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Original Research Paper

Environmental Persistence of Zoonotic Pathogens and Public Health Risk

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Key Words

Zoonotic pathogens, Environmental persistence, Public health Risk, Exposure modeling.

Abstract

The role of the environmental persistence of zoonotic pathogens in epidemiological frameworks is often underrepresented when considering the extent of its influence on the dynamics of transmission, the ability to detect outbreaks, and the risk to public health. This research designs a novel integrated framework to analyze the impact of the environmental persistence of pathogens on total human risk and the potential for outbreaks. This research integrates reported persistence ranges to decay modeling, potential exposure estimation, and risk characterization to describe the heterogeneity of persistence in soil, water, organic waste, aerosols, and fomites, and its strong dependence on climate. The study finds that environmental persistence is a non-linear risk amplifier, in which a small increase in the duration of pathogen survival can lead to a significant increase in total infection risk and facilitates prolonged transmission that is concealed. The study finds that persistence reservoirs hinder outbreak surveillance and obstruct the efficacy of interventions that focus on animal hosts. The study emphasizes the importance of the environmental persistence of pathogens and the need for novel surveillance and mitigation frameworks to assist in the early detection of public health zoonotic threats.

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Introduction

Zoonotic pathogens continue to contribute greatly to the global burden of infectious disease because their transmission does not remain limited to animal hosts or human populations alone. Instead, transmission often occurs within linked animal–environment–human systems. Many emerging infectious diseases are animal in origin, and a large share of them involve indirect transmission, where infection occurs without direct contact between humans and infected animals or their secretions (Elsohaby & Villa, 2023). In such cases, the environment is not simply a passive sink. The system can also serve as an active reservoir holding, moving, and reintroducing infectious agents into human populations. This role alters the environment, of the outbreak, and prolongs exposure, conceals transmission pathways, and reduces the efficacy of interventions that target the hosts alone.

The ability to survive in the environment for prolonged periods is therefore an important feature of many zoonotic pathogens. Environmental persistence allows pathogens to bridge the time and space between shedding and human exposure, making transmission possible even when host presence is low, irregular, or difficult to detect. Studies of zoonotic disease emergence show that persistence in soil, water, air, and contaminated surfaces commonly precedes human infection, especially in farming regions, peri-urban settings, and wildlife–human contact zones (Jones et al., 2008). These observations show that disease ecology cannot be understood by focusing only on the host. Environmental reservoirs must also be included in public health risk assessment.

The ability of a pathogen to persist in the environment is contingent on a mixture of its inherent biological characteristics and those of the surrounding environment. Structural features like viral envelopes, capsids, cell walls of bacteria, and the ability to form spores improve resistance to drying, ultraviolet radiation, oxidative stress, and electromagnetic radiation. Environmental conditions, like the moisture and temperature, and the characteristics of surfaces may modify the decay rate that a pathogen may exhibit in various places at various times (Oswin et al., 2021). This variation shows that persistence should not be treated as a fixed parameter in epidemiological models.

Pathogens are influenced by their surrounding environment and can stay alive longer or shorter depending on what the environment is like. Pathogens try to mask their true locations in many environments like water and soil. In water systems, pathogens are usually protected from being inactivated longer and are able to be transported over longer distances, and pathogen survival is longer. In soil systems, soil texture, moisture and composition helps mask them and in many studies, moist and organic-rich soils can prolong the environmental survival of pathogens (Bierque et al., 2020). These mechanisms demonstrate that environmental persistence depends not only on the pathogen itself, but also on the properties of the reservoir in which it is located.

Biofilm formation is another important mechanism that increases public health risk.

Microorganisms embedded in biofilms are protected by a polymeric matrix that reduces the effect of disinfectants, buffers environmental stress, and supports survival in slower metabolic states. Experimental and modeling studies have shown that pathogens in mixed-species biofilms survive longer than planktonic organisms, even under standard sanitation conditions (Wißmann et al., 2021). As a result, environmental settings that appear clean may still remain infectious.

