Global, regional, and national burden of meningitis, its risk factors, and aetiologies, 1990–2023: a systematic analysis for the Global Burden of Disease Study 2023

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Summary

Background: Meningitis remains the leading infectious cause of neurological disabilities globally, disproportionately affecting children under 5 and populations in the African meningitis belt. While previous global estimates focused on a limited number of pathogens, this study presents the most comprehensive analysis to date, assessing burden attributable to 17 causative pathogens using the Global Burden of Disease (GBD) Study 2023 framework.

Methods: GBD is a systematic, scientific effort aimed at quantifying the comparative magnitude of health loss caused by diseases, injuries, and risk factors across age groups, sexes, and geographical locations over time. We estimated meningitis mortality using the Cause of Death Ensemble model (CODEm) and morbidity using DisMod-MR 2.1, incorporating data from vital registration, verbal autopsy, surveillance, hospital data, and systematic reviews. Aetiology-specific estimates were generated using pathogen-linked CFRs and splined binomial regression models. Risk factor attribution was based on established risk–outcome pairs and population attributable fractions.

Findings: In 2023, there were 259,000 (95% UI 202,000–335,000) global deaths and 2.54 million (2.20–2.93) incident cases of meningitis. Children under 5 accounted for over one-third of deaths (86,600 [53,300–149,000]). *Streptococcus pneumoniae*, *Neisseria meningitidis*, non-polio enteroviruses, and other viruses were the leading causes of death, while non-polio enteroviruses caused the most cases. The four WHO-defined preventable meningitis pathogens of interest (*Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus influenzae,* and Group B streptococcus) contributed to 98,700 deaths (77,000–127,000) and 594,000 cases (514,000–686,000). Low birthweight, short gestation, and household air pollution were the top risk factors. Although mortality and incidence have declined significantly since 1990, progress is insufficient to meet WHO 2030 targets.

Interpretation: Despite marked progress in reducing bacterial meningitis via global vaccination campaigns, substantial meningitis burden persists, attributable both to common pathogens of great public health significance such as *Streptococcus pneumoniae* and *Neisseria meningitidis*, and to emerging non-bacterial pathogens like *Candida* spp. and drug-resistant fungi. The predominance of non-polio enteroviruses in incidence underscores the evolving landscape of meningitis in the post-*H influenzae* type b and pneumococcal vaccine era. Achieving WHO goals will require sustained investment in surveillance, vaccination, maternal screening, and health system strengthening, especially in high-burden settings.

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Research in Context

Evidence before this study

The global burden of meningitis and its aetiologies has been quantified by different groups, including WHO and the Maternal and Child Epidemiology Estimation Group (WHO-MCEE) and the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD). We conducted a PubMed search using the terms "meningitis" [MeSH] AND ("mortality" OR "incidence") AND "risk factors" AND "global" for studies published from database's inception to February 3, 2026. Of the 47 resulting studies, seven reported on one meningitis-causing pathogen, three reported on two pathogens, and one study examined three pathogens. None of these studies quantified meningitis attributable to *Acinetobacter* baumanni, *Candida* spp., coagulase-negative staphylococci, or non-polio enteroviruses. The most comprehensive study to date has been the GBD 2019 meningitis study, which estimated 2·51 million (2·11–2·99) cases of meningitis in 2019, and 236 000 deaths (204 000–277 000) attributable to meningitis globally.

Added value of this study

This study provides the most comprehensive global assessment of meningitis to date, expanding pathogen coverage from 10 to 17, including the first global quantification for non-polio enteroviruses (the leading cause of meningitis incidence), *Acinetobacter* baumanni, *Candida* spp., coagulase-negative staphylococci, the aggregate categories of other fungi, and other *Streptococcus* species. For the first time, we assessed meningitis deaths attributable to risk factors, including low birthweight, short gestation, and household air pollution, providing evidence to inform prevention strategies in maternal, child, and environmental health. The manuscript also assesses progress toward the WHO global roadmap by quantifying and trending the combined burden of the WHO priority preventable pathogens (*S. pneumoniae*, *N. meningitidis*, *H. influenzae*, Group B streptococcus) from 2015 onward, showing that current rates of decline are insufficient to meet global targets.

