

1 amr.watch – monitoring antimicrobial resistance trends from global genomics data

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8 Genomics and enabling data for the Surveillance of AMR

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36

37 **Abstract**

38

39 **Background**

40 Whole genome sequencing (WGS) is increasingly supporting routine pathogen surveillance
41 at local and national levels, providing comparable data that can inform on the emergence
42 and spread of antimicrobial resistance (AMR) globally. However, the potential for shared
43 WGS data to guide interventions around AMR remains under-exploited, in part due to
44 challenges in collating and transforming the growing volumes of data into timely insights. We
45 present an interactive platform, amr.watch (<https://amr.watch>), that enables interrogation of
46 AMR trends from public WGS data on an ongoing basis to support research and policy.

47

48 **Methods**

49 The amr.watch platform incorporates, analyses and visualises high-quality WGS data from
50 WHO-defined priority bacterial pathogens. Analytics are performed using
51 community-standard methods with bespoke species-specific curation of AMR mechanisms.

52

53 **Findings**

54 By 31 March 2025, amr.watch included data from 620,700 pathogen genomes with
55 geotemporal information, with highly variable representation of different species and
56 geographic regions. By integrating WGS data with sampling information, amr.watch enables
57 users to assess geotemporal trends among genotypic variants (e.g. sequence types) and
58 AMR mechanisms, with implications for interventions including antimicrobial prescribing and
59 drug and vaccine development.

60

61 **Interpretation**

62 amr.watch is an information platform for scientists and policy-makers delivering ongoing
63 situational awareness of AMR trends from genomic data. As broad adoption of WGS
64 continues, amr.watch is positioned to monitor both pathogen populations and our global
65 efforts in genomic surveillance, guiding control strategies tailored to each pathogen's
66 characteristics.

67

68 **Funding**

69 UK National Institute for Health Research & Gates Foundation.

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73 Research in context

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75 Evidence before this study

76 Whole genome sequencing (WGS) approaches enable us to track the spread of bacterial
77 pathogens and antimicrobial resistance (AMR) with high resolution at local, national and
78 global levels. To date, genomic studies assessing AMR dynamics have largely used
79 retrospective data collected for specific research agendas. However, the growing volumes of
80 publicly-shared WGS data, generated increasingly from routine surveillance, provide
81 improved power to detect novel trends and guide interventions. Efforts to collate public
82 bacterial genome data exist, such as AllTheBacteria, although these are aimed at the
83 research community and do not facilitate data usage and interpretation by non-genomics
84 experts, particularly those in public health. To our knowledge, no platforms exist for readily
85 interrogating AMR dynamics from continuously updated public genome data across the array
86 of different WHO priority bacterial pathogens. Of note, however, TyphiNet provides an
87 interactive online dashboard for examining AMR trends exclusively from *Salmonella* Typhi
88 using a periodically-updated data set from the Pathogenwatch platform. These findings are
89 based on searching PubMed without language restrictions from Jan 1 2000 to December 31
90 2024, using terms related to “genomic surveillance” and “antimicrobial resistance”.

91

92 Added value of this study

93 We have developed amr.watch which, to our knowledge, is the first platform that enables
94 ongoing analysis and visualisation of AMR trends from public genome data across the
95 spectrum of WHO priority bacterial pathogens via an accessible interface. Crucially, the
96 platform incorporates processed genome data via a live always-on stream, enabling insights
97 that are delayed only by the time to data deposition. We reviewed public genomes with
98 available geotemporal information up until 31 March 2025, providing a contemporary global
99 landscape of pathogen genome sequencing. While the number of genomes available
100 annually grew over five-fold globally between 2010 and 2018, we also revealed the extent of
101 differences in geographic representation, with 89.6% of genomes originating from
102 high-income countries and 89 countries contributing no genome data from the priority
103 pathogens.

104

105 Implications of all the available evidence

106 Rapid generation and sharing of global genome data enables us to more precisely track the
107 spread and define the characteristics of contemporary circulating resistant pathogens. The
108 amr.watch platform provides a solution for retrieving, curating and translating shared

109 genomic data into relevant insights that are accessible and actionable by diverse
110 stakeholders. It thereby forms a basis for monitoring progress in surveillance efforts, alerting
111 on ongoing population changes and guiding enhanced precision for surveillance and
112 interventional development. Additionally, our review of available bacterial genome data
113 highlights the need for additional efforts to increase and sustain the implementation of
114 genomic surveillance of AMR globally, and improve the timely sharing of WGS data and its
115 associated metadata.

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139 Introduction

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141 Antimicrobial resistance (AMR) is a major global health crisis projected to worsen. A recent
142 study estimated that 1.14 million deaths worldwide in 2021 were directly attributable to
143 bacterial AMR, a figure projected to rise to 1.91 million deaths by 2050¹. AMR extends to
144 numerous pathogens (including bacterial, viral and fungal), each with different biological and
145 epidemiological characteristics that necessitate tailored intervention strategies. Since 2017,
146 the World Health Organisation (WHO) has categorised different bacterial pathogens into
147 “critical”, “high” and “medium” priority groups to inform research and public health priorities
148 around AMR^{2,3}.

149

150 Whole genome sequencing (WGS) is being increasingly adopted by local and national
151 surveillance programmes to improve detection and monitor spread of bacterial pathogens
152 and AMR. In particular, the high resolution provided by WGS, as compared with previous
153 typing methods, enables enhanced detection of outbreaks and tracking of transmission
154 pathways. It also enables typing of bacterial genomes into genotypic variants using
155 community-adopted nomenclature systems (e.g. multi-locus sequence typing (MLST)),
156 identification of resistance and virulence mechanisms, as well as descriptions of other
157 species-specific genomic features that may affect disease outcomes or control strategies.
158 Substantial use of WGS in bacterial pathogen surveillance to date has been in retrospective
159 studies and for specific research agendas (with a particular focus on AMR), as reflected in
160 available data from public repositories⁴. However, there are widespread initiatives to
161 strengthen and broaden genomic capacity within routine surveillance programmes in order to
162 improve the public health response to contemporary threats. In particular, the WHO aims for
163 “all 194 WHO member states [to] have, or have access to, timely genomic sequencing for
164 pathogens with pandemic and epidemic potential” by 2032⁵.

