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Antimicrobial Resistance: Global Burden, Drivers and Stewardship Responses

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Abstract

Antimicrobial resistance is now among the leading infectious causes of death worldwide, and the epidemiological picture has changed in ways that matter for policy. This paper reviews the quantitative burden, the drivers that sustain it, and the evidence on interventions that reduce it. Successive systematic analyses place the annual toll at more than a million deaths directly attributable to bacterial resistance, with several million further deaths in which a resistant infection played a part, and current forecasts project substantial growth by the middle of the century. The age distribution of that burden has shifted markedly, with large reductions among young children set against still larger increases among older adults, a pattern that reflects success in child survival programmes alongside the ageing of populations and the expansion of invasive medical care. Drivers are examined across human medicine, animal production and the environment, with attention to the socioeconomic determinants that predict national resistance levels as strongly as consumption volume does. The paper then assesses the evidence on stewardship, where controlled evaluation shows meaningful reductions in resistant infection when prescribing interventions are combined with infection control, and on the antibiotic pipeline, where the economic problem has proved more intractable than the scientific one. The argument throughout is that resistance is a problem of health system capacity and of sanitation as much as of prescribing behaviour.

Keywords: Antimicrobial Resistance, Global Burden of Disease, Antibiotic Stewardship, One Health, Antibiotic Pipeline, Health Systems.

INTRODUCTION

Antimicrobial resistance occupies an unusual position in global health. It is not a disease but a property of pathogens that makes other diseases harder to treat, and its effects are therefore distributed across every infectious syndrome rather than concentrated in one. This diffuseness delayed serious quantification for decades. Early estimates of the death toll were derived from small national datasets and extrapolated with generous assumptions, and the figure of ten million annual deaths by 2050 that circulated widely after an influential policy review was a scenario projection rather than a measurement.[1]

Systematic quantification arrived with the application of burden of disease methods to resistance. The first comprehensive analysis estimated that in 2019 some 4.95 million deaths were associated with bacterial antimicrobial resistance and 1.27 million were directly attributable to it, placing resistance among the leading causes of death globally in that year.[2] A subsequent analysis extended the series

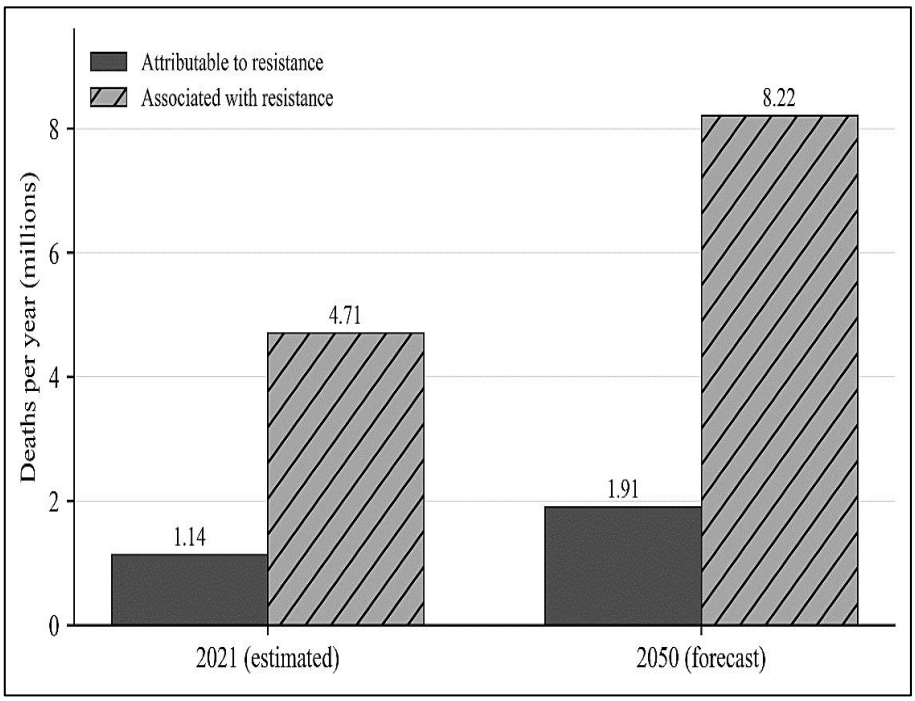
backwards to 1990 and forwards to 2050, and its findings are the empirical basis for much of what follows.[3]

This paper reviews that evidence, examines the drivers that sustain resistance in human medicine, animal production and the environment, and assesses what is known about the interventions available. The emphasis throughout falls on measured effects rather than projections, and on the distinction between what is known with confidence and what remains contested.