Implications of all available evidence

WHO has set a goal to reduce global vaccine-preventable bacterial meningitis cases by 50% and deaths by 70% by 2030, compared to a baseline year of 2015. Though there have been substantial improvements in reducing the morbidity and mortality of meningitis, the pace of progress is not currently on track to meet these goals by 2030. The two leading causes of meningitis mortality globally, *Streptococcus pneumoniae* and *Neiserria meningitidis,* are both vaccine-preventable bacterial species and serve as notable examples of the need for more comprehensive vaccine coverage programmes. In addition, viral meningitis poses a rising relative burden in the post-*H influenzae type b*, pneumococcal, and meningococcal vaccine era. We find that non-polio enteroviruses, which cause a less severe phenotype and a lower likelihood of mortality than bacteria are the number one cause of meningitis incidence and the third leading cause of meningitis death. Additionally, we have characterised for the first time the burden attributable to the rare *Candida* meningitis, emphasising the growing threat of health care–associated infections, which pose an increased risk of antimicrobial resistance. Continued efforts focused on vaccination, antibiotic stewardship, and advances in treatment access and equity can promote the continued prevention of disability and death due to meningitis.

Introduction

Meningitis, or inflammation of the meninges, is the leading infectious cause of neurological DALYs globally.[1,2] It is a heterogeneous infectious syndrome with numerous causative pathogens, including bacteria, viruses, and fungi. Compared to viral meningitis, bacterial meningitis has a higher fatality rate and a higher proportion of survivors with permanent disability.[3,4] A global systematic review published in 2024 estimated a worldwide bacterial meningitis case-fatality ratio of 18% (95% CI, 16% to 19%), or 15% (95% CI, 12% to 19%) when only including studies after 2010.[5] Another global systematic review published in 2010 estimated that one-fifth of people who recover from bacterial meningitis have lasting major sequelae, with the risk of sequelae being twice as high in the African and southeast Asian regions compared to the European region.[6] The most common meningitis sequela is hearing loss, with other sequelae including cognitive impairment, motor impairment, and seizures.[6] These disabilities can have downstream impacts on a patient's whole family, including education, caregiving burden, and economic means.[7]

Since 2000, widespread global vaccine rollout, first against *Haemophilus influenzae* type b (Hib) and later against *Streptococcus pneumoniae* and *Neisseria meningitidis*, has greatly reduced the incidence and mortality due to these infections in both high-income and low-income settings.[8,9] Despite these advances in vaccination, progress against meningitis lags behind when compared to other vaccine-preventable diseases.[10] Incidence of meningitis remains high, particularly in low-income countries where access to

health care and vaccination coverage are limited.[11] The African meningitis belt, spanning from Senegal to Ethiopia[12], experiences the highest incidence rates, with seasonal outbreaks exacerbating the burden.[13] In addition, key pathogens, including the leading causes of neonatal meningitis, Group B streptococcus (GBS) and *Escherichia coli*, remain without a licensed vaccine.[14]

Beyond these well-known pathogens, dozens more contribute to the global burden of meningitis. Non-polio enteroviruses, a family of over 100 serotypes that includes coxsackieviruses and echoviruses, comprise the most common cause of viral meningitis. In some populations, particularly among very young children in industrialised settings, enteroviral meningitis cases outnumber bacterial meningitis cases.[15,16] Gram-negative bacteria such as *Klebsiella pneumoniae and Acinetobacter baumanii* are particularly associated with hospitalized populations, including neonates, in low-income settings.[17] Some commensal skin flora, such as *Candida* species and coagulase-negative *Staphylococcus*, can cause health care–associated meningitis in certain populations, with high morbidity and mortality. *Candida* meningitis occurs particularly in post-neurosurgical patients, immunocompromised patients, or critically ill neonates with disseminated disease.[18,19] Similarly, coagulase-negative *Staphylococcus*, while a rare source of meningitis in the general population, is also a leading cause of meningitis in neurosurgical patients, especially those with implanted devices such as ventricular shunts.[20,21]

In 2021, the World Health Organization launched a global roadmap to defeat meningitis by 2030.[22] The roadmap aims to reduce vaccine-preventable bacterial meningitis cases by 50% and deaths by 70% compared to a baseline year of 2015, as well as eliminate epidemics and reduce meningitis-attributable disability. In their roadmap, the WHO defines vaccine-preventable meningitis as *S pneumoniae, N meningitidis, H influenzae,* and GBS*.* Although no licensed vaccine currently exists for the prevention of GBS, several candidates are in advanced stages of development; furthermore, mother-to-child transmission is considered partially preventable through interventions such as screening and antibiotic administration. Going forth in this study, we will follow the WHO convention, using the term vaccine-preventable to refer to these four pathogens of great public health interest. The roadmap includes 18 strategic goals within five key pillars: prevention and epidemic control, diagnosis and treatment, disease surveillance, support for people affected by meningitis, and advocacy and engagement. To assess progress towards the goals of the roadmap, a comprehensive assessment of meningitis incidence, mortality, and pathogen distribution is key. These country-specific and regional estimates are fundamental for evidence-based regional planning, allowing policymakers to identify the highest-burden countries requiring immediate intervention and allocate limited resources most effectively. Such data enables monitoring progress towards the WHO 2030 targets and can inform where accelerated efforts are most urgently needed.[23]

This study leverages the results from the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2023 to assess the incidence, mortality, and pathogen distribution of acute infectious meningitis in 204 countries and territories from 1990 to 2023. Notable improvements in GBD 2023 include the pathogen modelling of seven new meningitis aetiology categories: *A.* baumanni, *Candida* spp., coagulase-negative staphylococci, and non-polio enteroviruses – plus the splitting of the previous "other pathogen" category into other *Streptococcus* species, other fungi, other bacteria and other viruses. This manuscript was produced as part of the GBD Collaborator Network and in accordance with the GBD Protocol.