165

166 The value of rapidly generating and sharing genomic data to support evidence-based
167 policy-making was exemplified in the COVID-19 pandemic. In particular, sharing of
168 information on different SARS-CoV-2 variants (e.g. Alpha, Beta) greatly enhanced our
169 understanding of how the pathogen population was evolving and spreading at local, national
170 and international levels^{6,7}. This led to assessments of the public health risk of specific
171 variants, the instigation of counter-measures to delay the spread of high-risk variants to
172 unaffected areas, the (re-)design of vaccines towards dominant circulating variants and
173 changes to immunisation schedules. Similarly, collated genomic data on bacterial pathogens
174 provides significant power for understanding the geographic distribution and spread of

175 important bacterial variants (e.g. sequence types; STs), together with their associated AMR
176 mechanisms. These particular characteristics, when provided via community-adopted shared
177 nomenclatures, can offer crucial insights into pathogen epidemiology (e.g. routes and extent
178 of spread), expected infection severity, and the response of a pathogen to interventions (e.g.
179 antimicrobials, infection prevention measures and vaccines). Such insights can in turn inform
180 targeted interventions, for example relating to AMR surveillance approaches and
181 containment measures, antimicrobial treatment policies, and development strategies for
182 urgently-needed vaccines and antimicrobial drugs. However, the potential of bacterial
183 genomic data to inform all of these applications remains under-exploited, in part due to
184 challenges in collating the growing volumes of WGS data available in public data
185 repositories and translating these into relevant insights across the range of different
186 pathogens.

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188 To address this, we have developed the amr.watch platform (<https://amr.watch>), an
189 interactive web application that enables monitoring of the WHO-defined priority bacterial
190 pathogens and their associated AMR mechanisms, via ongoing daily curation and analysis
191 of available quality-assured public genomes. Here we describe the platform and summarise
192 the availability and representativeness of the shared public genomes to date. As the use of
193 genomics in routine surveillance grows globally, we show how shared data can be
194 interpreted via amr.watch to identify geographic and temporal trends with respect to both
195 variants (e.g. STs) and AMR mechanisms. It aims to provide insight to support diverse
196 stakeholders including scientists and policy-makers with AMR research prioritisation and
197 interventional strategy decisions.

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199

200 Materials and methods

201

202 Overview of the amr.watch application

203 amr.watch currently reports genome data from pathogens in the 2017 WHO priority
204 pathogen list². From the “critical” priority group, the supported pathogens include
205 *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and three *Enterobacteriaceae*
206 pathogens comprising *Escherichia coli*, *Klebsiella pneumoniae* and the *Enterobacter cloacae*
207 species complex. From the “high” priority group, we include *Enterococcus faecium*,
208 *Staphylococcus aureus*, *Campylobacter* spp. (*C. coli* and *C. jejuni*), *Salmonellae* (*Salmonella*
209 *enterica* subsp. *enterica* serovars Typhi, Enteritidis and Typhimurium; from here on referred
210 to as *Salmonella* Typhi, *Salmonella* Enteritidis and *Salmonella* Typhimurium) and *Neisseria*

211 *gonorrhoeae*. *Helicobacter pylori* is currently not included due to lack of appropriate AMR
212 mechanisms within the AMRFinderPlus database (used for identification of AMR
213 mechanisms; see below). From the “medium” group, we include *Streptococcus pneumoniae*,
214 *Haemophilus influenzae* and *Shigella* spp. (*S. sonnei* and *S. flexneri*).

215

216 In short, high-quality genomes that meet specific requirements are downloaded from the
217 public sequence archives and processed via our “always-on” pipeline within Pathogenwatch
218 (<https://pathogen.watch>), prior to import and visualisation within amr.watch. The component
219 steps of this pipeline are summarised below and detailed further in the **Supplementary**
220 **Methods**.

221

222 *Retrieval of WGS data and associated metadata from International Nucleotide Sequence*
223 *Database Collaboration (INSDC) databases*

224 As part of our “always-on” pipeline, metadata are retrieved from the European Nucleotide
225 Archive (ENA) via the ENA Portal API every four hours. We proceed with entries (samples)
226 from the above pathogens (based on the submitted classification; see **Supplementary**
227 **Table 1** for accepted taxon IDs) with an available sampling date from 2010 onwards that is
228 decodable to at least the year, as well as a sampling location decodable to at least the
229 country level. Among these entries, we then proceed with those annotated as
230 `library_strategy="WGS"`, `library_source="GENOMIC"`, `library_layout="PAIRED"` and
231 `instrument_platform= "ILLUMINA"` in the ENA metadata. Further checks are performed to
232 ensure the consistency and integrity of the data, including a requirement for two FASTQ files
233 per sample and for sequencing runs to possess at least 20x coverage. Sequence reads
234 fulfilling the above criteria are downloaded from the Sequence Read Archive (SRA) using
235 SRA-Toolkit `fastq-dump v3.1.0` (<https://hpc.nih.gov/apps/sratoolkit.html>). Further details on
236 the retrieval of WGS data and the filtering processes applied are available in the
237 **Supplementary Methods**.

