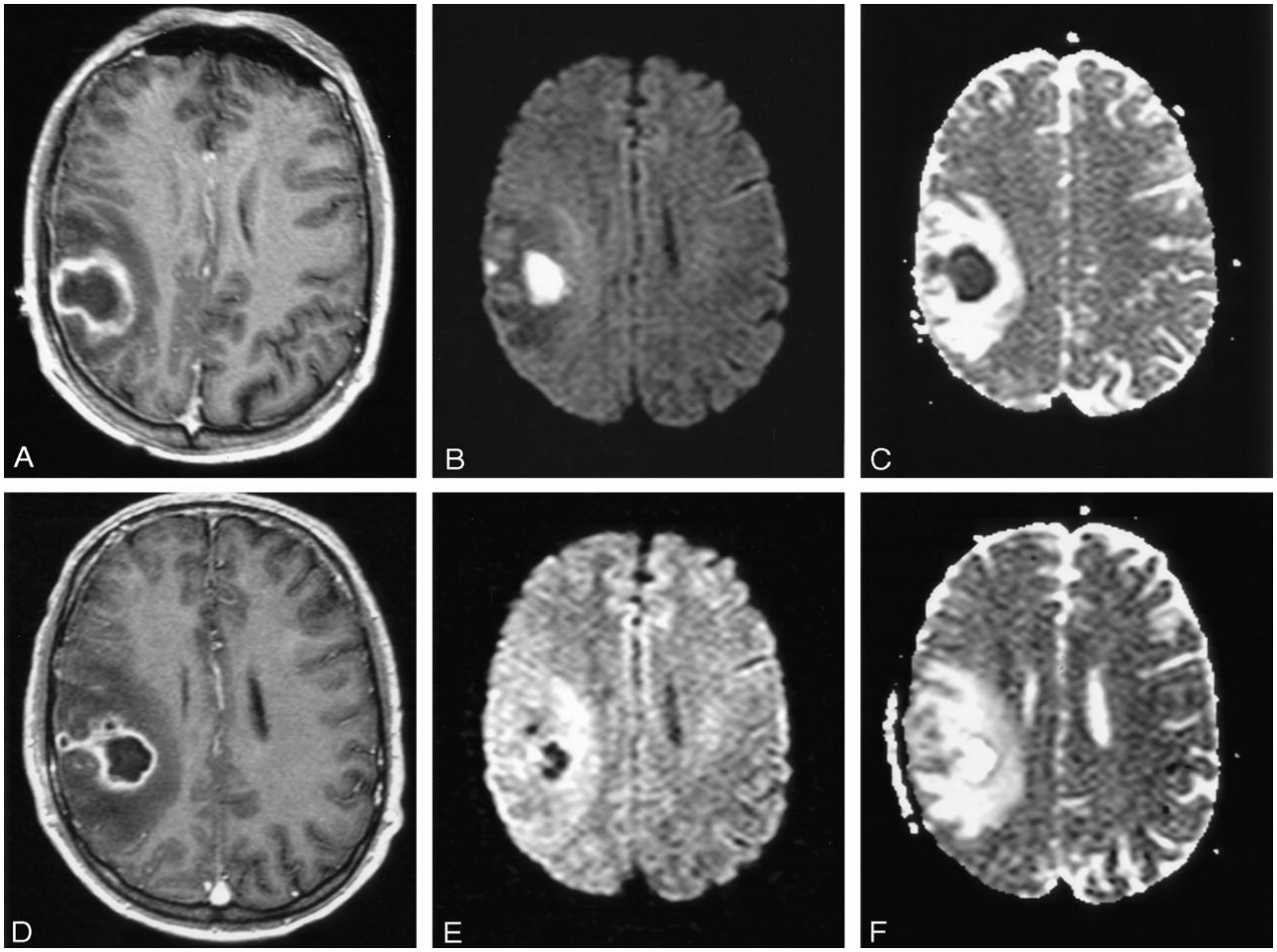


**Figure 1.** Axial contrast-enhanced T1-weighted imaging (peripheral enhancing lesion in the right parietal region) (A); Hyperintense DWI (B); a low ADC (C); decreased size of the abscess with hypo intensity at DWI and a high ADC (D-F)[40].

and *toxoplasmosis* until further diagnostic tests can be conducted[17].

***Duration of Antibiotic Therapy:*** It is advised that intravenous antibiotics should be continued for at least 6-8 weeks, followed by 2-3 months of oral antibiotics after the termination of intravenous therapy[44,47]. The European Society of Clinical Microbiology recommends a shorter duration of intravenous therapy, typically 1-2 weeks, for bacterial brain abscesses[37]. If the patient shows a good clinical response, a change to an appropriate oral regimen can be considered[37,47].

**Table 5.** The mortality rate of pyogenic brain abscess based on geographical location[48-50,54,59]**.**

| Geographical Location | Mortality rate (%) |
|---|---|
| **Europe** | 22 |
| **Americas** | 31 |
| **Australia** | 25 |
| **Asia** | 15 |

For immunocompromised patients, the duration of parenteral treatment for brain abscesses should generally be longer compared to immunocompetent patients. However, there is little consensus on the optimal duration of treatment for this population. The recommended range is typically between 12 weeks to 1 year, but the specific duration may vary depending on individual patient factors and the clinical judgment of healthcare professionals. Close clinical and imaging follow-up is important due to the uncertainty