Methods

Overview

GBD is a systematic, scientific effort aimed at quantifying the comparative magnitude of health loss caused by diseases, injuries, and risk factors across age groups, sexes, and geographical locations over time. The GBD geographical hierarchy encompasses 204 countries and territories, organised into 21 regions based on epidemiological similarities and geographical proximity. These regions are further consolidated into seven super-regions according to patterns of cause-specific mortality. Detailed methodologies for GBD have been published previously.[24–26] Aetiology morbidity and mortality were estimated through the Anti-Microbial Resistance (AMR) study.[27] In this study, we outline the methods and estimation strategies used for meningitis, including its associated risk factors and pathogens.

For this study, meningitis is defined as a disease caused by the inflammation of the meninges through bacterial, viral, or fungal agents. The Internal Classification of Diseases (ICD) codes that correspond to meningitis within the GBD framework are listed within the methods appendix 1 (p 4). Age-standardised estimates were calculated using age weights from GBD standard reference population.[28]

For information on input data and sources, the GBD Sources tool on the Global Health Data Exchange (GHDx) provides all metadata to identify which sources were used for any of the GBD estimates.[29] This research complies with the Guidelines for Accurate and Transparent Health Estimates Reporting (GATHER) statement (methods appendix 1 p 72).

Mortality estimation process

Meningitis mortality was estimated using the Cause of Death Ensemble model (CODEm) using data from vital registration, verbal autopsy, surveillance, and minimally invasive tissue sampling. CODEm creates an array of sub-models utilising combinations of different predictor covariates to estimate mortality rates or cause fractions.[30] The array of sub-models includes linear mixed-effects models with random intercepts at the super-region, region, and country levels, and spatiotemporal Gaussian process regression models. CODEm selects from the ensemble of models that perform best in out-of-sample predictive validity tests to use as our mortality estimates. Due to the large differences between the mortality trends for children under 5, they were modelled separately from those 5 years and older to adequately capture trends. For a complete list of the covariates used in the meningitis model, see methods appendix 1 pp 5-6.

Morbidity estimation process

Data used in the estimation processes of meningitis morbidity came from a systematic review of published studies, surveillance data, cause-specific mortality estimates, claims, and inpatient data (methods appendix 1 pp 8-10). This systematic review was conducted in the online tool DistillerSR and utilized the software's "DistillerSR Artificial Intelligence System" (DAISY). We used an initial training set of manually screened records to set up DAISY. The tool then prioritized remaining citations for inclusion or exclusion. We worked with DistillerSR's error prediction tool and audit function to run various checkpoints throughout the process. This allowed our reviewers to focus their time on discussing critical decisions and identify potential errors in screening and extractions. These data went through a standardised adjustment to make claims and surveillance data comparable with inpatient data (methods appendix 1 pp 10-11). Overall morbidity was estimated for meningitis using the Bayesian meta-regression tool DisMod-MR 2.1. A more detailed explanation of DisMod-MR 2.1 can be found in previous studies.[26]

Aetiology estimation process

We estimate mortality and incidence attributable to the following pathogen categories: *Acinetobacter baumannii*, *Candida* spp., coagulase-negative staphylococci, *Escherichia coli*, GBS, *Haemophilus influenzae*, *Klebsiella pneumoniae, Listeria monocytogenes, Neisseria meningitidis,* non-polio enteroviruses, other *Streptococcus* species, other fungi, other bacteria, other viruses, *Pseudomonas aeruginosa, Staphylococcus aureus*, and *Streptococcus pneumoniae*. "Other" categories are defined as residual, aggregate pathogen categories not otherwise modelled with more granularity; e.g., "other *Streptococcus* spp." refers to *Streptococcus* spp. other than GBS or S. *pneumoniae*. Data used in the aetiology estimation process included multiple causes of death data, hospital discharge, linkage, microbial data, literature studies, and mortality surveillance (Child Health and Mortality Prevention Surveillance [CHAMPS]) (methods appendix 1 page 16-17). All data were extracted at the most granular pathogen level available; pathogens with fewer than 300 cases were not estimated individually, and were modelled in aggregate categories, such as other fungi. It is important to note that the opportunistic fungi genera *Cryptococcus* and *Toxoplasma* were excluded from the other fungi category and from the current study, as deaths due to these pathogens are considered attributable to HIV. *Mycobacterium tuberculosis* was also excluded, as these deaths are attributed to tuberculosis. A more detailed explanation of the aetiology estimation process can be found in previous research,[27] and is also described in full in our methods appendix 1, pages 15-39. It should be noted that the research describes pathogen distributions for multiple infectious syndromes, of which meningitis is one. Although the different models share the same estimation methodologies, the data and results are independent by syndrome.