238

239 *Assembly and quality control (QC) of WGS data*

240 Sequence reads are assembled with a workflow
241 (<https://gitlab.com/cgps/ghru/pipelines/assembly>) that uses the SPAdes assembler v3.15.3⁸.
242 The quality of the resulting *de novo* assemblies is analysed using species-specific metrics
243 that assess contiguity, contamination and correctness (**Supplementary Table 1**).
244 Assemblies are excluded if they fail to meet one or more of the defined criteria. The
245 Speciator tool (v4.0.0) within Pathogenwatch (<https://pathogen.watch>) is used to verify the
246 species of the genome assemblies. The SISTR tool (v1.1.1)⁹, implemented within
247 Pathogenwatch, is additionally used to assign the serotype of *S. enterica* genomes. Genome

248 assemblies with a species and/or serotype identification that do not match defined taxon IDs
249 for each pathogen (see **Supplementary Table 1**) are excluded. Note that for each of *E. coli*,
250 *S. sonnei* and *S. flexneri*, we accept genomes annotated as either *E. coli* or *Shigella* in the
251 ENA, with the Speciator assignments used in subsequent processing. Further details on the
252 assembly and QC are available in the **Supplementary Methods**.

253

254 *Variant typing*

255 amr.watch displays the genotypic variants found among each pathogen using
256 community-based schemes implemented in Pathogenwatch. For most of the pathogens,
257 these comprise MLST schemes from PubMLST¹⁰. In addition to MLST, we also use Global
258 Pneumococcal Sequencing Cluster (GPSC) assignments for *S. pneumoniae*¹¹ and “clonal
259 group” assignments from the LIN code nomenclature for *K. pneumoniae*¹². For *Salmonella*
260 Typhi, we use the higher-resolution scheme, GenoTyphi¹³, rather than the MLST scheme.

261

262 *Identification of mechanisms associated with AMR*

263 amr.watch shows the genes and mutations associated with AMR among the represented
264 genomes, as identified using AMRFinderPlus v3.10.23, database version 2021-12-21.1¹⁴
265 (see **Supplementary Methods**). A list of curated genes and mutations included for each
266 pathogen and antimicrobial combination, obtained via a comprehensive literature review, is
267 provided in **Supplementary Table 2**. AMR mechanisms are identified for antimicrobial
268 classes defined in the 2017 WHO priority pathogen list². We additionally report mechanisms
269 for quinolone resistance in *E. coli* due its high global burden and clinical importance¹.

270

271 *amr.watch web application*

272 amr.watch is developed in JavaScript using the Next.js framework (<https://nextjs.org/>) and
273 React library (<https://reactjs.org/>). Geographic data are represented using Mapbox
274 (<https://www.mapbox.com>) and charts are visualised using the Apache ECharts library
275 (<https://echarts.apache.org/>). The amr.watch application incorporates the processed data
276 from Pathogenwatch on an ongoing basis following successful implementation of the
277 analytical steps described above. All genomes and associated metadata visualised in
278 amr.watch are also available within Pathogenwatch for further use by the community.

279

280 *Role of the funding source*

281 The funding sources played no role in the study design, in the collection, analysis and
282 interpretation of the data, in the writing of the manuscript or the decision to submit the
283 manuscript for publication.

284 Results

285

286 Monitoring AMR trends from global genomics data – the amr.watch platform

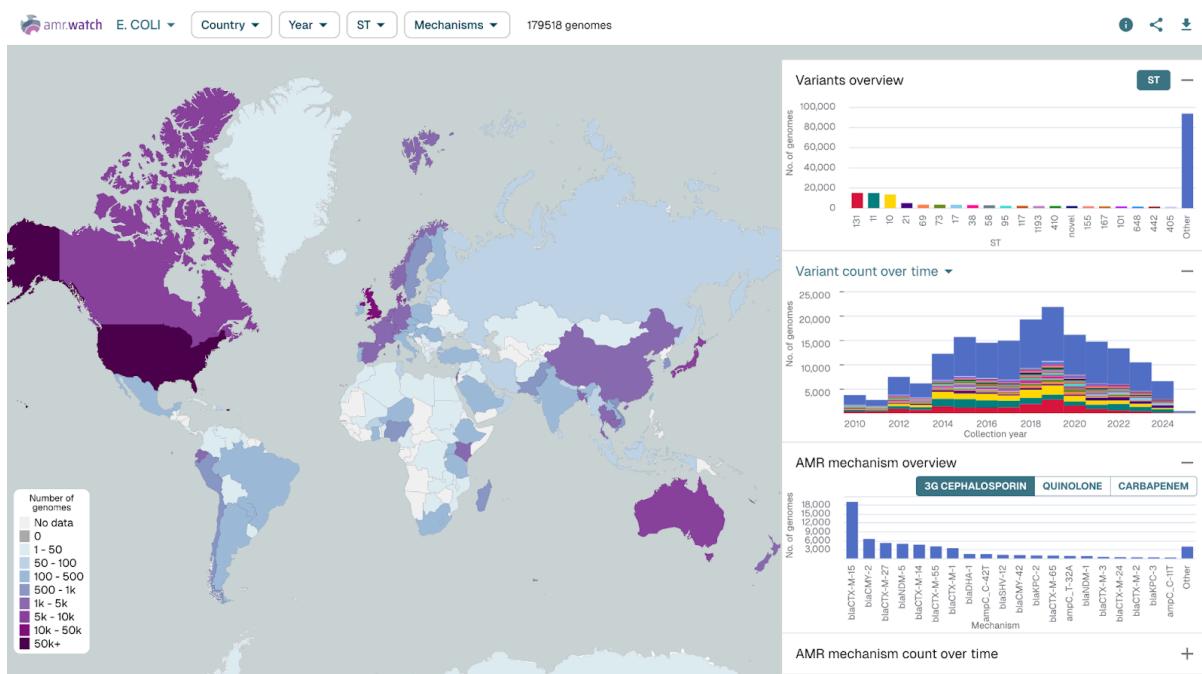
287 We developed amr.watch (<https://amr.watch>), an interactive web application that enables
288 monitoring of the WHO-defined priority bacterial pathogens and their associated AMR
289 mechanisms. As input, the platform incorporates processed genome data available via
290 Pathogenwatch on an ongoing basis on: 1) the variant type(s); and 2) any AMR mechanisms
291 for relevant antimicrobial classes, from all high-quality short-read (Illumina) sequenced
292 pathogen genomes with geotemporal information in the INSDC databases (see **Methods**
293 and **Supplementary Methods**). The platform can incorporate one or more forms of variant
294 nomenclature per pathogen, with 7-gene MLST schemes currently used for all pathogens
295 except *S. pneumoniae*, *K. pneumoniae* and *Salmonella* Typhi, for which we use additional
296 and/or alternative schemes (see **Methods**). We provide a live overview of all genomes
297 represented in amr.watch (<https://amr.watch/all>) and the filtering processes applied to
298 genomes of each pathogen from the public archives (<https://amr.watch/summary>).