Environmental persistence also creates indirect exposure pathways that are not directly linked to animal contact. Human exposure may continue after initial shedding through contaminated dust, resuspended dried feces, aerosols, and fomites. Research on airborne zoonotic pathogens has shown that, particularly in dry and windy environments, viable pathogens may remain in aerosols and settled dust at distances greater than those expected from simple droplet loss alone (Santarpia et al., 2020). These pathways help explain outbreaks in populations that have no obvious occupational or domestic animal exposure.

Water-mediated exposure is one of the best documented links between persistence and infection. Zoonotic pathogens can enter surface waters from animal waste, runoff, or from activities of wildlife and can remain viable to contaminate drinking waters, irrigation waters, and waters used for recreation. Although sunlight and higher temperature can reduce viability, many studies show that viable pathogens may still persist for long periods under favorable environmental conditions, turning short contamination events into chronic public health risks (Brouwer et al., 2018).

Leptospira is a clear example of how environmental persistence supports zoonotic transmission. Pathogenic Leptospira spp. can survive and in some cases multiply in soil and surface water, especially under warm and humid conditions. Environmental studies over many biogeographic regions have shown persisting organisms in wet soils and stagnant waters, and the persistence appears to be dependent on the availability of water and conditions of land use (Sayanthi & Susanna, 2024). Experimental work has also shown that waterlogged soils can sustain infection risk well beyond the period of fresh contamination (Yanagihara et al., 2022).

The environment is also important in zoonoses acquired by inhalation, where aerosol persistence plays a major role. Coxiella burnetii, the causative agent of Q fever, is highly resistant to drying and other environmental stressors. Field studies in livestock settings have shown that contaminated dust can remain viable for long periods and may spread infection across large areas under suitable climatic conditions (Oliveira, 2021). This kind of endemic expansion shows why outbreak containment is complex and why monitoring the environment should be considered in conjunction with occupational and community health monitoring (Woodford et al., 2024).

From a public health perspective, prolonged environmental persistence acts as a multiplier of risk because it increases the probability of repeated and cumulative exposure. Quantitative microbial exposure studies show that even a

small increase in environmental survival time can lead to a much larger infection risk when exposure occurs repeatedly across populations (Brouwer et al., 2018). At the same time, climate change, land-use change, and ecosystem disruption can alter persistence–risk relationships in ways that further increase zoonotic threats (Mahon et al., 2024). Although the importance of persistence is now more widely recognized, available persistence data are still not well integrated into operational risk assessment frameworks. Most studies describe survival in specific media or under specific conditions, but fewer studies connect persistence mechanisms directly to human exposure pathways in a structured risk framework. This gap provides the basis for the present study, which aims to connect environmental persistence with public health risk within a One Health perspective (Ghai et al., 2022).

Environmental Reservoirs and Persistence Mechanisms

Environmental reservoirs serve as the critical interface through which zoonotic pathogens move from being host-associated to becoming a population-level public health threat. Unlike direct transmission, these reservoirs separate the presence of a pathogen from immediate host contact, thereby enabling persistence and delayed exposure. Pathogens shed from animal hosts are redistributed through interconnected compartments such as soil, water, air, fomites, and organic waste, which together create environmental settings that support sustained human infection, as represented conceptually in figure 1.

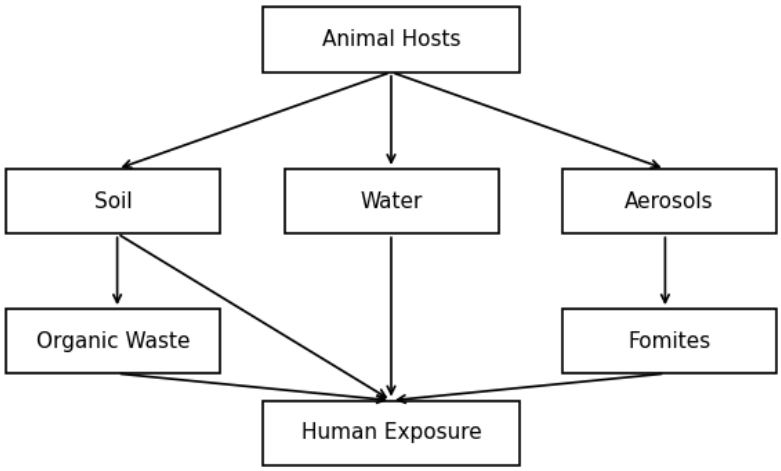

Figure 1: Schematic Representation of Environmental Reservoirs and Transmission Pathways of Zoonotic Pathogens