In summary, once data were extracted and processed, pathogen distributions were estimated using the multinomial estimation with partial and composite observations (MEPCO) modelling environment, allowing for the inclusion of covariates in the network analysis[27] and for Bayesian priors to be incorporated (methods appendix 1, pages 30-39). We estimated the incidence proportions attributable to viral, fungal, parasitic, and bacterial pathogens with this model, and we used modeled case-fatality ratios (CFRs), as described below, to maximally leverage mortality-only data sources to estimate implied cases for incidence estimation. Data that showed clear linkage between pathogen-specific disease incidence and deaths were used to create models for pathogen-specific CFRs for each age group and syndrome. A splined binomial regression was implemented using the RegMod modelling environment to estimate pathogen-specific CFRs as a function of the Healthcare Access and Quality (HAQ) Index and other covariates, as described in our methods appendix 1 pp 16-21. Finally, the estimated CFR was used to calculate mortality proportions from incidence proportions as modelled above. For more detailed methods, see our methods appendix 1 pp 15-39. To estimate the progress towards the WHO global roadmap Defeating Meningitis by 2030, we computed the totals of what WHO considers to be vaccine-preventable diseases: *S pneumoniae, N meningitidis, H influenzae,* and GBS.[22]

Risk attribution estimation process

Risk attribution for meningitis was calculated using risk–outcome pairs that were selected based on their convincing or probably causal relationship to meningitis. Relative risks for each risk–outcome pair were derived from published systematic reviews. Exposure levels for each risk factor were estimated using spatiotemporal Gaussian process regression, a Bayesian meta-regression tool (DisMod-MR 2.1), or other methods when applicable (methods appendix 1 pp 39-74). Exposure levels that equate to the theoretical minimum risk were determined through relevant data sources (methods appendix 1 pp 41-42). We calculated the number of meningitis deaths attributable to each risk factor by applying the population attributable fractions (PAFs) for each risk factor to the total number of meningitis deaths for each specific risk–outcome pair.

Role of the funding source
The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

Overall meningitis mortality and morbidity estimates

Globally, meningitis was responsible for 259,000 all-age deaths (95% UI 202,000–335,000) and an all-age mortality rate of 3.2 per 100,000 (2.5–4.1) in 2023 (Table 1; Figure 1). This is a decline of 25.4% (5.9–40.4) since 2015, and a 63.5% (51.0–72.8) decline between 1990 and 2023 in all-age mortality rate (Table 1). Across age groups, the largest burden of global deaths was in children under 5 years old, with 86,600 deaths (53,300–149,000) and a mortality rate of 13.5 per 100,000 (8.3–23.2) (Figure 1). For all ages globally in 2023, meningitis caused about the same number of deaths in females (130,000 [91,300–173,000]) and in males (129,000 [95,300–187,000]), and mortality rates between males (3.2 per 100,000 [2.4–4.6]) and females (3.2 [2.3–4.3]) were also nearly the same (results appendix 2 table 7).

Globally, in 2023, there were 2.54 million (95% UI 2.20–2.93) cases of meningitis and an all-age incidence rate of 31.5 per 100,000 (27.2–36.4) (table 2; results appendix 2 figure 1). The percent decline in incidence rate from 1990 to 2023 was 57.5 (54.5 to 60.5) while the decline was 18.5 (17.1 to 20.0) from 2015 to 2023 (Table 2). The largest burden of cases globally was in children under 5 (953,000 [780,000–1,140,000]), and the largest incidence rate also came from children under 5 (148.1 per 100,000 [121.3–177.4]) (results appendix 2 figure 1). For all ages, about the same number of cases of meningitis occurred in males (1.28 million [1.11–1.48]) and females (1.26 million [1.09–1.46]) (results appendix 2 table 8), and both males (31.6 [27.3–36.5]) and females (31.4 [27.2–36.3]) experienced a similar incidence rate of meningitis (results appendix 2 table 8). More detailed meningitis burden results by age and sex across locations and years are available in the GBD Results Tool.