THE MEASURED BURDEN

The most recent systematic analysis estimated 1.14 million deaths attributable to bacterial antimicrobial resistance in 2021, with 4.71 million deaths associated with resistance, and forecast 1.91 million attributable and 8.22 million associated deaths annually by 2050. Cumulatively, more than 39 million attributable deaths and 169 million associated deaths are projected between 2025 and 2050.[3] Figure 1 sets the current and forecast figures side by side.

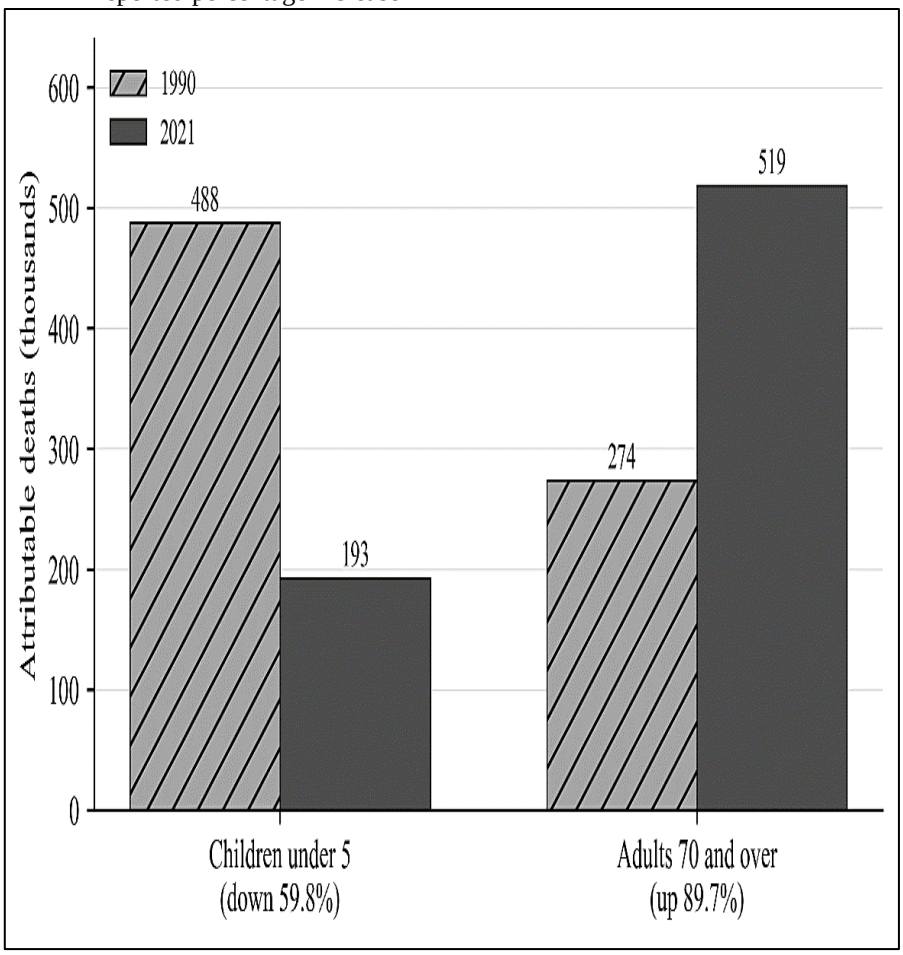
Figure 1: Deaths attributable to and associated with bacterial antimicrobial resistance, estimated for 2021 and forecast for 2050[3]



The two measures answer different questions and should not be conflated. The attributable figure counts deaths that would have been avoided had the infecting organism been susceptible rather than resistant. The associated figure counts deaths in which a resistant infection was present, whether or not resistance itself was decisive. The first is the smaller and more defensible number for evaluating interventions that target resistance specifically; the second is the more relevant number for evaluating investment in infection prevention, since preventing the infection avoids the whole of the associated burden.

The age structure of the burden has changed substantially, and this is the finding with the clearest policy implications. Attributable deaths among children under five fell by 59.8 per cent between 1990 and 2021, from roughly 488,000 to 193,000, while attributable deaths among people aged seventy and over rose by 89.7 per cent to about 519,000.[3] Figure 2 displays the contrast.

Figure 2: Deaths attributable to bacterial antimicrobial resistance by age group, 1990 and 2021. The 1990 value for adults aged 70 and over is that implied by the reported percentage increase[3]



The decline among young children is a genuine achievement, reflecting vaccination, improved water and sanitation, and better management of childhood pneumonia and diarrhoea. The increase among older adults reflects demography together with the expansion of surgery, chemotherapy, dialysis and intensive care, all of which depend on effective prophylaxis and all of which create opportunities for resistant infection. Resistance is therefore increasingly a problem of the health system itself rather than of community acquired infection, and interventions designed for the older pattern will address a shrinking share of the burden.

