

# Climate change and antimicrobial resistance

Erta Kalanxhi<sup>1</sup> & Ramanan Laxminarayan<sup>2,3</sup>  

## Abstract

Climate change is increasingly linked to a surge of extreme weather events, raising the risk of disease outbreaks and food insecurity. Meanwhile, an increase in emerging and re-emerging infectious diseases, many of which do not respond to available antibiotics owing to antimicrobial resistance (AMR), poses another great challenge to public health. Although some studies have shown that climate change and extreme weather events are associated with higher levels of AMR, much work remains to determine whether these are causal linkages or merely parallel reflections of an anthropogenic change. In this Review, we explore evidence on the relationship between climate and AMR, highlighting pathways through which rising temperatures and extreme weather events might intensify this pressing issue. Beyond existing ecological evidence demonstrating correlations between temperature and AMR prevalence in clinically important pathogens, a growing body of work suggests that the predominant impact of climate change on AMR manifests through an increase in infectious disease prevalence and a demand for antimicrobial use. Current evidence on the relationship between climate and AMR is insufficient in addressing issues related to temporality and causality, and underscores the need for further research to understand the nature of these complex relationships.

## Sections

Introduction

Infectious disease incidence and transmission

Increase in antimicrobial use

Rising temperatures and antibacterial resistance

Antifungal resistance

Climate change and host-related factors

Mitigation

Conclusion

<sup>1</sup>One Health Trust, Washington, DC, USA. <sup>2</sup>One Health Trust, Bangalore, India. <sup>3</sup>School of Public and International Affairs, Princeton University, Princeton, NJ, USA. ✉e-mail: [ramanan@onehealthtrust.org](mailto:ramanan@onehealthtrust.org)

## Introduction

The decreasing effectiveness of antibiotics owing to antimicrobial resistance (AMR) threatens the progress made in reducing infectious disease mortality since the drugs were first used in the 1940s<sup>1–3</sup>. About two-thirds of approximately 7.7 million deaths from bacterial infections each year are associated with AMR, with roughly one-sixth directly attributable to drug-resistant infections<sup>4</sup>. Meanwhile, invasive fungal infections affect 6.5 million people annually and are associated with 3.8 million deaths, many of which are owing to antifungal resistance<sup>5</sup>. Although AMR is a global challenge, its burden varies greatly across countries, partly owing to disparities in access to water, sanitation and hygiene (WASH) infrastructure, diagnostics, vaccines and antimicrobials<sup>6,7</sup>. Over the past three decades, the global AMR burden has been increasing at varying rates across countries and age groups, with low-income and middle-income countries (LMICs) bearing the greatest burden, and AMR-related deaths among adults aged 70 years and older increasing by 80%<sup>6</sup>.

Although AMR occurs naturally, it is accelerated by the exposure of pathogens to antimicrobials, with overuse and misuse increasing the selection pressure for mutations that enhance survival<sup>8</sup>. In the context of bacterial AMR, the transfer of antibiotic resistance genes (ARGs) through horizontal gene transfer (HGT) presents an opportunity for resistance to spread within bacterial populations<sup>9</sup>. Antimicrobial use in humans, animals and plants – shaped by the prevalence of infectious diseases and socioeconomic development – is a key driver of AMR<sup>8,10–12</sup>. However, emerging evidence on the planetary consequences of climate change, such as rising temperatures, extreme weather events and ecosystem disruptions, suggests that it may also contribute to the development and spread of AMR.

Climate change is another major global challenge, influenced by human behaviour and economic activity<sup>13,14</sup>. Although economic growth and industrialization have substantially reduced mortality from infectious diseases and malnutrition, these improvements have come at the cost of pollution and environmental degradation, which

now threaten to reverse those gains. Reliance on fossil fuels and deforestation (Box 1), along with a growing human population, have resulted in increases in atmospheric carbon content<sup>14–16</sup>. These large-scale environmental changes related to greenhouse gas (GHG) emissions, specifically carbon dioxide and methane, are linked to record-high temperatures and extreme weather (droughts, floods and storms), which have a demonstrable impact on human health<sup>17,18</sup>. In 2024, heat-related mortality among those over 65 years of age increased by 167%, compared with the 1990s, whereas rising temperatures and changing precipitation patterns are raising the number of people at risk of vector-borne diseases<sup>19</sup>. Climate-related hazards, such as warming temperatures, precipitation, floods, droughts and storms, are estimated to exacerbate more than half (58%) of human pathogenic diseases that are sensitive to climate<sup>17</sup>. Through more than 1,000 transmission pathways including various routes (vector-borne, water-borne, airborne, direct contact and food-borne), climatic hazards led to conditions that increased contact between humans and pathogens (expanded vector ranges and spillovers), improved pathogen traits (reproduction, life cycle and virulence), or reduced the defences of a host by impairing immunity, limiting health-care access and reducing food security<sup>17</sup>.

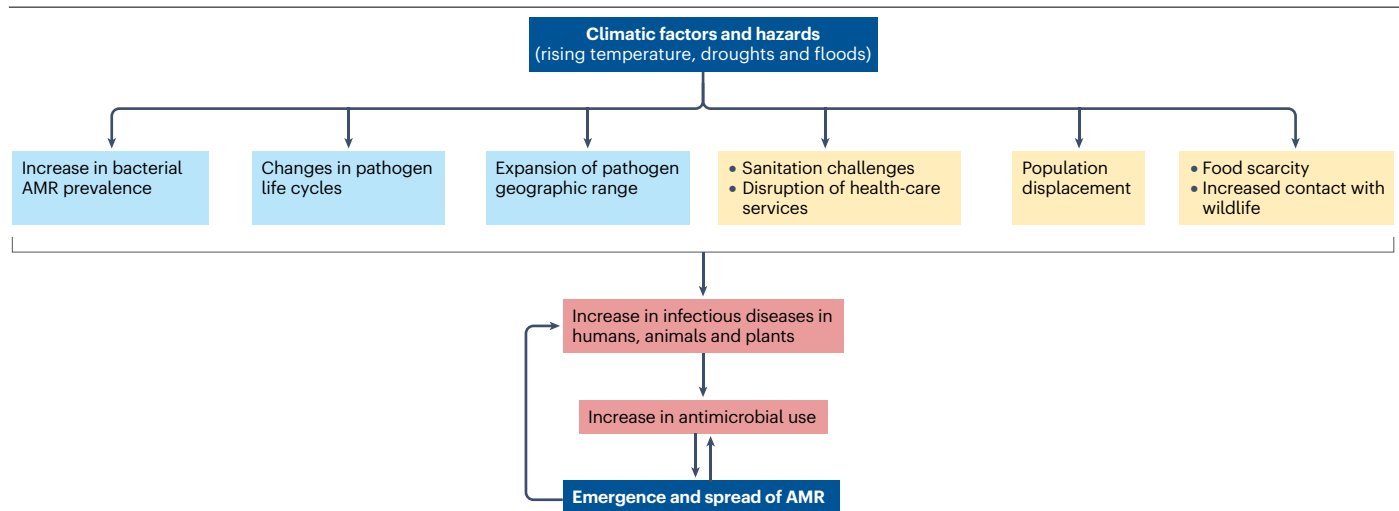
Several studies have demonstrated temperature-dependent increases in infections caused by drug-resistant pathogens in hospital and community settings<sup>20–23</sup>, and associations between higher ambient temperatures and the prevalence of AMR among clinically important bacterial and fungal pathogens<sup>24,25</sup>. Increased temperatures and thawing of permafrost have both been linked to a greater abundance and diversity of ARGs in water environments and permafrost-associated soils<sup>26</sup>. Furthermore, rising temperatures have been shown to increase the frequency of HGT events in pathogenic bacterial populations<sup>27,28</sup>. However, these associations between temperature and AMR or related factors do not necessarily imply correlation or causation. Economic growth and population increase, which drive both anthropogenic climate change and antibiotic consumption (a major driver of AMR),

## Box 1 | Global factors mediating the impact of climate change on antimicrobial resistance

The emergence and spread of infectious diseases and antimicrobial resistance (AMR) are influenced by many global factors that are sensitive to climate change<sup>199</sup>. Increasing pressure on food systems owing to changing climates and a growing global population leads to intensified farming practices and a higher risk of food-borne pathogen outbreaks<sup>110,200</sup>. Deforestation, primarily driven by agricultural expansion and urbanization, brings wildlife closer to human populations, raising the likelihood of zoonotic disease spillover<sup>33,34</sup>. Migration owing to poverty, conflicts and climate issues forces populations to live in overcrowded conditions, with limited access to health care, placing them at a higher risk of facing disease outbreaks and contributing to increased transmission of multidrug-resistant pathogens<sup>39,40,198</sup>. Food insecurity and malnutrition further exacerbate the global burden of disease and AMR by increasing susceptibility to infections while weakening immunity and the ability to fight these infections<sup>155,156,167,200</sup>. These interconnected factors create conditions that increase the frequency and severity of infectious disease outbreaks and the threat of AMR, which is further amplified by climate change. This underscores the urgent need to develop comprehensive global

strategies that address climate change and strengthen public health resilience.

The effects of climate change on health and AMR vary across regions and populations, as does the ability to mitigate these effects. Therefore, further research on how changing climates influence health outcomes is crucial to developing context-specific policies for action. To address the global burden of infectious diseases and AMR, priorities should include infection prevention and treatment, optimizing antibiotic use, and ensuring food security to prevent malnutrition. Access to clean water and health care remains crucial, especially for populations living in poverty and in conflict zones or affected by climate-driven migration<sup>7182</sup>. Improving diagnostic capabilities and enhancing disease and AMR surveillance can help manage outbreaks and reduce antibiotic misuse and overuse<sup>186–188</sup>. Expanding access to a broader range of antibiotics, particularly in low-resource settings, can reduce reliance on a few overprescribed drugs and ensure access to the right treatment at the right time<sup>103,105,106</sup>. Finally, promoting food security through sustainable agriculture and improved animal health can reduce antibiotic and pesticide use while addressing nutritional needs<sup>8,42,195</sup>.



**Fig. 1 | Warming climates and extreme weather events exacerbate infectious disease burden and antimicrobial use.** Rising temperatures, droughts and floods affect pathogens and their hosts through various pathways. Rising temperatures have been shown to increase the rate of bacterial infections<sup>20,21,50,52</sup> and the prevalence of antimicrobial resistance (AMR) in bacterial pathogens<sup>24,55,56</sup>. Increased thermotolerance can lead to changes in pathogen life cycles and expansion of pathogen ranges, resulting in unpredictable seasonal disease patterns and the emergence of novel pathogens<sup>25,135,138,145</sup>. Sanitation challenges during droughts and floods limit access to clean water and sanitation infrastructure, increasing the incidence and spread of infections<sup>66,72,197</sup>.

Health-care disruptions and population displacement increase the risk and transmission of infections, contributing to disease outbreaks and the spread of multidrug-resistant pathogens<sup>39,40,198</sup>. Furthermore, climate change-related food scarcity and land-use change can increase human interactions with wildlife, raising the risk of zoonotic disease spillover<sup>33,34,88</sup>. The resulting increase in infectious disease incidence and transmission is estimated to further increase the demand for antimicrobials, which are both drivers and consequences of AMR. Although overuse and misuse of antimicrobials increase the selection pressure for the development of resistance, AMR often results in their overuse. Please note that this is not an exhaustive list of factors and impacts.

may be common explanatory factors<sup>29,30</sup>. Nevertheless, plausible causal pathways for the links between climate change and AMR include climate-driven increases in the transmission of existing pathogens and the emergence of novel pathogens, which are heightened by increased land-use changes and greater contact between humans and wildlife<sup>31–34</sup>.