Aetiology results

Globally, the aetiology responsible for the most deaths in 2023 was *S pneumoniae* (41,400 deaths [95% UI 32,200–53,600]) followed by *N meningitidis* (34,400 [26,600–44,500]), non-polio enteroviruses (18,200 [13,700–23,900]), and other viral aetiologies (18,000 [14,000–23,100]) (results appendix 2 figure 2; results appendix 2 table 3). Across all bacterial aetiologies considered by WHO to be largely preventable—that is, *S pneumoniae, N meningitidis, H influenzae,* and GBS—we estimate a total of 98,700 deaths (77,000–127,000) in 2023, a 27.4% (9.6–43.4) decline since 2015, when there were 126,000 deaths (99,200–163,000) (results appendix 2 table 4). Likewise, for this same pathogen group, we estimate 594,000 cases (514,000–686,000) in 2023, a 16.3% (14.8–18.2) decrease from 653,000 cases (560,000–762,000) in 2015 (results appendix 2 Table 4). In 1990, the leading pathogen causing meningitis deaths was *N meningitidis* (79,900 deaths [62,100–99,100]), followed by *S pneumoniae* (60,600 [47,000–75,900]), non-polio enteroviruses (43,100 [33,200–54,100]), and *H influenzae* (40,300 [29,500–51,900]) (results appendix 2 figure 3). The pathogen responsible for the most cases of meningitis in 2023 was non-polio enteroviruses (870,000 cases [735,000–1,030,000]), followed by *N meningitidis* (232,000 [197,000–274,000]) (results appendix 2 figure 2; results appendix 2 table 5). In 1990, the largest number of cases also came from non-polio enteroviruses (1.70 million [1.38–2.06]), followed by *N meningitidis* (373,000 [302,000–456,000]) (results appendix 2 figure 3).

In children under the age of 5, the pathogen that was responsible for the most deaths in 2023 was *S pneumoniae* (14,000 deaths [95% UI 8630–23,800] with a mortality rate of 2.2 per 100 000 (1.3–3.7) (results appendix 2 figure 2; figure 2). *N meningitidis* had the second largest number of deaths in children under 5 (11,400 [7050–19,600] with a mortality rate of 1.8 per 100 000 (1.1–3.1) in this age group (figure 2; results appendix 2 Figure 2). GBS was the pathogen responsible for the third largest number of deaths in children under 5 (8540 [5420–14,500]) with a mortality rate of 1.3 per 100 000 (0.8–2.3) in this age group (figure 2; results appendix Figure 2). Among age groups within the under-five population, the GBS meningitis mortality rate was highest in the early neonatal age group (93.1 deaths per 100,000 [56.6–148.1]), followed by the late neonatal age group (22.1 deaths per 100,000 [13.5–36.9]) (results appendix 2 table 10).The pathogen responsible for the most meningitis cases in children under 5 was non-polio enteroviruses (327,000 [264,000–402,000] with an under 5 incidence rate of 50.9 per 100 000 [41.1–62.6]), followed by *N meningitidis* (88,200 [72,000–107,000] with an incidence rate of 13.7 per 100 000 [11.2–16.7]) and other viral aetiologies of meningitis (74,100 [60,400–90,400]) with an under 5 incidence rate of 11.5 per 100 000 (9.4–14.1) (figure 2; results appendix 2 figure 2).

Of the newly modelled fungal aetiologies of meningitis from GBD 2023, *Candida* spp. was responsible for 92,200cases (95% UI 77,200–108,000) and 13,700 deaths (10,600–17,700), and other fungi were responsible for 110,000 cases (90,700–141,000) and 13,300 deaths (9730–19,200) (results appendix 2 table 3; results appendix 2 figure 2).

Risk factors of meningitis

Globally, in 2023, the greatest risk factor contributing to meningitis deaths was low birthweight, responsible for 7660 deaths (95% UI 4940–12,300), followed by short gestation (3540 [2280–5720]), household air pollution (2690 [1700–4350]), and ambient particulate matter pollution (554 [348–917]) (table 3). Between males and females, the ranking of these risk factors in 2023 did not vary from the ranking for both sexes combined (results appendix 2 table 9). Meningitis mortality rate attributable to low birthweight has substantially decreased between 1990 and 2023, by 73.8% (49.7-85.2) (table 3). The mortality rates attributed to short gestation, household air pollution, and ambient particulate matter pollution have all declined rapidly from 1990 as well: short gestation has decreased by 72.1% (45.8-83.9) household air pollution by 70.5% (42.2-83.7) and ambient particulate matter pollution by 77.4% (57.7-87.8)

The meningitis belt

Across the countries of the meningitis belt in 2023, the country with the largest meningitis all-age mortality rate was Nigeria (30.2 per 100 000 [95% UI 21.5–41.1]) followed by Niger (30.0 per 100 000 [18.4–44.9]) and Chad (28.8 per 100 000 [18.4–44.6]) (figure 3; results appendix 2 Table 6); these countries also have the highest mortality rates globally. In Nigeria, the pathogen responsible for the most deaths in 2023 was *S. pneumoniae* (11,800 [8320–16,000]) (results appendix 2 table 3).