299

300 For each pathogen, an interactive map in amr.watch displays the geographic distribution of
301 available genomes based on the recorded sampling locations (**Fig. 1**). Floating panels
302 (right-hand side) show the top twenty most frequent variants (e.g. STs) and AMR
303 mechanisms, and their distributions over time. Users can create tailored visualisations by
304 using the filter menu at the top of the page or by using available filters in each of the panels.
305 This allows data exploration based on user-derived questions, for example, relating to the
306 distribution of particular variants or AMR mechanisms within an individual country/region
307 and/or over a designated time frame. When one or more filters are selected (i.e. year,
308 variants and/or AMR mechanisms), the map can also be optionally coloured to display the
309 proportion of genomes from each country matching the criteria. Visualisations with selected
310 filters can be saved and/or shared onwards via the generation of URLs. All raw data can be
311 downloaded by users in CSV format and includes the genome accession numbers.

312

313

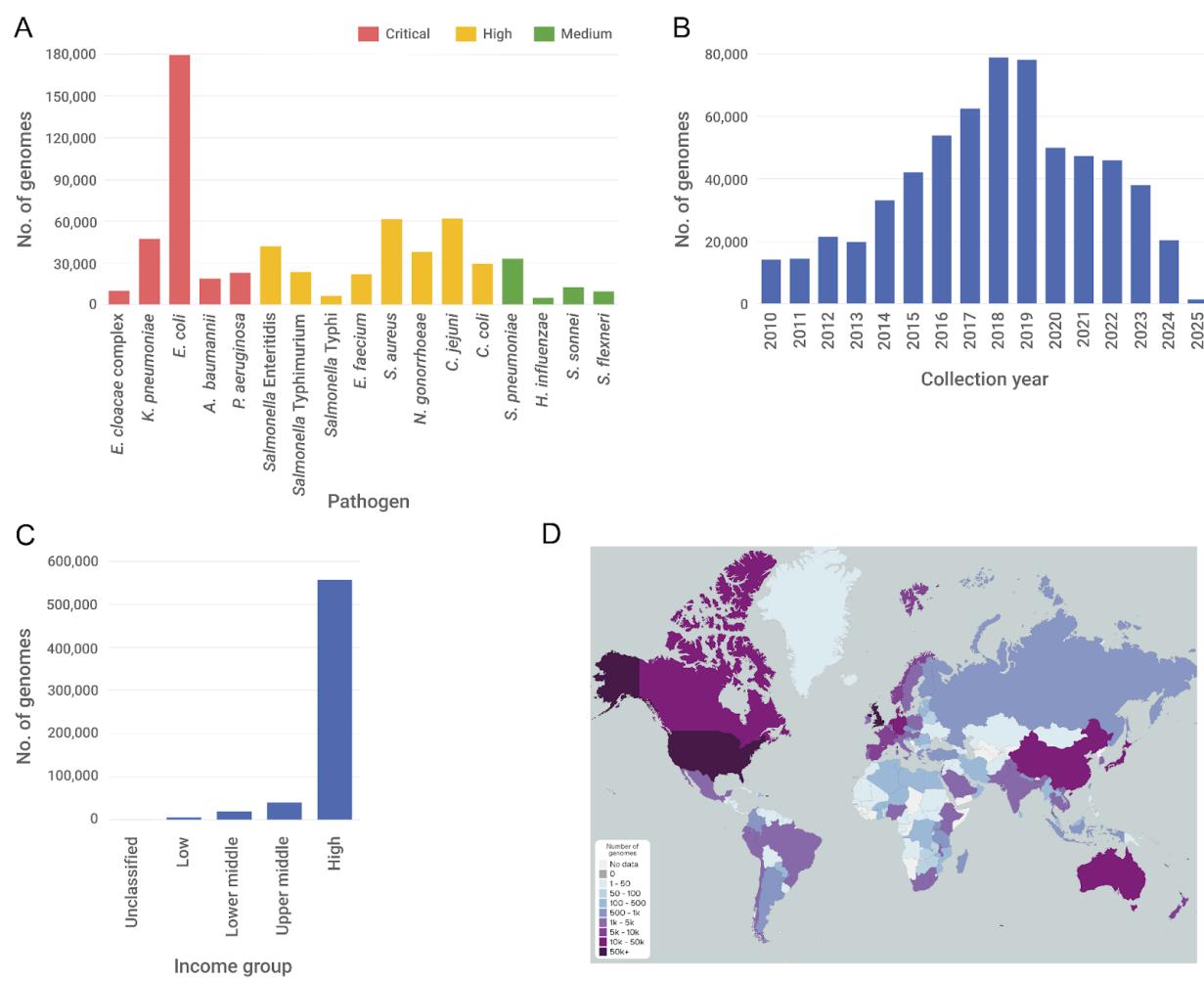


338 of the *H. influenzae* type b (Hib) vaccine into routine childhood immunisation schedules¹⁵.
339 The number of available genomes across all species largely increased year-on-year until
340 2018 (Fig. 2b), with a peak of 78,849 genomes possessing a sampling date within this year.
341 A subsequent decrease in the available genomes may be due to a lag in deposition times
342 from sampling to archiving and/or the large-scale focus on sequencing SARS-CoV-2
343 genomes from 2020.

