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POLICY BRIEF

Aerobiology in a Changing Climate

Particulate Matter · Airborne Viruses · Atmospheric Microbiome · Antifungal Resistance

ONE-Bridge in Health Webinar Series · June 2026

Executive Summary

Climate change does not only alter temperature and precipitation — it changes the biology of the air we breathe. This is not a hypothesis: it is documented reality with measurable consequences for viral transmissibility, pathogen survival, and the emergence of resistant microorganisms in the spaces where children, patients, and elderly people spend most of their day.

Four findings demand immediate policy action. Particulate matter is becoming a vehicle for viral transmission under climate pressure. Hospital air can warn of epidemic surges before they become clinically visible. Extreme weather events distribute microbial hazards into spaces that no regulation currently monitors. And Portuguese primary schools harbour drug-resistant Aspergillus fumigatus at rates four times higher than in agricultural soils — entirely invisible to the current legal monitoring framework.

7M+ Annual deaths attributable to air pollution globally	**18.5%** Thermotolerant Aspergillus fumigatus with potential azole resistance in Portuguese primary schools	**>80%** Resistant fungal isolates that active air sampling — the only legally required method — would have missed	**1,000–3,000 km** Distance microbial communities and resistance genes travel through the atmosphere

1. Particulate Matter and Viruses: A Climate-Amplified Transmission Mechanism

Particulate matter is not merely a pollutant — it is a potential vehicle for pathogens. When PM co-exists in air with respiratory viruses, particles adhere to viral surfaces through electrostatic and hydrophobic interactions, shielding them from UV inactivation and allowing them to remain infectious over longer distances and durations. Climate change amplifies this mechanism: it increases PM emissions from wildfires, alters the thermal stratification that keeps particles suspended, and creates humidity fluctuations that govern how respiratory droplets behave in the air.

- Patients with COPD show approximately twofold higher viral detection frequency — particularly Influenza A — during episodes of elevated ambient air pollution.
- The co-presence of PM converts localised respiratory infections into systemic inflammatory conditions, increasing ICU admissions and case fatality rates.

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- Preliminary measurements from school classrooms in Patras (2026) show positive correlations between PM2.5/PM10 concentrations and detection of Influenza A and SARS-CoV-2.
- Air pollution and infectious disease are not independent public health problems. Forecasting viral surges without PM data produces structurally incomplete predictions.

BOTTOM LINE	*Hospitals must receive advance warning when air pollution surges coincide with viral circulation — not after ICUs begin to fill. PM data must become a standard input to national respiratory disease surveillance models.*

2. Hospital Air: An Early Warning System We Have Not Built

Hospital indoor air is not neutral. It carries bacteria, fungi, viruses, and — critically — antimicrobial resistance genes, even at low concentrations. These do not come only from patients: the airborne microbiology of a ward reflects the entire building ecosystem — ventilation, surfaces, plumbing, windows. Twenty-eight weeks of simultaneous air and surface molecular surveillance in two hospitals produced a finding with direct policy implications: in some instances, viral RNA was detected in air or on surfaces before the corresponding peak in hospitalised cases. Environmental surveillance can precede clinical signals.

- Two geographically adjacent hospitals showed different AMR resistance profiles — different BLA-KPC gene variants. Local differences invisible to national clinical surveillance.
- Resistance genes were found in hospital air independently of sink drain biofilms — the airspace is an autonomous AMR signal carrier, not merely a reflection of plumbing reservoirs.
- Seasonal pathogen dynamics are faithfully captured in weekly aerobiological data: Influenza and RSV in winter, SARS-CoV-2 during admission waves.

BOTTOM LINE	*A real-time aerobiological surveillance system in hospital wards could function as a biological barometer, detecting signals weeks before clinical burden peaks. This capability exists. No European hospital currently deploys it routinely.*

3. Air Connects Everything: Long-Range Transport and Extreme Events

Microorganisms and resistance genes travel 1,000–3,000 kilometres through the atmosphere above the planetary boundary layer. They do not need a passport. Resistance that develops in livestock farms in one country can be detected in the air of another. Urban and agricultural sources leave distinct signatures in airborne AMR profiles — aminoglycoside and macrolide resistance genes dominate in urban air; tetracycline and sulfonamide genes dominate near livestock settings. The air remembers where it came from.