The country with the largest all-age incidence rate in 2023 was Nigeria (239.3 per 100 000 [95% UI 203.7–280.7]), followed by Chad (230.6 per 100 000 [198.6–265.3]) and Niger (222.9 per 100 000 [182.8–259.3]) (results appendix 2 table 6); these three countries also have the highest incidence rates globally. In Nigeria, the pathogen responsible for the most cases of meningitis in 2023 was non-polio enteroviruses (226,000 [187,000–267,000]) (results appendix 2 table 5).

In the meningitis belt, both mortality and incidence rates have declined substantially for most countries when comparing between 1990 and 2023 (figure 3). The countries with a percent decrease of mortality rates greater than 80% were Sudan, Rwanda, and Ethiopia (results appendix 2 table 6). Countries with a percent decrease of incidence rates greater than 80% were Sudan and Rwanda (results appendix 2 table 6).

Discussion

This study presents estimates of the meningitis burden attributable to a comprehensive set of 17 pathogen categories by age group and sex, across countries, regions, and the globe, from 1990 until 2023. Of these pathogen categories, five are newly modelled in this year's GBD study. Although mortality and incidence have declined substantially since 1990, progress since 2015 has slowed and remains insufficient to meet the WHO 2030 targets for vaccine-preventable meningitis. We estimate 259,000 deaths (95% UI 202,000–335,000) attributable to meningitis worldwide in 2023, including 86,600 deaths (53,300–149,000) in children under 5 years old. The burden of disease remained disproportionately high in low-income countries, particularly in the African meningitis belt, where Nigeria, Chad, and Niger recorded the highest mortality and incidence rates.

Across all studied pathogens, S *pneumoniae* and *N meningitidis* remain the leading causes of meningitis mortality, responsible for 41,400 (32,200–53,600) and 34,400 (26,600–44,500) deaths, respectively, in 2023.These vaccine-preventable bacterial species present with high fatality and complication rates. The WHO Global Roadmap to Defeat Meningitis by 2030 targets a reduction in vaccine-preventable bacterial meningitis cases by 50% and deaths by 70% compared to 2015, requiring annualised decreases of approximately 8.0% for deaths and 4.6% for incidence. These vaccine-preventable aetiologies, as defined in the roadmap—*S pneumoniae, N meningitidis, H influenzae,* and GBS—were collectively responsible for an estimated 98,700 deaths (95% UI 77,000–127,000) and 594,000 cases (514,000–686,000) in 2023. Despite substantial progress in vaccination and health systems strengthening over past decades, the annualised rate of decline across the four aetiologies combined was 4.1% for deaths and 2.2% for incidence between 2015 and 2023, underscoring the need for accelerated efforts to further reduce the global burden of vaccine-preventable bacterial meningitis and achieve the ambitious benchmarks set by WHO.[31] Progress in the 2000s and 2010s has largely been driven by highly successful vaccination campaigns, including the MenAfriVac campaign, which virtually eliminated *N meningitidis* serogroup A in the meningitis belt, as well as the global introduction of pneumococcal and *H influenzae* type b vaccinations into routine childhood immunisation schedules.[8,32]

However, due to serogroup and serotype replacement, non-vaccine serotype meningitis incidence has, in relative terms, risen, inhibiting progress towards benchmarks set by the WHO.[33–35] Non-typeable *H influenzae*, traditionally regarded as non-invasive, has been increasingly detected as a cause of meningitis in the post-vaccine era,[36,37] though its incidence remains much lower than that of Hib before the rollout of its immunization.[38] In the WHO global invasive bacterial vaccine-preventable disease network, more than half (52.9%) of pneumococcal meningitis cases identified in post-PCV vaccine years were non-PCV13 strains, and nearly half (49.4%) of global meningococcal cases were serogroup B, though Y and W were most commonly detected in the African region.[35] These findings further reinforce the importance of accurate diagnostics, not only for accurate patient treatment, but also for robust pathogen surveillance that can drive future vaccination development and policy.