Pathogen concentration is another consistent finding. Six organisms, namely Escherichia coli, Staphylococcus aureus, Klebsiella pneumoniae, Streptococcus pneumoniae, Acinetobacter baumannii and Pseudomonas aeruginosa, accounted for roughly 930,000 of the 1.27 million attributable deaths in 2019, and methicillin resistant S. aureus was the single pathogen and drug combination responsible for more than 100,000 attributable deaths.[2] Concentration of this kind is useful, because it means that vaccines, diagnostics and treatments directed at a small number of organisms could address a large share of the burden.

PRIORITY SETTING

Prioritisation exercises translate burden into research and development targets. The current bacterial priority pathogens list covers twenty four antibiotic resistant pathogens grouped into three tiers by multicriteria decision analysis, with mortality, transmissibility, treatability and preventability among the criteria.[4] Table 1 reproduces the composition of the tiers.

Table 1. Composition of the WHO bacterial priority pathogens list, 2024, by priority tier[4]

Priority tier	Pathogen and resistance phenotype
Critical	Carbapenem resistant Acinetobacter baumannii
Critical	Carbapenem resistant Enterobacterales
Critical	Third generation cephalosporin resistant Enterobacterales
Critical	Rifampicin resistant Mycobacterium tuberculosis
High	Fluoroquinolone resistant Salmonella enterica serotype Typhi
High	Fluoroquinolone resistant Shigella species
High	Vancomycin resistant Enterococcus faecium
High	Carbapenem resistant Pseudomonas aeruginosa
High	Fluoroquinolone resistant non typhoidal Salmonella
High	Neisseria gonorrhoeae resistant to third generation cephalosporins or fluoroquinolones
High	Methicillin resistant Staphylococcus aureus
Medium	Macrolide resistant group A streptococci
Medium	Macrolide resistant Streptococcus pneumoniae
Medium	Ampicillin resistant Haemophilus influenzae
Medium	Penicillin resistant group B streptococci

Two features of the list deserve comment. The critical tier is dominated by Gram negative organisms, for which the outer membrane presents a permeability barrier that has defeated most recent discovery programmes, and it now includes rifampicin resistant tuberculosis, which had previously been handled through separate policy channels despite carrying a very large share of the resistance burden. The inclusion signals a move towards treating drug resistant tuberculosis as part of the same problem rather than as an adjacent one.

DRIVERS

Consumption in Human Medicine

Antibiotic consumption rose sharply in the first two decades of this century. Analysis of sales data from seventy six countries found substantial global growth between 2000 and 2015, with the steepest increases in low and middle income countries, whose consumption rates converged towards and in some cases exceeded those of high income countries.[5] A separate spatial modelling study covering 204 countries reported a comparable trajectory for the period to 2018.[6] Growth in consumption is not in itself evidence of misuse. Much of the increase in lower income settings reflects improved access to drugs that were previously unavailable, and lack of access still kills more people than excess use does in the poorest countries.

Inappropriate prescribing nevertheless remains widespread. A systematic review of primary care in low and middle income countries found that around half of patients received at least one antibiotic, a proportion far above any plausible estimate of bacterial infection prevalence in those settings.[7] The determinants are not primarily ignorance. Diagnostic uncertainty in the absence of point of care testing, patient expectation, the absence of follow up, and the financial structure of dispensing all favour prescription, and prescribing behaviour within hospitals is shaped by hierarchical convention as much as by guidelines.[8]

Animal Production

A substantial share of global antimicrobial use occurs in food animals, much of it for growth promotion and disease prevention rather than treatment. Surveillance of resistance in animals across low and middle income countries found that the proportion of antimicrobial compounds with resistance above fifty per cent rose from 0.15 to 0.41 in chickens and from 0.13 to 0.34 in pigs between 2000 and 2018, with resistance hotspots concentrated in China and India and emerging in Brazil and Kenya.[9] The transmission pathway from animal to human resistance is real but its magnitude is contested, and it varies by organism. It is well documented for non typhoidal Salmonella and Campylobacter, and much less clear for the Gram negative hospital pathogens that dominate the mortality burden.

Environment and Sanitation

The environmental dimension has moved from a peripheral concern to a central one. Resistance genes are ancient and ubiquitous in soil and water, but selection pressure from pharmaceutical manufacturing effluent, hospital wastewater and agricultural runoff concentrates resistant organisms and mobile genetic elements in ways that favour transfer to human pathogens.[10] Metagenomic analysis of urban sewage across sixty countries showed that resistance gene abundance correlated strongly with sanitation and health system indicators, and that the geographical pattern of environmental resistance mirrored the pattern of clinical resistance.[11]

This finding supports one of the more important arguments in the field. Analysis across 103 countries found that indicators of governance, sanitation infrastructure and public health expenditure predicted national resistance levels more strongly than antibiotic consumption did.[12] If that relationship is causal even in part, then investment in water, sanitation and infection control may reduce resistance more efficiently in many settings than prescribing reform alone, because it reduces the transmission of resistant organisms rather than only the selection pressure that produces them.