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349 **Figure 2.** Distribution of pathogen genomes represented in amr.watch as of 31 March 2025. (A) Number of genomes belonging to each pathogen grouped by WHO priority listing². (B) Number of genomes by collection year across all pathogens. (C) Number of genomes by country income group¹⁶ across all pathogens. (D) Geographic distribution of all genomes by country. A live overview of all genomes represented in amr.watch with similar visualisations is available at <https://amr.watch/all>

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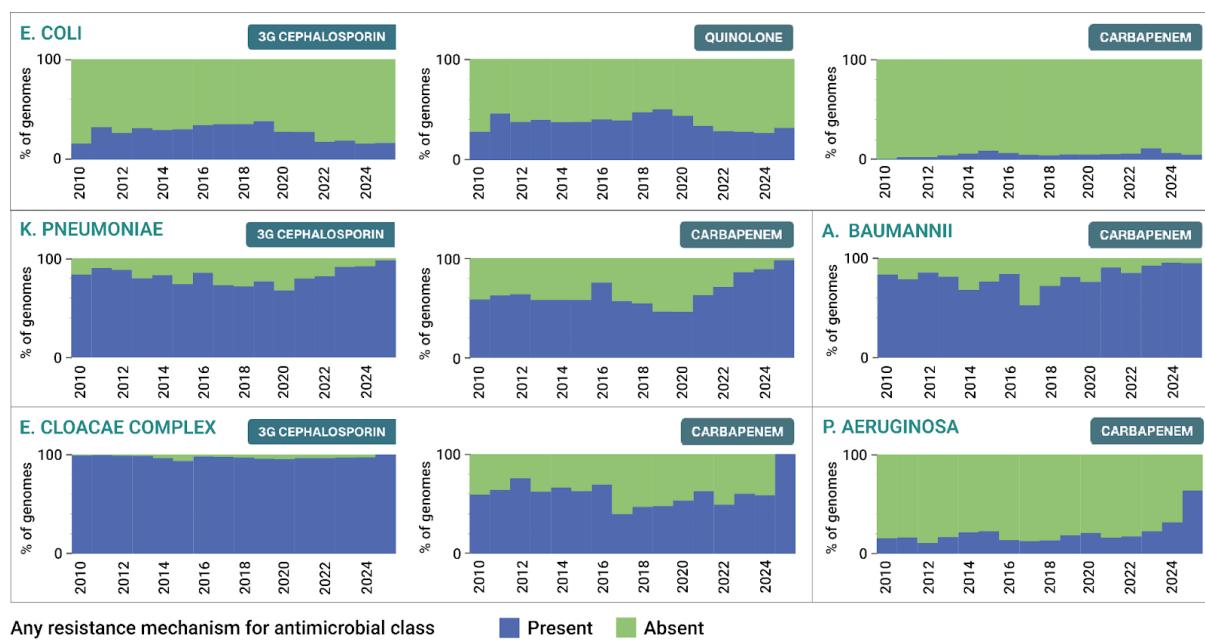
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356 We found that the vast majority (556,307 of 620,700; 89.6%) of genomes incorporated into
357 amr.watch originated from high-income countries, while only 39,843 (6.4%), 18,988 (3.1%)
358 and 5422 (0.9%) were from upper middle-, lower middle- and low-income countries,
359 respectively (**Fig. 2c**). Among genomes from high-income countries, there was also a large
360 skew with 243,644 of 556,307 (43.8%) originating from the USA and a further 122,440
361 (22.0%) from the UK (**Fig. 2d**). The third largest contributing high-income country was
362 Australia, accounting for 33,728 (6.1%) genomes. We found significant geographic gaps
363 globally, with 89 countries (from 249 countries with officially-assigned ISO 3166-1 codes)
364 contributing no genome data that met our defined criteria from 2010 onwards for any of the
365 included pathogens (**Fig. 2d**). This rose to 146 countries when assessing genomes sampled
366 from 2020 onwards. The uneven distribution of data between countries was particularly stark
367 for some pathogens including *Salmonella* Enteritidis, associated with gastroenteritis, for
368 which 35,700 of the 41,779 (85.4%) genomes were from the UK (24,206; 57.9%) or USA
369 (11,494; 27.5%). Notably, 3225 of 6157 (52.4%) genomes belonging to *Salmonella* Typhi
370 were from isolates collected in the UK, a non-endemic region for typhoid fever, and likely
371 associated with travel to endemic regions in Africa, Asia and South America^{17,18}. For many
372 individual countries, there was also high variability in the number of genomes contributed
373 across different species. For example, of 243,644 genomes from the USA, approximately
374 half (50.7%) belonged to either *E. coli* (65,735; 27.0%), *C. jejuni* (33,924; 13.9%) or *C. coli*
375 (23,802; 9.8%).

376

377 We reviewed the proportion of genomes from each pathogen that carried one or more AMR
378 genes and/or mutations conferring resistance to relevant antimicrobial classes, with
379 substantial variability observed across different pathogen-antimicrobial combinations (**Fig.**
380 **3**). This may be due to a combination of factors including true differences in global
381 resistance rates, differences in the extent to which resistance phenotypes can be explained
382 by AMR mechanisms reported in amr.watch, and different degrees of bias among
383 sequenced genomes. Comparison of the genomic data with estimates of antimicrobial
384 resistance from surveillance studies suggested that genome sequences are biased toward
385 resistant isolates for many of the pathogens. For example, 60.5% (28,577 of 47,233) of *K.*
386 *pneumoniae* genomes carried one or more carbapenemase genes that are associated with
387 non-susceptibility to carbapenems, compared with the estimated 28.7% of nosocomial *K.*
388 *pneumoniae* infections with carbapenem resistance globally¹⁹.

389



390 Any resistance mechanism for antimicrobial class ■ Present ■ Absent

391

392 **Figure 3.** Proportion of genomes by year from the five critical-level priority pathogens carrying one or
393 more AMR mechanisms for relevant antimicrobial classes, as displayed in the “AMR mechanism
394 proportion over time” panel within amr.watch. These proportions are based on data from 31 March
395 2025.