For the first time, we estimate the global incidence and mortality of non-polio enteroviral (NPEV) meningitis. NPEVs were responsible for the most worldwide meningitis cases in 2023 and were also the third-leading pathogen cause of meningitis mortality. NPEVs are a diverse group of pathogens responsible for a wide range of clinical syndromes, from asymptomatic infections to serious conditions , including meningitis.[39] Although the current study does not estimate viral serotype distribution, a 2019 systematic review estimated that echovirus 30 was the commonest global serotype[40] Echovirus 30 outbreaks typically occur over large geographical areas and are common in Europe, the USA, Asia, and South America.[41] This finding highlights a pressing need for surveillance frameworks and diagnostic readiness in LMICs, where enterovirus outbreaks often go undetected. While there is no known global surveillance network for NPEVs[42] , regional networks such as the European Non-Polio Enterovirus Network (ENPEN) and the Asia-Pacific Network for Enterovirus Surveillance (APNES) can serve as global examples for early detection of enterovirus outbreaks.[43,44]

Antimicrobial resistance (AMR) poses a major barrier to achieving WHO goals for meningitis control. *N meningitidis* isolates resistant to penicillin and fluoroquinolones have become widespread over the past decade.[45,46] A global systematic review noted the highest fluoroquinolone (ciprofloxacin) *N meningitidis* resistance in Africa (30.3% [14.1-53.5]) followed by Asia (6.3% [0.2-73.3]), though most studies from these continents used the disk diffusion method, which may substantially overestimate the resistance rate.[46] Resistance to cephalosporins remains rare [45,46] but highly concerning as these antimicrobials are the first-line therapy for adult and child meningitis worldwide. *S pneumoniae* resistance displays a similar pattern, with frequent resistance to penicillin, and rare but worrisome resistance to cephalosporins. The ATLAS study estimated global resistance rates of *S pneumoniae* to penicillin, ceftriaxone, and ceftaroline at 36.6%, 6.0% and 0.4%, respectively.[47] Substantial geographic variability was observed, with ceftriaxone resistance up to 34% in China and South Korea, while North America and Europe maintain resistance rates lower than 5%.[47] Despite this rising proportion of resistant isolates, the GBD AMR study estimates that the total number of deaths attributable to AMR in *S pneumoniae* across all sites of infection has fallen over time, from an estimated 258 000 (179 000–336 000) attributable deaths in 1990 to 155 000 (122 000–188 000) attributable deaths in 2021.[27] This is most likely due to a decline in overall *S pneumoniae* infections following the rollout of global vaccination. Amongst non-vaccine preventable, often healthcare-associated pathogens, including *K pneumoniae* and *S aureus*, AMR poses an even greater threat. Carbapenem-resistant Enterobacterales, including *K pneumoniae*, are classified by the WHO as "critical" priority pathogens representing one of the greatest threats to public health.[48] Ultimately, AMR jeopardizes common treatments and increases fatality of meningitis. Strategies to address this threat include a global focus on drug development, ensuring quality and availability of full antibiotic courses, and robust antibiotic stewardship alongside bolstering existing vaccine frameworks and novel vaccine development.

Although no licensed vaccines against GBS are currently available commercially, it is still considered a preventable infection, as the incidence of invasive GBS in neonates is substantially reduced in settings that administer intrapartum antibiotics for women who screen GBS-positive during pregnancy.[49,50] A recent global systematic review has shown that policies targeting all women who screen positive for GBS, rather than risk-based approaches, are associated with the largest reduction in neonatal early-onset GBS infection without an appreciable risk in first-line antibiotic resistance.[50] However, implementing screening at 36-37 weeks, as is done in the United States, may be impractical in low-resource settings, as regular access to antenatal care and accurate pregnancy dating are not always available.[51] A potential solution is screening during labor, although this risks the infant being born before antibiotics can be administered.[52] A maternal GBS vaccine serves as a potential solution to these challenges, and several promising vaccines are in development.[53,54] After vaccine approval, challenges in rollout, equity, and vaccine acceptance, such as those which have been seen with the recent approval of the maternal respiratory syncytial virus vaccine, could be the next frontier for GBS prevention.[55,56]

To our knowledge, this is the first study to systematically estimate the global incidence and mortality attributable to meningitis from the following pathogens: *Candida* spp., coagulase-negative staphylococci, non-polio enteroviruses, other fungi, and other *Streptococcus* species. We found that *Candida* spp. was responsible for 92,200 meningitis cases (95% UI 77,200–108,000) and 13,700 deaths (10,600–17,700) in 2023. Although no comprehensive review exists on the leading meningitis-causing *Candida* species, case series suggest that *Candida albicans* is the most common cause in both neonatal and post-surgical patient populations.[57,58] This species was named by WHO in 2022 as one of four critical fungal priority pathogens because of its global ubiquity and high case-fatality ratio for invasive disease, with an estimated case-fatality ratio of 20–50% despite appropriate antifungal treatment.[59] Across all invasive *Candida* infections, the incidence of previously rare *Candida* spp., including the drug-resistant *Candida auris*, is on the rise.[60] Candidal meningitis is particularly difficult to treat, as several antifungals cannot penetrate the blood–brain barrier, requiring regimens with the powerful antifungal amphotericin B. Concerningly, amphotericin B isolates of *C auris* have been detected in invasive infections.[61] This underscores the importance of both continued development of novel antifungal and antimicrobial agents, as well as infection prevention across hospital systems. In high-risk post-neurosurgical patients, a common demographic for *Candida* as well as coagulase-negative *Staphylococcus* infection, reducing the duration of drain placement and avoiding unnecessary drain manipulation are key to meningitis prevention.[62] In infants, preventing risk factors for invasive infection, such as preterm birth and very low birthweight, can help reduce invasive meningitis.[57]