Climate-induced sanitation challenges, such as water source contamination during floods and droughts, can amplify the spread of drug-resistant pathogens and ARGs between humans, animals and the environment<sup>26,35–37</sup>. Furthermore, ecosystem disruptions have led to increased food insecurity, population displacement, more frequent disease outbreaks, and reduced access to essential health services<sup>38–40</sup>. The overall rise in infectious disease incidence and increased reliance on antimicrobials for treatment and prevention are raising the risk of AMR and making populations more vulnerable to its impact<sup>41</sup>. In agriculture, water scarcity, food insecurity and the intensification of livestock production systems are estimated to lead to increased antibiotic use in food-producing animals, affecting both animal and human health<sup>11,42</sup>. Additionally, changes in temperature, humidity and precipitation tend to alter the growth rates and geographic ranges of plant pathogens and insect pests, raising the risk of disease development and increasing pesticide use<sup>43–45</sup>. Many of the factors that affect climate change and AMR are interdependent. Research shows that climate change and AMR may have shared drivers, such as intensified agricultural practices, land use and deforestation, and shared solutions, including agricultural transformation and biotechnological innovations that make crops more resistant to climate change or provide antibiotic alternatives for human and animal health<sup>46</sup>.

In this Review, we examine the evidence supporting the links between climate change and AMR. We highlight opportunities for

action and identify areas wherein further research would help understand the strength of association and causality when it seems plausible and is supported by evidence (Box 1). We explore the associations between temperature and the incidence of bacterial infections and the prevalence of AMR among human bacterial pathogens, as well as potential biological mechanisms and environmental factors that may affect these associations. Additionally, we discuss the impact of climate change on the rise of other infections – viral, parasitic, fungal and vector-borne – in human health and agriculture, as well as the demand for antimicrobials. Furthermore, we discuss how temperature may directly influence the development and spread of AMR through genetic mutations and HGT. Highlighting the importance of biological and host-related factors, we review how rising temperatures and extreme weather events affect host immunity and how socioeconomic determinants of health may influence the development and mitigation of AMR. Finally, we conclude by highlighting knowledge gaps and research needs and suggesting actions to mitigate the impact of climate change on AMR and its effects on human health. Although we acknowledge the complexity of the relationship between climate, health and AMR, this Review will not focus on the role of non-communicable diseases in the climate–AMR nexus.

## Infectious disease incidence and transmission

### Rising temperatures increase bacterial infection rates

Climatic factors and climate-related hazards affect both pathogens and their hosts, and have been shown to increase the burden of infectious diseases and related antimicrobial use, which are both key drivers and consequences of AMR<sup>47,48</sup> (Fig. 1). A recent analysis has revealed that human pathogenic bacteria have been identified in 88% of over

1 million sequenced microbial communities from animals, plants, soil and aquatic environments worldwide<sup>49</sup>. These bacterial populations were influenced by climate factors, such as higher temperatures and increased precipitation, as well as anthropogenic activities<sup>49</sup>. Numerous studies have explored the relationship between temperature or seasonal variations and the prevalence of bacterial infections in hospital and community settings, with most indicating an association between temperature and Gram-negative bacterial infections<sup>20,21,50–54</sup>. Gram-negative bacterial infections have been shown to follow a seasonal, temperature-dependent trend, wherein increases in outdoor temperatures correlate with increased occurrences of bloodstream infections caused by pathogens such as *Escherichia coli*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae* and *Acinetobacter* spp.<sup>20,21,50,52</sup>. Other studies have found associations between higher temperatures and increased community-onset cases of *Acinetobacter baumannii* complex and *E. coli* infections, but not with hospital-acquired infections<sup>22,23</sup>. Behavioural, geographical and socioeconomic factors may also affect the incidence of bacterial infections. For instance, proximity to the equator and lower health-care spending as a proportion of the gross domestic product (GDP) were associated with a greater likelihood of Gram-negative bloodstream infections<sup>51</sup>.

## Temperature affects the prevalence of antibiotic resistance among bacterial pathogens

Rising temperatures may increase not only bacterial infection rates but also the risk of antibiotic resistance in bacterial pathogens (Fig. 1). In the USA, a 10 °C rise in ambient temperature across different regions was associated with 2.2% to 4.2% increases in antibiotic resistance prevalence among pathogens such as *E. coli*, *K. pneumoniae* and *Staphylococcus aureus*<sup>24</sup>. Similarly, in China, AMR trends between 2014 and 2020 revealed that a 1 °C rise in annual average temperature was associated with 4.7% and 10.7% increases in third-generation cephalosporin-resistant and carbapenem-resistant *K. pneumoniae* isolates, respectively<sup>55</sup>. Another study supports these findings by demonstrating that, after accounting for variables such as antibiotic use, population density and minimum ambient temperature, European countries experiencing greater temporal variations in minimum ambient temperatures between 2000 and 2016 had higher AMR prevalence among *E. coli* and *K. pneumoniae* isolates and declines in resistance among methicillin-resistant *S. aureus* isolates<sup>56</sup>. Similarly, a large ecological study in China has observed that a 1 °C increase in average ambient temperature was associated with a 1.14-fold rise in carbapenem-resistant *K. pneumoniae* and a 1.06-fold rise in carbapenem-resistant *P. aeruginosa* prevalence<sup>57</sup>.

However, the relationship between temperature and resistance development is complex and may not follow a linear trend. A study investigating antibiotic resistance to broad-spectrum fluoroquinolones in *E. coli* strains at various temperatures ranging from 22 °C to 42 °C has found that resistance increased 256-fold at 27 °C but declined at 42 °C<sup>58</sup>. In addition to the complex interplay of these physiological factors, population-level factors have been shown to modulate the association between temperature and AMR prevalence. In China, the impact of rising ambient temperature on the prevalence of antibiotic resistance in *E. coli* isolates was more pronounced in regions with lower socioeconomic status, characterized by lower income, fewer health-care resources, and lower hospital admission rates<sup>59</sup>. By contrast, a large study across 30 European countries has revealed that, after adjusting for governance and economic indicators, factors such as antibiotic consumption, population density and the governance

index, rather than temperature change, were correlated with AMR prevalence<sup>59,60</sup>. Ecological studies examining the association between temperature and the prevalence of bacterial infections and antibiotic resistance offer valuable insights at the population level. However, these studies often lack information about individual patient groups, demographics and socioeconomic factors, which makes it challenging to draw inferences at the individual level or to establish clear cause-and-effect relationships.

## Bacterial responses to temperature changes and antibiotics

Research suggests that the impact of temperature on AMR prevalence in bacterial pathogens may be orchestrated by overlapping bacterial physiological and genetic responses to both heat and antibiotic exposure<sup>61</sup>. Antibiotics that target ribosome functions and induce protein folding stress have been shown to elicit cellular responses resembling those of heat-shock or cold-shock<sup>62</sup>. For example, the sigma factor  $\sigma$ 32 regulon in *E. coli* regulates the response to heat-shock, but it can also be induced by antibiotics such as aminoglycosides, cephalosporins and fluoroquinolones<sup>63</sup>. Through stressor interaction network analysis, researchers have demonstrated an overlap between bacterial physiological responses to temperature and antibiotics, with bacteria adapted to high temperatures being more sensitive to antibiotics that elicit responses similar to those of cold-shock<sup>61</sup>. Therefore, it is plausible that bacterial adaptation to warmer temperatures, or thermotolerance, could lead to increased resistance to certain antibiotics. For example, *A. baumannii* cells pretreated at higher temperatures were better at surviving streptomycin exposure than cells pretreated at normal physiological temperatures<sup>64</sup>. Additionally, exposure of a multidrug-resistant strain of *A. baumannii* to various antibiotics induced the heat-shock protein DnaK<sup>64</sup>. Moreover, antibiotics themselves have been shown to shift and reduce the optimal growth temperature range of the bacteria<sup>65</sup>.

## Extreme weather events increase disease transmission

Extreme weather events such as floods, droughts and storms cause important sanitation challenges (Fig. 1). These include water shortages, contamination and damage to WASH infrastructure<sup>66–68</sup>. Moreover, such events often lead to population displacement and reduce access to health-care services<sup>19,38,69</sup>. These disruptions increase the risk of infectious disease outbreaks and weaken health-care systems, contributing to the spread of drug-resistant infections and a greater reliance on antimicrobials<sup>70,71</sup>. For instance, flooding can spread bacteria from wastewater, whereas water scarcity during droughts concentrates bacteria in water sources and limits access to clean drinking water<sup>72</sup>. During frequent cholera outbreaks, the extensive use of antibiotics, combined with the propensity for HGT between *Vibrio cholerae* strains, contributes to the emergence and spread of antibiotic-resistant *V. cholerae*<sup>73,74</sup>. Furthermore, the widespread use of antibiotics during a single disease outbreak can compromise their effectiveness in future outbreaks. In 2018, high resistance rates (240 out of 241 cases) to ceftriaxone among *V. cholerae* O1 serotype Ogawa isolates in Harare, Zimbabwe, were probably linked to the extensive use of extended-spectrum cephalosporins during a previous typhoid outbreak<sup>75</sup>.

## Climate change affects pathogen seasonality and vector range

Climate-induced changes in the epidemiology and seasonality of viral and parasitic infections may also have implications for AMR, as persistent infections increase the susceptibility of a host to bacterial co-infections<sup>76–79</sup> (Fig. 1). Additionally, in low-resource settings,

misdiagnosis of viral infections often leads to frequent and inappropriate antibiotic use<sup>80,81</sup>. Traditionally, peaks in viral respiratory infections have been linked to seasonal variations in temperature, humidity and behavioural factors; however, climate change could make these seasonal cycles more unpredictable, expand the geographical range of pathogens, and increase the risk of cross-species viral transmission<sup>32,82</sup>. For example, vector-borne parasitic infections such as malaria, dengue, West Nile fever, leishmaniasis, Chagas disease and amoebic dysentery are predominantly found in tropical and subtropical regions; however, rising temperatures and increased humidity could expand the distribution of the disease-causing pathogens to new areas<sup>83,84</sup>. Projections indicate that the prevalence and geographical range of these vector-borne diseases will probably increase globally, particularly in Europe and North America<sup>18,84</sup>. The rise in viral and vector-borne infections is expected to amplify the risk of zoonotic disease transmission and increase reliance on antimicrobials, which, in turn, drives the development of resistance<sup>71,82</sup>.