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398 Altogether, these findings reflect the varying availability of WGS across different geographic
399 regions to date as well as varying regional public health and research priorities, with a
400 particular ongoing bias towards sequencing of resistant pathogens. The predominance of
401 pathogens such as *Salmonella* Enteritidis, *E. coli* and *Campylobacter* spp. among genomes
402 from the USA and UK also reflects recent adoption of WGS by foodborne pathogen
403 surveillance networks (e.g. GenomeTrackr²⁰, PATH-SAFE²¹) coordinated by national public
404 health laboratories with streamlined data deposition protocols. Notably, the lower proportion
405 of genomes with AMR mechanisms from these pathogens (e.g. *E. coli*, of which 29.8%
406 (53,579 of 179,518) and 39.6% (71,130 of 179,518) of genomes carry third-generation
407 cephalosporin and quinolone resistance mechanisms, respectively), likely also reflects a shift
408 towards broadened surveillance without pre-selection for AMR traits.

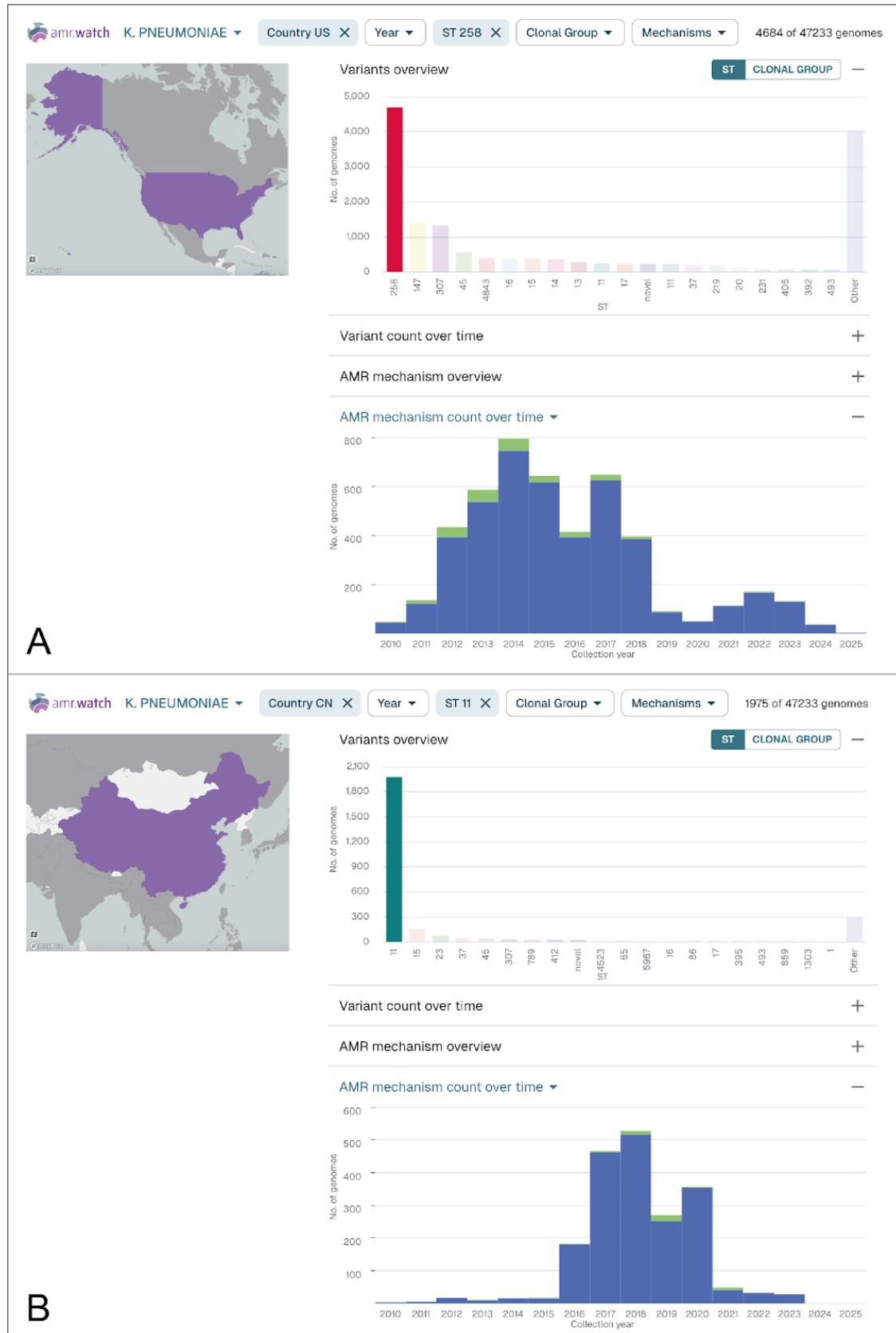
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410 **Identifying geotemporal trends in AMR – towards public health insights**

411 While clear biases and gaps remain in public bacterial genome data, we have found that
412 some evidenced historical trends in AMR can nevertheless be observed using amr.watch.
413 This suggests that available public data may be interrogated, albeit with user-awareness of
414 the data limitations, to explore ongoing trends that could be followed up with further

415 investigation. For example, across many of the pathogens, we can observe that the
416 genomes available to date (as of 31 March 2025) are frequently dominated by a small
417 proportion of the total variants (i.e. STs) observed, especially when filtered by those carrying
418 AMR mechanisms. This reflects a common tendency for a small number of highly-adapted
419 (“high-risk”) variants within a bacterial species to cause the majority of disease cases and/or
420 cases associated with AMR, and thus be prioritised for sequencing within public health and
421 research agendas. This is exemplified by *K. pneumoniae*, where a small number of
422 hospital-associated STs, including ST258, ST11, ST147, ST307 and ST15, are known to
423 dominate drug-resistant infections globally²², and indeed comprise 17,601 of 47,233 (37.3%)
424 of all *K. pneumoniae* genomes from amr.watch. Reports also show the prevalence of these
425 STs varies strongly by country²², consistent with amr.watch data. For example, the majority
426 of *K. pneumoniae* genomes from China belonged to ST11 (1975 of 2827 (69.9%)) (**Fig. 4A**),
427 a persistent trend over several years which is supported by extensive literature on the impact
428 of this variant in China²³. Meanwhile, ST258 is prevalent among *K. pneumoniae* genomes
429 from the USA (4684 of 15,443 (30.3%)) (**Fig. 4B**) although its relative proportion has
430 declined in recent years, in line with reports showing an increasing prevalence of other
431 variants such as ST147²⁴. By contrast, there are some pathogens in amr.watch that exhibit
432 higher diversity, such as *C. jejuni*, for which variants (STs) outside of the top twenty account
433 for over half of the genomes (32,761 of 61,947 (52.9%)). This is in line with the epidemiology
434 of *C. jejuni* infections, which are typically sporadically acquired from eating raw or uncooked
435 meat (primarily poultry) from colonised livestock.