Extreme weather events sharply intensify this dynamic. The Valencia flood (2024) mixed sewage, agricultural sediments, industrial waste, and pathogenic microorganisms into an urban environment. After the water receded, contaminated sediments dried and became inhalable dust

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during reconstruction — months later. Sampling during the reconstruction phase found exceptionally rich microbial loads in air and soil, including organisms with resistance genes and potential public health relevance.

- Hospitals, elderly care centres, and schools have distinct indoor airborne microbial fingerprints shaped by occupants, ventilation, building design, and outdoor imprint.
- Heat resistance genes were detected at unexpectedly high numbers in indoor environments — direct evidence of microbial adaptation to rising temperatures.
- Floods do not end when the water recedes. Their aerobiological footprint persists through reconstruction. Monitoring must start within weeks and continue for months.

BOTTOM LINE	*Post-disaster aerobiological surveillance must be a standard protocol in flood, wildfire, and dust storm response plans — not an afterthought. The Valencia data provide the methodological template.*

4. Drug-Resistant Aspergillus in School Classrooms: A Regulatory Failure

In Portuguese primary schools, 18.5% of Aspergillus fumigatus detected shows potential resistance to azole antifungals — the primary drugs for treating invasive aspergillosis. This rate is four times higher than typically reported in southern European agricultural soils. Of the resistant isolates, 75% show multi-drug resistance to two or more azoles simultaneously. Over half have itraconazole MIC values above 8 mg/L — far beyond clinically achievable blood concentrations of 1–2 µg/L. These isolates would not respond to first-line antifungal treatment. If they infect an immunocompromised patient, treatment fails.

This resistance did not develop in hospitals. It developed in agricultural fields through the use of azole-class DMI fungicides — the same chemical class used in medicine. It entered indoor spaces through spores deposited in settled dust. Portugal, with extensive vineyards, olive groves, and cereal cultivation, provides strong selective pressure. Climate change is making it worse: longer sporulation seasons, higher outdoor spore loads, more crop disease driving more fungicide use.

- Aspergillus fumigatus causes 30–90% mortality in immunocompromised patients. The WHO placed it at critical priority in its first Fungal Priority Pathogens List (2022), explicitly recognising its One Health dimension.
- Over 80% of resistant isolates were recovered from dust — not from active air sampling. Active air sampling is the only method currently required by Portuguese law. It would have missed more than 80% of the evidence.
- Children spend 6–9 hours per day in these classrooms — an occupational-level exposure with no occupational health monitoring whatsoever.
- The ventilation paradox worsens with climate change: schools close windows during heatwaves to stay cool, reducing air exchange and allowing indoor spore accumulation to grow.

REGULATORY FAILURE	*Current Portuguese indoor air quality legislation (2013) requires only total fungal CFU/m³ counts — not species identification, not thermal tolerance testing, not resistance profiling, not dust sampling. A classroom with 500 CFU/m³ of azole-*

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resistant A. fumigatus and one with 500 CFU/m³ of harmless Cladosporium are treated as identical. The science has advanced considerably since 2013. The regulation has not.

5. Policy Recommendations

5.1 Air Pollution and Infectious Disease Surveillance

1. **REC 1:** Integrate PM2.5 and PM10 data as mandatory co-variables into national respiratory disease surveillance models.

Forecasting viral surges without concurrent air quality data produces structurally incomplete predictions. National public health surveillance systems should issue automated alerts to hospitals when converging PM surges and viral circulation are projected — enabling pre-emptive scaling of capacity before clinical demand peaks.

2. **REC 2:** Update indoor air quality standards for buildings housing vulnerable populations to include climate-sensitive biological parameters.

European standards (EN/ISO) for indoor air in hospitals, schools, and elderly care facilities must define seasonal PM thresholds, documented air purification protocols, and ventilation requirements that adapt to outdoor pollutant concentrations in real time — not fixed benchmarks that ignore season, climate, and occupant vulnerability.

5.2 Hospital Infection Prevention

3. **REC 3:** Implement routine integrated molecular aerobiological surveillance in ICUs, emergency departments, and infectious disease wards.

Weekly air and surface sampling using qPCR and metagenomics — covering respiratory viruses, nosocomial bacteria, fungi, and AMR markers simultaneously — should be established as standard practice. Environmental signals appearing before clinical peaks should trigger proportionate infection control responses. The methodology is validated and operationally feasible.

4. **REC 4:** ECDC should initiate a pilot air-based AMR surveillance programme across hospital networks in multiple member states.

Airborne AMR gene profiles reflect local antimicrobial pressures not captured in clinical isolate surveillance alone. Two adjacent hospitals may have entirely different environmental AMR fingerprints. A coordinated European pilot, with open data publication requirements, would generate the evidence base needed to scale this approach.