## Extreme weather increases disease risk in agriculture

Extreme weather events can increase the prevalence of infectious diseases in animals and have an effect on human health<sup>85–87</sup>. Depending on their severity and duration, heatwaves can cause metabolic disruptions and immune suppression in animals, making them more susceptible to infections<sup>86</sup>. Furthermore, climate-induced disruptions lead to land-use changes, which can increase the risk of zoonotic disease transmission and the spread of mobile genetic elements that confer resistance<sup>88</sup>. Additionally, changing temperature and precipitation patterns alter the geographic range and transmission potential of pathogens and their vectors, thereby increasing the number of animals at risk<sup>89</sup>. Similar vulnerabilities are observed in aquaculture, wherein environmental stressors, including temperature fluctuations, salinity changes and low pH, combined with high-density farming conditions, heighten susceptibility to disease, as well as the risk and spread of infections<sup>90</sup>. In addition to environmental dissemination, the consumption of contaminated animal products and direct contact between humans and animals provide additional pathways for transmitting drug-resistant pathogens<sup>91</sup>. Although the reduction of antibiotic use in animals can reduce the selection pressure for AMR, more research is needed to understand and quantify this effect at the human–animal health interface<sup>92–94</sup>.

Antifungals, antibiotic and other biocides are critical inputs in agriculture, including horticulture. Although antibiotic use in agriculture is generally considered to be lower than in human and animal health, the lack of comprehensive global monitoring suggests that it may be more widespread than previously thought<sup>95</sup>. The effects of temperature, humidity and CO<sub>2</sub> levels on plant pesticide use are not fully understood, mainly owing to the variability in optimal conditions for different plant and pathogen combinations<sup>45</sup>. However, it is projected that global warming will alter the regional and temporal patterns of crop susceptibility to certain pathogens<sup>45</sup>. This, combined with the potential for pathogens to develop thermotolerance, could lead to more frequent pesticide applications and increased selection pressure for resistance<sup>45</sup>. For instance, warmer temperatures are expected to accelerate metabolic and growth rates in some insects, resulting in higher plant consumption by insects and greater damage to crops<sup>43</sup>. Additionally, climate change may facilitate year-round survival for some agricultural pests such as the diamondback moth (*Plutella xylostella*), the European corn borer (*Ostrinia nubilalis*), the codling moth (*Cydia pomonella*), the peach twig borer (*Anarsia lineatella*), and the oriental fruit moth (*Grapholita molesta*), necessitating

repeated pesticide exposure and increasing the pressure for AMR emergence<sup>44,96,97</sup>. Although changes in environmental conditions may have different effects on plant health and pathogen virulence, the overall impact probably depends on a balance between pathogen growth and virulence, plant susceptibility and the effectiveness of pesticides<sup>98</sup>.

## Increase in antimicrobial use Antibiotic consumption in human health

The projected increase in infectious disease burden and exacerbations owing to climate change is expected to lead to greater reliance on antimicrobials and heightened selection pressure for AMR. Specifically, antibiotic consumption in humans increased by 10.6% between 2016 and 2023, with the increase potentially reaching 52.3% by 2030 (ref. 99). Notably, a substantial portion of this increase, estimated at around 37%, is owing to inappropriate antibiotic use, a pattern that persists across regions regardless of the income levels<sup>100</sup>. Additionally, climate-induced shifts in the seasonal cycles of infectious diseases, such as influenza outbreaks, can further increase overall antibiotic consumption and inappropriate use, ultimately contributing to the development of antibiotic resistance in both community and clinical settings<sup>79,101</sup>. Adding another layer of complexity to the projected increase in antibiotic consumption is the overreliance on a few antibiotics and the lack of access to others, which complicates efforts to mitigate the health impacts of climate change. For example, rising temperatures are associated with increases in rates of Gram-negative bacterial infections, which are increasingly challenging to treat owing to their propensity for antibiotic resistance<sup>20,21,51,52,54,102</sup>. However, in many LMICs with high infectious disease burden, limited access to antibiotics complicates the treatment of these infections<sup>103–105</sup>. A recent study of antibiotic consumption in 11 African countries has found that facility-level antibiotic use was dominated by only 10% of the Access category of antibiotics recommended by the WHO, possibly suggesting a lack of access to other antibiotics<sup>106</sup>.

## Antibiotic consumption in animal health

The rising demand for animal protein production has important impacts on both animal and environmental health<sup>93,107,108</sup>. Intensified livestock production systems developed to meet this demand increase the risk of infectious diseases among animals, whereas animal protein production contributes 12% of total GHG emissions from human activities<sup>108,109</sup>. Crowded and unsanitary farming conditions enhance disease incidence and transmission and weaken animal immune systems, leading to higher antibiotic use<sup>110,111</sup>. Notably, antibiotic use in animals constitutes approximately 70% of total antibiotic consumption and is projected to reach 107,500 tonnes by 2030, an 8% increase from 2020 baseline levels<sup>30,112</sup>. Additionally, the use of medically relevant antibiotics in aquaculture is expected to grow by 33% between 2017 and 2030 globally, with the Asia-Pacific region accounting for 93.8% of this consumption<sup>113</sup>. These estimates do not consider climate-related increases in antibiotic demand, which suggests that actual future use may be underestimated. Although research within the One Health framework remains limited, existing studies indicate a connection between agricultural antibiotic use and AMR in humans. A recent global modelling study has identified a significant correlation between animal antibiotic consumption and AMR in human health<sup>11</sup>. Similarly, a meta-analysis has found a relationship between antibiotic resistance in aquaculture and clinical bacteria in humans<sup>90</sup>. In plant agriculture, the use of azole pesticides has been linked to antifungal resistance in clinical settings<sup>114</sup>.

## Rising temperatures and antibacterial resistance

### Bacterial mutation rates

Temperature can exert direct effects on AMR through modulation of pathogen mutation rates and HGT (Fig. 2). For example, an in vitro study has found that *E. coli* isolates exposed to ciprofloxacin and rifampicin had higher resistance mutation rates at 40 °C than at 37 °C, whereas exposure to ampicillin resulted in lower mutation rates at the higher temperature<sup>115</sup>. A potential mechanism for the overall increase in mutation rates includes the enhanced activity of error-prone DNA polymerases during heat-shock<sup>116</sup>. As previously noted, heat stress and antibiotics may affect similar cellular functions. Consequently, mutations that confer thermotolerance may also lead to antibiotic resistance<sup>61</sup>. For example, *E. coli* cultured over 2,000 generations at increased temperatures developed mutations in RNA polymerase (a shared target for antibiotic resistance and heat stress), which subsequently conferred rifampicin resistance even without prior antibiotic exposure<sup>117</sup>.

### Horizontal gene transfer

Plasmid-mediated ARG transfer via HGT is a well-established pathway for the spread of antibiotic resistance in both environmental and clinical settings<sup>27,118</sup>. Although the effect of temperature on HGT-driven ARG dissemination is not fully understood, some studies suggest that higher temperatures can increase the frequency of conjugation events among clinically relevant pathogens in environmental models<sup>118,119</sup>. However, this effect seems to be nonlinear and may vary between species, including both donor and recipient strains. For example, conjugative transfer of the New Delhi metallo- $\beta$ -lactamase I gene, *bla*<sub>NDM-1</sub>, from environmental bacteria to *E. coli*, *Salmonella enterica* serotype Enteritidis and *Shigella sonnei* more frequently at 30 °C than at 25 °C or 37 °C, with transfer to *E. coli* being the most efficient across all temperatures<sup>119</sup>. These variations tend to be influenced by factors such as donor–recipient compatibility and other environmental conditions<sup>120</sup>. These findings, combined with those

from another study showing stronger associations between temperature and resistance to fluoroquinolone and beta-lactam antibiotics (wherein plasmid-mediated resistance is an important mechanism for AMR), support the hypothesis that temperature may modulate specific pathways of antibiotic resistance dissemination<sup>24,121,122</sup>.

### Antibiotic resistance genes in the environment

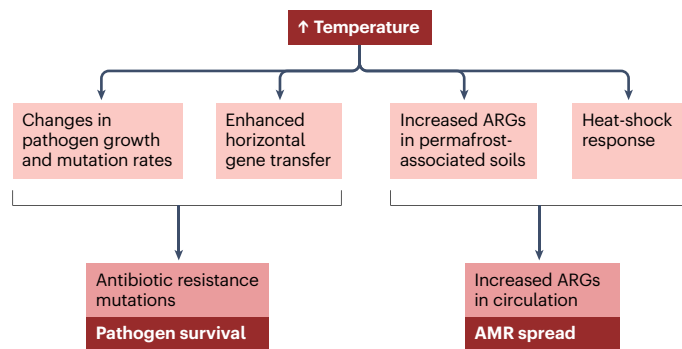
In environmental settings, increased temperatures can have an impact on the abundance and diversity of ARGs<sup>123,124</sup>. A metagenomic study in the Yellow River in China has shown that increased water temperature led to a decline in diversity but increased abundance of ARGs in water samples<sup>124</sup>. However, a study modelling river biofilm communities (which limit the spread of foreign ARGs by competing for resources) has shown that at higher temperatures, naturally occurring ARGs increased in abundance, whereas foreign ARGs that originated from wastewater displayed a reduced ability to establish within the river microbial communities<sup>123</sup>. Other studies assessing ecosystem disturbances resulting from the thawing of permafrost layers highlight the potential risks associated with the release of ARGs into permafrost-associated soils and water environments, such as thermokarst lakes<sup>125–127</sup>. One study has shown that a high proportion of bacterial isolates from disturbance-induced thaw gradients in Interior Alaska were resistant to at least one antibiotic, with the highest prevalence being against ampicillin<sup>128</sup>. Another study has shown that thermokarst lakes, formed by the accelerated degradation of permafrost, shared similar ARG profiles with surrounding soils, indicating that the proliferation of such lakes owing to climate change could amplify the reservoir and potential spread of ARGs in remote environments<sup>126</sup>.

### Antifungal resistance

Invasive fungal infections are associated with very high mortality, particularly in immunocompromised populations, resulting in more than 3.7 million deaths each year<sup>5</sup>. For context, the mortality rate for multidrug-resistant (MDR) *Candida auris* ranges between 30% and 60%, whereas mortality from triazole-resistant *Aspergillus fumigatus* infections is associated with rates of 50% to 100%<sup>129–131</sup>. The treatment of severe fungal infections is complicated by limited therapeutic options with considerable adverse effects and increasing antifungal resistance<sup>132–134</sup>. Although systematic surveillance of antifungal resistance is lacking, available evidence shows antifungal resistance against all classes of antifungals<sup>131,133,135,136</sup>. Antifungal resistance does not spread through mobile genetic elements and can develop de novo in patients undergoing prolonged antifungal therapy<sup>133</sup>. However, warming climates and the environmental use of fungicides have been implicated in the rise of antifungal resistance and the emergence of novel MDR fungal pathogens<sup>131</sup>.