436



437

438 **Figure 4.** *K. pneumoniae* genomes represented in amr.watch as of 31 March 2025 from the USA (A) 439 and China (B) filtered by the most frequent ST from each country (ST258 and ST11, respectively). 440 The “Variants overview” panel shows the twenty most frequent variants (STs) in each country, while

441 the “AMR mechanism count over time” panel shows the number of genomes from the selected variant
442 (ST258/ST11) by year and the number with (blue) and without (green) any carbapenem resistance
443 mechanisms. Updated visualisations with the same filters can be found at:
444 <https://amr.watch/organism/573?charts=1,0,0,1&Country+Code=US&ST=258> and
445 <https://amr.watch/organism/573?charts=1,0,0,1&Country+Code=CN&ST=11>

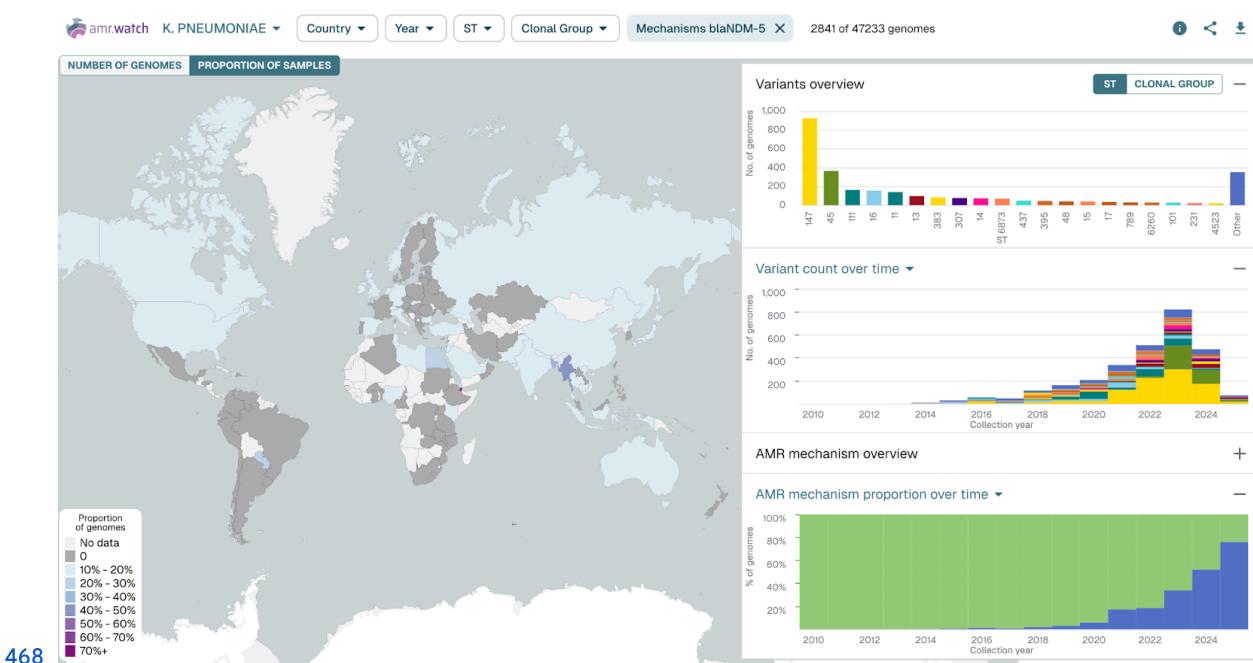
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448 As with variants, we found that there was typically a small number of AMR mechanisms for
449 each antimicrobial class that dominated among the different pathogens, with their distribution
450 also strongly influenced by geographic region. For example, most *K. pneumoniae* genomes
451 with one or more carbapenem resistance mechanisms from China carried the *bla*_{KPC-2} gene
452 (2129 of 2409; 88.4%), while the majority of those from Thailand carried *bla*_{NDM-1} (548 of 893;
453 61.4%), and most from Spain carried *bla*_{OXA-48} (1193 of 1899; 62.8%). We also observed a
454 rise in the proportion of genomes with *bla*_{NDM-5} in *K. pneumoniae* from 2013 onwards (**Fig. 5**),
455 a trend which is also visible among the *E. coli* genomes and in line with the increasing
456 frequency of detection of *bla*_{NDM-5} observed in other surveillance studies²⁵. The identification
457 of these different carbapenemase genes, which belong to carbapenemase classes A
458 (*bla*_{KPC-2}), B (*bla*_{NDM-1} and *bla*_{NDM-5}) and D (*bla*_{OXA-48}), together with scrutiny of their trends, is
459 vital as their different properties can affect interventions. For example, class B
460 carbapenemases render the bacteria non-susceptible to newer antimicrobials such as
461 cefidericol and beta-lactamase/beta-lactamase inhibitor combinations (e.g.
462 ceftazidime-avibactam) which are active against other carbapenemase types. Furthermore,
463 the different gene types produce enzymes with varying hydrolytic activities against
464 carbapenems, with *bla*_{OXA-48} (and other closely-related variants) in particular associated with
465 low MICs which can increase the difficulty of detection by standard laboratory methods²⁶.