WHAT WORKS

Stewardship

Antibiotic stewardship comprises the interventions that improve the selection, dose, route and duration of antimicrobial treatment. The strongest evidence comes from a systematic review and meta analysis of thirty two studies covering more than nine million patient days, which found substantial reductions in the incidence of infection and colonisation with resistant organisms following stewardship implementation.[13] Table 2 gives the principal effect estimates.

Table 2. Reductions in incidence of resistant infection and colonisation following antibiotic stewardship, from meta analysis of 32 studies[13]

Outcome	Reported reduction in incidence
Multidrug resistant Gram negative bacteria	51 per cent
Extended spectrum beta lactamase producing Gram negative bacteria	48 per cent
Methicillin resistant Staphylococcus aureus	37 per cent
Clostridioides difficile infection	32 per cent
Stewardship combined with hand hygiene compared with stewardship alone	Larger effect than stewardship implemented alone

The final row carries the most practical message. Stewardship was consistently more effective when implemented alongside infection control measures, and particularly alongside hand hygiene interventions, than when implemented on its own. Reducing selection pressure and interrupting transmission are complementary rather than alternative strategies, and programmes that fund one without the other forgo much of the available benefit. Independent evidence from controlled trials of prescribing interventions in hospital inpatients supports the same conclusion, with persuasive and restrictive interventions both improving guideline compliance and reducing duration of therapy without evidence of harm to patients.[14]

The pipeline

The supply of new antibiotics has not kept pace with resistance, and the obstacle is economic rather than scientific. A new antibiotic that works against a critical priority pathogen will, if stewardship functions as intended, be held in reserve and used sparingly, which is precisely the opposite of the high volume use that recoups development costs. Several companies that successfully brought novel agents to market have subsequently failed commercially.[15] Analysis of the preclinical pipeline finds a reasonable number of candidate projects but a marked shortage of genuinely novel classes active against Gram negative organisms, and a heavy dependence on small enterprises and public funding at the stage where attrition is highest.[16]

Proposed remedies separate the reward for developing an antibiotic from the volume sold. Subscription arrangements, market entry rewards and transferable exclusivity vouchers have all been

trialled or legislated in various jurisdictions. The evidence on their effectiveness remains thin, and the design problem is genuinely difficult, since a reward large enough to attract investment is also large enough to attract political objection.

System Capacity

The interventions that have historically done most to reduce the burden of resistant infection are not specific to resistance at all. Vaccination removes infections that would otherwise be treated, sanitation reduces transmission, and diagnostic capacity permits treatment to be targeted. In low and middle income countries, the binding constraint is frequently the absence of a microbiology laboratory able to identify an organism and test its susceptibility, without which stewardship guidelines cannot be locally calibrated and surveillance data cannot be generated.[17] The global action plan on resistance accordingly places surveillance and infection prevention alongside stewardship as its operational objectives, though implementation has been uneven and national action plans have frequently been produced without the financing to execute them.[18]

LIMITATIONS OF THE EVIDENCE

Three limitations qualify the figures presented here. The first is data sparsity. Burden estimates rest on modelled extrapolation from surveillance systems that are dense in Europe and North America and thin across much of sub Saharan Africa and South Asia, precisely where the modelled burden is highest, so the uncertainty intervals are wide and the central estimates depend on structural assumptions in the models.[2,3]

The second is the counterfactual. Attributable burden requires an estimate of what would have happened had the organism been susceptible, which is not directly observable and must be inferred from studies comparing outcomes in resistant and susceptible infections, studies that are vulnerable to confounding by indication and by severity.

The third is forecasting. The projections to 2050 assume the continuation of current trends in demography, health system development and resistance emergence. They are useful for indicating direction and order of magnitude, and they should not be read as predictions with the precision their decimal places imply.[3]

CONCLUSION

The measured burden of antimicrobial resistance is large, comparable to that of major established causes of death, and its distribution is shifting from young children towards older adults receiving invasive medical care. The drivers extend well beyond prescribing to encompass animal production, environmental contamination and, most consistently, the state of sanitation and health system infrastructure.[12] The interventions with the best evidence behind them are stewardship combined with infection control, which together reduce resistant infection substantially in hospital settings,[13] and the unglamorous measures of vaccination, water and sanitation that prevent infection from occurring. The pipeline problem is real but it is a problem of market design rather than of microbiology, and it will not be solved by exhortation.[15,16] A response proportionate to the burden would fund laboratory capacity and infection prevention in the settings that carry most of it, rather than concentrating attention on prescribing behaviour in the settings that carry least.

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