### Spread and emergence of fungal pathogens and antifungal resistance

A human body temperature averaging 37 °C is an important defence against most fungal species, which typically thrive between 20 °C and 30 °C. Few pathogenic fungi can tolerate human body temperatures<sup>137</sup>. However, this is predicted to change with the rise in global temperatures and the development of thermotolerance in additional fungal species<sup>31,138,139</sup>. For example, it is estimated that in a high-warming climate scenario, the number of regions endemic for coccidioidomycosis or Valley fever, an invasive fungal infection, will double by the year 2100 in the USA<sup>140</sup>. These concerning projections are further complicated by the limited antifungal treatment options and increasing antifungal



**Fig. 2 | Direct effects of temperature on the emergence and spread of antimicrobial resistance.** Temperature can directly affect pathogen growth and mutation rates, and increase the exchange of antibiotic resistance genes through horizontal gene transfer<sup>27,34,115</sup>. Environmental consequences of warming climates such as thawing of permafrost increase the concentration of antibiotic resistance genes (ARGs) in permafrost-associated soils<sup>26,126,128</sup>. In addition, bacterial adaptation to warmer temperatures, or thermotolerance, could lead to increased resistance to certain antibiotics that elicit cellular responses resembling heat-shock responses<sup>61,63,64</sup>. These effects in turn lead to increased antibiotic resistance mutations and increase the prevalence of ARGs in circulation, which promotes pathogen survival and the spread of antimicrobial resistance (AMR). Please note that this is not an exhaustive list of factors and impacts.

resistance against therapies for Valley fever<sup>141,142</sup>. In addition to the climate-driven geographic expansion of endemic mycoses, rising global temperatures have also been implicated in the emergence and rapid spread of a highly drug-resistant fungal pathogen, *C. auris*<sup>135,143,144</sup>. *C. auris* was first identified in 2009 in Japan, but by 2015, drug-resistant strains belonging to unique clades with significant genetic diversity were detected on three continents<sup>144</sup>. *C. auris* isolates are more tolerant to high temperatures than other *Candida* spp., implicating a changing climate as an important factor facilitating their emergence and spread<sup>25,145</sup>. Increased thermotolerance may not lead to antifungal resistance; however, it can facilitate transmission in warming climates<sup>131</sup>. For example, thermotolerance in *C. auris* has been noted as a key trait that enables it to survive on various environmental surfaces, at temperatures relevant to hosts, and as high as 42 °C, which enables it to persist on different surfaces and spread more easily<sup>143,146</sup>. Meanwhile, agricultural use of antifungals may intensify the selection pressure for resistant strains that are also thermotolerant<sup>147</sup>.

## Warming climates increase antifungal use in agriculture

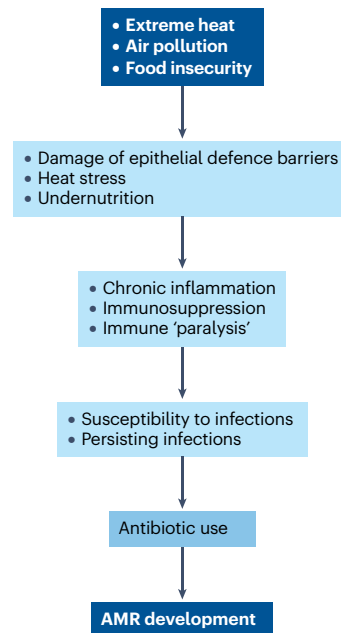
Antifungals and other biocides are essential in agriculture, especially in horticulture. As temperatures rise, crops are expected to become more susceptible to pests, which tends to lead to increased pesticide exposure and selection for novel, drug-resistant pathogens<sup>43,44</sup>. This scenario is particularly relevant when antifungals with the same mode of action are utilized in both agricultural and clinical settings, as is the case with *A. fumigatus* and its resistance to azole antifungals<sup>114,148</sup>. *A. fumigatus*, a fungus that is commonly found in decaying vegetation and soil, is also a human pathogen that causes allergic, chronic and invasive forms of aspergillosis<sup>149</sup>. Although azole antifungals are used to treat aspergillosis in clinical settings, azole pesticides are frequently used in agricultural practices<sup>114,150</sup>. In vivo azole resistance has been shown to arise in patients with chronic aspergillosis; however, accumulating evidence implicates azole resistance of environmental origin in clinical cases<sup>114,151–153</sup>. *A. fumigatus* can survive increased temperatures above 37 °C and harsh environmental conditions<sup>149,154</sup>. Together, these findings suggest that rising temperatures could facilitate the development of resistant fungal pathogens. Conversely, the use of antifungals in the environment might provide a selective advantage to fungal strains that are both resistant and able to tolerate higher temperatures.

## Climate change and host-related factors

### Effects on the immune system

The overall negative impact of climate stressors on human immunity and the ability to fight infections is well-documented, with extreme heat, air pollution and malnourishment increasing the risk and frequency of infections<sup>155,156</sup> (Fig. 3). Bacterial density, antibiotic dosage and immune responses all interact and affect clinical outcomes following infections<sup>157,158</sup>. Although the immune system has a critical role in fighting infections<sup>159,160</sup>, in immunocompromised individuals, persisting infections and prolonged treatments can increase the risk of development and spread of AMR<sup>161,162</sup>. Animal models show that even in the absence of AMR, as defined in a biological and phenotypical sense, immunosuppression is linked to antibiotic failure, that is, the inability of antibiotics to clear the infection<sup>163</sup>.

Extreme heat and air pollution are two consequences of warming climates that impair the integrity of epithelial defence barriers and overstimulate innate and adaptive immune responses, resulting in acute and chronic inflammatory conditions that damage tissues and heighten susceptibility to drug-resistant infections<sup>156,158,164,165</sup>.



**Fig. 3 | Effects of climate change on host immunity.** The consequences of climate change, including air pollution, extreme heat and food insecurity, create conditions that weaken defence barriers and compromise immune responses, increasing susceptibility to infections and antimicrobial resistance (AMR)<sup>155</sup>. Extreme heat and air pollution damage epithelial barriers and dysregulate immune processes, promoting chronic inflammation and susceptibility to drug-resistant infections<sup>166</sup>. Malnutrition owing to climate-driven food insecurity can cause immune suppression and lead to prolonged antibiotic exposure, particularly in vulnerable populations<sup>167–169,171</sup>. The increases in the risk of infections and antibiotic use can contribute to AMR development. Of note, socioeconomic factors moderate the relationships between climate change, host-related factors and AMR, with stronger AMR associations often linked to antibiotic use, pollution and socioeconomic indicators rather than climate variables alone<sup>60,176,177</sup>. Please note that this is not an exhaustive list of factors and impacts.

Additionally, climate change-related environmental stressors are linked to immune dysregulations, which have increased the incidence of conditions such as asthma, cancers and autoimmune disorders, often requiring extensive use of antibiotics for both prophylactic and therapeutic purposes<sup>166</sup>.

### Extreme weather increases food insecurity and malnutrition

The adverse effects of climate change on food insecurity could increase the global rates of malnutrition, particularly in sub-Saharan Africa, Asia and Central America<sup>18</sup>. Childhood malnutrition is linked to mucosal barrier dysfunction, alterations in the intestinal microbiota, immune deficiencies, increased susceptibility to frequent infections, and prolonged antibiotic use<sup>167,168</sup>. Illustrating a more direct connection between malnutrition and AMR, a mouse model exploring the roles of malnutrition and dehydration on susceptibility to methicillin-resistant *S. aureus* (MRSA) describes a reversible ‘immune paralysis’, whereby immune cells recruited to engulf pathogens fail to function normally, rendering the host hypersusceptible to MRSA<sup>169</sup>. Although the use of antibiotics for uncomplicated acute malnourishment is debated, broad-spectrum antibiotics are recommended in treatment guidelines

for severe cases, further increasing the vulnerability to AMR among fragile populations<sup>170,171</sup>.

## Socioeconomic factors

Socioeconomic factors and population density have important roles in the prevalence of infectious disease burden<sup>172,173</sup>. The transmission potential of infectious diseases tends to be higher in densely populated areas, whereas low social and economic development limits the ability of populations to prevent, detect and mitigate infectious disease burden<sup>47,174</sup>. However, the impact of climate-related and socioeconomic factors on AMR has been shown to be heterogeneous in both magnitude and direction, reflecting the complexity of the relationship and the lack of robust data to enable dissection of their effects<sup>175</sup>. A large study of 30 European countries has found that the correlation between temperature change and AMR disappeared after adjusting for GDP per capita and governance<sup>60</sup>. Similarly, a study from China analysing antimicrobial susceptibility data from more than 20 million isolates collected from hospitals across 31 provincial-level divisions has identified a positive correlation between AMR and factors such as human antibiotic use, veterinary antibiotic use, particulate matter, population density and GDP per capita<sup>176</sup>. However, climate indices such as temperature, humidity and precipitation showed no such correlation<sup>176</sup>. Another comprehensive study covering 101 countries has revealed that temperature changes and environmental pollution are associated with AMR prevalence<sup>177</sup>. Importantly, it has highlighted that disparities in AMR between high-income and low-income countries are driven by poverty, limited health-care access and inadequate sanitation<sup>177</sup>. Population density was also cited as a factor exacerbating AMR spread in urbanized areas<sup>177</sup>. Other factors, such as patient demographics, also have a role. One study has found differences in seasonal variations of MRSA infections, wherein community-acquired cases show seasonal patterns across both adult and paediatric populations, whereas hospital-acquired MRSA infections demonstrate seasonal variation only in children<sup>178</sup>. Socioeconomic disparities exacerbate the infectious disease burden and hinder efforts to mitigate climate-related effects, thereby challenging prevention and response strategies. For example, in Africa, climate change is shown to exacerbate the AMR issue because many of the infectious diseases that cause substantial mortality are sensitive to climate change<sup>179,180</sup>.

## Mitigation

### Reducing the incidence of infections through prevention and treatment

To address the impact of climate change on AMR, it is essential to focus on lowering infection rates through sustainable development strategies and improved access to effective antibiotics. Recent trends show that global contributions to GHG emissions are shifting from high-income countries (HICs) to LMICs, whereas the least developed nations, responsible for only 3.8% of total emissions, are expected to face the most severe consequences of climate change<sup>15,181</sup>. Similarly, although AMR is a global concern, its impact varies across countries, driven by limited access to quality antibiotics, inadequate clean water and sanitation services, weak infection prevention and control measures, low vaccination rates, and limited health-care resources<sup>6,182–184</sup>. Collectively, these challenges disproportionately affect the most vulnerable populations who bear the brunt of climate change effects and AMR burden (Box 1).

Reducing the burden of infectious diseases in human and animal health through vaccines and improved WASH infrastructure can address AMR and mitigate climate change impacts<sup>72,185</sup>. Overall, better

WASH infrastructure alone could prevent approximately 250,000 AMR-associated deaths annually in LMICs, whereas increasing paediatric immunization coverage could avert an additional 180,000 AMR-associated deaths annually<sup>7</sup>. These interventions are especially urgent for populations facing poverty, conflicts and climate-forced migration, wherein WASH and health-care disparities are most pronounced (Box 1).

### Improving antibiotic use through diagnostics and surveillance

Improving the diagnosis and treatment of infections is crucial, as gaps in surveillance and access to the right treatment hinder effective responses to AMR<sup>186,187</sup>. Rapid testing technologies for vector-borne and other diseases could facilitate the appropriate use of antimicrobials and improve disease control during outbreaks, including those occurring during extreme weather scenarios<sup>188</sup>. At the population level, early warning systems integrating disease and genomic wastewater surveillance with climate forecasting could guide appropriate interventions to prevent outbreaks and reduce reliance on antimicrobials<sup>36,189,190</sup>.