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467



477 Discussion

478

479 Genomic-based surveillance of bacterial pathogens has the potential to play a vital role in
480 shaping the global response to AMR. Here we present the amr.watch platform, which
481 automatically extracts and curates key information from the growing volumes of
482 publicly-shared genomes, enabling visual exploration and monitoring of ongoing AMR trends
483 among WHO-defined priority bacterial pathogens. While currently available data lacks broad
484 geographic coverage and has largely been generated for specific research agendas, WGS is
485 increasingly being adopted into routine surveillance systems worldwide. The ability to
486 interrogate geotemporal trends around AMR from shared WGS data offers a key opportunity
487 to more rapidly and precisely define the characteristics of contemporary circulating resistant
488 pathogens. This will allow us to more accurately quantify AMR burden across different
489 populations, as compared with current species-based estimates¹, and thereby prioritise and
490 tailor our interventions more effectively. Such advancements will be important globally but
491 especially in resource-limited settings which are disproportionately affected by AMR¹. Our
492 aim is that information harnessed using amr.watch can be increasingly used by a broad
493 range of stakeholders including those involved in AMR surveillance, vaccine and drug
494 development, and clinical and public health policy. However, we recommend that users
495 remain vigilant to the data limitations and advise that identified trends are followed up with
496 additional investigation.

497

498 To accelerate towards the goal of global AMR surveillance, we encourage all efforts to
499 increase and sustain the implementation of WGS in national and international surveillance
500 systems. This necessitates local investment in infrastructure and capacity-building by
501 governments and public health agencies, driven by evidence of public health value and
502 favourable cost-benefit assessments. However, other common hurdles are also impeding
503 broad implementation, especially in resource-limited settings. These include the
504 procurement, affordability and maintenance of sequencing hardware and reagents, and the
505 acquisition and retainment of personnel with the required laboratory and bioinformatics
506 skills^{27,28}. Such factors have led to increasing recognition that local efforts must also be
507 supported through a global genomics strategy, as outlined by the WHO⁵. Community-led
508 consortia have also taken on vital roles, such as the Public Health Alliance for Genomic
509 Epidemiology (<https://pha4ge.org/>) who are developing accessible resources for different
510 components of genomic surveillance (e.g. data collection, sequencing, analytics,
511 interpretation and data sharing), and thereby aiming to reduce the barriers to entry for local
512 data generators.

513

514 For global genomic surveillance to enable actionable responses to contemporary AMR
515 threats, we also advocate for the timely sharing of data, with ongoing consideration into how
516 this can be incentivised and encouraged. Rapid sharing of data from pathogens associated
517 with other health emergencies (e.g. COVID-19, influenza and Mpox), for example via the
518 Global Initiative on Sharing All Influenza Database (GISAID) database, has shown this is
519 achievable. We found that many publicly-available bacterial genomes were deposited in the
520 sequence archives several years after sampling, although a shift towards rapid, automated
521 data deposition protocols by some national public health agencies and international
522 foodborne surveillance networks is apparent. We also support efforts to improve the
523 availability of metadata, such as the recently-introduced requirement from the INSDC for
524 mandatory spatio-temporal information to be submitted with all sequence data (from May
525 2023). Ongoing efforts within the public health and research communities to develop
526 streamlined metadata guidelines for individual pathogens, including on the provision of
527 information relating to the purpose of sampling, will also further enhance the re-usability of
528 genome data.

529

530 Furthermore, varying gaps also remain in our understanding of how genomic markers
531 translate to resistance phenotypes across different bacteria. While resistance can be
532 predicted with high accuracy from some pathogen genomes, for example with a consistency
533 rate of 98.4% among *S. enterica*, *Campylobacter* spp. and *E. coli*²⁹, other pathogens such as
534 *P. aeruginosa* have proved more challenging, even using additional gene expression data³⁰.
535 As a result, additional research is required to further identify genomic (and gene expression)
536 changes driving AMR development that can be incorporated into resistance prediction tools
537 and monitored within a public health framework.

538

539 In summary, we have developed the amr.watch platform that can act on top of established
540 pathogen surveillance systems to further augment understanding of global AMR dynamics
541 and promote effective prioritisation and use of tailored interventions. Crucially, amr.watch can
542 be readily adapted to include additional pathogens and AMR mechanisms (and other data
543 types), in line with the evolving pathogen landscape. We also aim to incorporate curated
544 genome collections (in addition to the full public collections), enabling interrogation of data
545 generated using defined sampling frameworks. Finally, we urge for widespread investment in
546 local and national AMR surveillance and genomics capacity, alongside other fundamental
547 measures including antimicrobial stewardship, sanitation and infection prevention and
548 control, in response to the rapidly growing crisis of AMR.

549

550 Contributors

551 DMA conceptualised the study. SD, JDC, NC, KA, NFA, AM, SA and DMA designed the
552 amr.watch application interface and features. HMS, PMA, HG, MTGH, EJF, SBS, PDG, RKL
553 and INO provided guidance and feedback on the application design. SD, JDC, NC, NFA,
554 PMA and SA determined the analytical procedures and reviewed the input data for the
555 application. JDC, NC, SA and NFA curated the genome QC thresholds. JDC, NC and SA
556 curated the AMR mechanism library. JDC, KA, NFA, CY and AU developed the pipeline for
557 automated download, genome assembly, QC and analysis of sequence data from the public
558 archives. KA developed the website. DC managed the project. SD, JDC, NC and DMA wrote
559 the article. All authors had full access to all data used in the study, and revised and approved
560 the final version of the manuscript.

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563 Data sharing

564 All data represented in amr.watch are available for download within the application. The
565 assembled genomes and associated metadata can be accessed via Pathogenwatch
566 (<https://next.pathogen.watch>), together with additional genotypic data. Raw sequence reads
567 are available in the ENA/SRA.

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570 Declaration of interests

571 We declare no competing interests.

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