Soils have the potential to act as heterogeneous reservoirs for a wide range of zoonotic pathogens. Pathogens can stick around for long amounts of time due to the variety of their substrates, their ability to retain moisture, and their content of organic materials. A higher concentration of organic materials and clay contributes to the retention of pathogens and moisture. Soils also provide some protection from UV radiation. Survival is also strongly

affected by moisture content, and the higher moisture content the longer the pathogens will probably persist. These properties especially after a natural man-made event greatly increase the risk of soil- mediated transmission.

Pathogens from wildlife and livestock are constantly entering surface water through the runoff, and because of the water, these pathogens can survive and spread over large areas. Survival is influenced by temperature, solar radiation, salinity, and turbidity, with cold, nutrient-rich and turbid waters often supporting longer persistence and reduced photoinactivation. Therefore, water bodies may become important convergence points linking pathogen shedding to human exposure via contaminated drinking water, irrigation, and recreational activities.

Aerosols and airborne particulates represent a related but highly variable and epidemiologically important reservoir for zoonotic agents that can survive outside liquid environments. During animal movement, waste handling, and wind-driven resuspension of dust, contaminated particles may become airborne. The ability of pathogens to remain viable in aerosols depends on particle size, humidity, and temperature. Conditions of low humidity and moderate temperature can support longer airborne survival for some pathogens, thereby increasing the possibility of prolonged exposure in populations without direct animal contact or surface contamination.

Fomites are short- to medium-term reservoirs that connect human behavior with environmental contamination. Tools, equipment, clothing, and surfaces may retain viable pathogens for variable periods depending on the surrounding conditions and the properties of the material. On non-porous surfaces, viruses and bacteria may survive longer, while porous surfaces may trap microorganisms and diminish the efficacy of cleaning agents. Successive human intervention with infected surfaces can be a result of ongoing transmission despite the lack of visibility of contamination.

Animal organic waste such as manure and bedding is heavily contaminated and is one of the most concentrated environmental reservoirs. Storage conditions are also really important because low temperatures with high organic content and a lack of oxygen can actually slow down the inactivation of the pathogens which is a really important factor. During handling, transport, and land application, organic waste can also act as a secondary source that redistributes pathogens into soil, water, and aerosol reservoirs, thereby extending exposure pathways.

Desiccation resistance is one of the most important adaptations that enhance the survival of organisms in environments that are drying repeatedly. Such adaptations include robust viral capsids, thick bacterial cell walls, and resistant outer membranes. Reduced water loss and preservation of infectivity under low humidity are all important. These organisms with strong resistance to desiccation are extremely significant with regard to the transmission of viral aerosols and fomites because of their ability to persist in environments that are very hostile to organisms.

Another mechanism that biofilms use to form and persist in environments that are natural and also ones that are man-made is their ability to

form and persist in built and natural environments. Biofilms form and persist in built and natural environments by building a biofilm that consists of a thick, protective, and complex matrix, which protects them from disinfectants and environmental conditions that are hostile to their survival, and also helps them survive in a collective defense mechanism. Biofilms form protective, thick, and complex biofilms and aid in the long survival of microorganisms in soil and water, and even in man-made water structures.

Microorganisms in a dormant state are able to survive for a long time. Spores from some microorganisms are even able to survive in extremely hot temperatures, and some microorganisms in soil and waste can survive in a dry state. Microorganisms that are present and are metabolically inactive can survive through conditions that are not favorable for a long time. There is also a great risk for infection from the microorganisms that are present in their inactive state are pathogens that are present, and hide the risk of infection.

Temperature and humidity act as integrative controls across all major environmental reservoirs. Rather than acting independently, these variables interact with reservoir properties to influence decay kinetics, aerosol stability, and surface persistence. Seasonal and diurnal changes may therefore shift the dominant reservoirs and exposure pathways over time, highlighting the need to treat environmental persistence as a system-level process. Taken together, these reservoir-specific mechanisms show why environmental persistence is central to zoonotic transmission and why it must be included in sound public health risk assessment.