### Improving health-care and antibiotic access

In LMICs, the unregulated sale and inappropriate use of antibiotics obscure a larger access problem<sup>103</sup>. Although the antibiotic access issue has been challenging to quantify, there is accumulating evidence of disparities in access to antibiotics treating bacterial infections, including those caused by Gram-negative bacteria<sup>104,191</sup>. Furthermore, a recent study has shown that reducing antibiotic consumption by 50% globally could lower AMR prevalence by 2.1%, whereas combining sustainable development strategies, such as reducing out-of-pocket health-care costs, increasing immunization coverage, and health investments, could result in a 5.1% reduction in AMR<sup>177</sup>. These findings support the notion that better access to health-care services and preventive measures, combined with access to the right antibiotics at the right time, may have a greater impact on AMR than total reductions in antibiotic consumption.

### Increasing food security through sustainable agricultural practices

Ensuring food security and eliminating malnutrition can substantially enhance our ability to mitigate climate-induced increases in infectious diseases and the burden of AMR, as malnourished populations are more susceptible to infections and may require prolonged antimicrobial treatments<sup>19,167,169</sup>. Additionally, food scarcity can prompt individuals to seek unsafe food sources to meet their nutritional needs, increase interactions with wildlife, and heighten the risk of disease spillover<sup>88</sup>. Sustainable agricultural practices, the use of antibiotic alternatives, and enhanced animal health through vaccination can have an important role in preventing disease emergence and spillovers while also enhancing livestock productivity and farming sustainability<sup>192–195</sup>. Furthermore, developing and adopting global and local guidelines for pesticide use, while assessing the risk of cross-resistance, is critical in reducing the risk of pesticide resistance and its impact on human health.

## Conclusion

The intricate relationship between climate change and AMR highlights the urgent need for coordinated action to investigate and tackle these two global health crises. Rising temperatures, extreme weather events, and associated environmental changes are amplifying the prevalence and spread of drug-resistant pathogens, while simultaneously increasing the demand for antimicrobials and weakening host immunity.

There are multiple pathways – from enhanced pathogen thermotolerance and horizontal gene transfer to climate-driven zoonotic transmission and intensified agricultural antimicrobial use – through which climate change may exacerbate AMR. Evidence suggests that climate change could worsen the burden of infectious diseases and increase our reliance on antimicrobials. Nonetheless, further research and epidemiological evidence are needed to dissect the impact of climate from other leading determinants.

To develop effective strategies for mitigating AMR amid changing climates, more research is required to determine the impact of climate change on AMR. It is important to note that the majority of evidence on the association between rising temperatures and AMR is derived from ecological studies, which lack specificity and the ability to control for confounders that can obscure or modulate the association. These studies cannot determine whether the increase in temperature precedes the increase in AMR prevalence, complicating the exploration of a causal relationship between the two. Research conducted at the cellular or molecular level reveals that the association between temperature and AMR is organism-specific and antibiotic-specific, and that increasing temperature does not always correlate with higher antibiotic resistance. The lack of evidence of a dose–response relationship from these studies further challenges the inference of a causal relationship. Moreover, AMR detection at the population level is affected not only by methodological factors but also by population size and its characteristics. Therefore, AMR measurement bias is another important factor that must be considered when comparing results across studies. For example, AMR surveillance data from a specific population subgroup (that is, hospitalized patients from a tertiary-level hospital) may not reflect AMR levels in the community.

In addition, our understanding of the emergence and transmission of AMR between microbiomes suggests that climate-driven increases in AMR in agriculture may have consequences for AMR in human health. For example, antibiotic residues from antibiotics applied to crops may lead to unintended outcomes, such as drug resistance and cross-resistance in soil bacteria and zoonotic pathogens, which often share the same environment<sup>196</sup>. However, how and to what extent this could affect human health are currently unclear. Similarly, the increase in circulating ARGs owing to the thawing of permafrost could increase the risk of soil bacteria acquiring antibiotic resistance, but the risk to humans and animals remains unknown.

Finally, a substantial number of studies addressing the connection between climate and AMR are primarily conducted in HICs. LMICs have a higher incidence of infectious diseases that are sensitive to changing climates, weaker health-care systems, and limited capacity to address climate change; all of which may greatly impact the relationship between climate and AMR. These disparities may be even more pronounced in regions affected by conflict and displacement.

Given that climate change is already underway and opportunities for adaptation are shrinking, future efforts must focus on understanding the relationship between climate change, health and AMR. To this end, more primary research and epidemiological evidence on the impact of climate on AMR is needed; such studies should include various pathogens such as parasites and fungi and should also focus on the role of host-related factors such as gender, age and immunity. In addition, ecological and individual-level studies across different geographic, climatic and socioeconomic contexts are needed to disentangle the role of socioeconomic factors in the climate and AMR interface. Finally, we require more research to understand how climate influences the emergence and transmission of AMR at One

Health interfaces, and how the increase of AMR in agriculture affects human health.

Although climate change and AMR burden may be growing concurrently, tackling this dual challenge requires fundamental yet critical interventions, such as improving WASH infrastructure, expanding vaccination programmes, enhancing diagnostic capabilities, and increasing access to effective antibiotics. Addressing socioeconomic disparities, combating malnutrition and improving health-care access are all part of broader strategies to improve well-being and strengthen our ability to fight infections, consequently increasing resilience to climate change.

Published online: 06 February 2026

## References

1. Cook, M. A. & Wright, G. D. The past, present, and future of antibiotics. *Sci. Transl. Med.* **14**, eabo7793 (2022).
2. Aminov, R. I. A brief history of the antibiotic era: lessons learned and challenges for the future. *Front. Microbiol.* **1**, 134 (2010).
3. Armstrong, G. L., Conn, L. A. & Pinner, R. W. Trends in infectious disease mortality in the United States during the 20th century. *JAMA* **281**, 61–66 (1999).
4. Okeke, I. N. et al. The scope of the antimicrobial resistance challenge. *Lancet* **403**, 2426–2438 (2024).
5. Denning, D. W. Global incidence and mortality of severe fungal disease. *Lancet Infect. Dis.* **24**, e428–e438 (2024).
6. GBD 2021 Antimicrobial Resistance Collaborators. Global burden of bacterial antimicrobial resistance 1990–2021: a systematic analysis with forecasts to 2050. *Lancet* **404**, 1199–1226 (2024).
7. Lewnard, J. A. et al. Burden of bacterial antimicrobial resistance in low-income and middle-income countries avertible by existing interventions: an evidence review and modelling analysis. *Lancet* **403**, 2439–2454 (2024).
8. Holmes, A. H. et al. Understanding the mechanisms and drivers of antimicrobial resistance. *Lancet* **387**, 176–187 (2016).
9. Reygaert, W. C. An overview of the antimicrobial resistance mechanisms of bacteria. *AIMS Microbiol.* **4**, 482–501 (2018).
10. Sriram, A. et al. *State of the World's Antibiotics 2021: A Global Analysis of Antimicrobial Resistance and Its Drivers* (Center for Disease Dynamics Economics & Policy, 2021).
11. Allel, K. et al. Global antimicrobial-resistance drivers: an ecological country-level study at the human-animal interface. *Lancet Planet. Health* **7**, e291–e303 (2023).
12. Laxminarayan, R. et al. Antibiotic resistance — the need for global solutions. *Lancet Infect. Dis.* **13**, 1057–1098 (2013).
13. Burnham, J. P. Climate change and antibiotic resistance: a deadly combination. *Ther. Adv. Infect. Dis.* **8**, 2049936121991374 (2021).
14. Zhang, Z. et al. Anthropogenic emission is the main contributor to the rise of atmospheric methane during 1993–2017. *Natl. Sci. Rev.* **9**, nwab200 (2022).
15. United Nations Environment Programme. *Emissions Gap Report 2023: Broken Record — Temperatures Hit New Highs, Yet World Fails to Cut Emissions (Again)* (UNEP, 2023).
16. Liu, H., Lei, M., Zhang, N. & Du, G. The causal nexus between energy consumption, carbon emissions and economic growth: new evidence from China, India and G7 countries using convergent cross mapping. *PLoS ONE* **14**, e0217319 (2019).
17. Mora, C. et al. Over half of known human pathogenic diseases can be aggravated by climate change. *Nat. Clim. Change* **12**, 869–875 (2022).
18. World Meteorological Organization. *2023 State of Climate Services* (WMO, 2023).
19. Romanello, M. et al. The 2024 report of the *Lancet* Countdown on health and climate change: facing record-breaking threats from delayed action. *Lancet* **404**, 1847–1896 (2024).
20. Eber, M. R., Shardell, M., Schweizer, M. L., Laxminarayan, R. & Perencevich, E. N. Seasonal and temperature-associated increases in Gram-negative bacterial bloodstream infections among hospitalized patients. *PLoS ONE* **6**, e25298 (2011).
21. Schwab, F., Gastmeier, P. & Meyer, E. The warmer the weather, the more Gram-negative bacteria — impact of temperature on clinical isolates in intensive care units. *PLoS ONE* **9**, e91105 (2014).
22. Feldman, S. F. et al. Effect of temperature on *Escherichia coli* bloodstream infection in a nationwide population-based study of incidence and resistance. *Antimicrob. Resist. Infect. Control* **11**, 144 (2022).
23. Kim, Y. A., Kim, J. J., Won, D. J. & Lee, K. Seasonal and temperature-associated increase in community-onset *Acinetobacter baumannii* complex colonization or infection. *Ann. Lab. Med.* **38**, 266–270 (2018).
24. MacFadden, D. R., McGough, S. F., Fisman, D., Santillana, M. & Brownstein, J. S. Antibiotic resistance increases with local temperature. *Nat. Clim. Change* **8**, 510–514 (2018).
25. Ellwanger, J. H. & Chies, J. A. B. *Candida auris* emergence as a consequence of climate change: impacts on Americas and the need to contain greenhouse gas emissions. *Lancet Reg. Health Am.* **11**, 100250 (2022).
26. Saleem, M. M. et al. The Petri dish under the ice: permafrost pathogens and their impact on global healthcare and antibiotic resistance. *Ann. Med. Surg.* **86**, 7193–7201 (2024).