5.3 Extreme Weather Events

5. **REC 5:** Mandate aerobiological surveillance as a standard component of disaster response plans for floods, wildfires, and dust storms.

Sampling should begin within weeks of an extreme event and continue through reconstruction. During the drying and rebuilding phase — when contaminated sediments become airborne — preventive respiratory protection measures must be activated for workers and residents in affected areas. The Valencia flood (2024) provides both the evidence base and the methodological template.

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5.4 Antifungal Resistance in Indoor Environments

6. **REC 6:** Update indoor air quality regulation to require Aspergillus species identification, thermal tolerance testing at 50°C, and dust matrix sampling alongside active air sampling.

A total CFU count is insufficient. Azole-resistant A. fumigatus and harmless Cladosporium register identically under the current framework. The Portuguese school data demonstrate that active air sampling alone misses over 80% of resistant isolates. Electrostatic dust collectors (30-day integrated sampling) and settled dust sampling must become mandatory parallel methods. This requires a regulatory update — not additional resources.

7. **REC 7:** Include indoor educational environments in European AoR surveillance networks.

Existing European azole-resistance surveillance networks focus on clinical settings and outdoor agricultural environments. They must be extended to schools and other indoor spaces where immunocompromised individuals spend extended periods. Portugal, with documented AoR in primary schools and extensive DMI fungicide use, should position itself as an active contributor to European environmental surveillance — not a passive observer.

8. **REC 8:** Clinicians treating immunocompromised patients should be informed that educational environments represent a potential source of primary azole resistance.

Immunosuppressed individuals — transplant recipients, patients on chemotherapy, HIV-positive individuals — who attend or work in primary schools are at elevated risk of exposure to azole-resistant A. fumigatus. Empirical antifungal treatment decisions for patients with plausible school-based exposure should factor in the possibility of primary resistance. Waiting for treatment failure is not an acceptable clinical strategy.

9. **REC 9:** Develop a One Health framework connecting agricultural fungicide use, indoor dust AMR accumulation, and clinical resistance outcomes.

The transmission chain — agricultural DMI fungicides → environmental spore selection → indoor dust deposition → human exposure → potential clinical treatment failure — is now molecularly documented. A European programme linking agricultural fungicide use data, environmental sampling (soil, outdoor air, indoor dust), and clinical isolate genomics through a shared database would complete this chain and enable targeted intervention at the point of greatest leverage.

6. Conclusion

Air is the invisible connective tissue of One Health. It links agricultural fields to school classrooms, industrial emissions to hospital wards, and climate systems to human lungs. It carries pathogens across national boundaries, warns of epidemic surges before hospitals notice them, and deposits drug-resistant fungi in spaces where children spend their school day — entirely below the threshold of current regulatory detection.

The surveillance tools exist. The scientific evidence is accumulating. What is absent is the regulatory and institutional will to treat airborne biological risk with the same seriousness applied to waterborne or foodborne hazards. The nine recommendations in this brief do not require new science. They require political decision.

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FINAL MESSAGE	*18.5% azole-resistant Aspergillus in school classrooms is invisible under current law. Viral RNA in hospital air precedes clinical peaks — but no system monitors it. Resistance genes travel thousands of kilometres through the atmosphere — but no regulatory framework addresses it. The knowledge exists. The action is overdue.*

Sources

Based exclusively on the ONE-Bridge in Health Webinar: 'Aerobiology in a Changing Climate' (June 2026). Scientific findings from: A. Chatziantoniou (University of Patras, Lab of Public Health, Epidemiology and Quality of Life) — PM-virus interaction and climate change; K.A. Koukouvini (University of Patras) — hospital indoor aerobiology, 2010 and 2026 case studies; Dr. S. Pozdniakova (ISGlobal Barcelona, AirLab, Environmental Microbiology) — atmospheric microbiome, aircraft sampling campaigns, Valencia flood post-disaster sampling (2024), TWIN AIR project; Dr. R. Cervantes (Instituto Politecnico de Lisboa / Nova University Lisbon, Health and Technology Research Center / Public Health Research Center) — azole-resistant Aspergillus fumigatus in Portuguese primary schools, IN-CHILD project. Webinar host: Laboratory of Public Health, Epidemiology and Quality of Life, Medical School, University of Patras. ONE-Bridge consortium: University of Patras, Technical University Dresden, CNRS France, ISGlobal Spain, EMVIO.

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