This study has several limitations. First, meningitis data are limited, with data gaps that are particularly pronounced in low-resource settings where meningitis cases and deaths often go undocumented. This contributes to wide uncertainty intervals for estimates that reflect burden concentrated in low-income locations. With more robust data, uncertainty intervals would narrow substantially, leading to more stable estimates. Second, meningitis is difficult to diagnose, particularly in neonates and infants. Its symptoms often overlap with those of other conditions, including encephalitis and neonatal sepsis. [63] This may affect the accuracy of the meningitis burden estimates, especially in locations where data are sparse or where sensitive, accurate diagnostic methods are unavailable. Third, viral pathogens are not often included in surveillance networks , are more difficult to detect using conventional culture methods, and tend to cause milder disease that may be less likely to come to medical attention. This may contribute to an underestimation of viral meningitis. We work to address this limitation by supplementing surveillance data with a variety of data sources from different settings, including hospital data and insurance claims. Fourth, we directly apply meningitis aetiology proportions from our pathogen distribution models to our overall estimates of meningitis deaths, even though the two methodologies have slightly different definitions of meningitis. More specifically, the GBD definition of meningitis deaths includes only instances in which meningitis was the underlying cause of death, whereas the pathogen distribution model definition includes any instance where meningitis was present in the causal chain, irrespective of the underlying cause of death. This one-cause-per-death approach additionally poses its own limitations, as most deaths, including those in children, often have multiple addressable conditions in the causal chain; a recent study using CHAMPS data resulted in a 16-fold increase in estimated infant meningitis deaths when including all causes along the chain. This has implications for resource allocation, as deaths for which meningitis is in the causal chain, even if it is not the underlying cause of death, may be preventable with proper meningitis treatment. Fifth, estimates for newly modelled pathogens, including *Candida* spp. and NPEV, are model-dependent and should be compared to output from further research for validation. Sixth, the current study does not incorporate serotype data for any pathogens, limiting its utility to track specific meningitis-causing strains. Seventh, these annual estimates do not account for seasonal and regional outbreak patterns, which, especially in areas like the African meningitis belt, can hide true peaks and limit the ability to assess control measures' efficacy. Eighth, a key limitation of DALY methodology is that prevalence-based YLD calculations capture disability at a single time point, rather than over survivors' lifetimes. Because people who recover from meningitis often live many years with disability, these point-in-time estimates may underestimate the true population impact.

In summary, while global vaccination campaigns have driven substantial declines in meningitis cases and deaths caused by vaccine preventable bacterial pathogens, progress remains insufficient to meet the ambitious WHO Roadmap targets for 2030. Accelerated efforts—including expanding immunisation, improving access to care, and strengthening diagnostics and surveillance—are essential to achieve these targets. Additionally, we have shown that meningitis, including viral meningitis, still poses a substantial burden. Non-polio enteroviruses, which cause a less severe phenotype and a lower likelihood of mortality than bacterial pathogens, are the leading pathogen of meningitis incident cases, both in 1990 and in 2023. Furthermore, we have characterised for the first time the burden attributable to the rare but highly hazardous *Candida* meningitis, emphasising the growing threat of antimicrobial resistance, particularly in health care–associated infections or in immunocompromised patients. Targeted investment in WHO pillars, including expanded vaccination coverage, new vaccine development, antibiotic stewardship, region-specific outbreak preparedness, and advances in treatment access and equity could prevent meningitis disability and mortality.

List of Tables and Figures

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Authors' Contributions

The corresponding author had full access to the data in the study and had final responsibility for the decision to submit for publication. HHK, SS, RGB, RMVD, and AV accessed and verified the underlying data reported in this study. Please see the collaborator appendix 3 (pp 40-55) for more detailed information about individual author contributions to the research, divided into the following categories: managing the overall research enterprise; writing the first draft of the manuscript; primary responsibility for applying analytical methods to produce estimates; primary responsibility for seeking, cataloguing, extracting, or cleaning data; designing or coding figures and tables; providing data or critical feedback on data sources; developing methods or computational machinery; providing critical feedback on methods or results; drafting the manuscript or revising it critically for important intellectual content; and managing the estimation or publications process.

Data sharing

To download the data used in these analyses, please visit the Global Health Data Exchange website at: https://ghdx.healthdata.org/gbd-2023

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