27. Lermينياux, N. A. & Cameron, A. D. S. Horizontal transfer of antibiotic resistance genes in clinical environments. *Can. J. Microbiol.* **65**, 34–44 (2019).
28. Warnes, S. L., Highmore, C. J. & Keevil, C. W. Horizontal transfer of antibiotic resistance genes on abiotic touch surfaces: implications for public health. *mBio* **3**, e00489-12 (2012).
29. Dissanayake, H. et al. Nexus between carbon emissions, energy consumption, and economic growth: evidence from global economies. *PLoS ONE* **18**, e0287579 (2023).
30. Klein, E. Y. et al. Global increase and geographic convergence in antibiotic consumption between 2000 and 2015. *Proc. Natl Acad. Sci. USA* **115**, E3463–E3470 (2018).
31. Casadevall, A. Climate change brings the specter of new infectious diseases. *J. Clin. Invest.* **130**, 553–555 (2020).
32. Carlson, C. J. et al. Climate change increases cross-species viral transmission risk. *Nature* **607**, 555–562 (2022).
33. Mahon, M. B. et al. A meta-analysis on global change drivers and the risk of infectious disease. *Nature* **629**, 830–836 (2024).
34. Rzymyski, P., Gwenz, W., Poniedzialek, B., Mangul, S. & Fal, A. Climate warming, environmental degradation and pollution as drivers of antibiotic resistance. *Environ. Pollut.* **346**, 123649 (2024).
35. Ching, C., Sutradhar, I. & Zaman, M. H. Understanding the impacts of temperature and precipitation on antimicrobial resistance in wastewater: theory, modeling, observation, and limitations. *mSphere* **10**, e0094724 (2025).
36. Larsson, D. G. J., Flach, C. F. & Laxminarayan, R. Sewage surveillance of antibiotic resistance holds both opportunities and challenges. *Nat. Rev. Microbiol.* **21**, 213–214 (2023).
37. Zambrano, M. M. Interplay between antimicrobial resistance and global environmental change. *Annu. Rev. Genet.* **57**, 275–296 (2023).
38. Marcus, H. et al. Climate change and the public health imperative for supporting migration as adaptation. *J. Migr. Health* **7**, 100174 (2023).
39. Khalid, A., Babry, J. A., Vearey, J. & Zenner, D. Turning up the heat: a conceptual model for understanding the migration and health in the context of global climate change. *J. Migr. Health* **7**, 100172 (2023).
40. McMichael, C., Schwerdtle, P. N. & Ayeb-Karlsson, S. Waiting for the wave, but missing the tide: case studies of climate-related (im)mobility and health. *J. Migr. Health* **7**, 100147 (2023).
41. Shafaati, M., Salehi, M. & Zare, M. The twin challenges of longevity and climate change in controlling antimicrobial resistance. *J. Antibiot.* **77**, 399–402 (2024).
42. Acosta, A. et al. The future of antibiotic use in livestock. *Nat. Commun.* **16**, 2469 (2025).
43. Deutsch, C. A. et al. Increase in crop losses to insect pests in a warming climate. *Science* **361**, 916–919 (2018).
44. Ma, C.-S. et al. Climate warming promotes pesticide resistance through expanding overwintering range of a global pest. *Nat. Commun.* **12**, 5351 (2021).
45. Velasquez, A. C., Castroverde, C. D. M. & He, S. Y. Plant-pathogen warfare under changing climate conditions. *Curr. Biol.* **28**, R619–R634 (2018).
46. van Bavel, B. et al. Intersections between climate change and antimicrobial resistance: a systematic scoping review. *Lancet Planet. Health* **8**, e1118–e1128 (2024).
47. Jones, K. E. et al. Global trends in emerging infectious diseases. *Nature* **451**, 990–993 (2008).
48. Cunningham, A. A., Daszak, P. & Wood, J. L. N. One Health, emerging infectious diseases and wildlife: two decades of progress? *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **372**, 20160167 (2017).
49. Geng, Y. et al. Anthropogenic activity and climate change exacerbate the spread of pathogenic bacteria in the environment. *Sci. Adv.* **11**, eads4355 (2025).
50. Chazan, B., Colodner, R., Edelstein, H. & Raz, R. Seasonal variation in *Escherichia coli* bloodstream infections in northern Israel. *Clin. Microbiol. Infect.* **17**, 851–854 (2011).
51. Fisman, D. et al. Geographical variability in the likelihood of bloodstream infections due to Gram-negative bacteria: correlation with proximity to the equator and health care expenditure. *PLoS ONE* **9**, e114548 (2014).
52. Kolonitsiou, F. et al. Trends of bloodstream infections in a university Greek hospital during a three-year period: incidence of multidrug-resistant bacteria and seasonality in Gram-negative predominance. *Pol. J. Microbiol.* **66**, 171–180 (2017).
53. Anthony, C. A., Peterson, R. A., Polgreen, L. A., Sewell, D. K. & Polgreen, P. M. The seasonal variability in surgical site infections and the association with warmer weather: a population-based investigation. *Infect. Control Hosp. Epidemiol.* **38**, 809–816 (2017).
54. Blot, K., Hammami, N., Blot, S., Vogelaers, D. & Lambert, M. L. Gram-negative central line-associated bloodstream infection incidence peak during the summer: a national seasonality cohort study. *Sci. Rep.* **12**, 5202 (2022).
55. Zeng, Y. et al. The association between ambient temperature and antimicrobial resistance of *Klebsiella pneumoniae* in China: a difference-in-differences analysis. *Front. Public Health* **11**, 1158762 (2023).
56. McGough, S. F., MacFadden, D. R., Hattab, M. W., Molbak, K. & Santillana, M. Rates of increase of antibiotic resistance and ambient temperature in Europe: a cross-national analysis of 28 countries between 2000 and 2016. *Euro Surveill.* **25**, 1900414 (2020).
57. Li, W. et al. Association between antibiotic resistance and increasing ambient temperature in China: an ecological study with nationwide panel data. *Lancet Reg. Health West. Pac.* **30**, 100628 (2023).
58. Zhao, W., Zheng, S., Ye, C., Li, J. & Yu, X. Nonlinear impacts of temperature on antibiotic resistance in *Escherichia coli*. *Environ. Sci. Ecotechnol.* **22**, 100475 (2024).
59. Li, W. et al. Estimating the effect of increasing ambient temperature on antimicrobial resistance in China: a nationwide ecological study with the difference-in-differences approach. *Sci. Total Environ.* **882**, 163518 (2023).
60. Maugeri, A., Barchitta, M., Magnano San Lio, R. & Agodi, A. Socioeconomic and governance factors disentangle the relationship between temperature and antimicrobial resistance: a 10-year ecological analysis of European countries. *Antibiotics* **12**, 777 (2023).
61. Cruz-Loya, M. et al. Stressor interaction networks suggest antibiotic resistance co-opted from stress responses to temperature. *ISME J.* **13**, 12–23 (2019).
62. Roemhild, R., Bollenbach, T. & Andersson, D. I. The physiology and genetics of bacterial responses to antibiotic combinations. *Nat. Rev. Microbiol.* **20**, 478–490 (2022).
63. Brand, C., Newton-Foot, M., Grobbelaar, M. & Whitelaw, A. Antibiotic-induced stress responses in Gram-negative bacteria and their role in antibiotic resistance. *J. Antimicrob. Chemother.* **80**, 1165–1184 (2025).
64. Cardoso, K. et al. DnaK and GroEL are induced in response to antibiotic and heat shock in *Acinetobacter baumannii*. *J. Med. Microbiol.* **59**, 1061–1068 (2010).
65. Cruz-Loya, M. et al. Antibiotics shift the temperature response curve of *Escherichia coli* growth. *mSystems* **6**, e0022821 (2021).
66. Furlan, J. P. R. et al. Catastrophic floods and antimicrobial resistance: interconnected threats with wide-ranging impacts. *One Health* **19**, 100891 (2024).
67. Hartinger, S. M. et al. The 2023 Latin America report of the *Lancet* Countdown on health and climate change: the imperative for health-centred climate-resilient development. *Lancet Reg. Health Am.* **33**, 100746 (2024).
68. Romanello, M. et al. The 2023 report of the *Lancet* Countdown on health and climate change: the imperative for a health-centred response in a world facing irreversible harms. *Lancet* **402**, 2346–2394 (2023).
69. Dewi, S. P., Kasim, R., Sutarsa, I. N. & Dykgraaf, S. H. A scoping review of the impact of extreme weather events on health outcomes and healthcare utilization in rural and remote areas. *BMC Health Serv. Res.* **24**, 1333 (2024).
70. Suk, J. E., Vaughan, E. C., Cook, R. G. & Semenza, J. C. Natural disasters and infectious disease in Europe: a literature review to identify cascading risk pathways. *Eur. J. Public Health* **30**, 928–935 (2020).
71. Wang, Z., Pei, S., Cui, H., Zhang, J. & Jia, Z. Zoonotic spillover and extreme weather events drive the global outbreaks of airborne viral emerging infectious diseases. *J. Med. Virol.* **96**, e29737 (2024).
72. Ahmed, A. K. et al. Cholera rages in Africa and the Middle East: a narrative review on challenges and solutions. *Health Sci. Rep.* **7**, e2013 (2024).
73. Das, B., Verma, J., Kumar, P., Ghosh, A. & Ramamurthy, T. Antibiotic resistance in *Vibrio cholerae*: understanding the ecology of resistance genes and mechanisms. *Vaccine* **38**, A83–A92 (2020).
74. Mhalu, F., Mmari, P. & Ijumba, J. Rapid emergence of El Tor *Vibrio cholerae* resistant to antimicrobial agents during first six months of fourth cholera epidemic in Tanzania. *Lancet* **313**, 345–347 (1979).
75. Mashe, T. et al. Highly resistant cholera outbreak strain in Zimbabwe. *N. Engl. J. Med.* **383**, 687–689 (2020).
76. Gonçalves, B. C. et al. Antiviral therapies: advances and perspectives. *Fundam. Clin. Pharmacol.* **35**, 305–320 (2021).
77. Feldman, C. & Anderson, R. The role of co-infections and secondary infections in patients with COVID-19. *Pneumonia* **13**, 5 (2021).
78. Arnold, F. W. & Fuqua, J. L. Viral respiratory infections: a cause of community-acquired pneumonia or a predisposing factor? *Curr. Opin. Pulm. Med.* **26**, 208–214 (2020).
79. Klein, E. Y. et al. The impact of influenza vaccination on antibiotic use in the United States, 2010–2017. *Open Forum Infect. Dis.* **7**, ofaa223 (2020).
80. Lewnard, J. A., Rogawski McQuade, E. T., Platts-Mills, J. A., Kotloff, K. L. & Laxminarayan, R. Incidence and etiology of clinically-attended, antibiotic-treated diarrhea among children under five years of age in low- and middle-income countries: evidence from the global enteric multicenter study. *PLoS Negl. Trop. Dis.* **14**, e0008520 (2020).
81. Jansen, K. U., Knirsch, C. & Anderson, A. S. The role of vaccines in preventing bacterial antimicrobial resistance. *Nat. Med.* **24**, 10–19 (2018).
82. He, Y., Liu, W. J., Jia, N., Richardson, S. & Huang, C. Viral respiratory infections in a rapidly changing climate: the need to prepare for the next pandemic. *eBioMedicine* **93**, 104593 (2023).
83. Filho, L., May, W., May, J., Nagy, M. & Gustavo, J. Climate change and malaria: some recent trends of malaria incidence rates and average annual temperature in selected sub-Saharan African countries from 2000 to 2018. *Malar. J.* **22**, 248 (2023).
84. Thomson, M. C. & Stanberry, L. R. Climate change and vectorborne diseases. *N. Engl. J. Med.* **387**, 1969–1978 (2022).
85. Kayendeke, M. et al. Pharmaceuticalised livelihoods: antibiotics and the rise of ‘quick farming’ in peri-urban Uganda. *J. Biosoc. Sci.* **55**, 995–1014 (2023).
86. Lacetera, N. Impact of climate change on animal health and welfare. *Anim. Front.* **9**, 26–31 (2019).
87. Duchenne-Moutien, R. A. & Neetoo, H. Climate change and emerging food safety issues: a review. *J. Food Prot.* **84**, 1884–1897 (2021).
88. Magnano San Lio, R., Favara, G., Maugeri, A., Barchitta, M. & Agodi, A. How antimicrobial resistance is linked to climate change: an overview of two intertwined global challenges. *Int. J. Environ. Res. Public Health* **20**, 1681 (2023).
89. Bett, B. et al. Effects of climate change on the occurrence and distribution of livestock diseases. *Prev. Vet. Med.* **137**, 119–129 (2017).
90. Reverter, M. et al. Aquaculture at the crossroads of global warming and antimicrobial resistance. *Nat. Commun.* **11**, 1870 (2020).