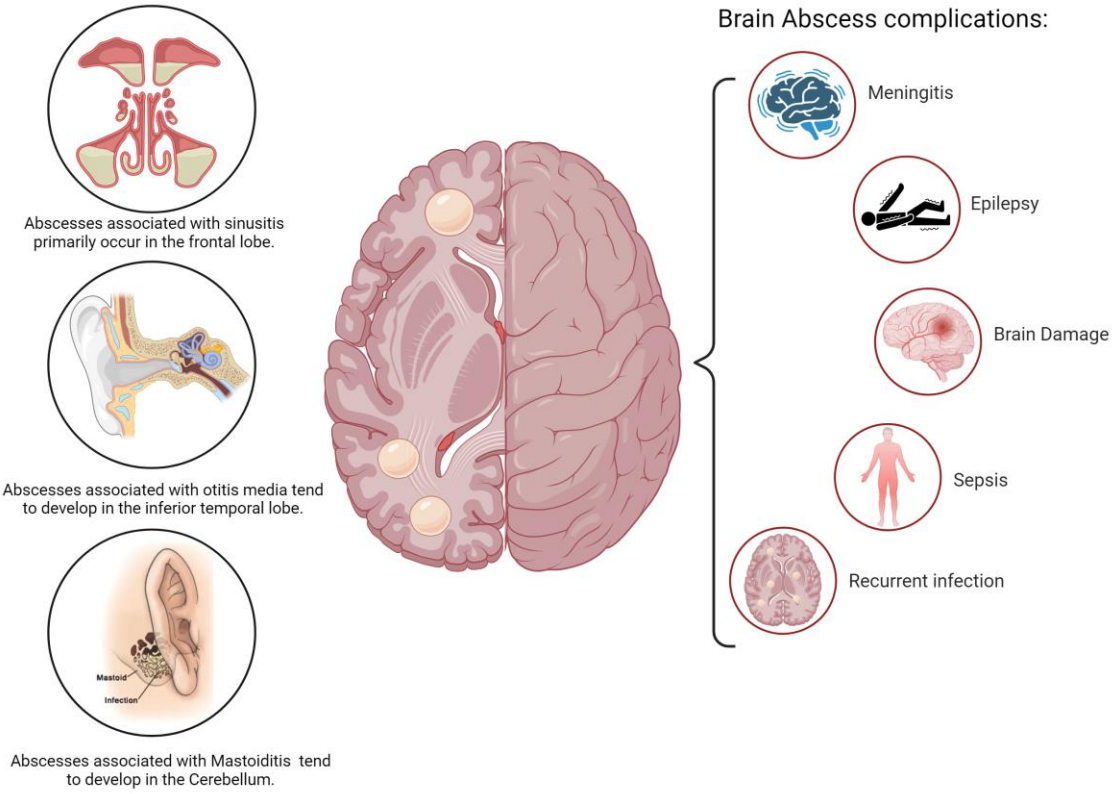


**Figure 2.** Brain Abscesses associations and complications.

surrounding the required duration of therapy in each case[37].

**Outcome and prognosis:** The prevalence of mortality is summarized in Table 5. With advancements in cranial imaging, microbiological technology, minimally invasive neurosurgical procedures, and improved antibiotic treatment, the overall outcome of patients with brain abscesses has gradually improved over the past 20 years[48-50].

When the diagnosis is made 'in time' and basic management rules are applied, the cure rate will be > 90%[51]. However, antibacterial resistance can make poor prognosis more challenging[52,53]. 70% of patients with brain abscesses have a good outcome, with no or minimal neurologic sequelae[54]. The key prognostic factors were level of consciousness at admission and presence of comorbidity[34], and a much poorer prognosis was reported for patients presenting with lower Glasgow Coma Scale (GCS) scores[34,54]. Long-term sequelae occur in about one-third of patients and include mental retardation, seizures, and focal neurologic deficits[55,56]. The frequency of neurological sequelae in survivors is 20–79% and relates to how quickly diagnosis is made and antimicrobials are administered[37]. Seizures are a long-term risk in a significant proportion of patients with brain abscesses, ranging from 30% to 50%[55,56]. The latency period, or the time between the resolution of the abscess and the occurrence of seizures, can be as long as five years but is typically shorter in older patients[57,58]. The use of prophylactic anticonvulsant drugs in brain abscesses has not been specifically evaluated. However, in other space-occupying lesions in the brain, such as tumours, prophylactic anticonvulsant treatment did not show a significant decrease in seizure rates[55,58]. In cases of tumoral lesions, including brain abscesses, where antiepileptic treatment is initiated after a seizure, antiepileptic prophylaxis should be started immediately and continued for at least one year due to the high risk of subsequent seizures[55,58].

## Conclusion

Pyogenic brain abscess remains a significant challenge in modern medicine despite advancements in diagnostic techniques, neurosurgical procedures, and antimicrobial therapy. Early recognition, prompt initiation of appropriate antibiotics, and timely neurosurgical intervention are crucial for improving patient outcomes and minimizing neurological sequelae. Managing pyogenic brain abscesses requires a multidisciplinary approach involving neurosurgeons, infectious disease specialists, and radiologists. The choice of antibiotic therapy should be based on the suspected pathogens, the patient's immune status, and the primary source of infection. Empiric antibiotic therapy should cover aerobic and anaerobic bacteria, adjusting based on culture results and clinical response. The duration of antibiotic treatment typically ranges from 4 to 8 weeks, with a combination of intravenous and oral therapy. Surgical intervention, either through stereotactic aspiration or open craniotomy, Surgical intervention plays a vital role in the management of pyogenic brain abscesses. The specific surgical approach chosen depends on several factors, including the size, location, and multiplicity of the abscesses and the patient's clinical status.

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## Conflict of interest

The authors further declare that they have no conflict of interest.

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