91. Marshall, B. M. & Levy, S. B. Food animals and antimicrobials: impacts on human health. *Clin. Microbiol. Rev.* **24**, 718–733 (2011).
92. Tang, K. L. et al. Restricting the use of antibiotics in food-producing animals and its associations with antibiotic resistance in food-producing animals and human beings: a systematic review and meta-analysis. *Lancet Planet. Health* **1**, e316–e327 (2017).
93. Van Boeckel, T. P. et al. Reducing antimicrobial use in food animals. *Science* **357**, 1350–1352 (2017).
94. Emes, D., Naylor, N., Waage, J. & Knight, G. Quantifying the relationship between antibiotic use in food-producing animals and antibiotic resistance in humans. *Antibiotics* **11**, 66 (2022).
95. Haynes, E., Ramwell, C., Griffiths, T., Walker, D. & Smith, J. *Review of Antibiotic Use in Crops, Associated Risk of Antimicrobial Resistance and Research Gaps*. Report to Department for Environment, Food and Rural Affairs (Defra) & The Food Standards Agency (FSA). (Fera Science Ltd, 2020).
96. Zuo, Y., Ji, M., Yang, J., Li, Z. & Wang, J. Risk assessment of corn borer based on feature optimization and weighted spatial clustering: a case study in Shandong Province. *China Sci. Rep.* **15**, 28036 (2025).
97. Jha, P. K. et al. Climate change impacts on insect pests for high value specialty crops in California. *Sci. Total Environ.* **906**, 167605 (2024).
98. Sundin, G. W. & Wang, N. Antibiotic resistance in plant-pathogenic bacteria. *Annu. Rev. Phytopathol.* **56**, 161–180 (2018).
99. Klein, E. Y. et al. Global trends in antibiotic consumption during 2016–2023 and future projections through 2030. *Proc. Natl Acad. Sci. USA* **121**, e2411919121 (2024).
100. Mulchandani, R. et al. Global trends in inappropriate use of antibiotics, 2000–2021: scoping review and prevalence estimates. *BMJ Public Health* **3**, e002411 (2025).
101. Sun, L., Klein, E. Y. & Laxminarayan, R. Seasonality and temporal correlation between community antibiotic use and resistance in the United States. *Clin. Infect. Dis.* **55**, 687–694 (2012).
102. Macesic, N., Uhlemann, A. C. & Peleg, A. Y. Multidrug-resistant Gram-negative bacterial infections. *Lancet* **405**, 257–272 (2025).
103. Laxminarayan, R. et al. Access to effective antimicrobials: a worldwide challenge. *Lancet* **387**, 168–175 (2016).
104. Mishra, A., Dwivedi, R., Faure, K., Morgan, D. J. & Cohn, J. Estimated undertreatment of carbapenem-resistant Gram-negative bacterial infections in eight low-income and middle-income countries: a modelling study. *Lancet Infect. Dis.* **25**, 1011–1019 (2025).
105. Mendelson, M. et al. Maximising access to achieve appropriate human antimicrobial use in low-income and middle-income countries. *Lancet* **387**, 188–198 (2016).
106. de Jong, Y. et al. Antibiotic consumption in 14 countries of sub-Saharan Africa: findings from a retrospective analysis. *PLoS ONE* **20**, e033842 (2025).
107. Henchion, M., Hayes, M., Mullen, A. M., Fenelon, M. & Tiwari, B. Future protein supply and demand: strategies and factors influencing a sustainable equilibrium. *Foods* **6**, 53 (2017).
108. Food and Agriculture Organization of the United Nations. *Pathways Towards Lower Emissions — A Global Assessment of the Greenhouse Gas Emissions and Mitigation Options from Livestock Agrifood Systems* (FAO, 2023).
109. Gilbert, W., Thomas, L. F., Coyne, L. & Rushton, J. Review: Mitigating the risks posed by intensification in livestock production: the examples of antimicrobial resistance and zoonoses. *Animal* **15**, 100123 (2021).
110. Bartlett, H. et al. Understanding the relative risks of zoonosis emergence under contrasting approaches to meeting livestock product demand. *R. Soc. Open Sci.* **9**, 211573 (2022).
111. Albernaz-Goncalves, R., Olmos Antillon, G. & Hotzel, M. J. Linking animal welfare and antibiotic use in pig farming — a review. *Animals* **12**, 216 (2022).
112. Zhao, C., Wang, Y., Mulchandani, R. & Van Boeckel, T. P. Global surveillance of antimicrobial resistance in food animals using priority drugs maps. *Nat. Commun.* **15**, 763 (2024).
113. Schar, D., Klein, E. Y., Laxminarayan, R., Gilbert, M. & Van Boeckel, T. P. Global trends in antimicrobial use in aquaculture. *Sci. Rep.* **10**, 21878 (2020).
114. Snelders, E. et al. Triazole fungicides can induce cross-resistance to medical triazoles in *Aspergillus fumigatus*. *PLoS ONE* **7**, e31801 (2012).
115. Van Eldijk, T. J. B. et al. Temperature dependence of the mutation rate towards antibiotic resistance. *JAC Antimicrob. Resist.* **6**, dlae085 (2024).
116. Rodriguez-Verdugo, A., Lozano-Huntelman, N., Cruz-Loya, M., Savage, V. & Yeh, P. Compounding effects of climate warming and antibiotic resistance. *iScience* **23**, 101024 (2020).
117. Rodriguez-Verdugo, A., Gaut, B. S. & Tenailon, O. Evolution of *Escherichia coli* rifampicin resistance in an antibiotic-free environment during thermal stress. *BMC Evol. Biol.* **13**, 50 (2013).
118. Pallares-Vega, R. et al. Temperature and nutrient limitations decrease transfer of conjugative IncP-1 Plasmid pKJK5 to wild *Escherichia coli* strains. *Front. Microbiol.* **12**, 656250 (2021).
119. Walsh, T. R., Weeks, J., Livermore, D. M. & Toleman, M. A. Dissemination of NDM-1 positive bacteria in the New Delhi environment and its implications for human health: an environmental point prevalence study. *Lancet Infect. Dis.* **11**, 355–362 (2011).
120. Alderliesten, J. B. et al. Effect of donor-recipient relatedness on the plasmid conjugation frequency: a meta-analysis. *BMC Microbiol.* **20**, 135 (2020).
121. Strahilevitz, J., Jacoby, G. A., Hooper, D. C. & Robicsek, A. Plasmid-mediated quinolone resistance: a multifaceted threat. *Clin. Microbiol. Rev.* **22**, 664–689 (2009).
122. Canton, R. & Coque, T. M. The CTX-M  $\beta$ -lactamase pandemic. *Curr. Opin. Microbiol.* **9**, 466–475 (2006).
123. Bagra, K. et al. Contrary effects of increasing temperatures on the spread of antimicrobial resistance in river biofilms. *mSphere* **9**, e0057323 (2024).
124. Yu, Q. et al. Metagenomics reveals the response of antibiotic resistance genes to elevated temperature in the Yellow River. *Sci. Total Environ.* **859**, 160324 (2023).
125. Kim, H., Kim, M., Kim, S., Lee, Y. M. & Shin, S. C. Characterization of antimicrobial resistance genes and virulence factor genes in an Arctic permafrost region revealed by metagenomics. *Environ. Pollut.* **294**, 118634 (2022).
126. Ren, Z., Zhang, C., Li, X. & Luo, W. Thermokarst lakes are hotspots of antibiotic resistance genes in permafrost regions on the Qinghai-Tibet Plateau. *Environ. Pollut.* **344**, 123334 (2024).
127. Vishnupriya, S., Jabir, T., Akhil Prakash, E. & Mohamed Hatha, A. A. Antibiotic resistance of heterotrophic bacteria from the sediments of adjoining high Arctic fjords, Svalbard. *Braz. J. Microbiol.* **55**, 2371–2383 (2024).
128. Haan, T. J. & Drown, D. M. Unearthing antibiotic resistance associated with disturbance-induced permafrost thaw in interior Alaska. *Microorganisms* **9**, 116 (2021).
129. Egger, N. B. et al. The rise of *Candida auris*: from unique traits to co-infection potential. *Microb. Cell* **9**, 141–144 (2022).
130. Bradley, K. et al. Fatal fungicide-associated triazole-resistant *Aspergillus fumigatus* infection, Pennsylvania, USA. *Emerg. Infect. Dis.* **28**, 1904–1905 (2022).
131. Lockhart, S. R., Chowdhary, A. & Gold, J. A. W. The rapid emergence of antifungal-resistant human-pathogenic fungi. *Nat. Rev. Microbiol.* **21**, 818–832 (2023).
132. Roemer, T. & Krysan, D. J. Antifungal drug development: challenges, unmet clinical needs, and new approaches. *Cold Spring Harb. Perspect. Med.* **4**, a019703 (2014).
133. Fisher, M. C. et al. Tackling the emerging threat of antifungal resistance to human health. *Nat. Rev. Microbiol.* **20**, 557–571 (2022).
134. Gow, N. A. R. et al. The importance of antimicrobial resistance in medical mycology. *Nat. Commun.* **13**, 5352 (2022).
135. George, M. E. et al. The impact of climate change on the epidemiology of fungal infections: implications for diagnosis, treatment, and public health strategies. *Ther. Adv. Infect. Dis.* **12**, 20499361251313841 (2025).
136. Perlin, D. S., Rautemaa-Richardson, R. & Alastruey-Izquierdo, A. The global problem of antifungal resistance: prevalence, mechanisms, and management. *Lancet Infect. Dis.* **17**, e383–e392 (2017).
137. Robert, V. A. & Casadevall, A. Vertebrate endothermy restricts most fungi as potential pathogens. *J. Infect. Dis.* **200**, 1623–1626 (2009).
138. Robert, V., Cardinali, G. & Casadevall, A. Distribution and impact of yeast thermal tolerance permissive for mammalian infection. *BMC Biol.* **13**, 18 (2015).
139. Seidel, D. et al. Impact of climate change and natural disasters on fungal infections. *Lancet Microbe* **5**, e594–e605 (2024).
140. Gorris, M. E., Treseder, K. K., Zender, C. S. & Randerson, J. T. Expansion of coccidioidomycosis endemic regions in the United States in response to climate change. *GeoHealth* **3**, 308–327 (2019).
141. Thompson, G. R. 3rd, Barker, B. M. & Wiederhold, N. P. Large-scale evaluation of in vitro amphotericin B, triazole, and echinocandin activity against *Coccidioides* species from U.S. institutions. *Antimicrob. Agents Chemother.* **61**, e02634-16 (2017).
142. Galgiani, J. N. et al. 2016 Infectious Diseases Society of America (IDSA) Clinical Practice Guideline for the Treatment of Coccidioidomycosis. *Clin. Infect. Dis.* **63**, e112–e146 (2016).
143. Jackson, B. R. et al. On the origins of a species: what might explain the rise of *Candida auris*? *J. Fungi* **5**, 58 (2019).
144. Lockhart, S. R. et al. Simultaneous emergence of multidrug-resistant *Candida auris* on 3 continents confirmed by whole-genome sequencing and epidemiological analyses. *Clin. Infect. Dis.* **64**, 134–140 (2017).
145. Garcia-Bustos, V. et al. Climate change, animals, and *Candida auris*: insights into the ecological niche of a new species from a One Health approach. *Clin. Microbiol. Infect.* **29**, 858–862 (2023).
146. Allert, S. et al. From environmental adaptation to host survival: attributes that mediate pathogenicity of *Candida auris*. *Virulence* **13**, 191–214 (2022).
147. van Rhijn, N. & Rhodes, J. Evolution of antifungal resistance in the environment. *Nat. Microbiol.* **10**, 1804–1815 (2025).
148. Verweij, P. E. et al. Dual use of antifungals in medicine and agriculture: how do we help prevent resistance developing in human pathogens? *Drug Resist. Updates* **65**, 100885 (2022).
149. Arastehfar, A. et al. *Aspergillus fumigatus* and aspergillosis: from basics to clinics. *Stud. Mycol.* **100**, 100115 (2021).
150. Denning, D. W. et al. Chronic pulmonary aspergillosis: rationale and clinical guidelines for diagnosis and management. *Eur. Respir. J.* **47**, 45–68 (2016).
151. Kang, S. E. et al. Evidence for the agricultural origin of resistance to multiple antimicrobials in *Aspergillus fumigatus*, a fungal pathogen of humans. *G3* **12**, jkab427 (2022).
152. Rhodes, J. et al. Population genomics confirms acquisition of drug-resistant *Aspergillus fumigatus* infection by humans from the environment. *Nat. Microbiol.* **7**, 663–674 (2022).
153. Arendrup, M. C. et al. Development of azole resistance in *Aspergillus fumigatus* during azole therapy associated with change in virulence. *PLoS ONE* **5**, e10080 (2010).
154. Berger, S., El Chazli, Y., Babu, A. F. & Coste, A. T. Azole resistance in *Aspergillus fumigatus*: a consequence of antifungal use in agriculture? *Front. Microbiol.* **8**, 1024 (2017).
155. Imberti, L., Tiecco, G., Logiudice, J., Castelli, F. & Quiros-Roldan, E. Effects of climate change on the immune system: a narrative review. *Health Sci. Rep.* **8**, e70627 (2025).

156. Skevaki, C. et al. Impact of climate change on immune responses and barrier defense. *J. Allergy Clin. Immunol.* **153**, 1194–1205 (2024).
157. Gjini, E. & Brito, P. H. Integrating antimicrobial therapy with host immunity to fight drug-resistant infections: classical vs. adaptive treatment. *PLoS Comput. Biol.* **12**, e1004857 (2016).
158. Ankomah, P. & Levin, B. R. Exploring the collaboration between antibiotics and the immune response in the treatment of acute, self-limiting infections. *Proc. Natl Acad. Sci. USA* **111**, 8331–8338 (2014).
159. Wheatley, R. et al. Rapid evolution and host immunity drive the rise and fall of carbapenem resistance during an acute *Pseudomonas aeruginosa* infection. *Nat. Commun.* **12**, 2460 (2021).
160. Garcia-Patino, M. G., Garcia-Contreras, R. & Licona-Limon, P. The immune response against *Acinetobacter baumannii*, an emerging pathogen in nosocomial infections. *Front. Immunol.* **8**, 441 (2017).
161. Huo, W. et al. Immunosuppression broadens evolutionary pathways to drug resistance and treatment failure during *Acinetobacter baumannii* pneumonia in mice. *Nat. Microbiol.* **7**, 796–809 (2022).
162. Huemer, M., Mairpady Shambat, S., Brugger, S. D. & Zinkernagel, A. S. Antibiotic resistance and persistence — implications for human health and treatment perspectives. *EMBO Rep.* **21**, e51034 (2020).
163. de la Fuente-Nunez, C., Cesaro, A. & Hancock, R. E. W. Antibiotic failure: beyond antimicrobial resistance. *Drug Resist. Updates* **71**, 101012 (2023).
164. Wlodarska, M. & Finlay, B. B. Host immune response to antibiotic perturbation of the microbiota. *Mucosal Immunol.* **3**, 100–103 (2010).
165. North, J. R. et al. A novel approach for emerging and antibiotic resistant infections: innate defense regulators as an agnostic therapy. *J. Biotechnol.* **226**, 24–34 (2016).
166. Agache, I. et al. Immune-mediated disease caused by climate change-associated environmental hazards: mitigation and adaptation. *Front. Sci.* <https://doi.org/10.3389/fsci.2024.1279192> (2024).
167. Ibrahim, M. K., Zambruni, M., Melby, C. L. & Melby, P. C. Impact of childhood malnutrition on host defense and infection. *Clin. Microbiol. Rev.* **30**, 919–971 (2017).
168. Unger, S. A., Mark, H. & Pagliari, C. Nutrition: the missing link in the battle against microbial resistance? *J. Glob. Health* **9**, 010321 (2019).
169. Lacey, K. A. et al. Dietary and water restriction leads to increased susceptibility to antimicrobial resistant pathogens. *Sci. Adv.* **10**, eadi7438 (2024).
170. Isanaka, S. et al. Routine amoxicillin for uncomplicated severe acute malnutrition in children. *N. Engl. J. Med.* **374**, 444–453 (2016).
171. Trehan, I. et al. Antibiotics as part of the management of severe acute malnutrition. *N. Engl. J. Med.* **368**, 425–435 (2013).
172. Yu, H., Stauss, H. & Persad, A. Use of population weighted density index for coronavirus spread in the United States. *J. Health Econ. Outcomes Res.* **11**, 1–8 (2024).
173. Smith, T. P. et al. Temperature and population density influence SARS-CoV-2 transmission in the absence of nonpharmaceutical interventions. *Proc. Natl Acad. Sci. USA* **118**, e2019284118 (2021).
174. Santos-Vega, M., Bouma, M. J., Kohli, V. & Pascual, M. Population density, climate variables and poverty synergistically structure spatial risk in urban malaria in India. *PLoS Negl. Trop. Dis.* **10**, e0005155 (2016).
175. Yang, L. et al. Climate change and antimicrobial resistance in the Western Pacific: a mixed-methods systematic analysis. *Lancet Reg. Health West. Pac.* **67**, 101772 (2026).
176. Zhou, W. et al. Factors associated with clinical antimicrobial resistance in China: a nationwide analysis. *Infect. Dis. Poverty* **14**, 27 (2025).
177. Li, W. et al. Changing climate and socioeconomic factors contribute to global antimicrobial resistance. *Nat. Med.* **31**, 1798–1808 (2025).
178. Mermel, L. A., Machan, J. T. & Parenteau, S. Seasonality of MRSA infections. *PLoS ONE* **6**, e17925 (2011).
179. Roth, G. A. et al. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet* **392**, 1736–1788 (2018).
180. El-Maradny, Y. A. et al. The impact of socioeconomic disparities climate factors and antimicrobial stewardship on antimicrobial resistance in Africa. *Discov. Public Health* **22**, 247 (2025).
181. Hallegatte, S. et al. *Shock Waves: Managing the Impacts of Climate Change on Poverty*. Climate Change and Development Series <https://doi.org/10.1596/978-1-4648-0673-5> (World Bank, 2016).
182. Loosli, K., Davis, A., Muwonge, A. & Lembo, T. Addressing antimicrobial resistance by improving access and quality of care — a review of the literature from East Africa. *PLoS Negl. Trop. Dis.* **15**, e0009529 (2021).
183. UNICEF. Drinking water, sanitation and hygiene (WASH) estimates. *UNICEF* <https://data.unicef.org/topic/water-and-sanitation/drinking-water/> (2023).
184. Rachlin, A., Danovaro-Holliday, M. C., Murphy, P., Sodha, S. V. & Wallace, A. S. Routine vaccination coverage — worldwide, 2021. *MMWR Morb. Mortal. Wkly Rep.* **71**, 1396–1400 (2022).
185. Mashe, T. et al. Descriptive epidemiology of the cholera outbreak in Zimbabwe 2018–2019: role of multi-sectorial approach in cholera epidemic control. *BMJ Open* **13**, e059134 (2023).
186. Laxminarayan, R. et al. Expanding antibiotic, vaccine, and diagnostics development and access to tackle antimicrobial resistance. *Lancet* **403**, 2534–2550 (2024).
187. Freeman Weiss, Z., Leon, A. & Koo, S. The evolving landscape of fungal diagnostics, current and emerging microbiological approaches. *J. Fungi* **7**, 127 (2021).
188. Osorio, L. et al. A scoping review on the field validation and implementation of rapid diagnostic tests for vector-borne and other infectious diseases of poverty in urban areas. *Infect. Dis. Poverty* **7**, 87 (2018).
189. Sophia, Y. et al. Dengue dynamics, predictions, and future increase under changing monsoon climate in India. *Sci. Rep.* **15**, 1637 (2025).
190. Ssempiira, J. et al. Interactions between climatic changes and intervention effects on malaria spatio-temporal dynamics in Uganda. *Parasite Epidemiol. Control* **3**, e00070 (2018).
191. Browne, A. J. et al. Global antibiotic consumption and usage in humans, 2000–18: a spatial modelling study. *Lancet Planet. Health* **5**, e893–e904 (2021).
192. Mulchandani, R., Wang, Y., Gilbert, M. & Van Boeckel, T. P. Global trends in antimicrobial use in food-producing animals: 2020 to 2030. *PLoS Glob. Public Health* **3**, e0001305 (2023).
193. Schoustra, S. E. et al. Environmental hotspots for azole resistance selection of *Aspergillus fumigatus*, the Netherlands. *Emerg. Infect. Dis.* **25**, 1347–1353 (2019).
194. Laxminarayan, R. et al. Unlock the potential of vaccines in food-producing animals. *Science* **384**, 1409–1411 (2024).
195. Gleason, A. et al. Global vaccination coverage and disease incidence in cattle, pigs, and poultry. *Proc. Natl Acad. Sci. USA* **122**, e2515557122 (2025).
196. Miller, S. A., Ferreira, J. P. & LeJeune, J. T. Antimicrobial use and resistance in plant agriculture: a One Health perspective. *Agriculture* **12**, 289 (2022).
197. Alvarez-Uria, G., Gandra, S. & Laxminarayan, R. Poverty and prevalence of antimicrobial resistance in invasive isolates. *Int. J. Infect. Dis.* **52**, 59–61 (2016).
198. McMichael, C. Climate change-related migration and infectious disease. *Virulence* **6**, 548–553 (2015).
199. Lindahl, J. F. & Grace, D. The consequences of human actions on risks for infectious diseases: a review. *Infect. Ecol. Epidemiol.* **5**, 30048 (2015).
200. Myers, S. S. et al. Climate change and global food systems: potential impacts on food security and undernutrition. *Annu. Rev. Public Health* **38**, 259–277 (2017).

## Author contributions

The authors contributed equally to all aspects of the article.

## Competing interests

The authors declare no competing interests.

## Additional information

**Peer review information** *Nature Reviews Microbiology* thanks Lianping Yang and the other, anonymous, reviewer(s) for their contribution to the peer review of this work.

**Publisher's note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Springer Nature or its licensor (e.g. a society or other partner) holds exclusive rights to this article under a publishing agreement with the author(s) or other rightsholder(s); author self-archiving of the accepted manuscript version of this article is solely governed by the terms of such publishing agreement and applicable law.

© Springer Nature Limited 2026