Methodology

This study was designed to examine how long zoonotic pathogens can remain in different environmental media and how that persistence contributes to public health risk. The model includes all aspects of the environment focus data that includes decay behavior, transfers between different media, human exposure, and risk of infection. In other models, risk of infection has been calculated without considering the environmental decay behavior. In this model, infection risk is estimated considering the amount of the environment and human interaction along with behavior of the pathogen.

The persistence and quantity of pathogens were obtained from three major sources: environment studies of pathogen persistence and quantity, active surveillance studies, and laboratory studies of pathogen persistence that are peer reviewed. Environmental study sampling gave pathogen quantitation located in soil, water, aerosol, fomites, and organic waste in agricultural, peri-urban wild life interfaces. Active surveillance studies gave information about infection and shedding of the pathogen as well as the spatial and temporal distribution of the contaminant. Laboratory studies of persistence gave information decay constants and survival functions for pathogens at controlled temperature, humidity and moisture. These studies all contributed to quantitatively modeling pathogen behavior both in the laboratory and in the field.

Before modeling, all data were harmonized before modeling to facilitate comparison across studies. When a study measured concentration of a quantity, unit and medium were converted into the same. There were studies that measured the presence of a mole, but did not confirm that the mole was alive. These data points were kept, but in strain studies risk was estimated. This step ensured that the modeled environmental loads reflected both the documented presence of pathogens and the limitations of the original data sources.

Persistence was modeled using either a first-order decay model or a biphasic decay model, depending on the survival pattern reported for each pathogen–medium combination. For simple exponential decline, the first-order model was used:

$$C_t = C_0 e^{-kt}$$

Where C_t is the pathogen concentration at time t, C_0 is the initial concentration and k is the decay constant. This form was used when survival declined continuously over time without evidence of a protected residual fraction.

When the data showed an initial rapid decline followed by a slower persistent phase, a biphasic model was used:

$$C_t = C_0[fe^{-k_1 t} + (1-f)e^{-k_2 t}]$$

Where f is the fraction of the rapidly decaying population, k_1 is the fast decay constant and k_2 is the slow decay constant. This model was used to represent protected subpopulations, biofilm-associated organisms, or other persistence mechanisms that produce tailing behavior.

Because decay is strongly influenced by environmental conditions, the decay constant was not treated as fixed. Instead, it was adjusted using matrix-specific and climate-related modifiers. The effective decay rate was defined as

$$k_{\text{eff}} = k_0 \times M_T \times M_H \times M_E$$

Where k_0 is the baseline decay constant, M_T is the temperature modifier, M_H is the humidity or moisture modifier and M_E is a medium-specific environmental modifier. For soil and organic waste, M_E reflected organic matter and moisture content. For water, it reflected turbidity and solar exposure. For fomites and aerosols, it reflected surface type and relative humidity. This allowed the model to account for environmental heterogeneity across different reservoirs.

The persistence ranges shown in table 1 were not used directly as fixed model inputs. Instead, they were used as empirical reference ranges to check whether the simulated outputs remained within biologically realistic limits. If a modeled persistence time fell far outside the reported literature range for that pathogen and medium, the related parameters were rechecked and included in later sensitivity analysis.

Pathogen movement between environmental media was represented using simplified transfer coefficients. These coefficients did not attempt to reproduce full hydrodynamic or aerosol transport processes, but they allowed the framework to capture the main pathways of redistribution. The transferred concentration from medium $i$to medium $j$was defined as

$$T_{ij} = \alpha_{ij} C_i$$

Table 1: Reported Environmental Persistence Ranges of Representative Zoonotic Pathogens Across Media

Zoonotic Pathogen	Reported Persistence Range (environmental)	Dominant Environmental Reservoir	Governing Persistence Mechanisms
Leptospira interrogans	Days to > 6 weeks	Moist soil and surface water	Moisture tolerance, metabolic dormancy
Salmonella enterica	Weeks to months	Soil and organic waste	Biofilm formation, organic matter protection
Campylobacter jejuni	Days to weeks	Water and animal waste	Viable-but-non-culturable state
Coxiella burnetii	Months to years	Dust-contaminated soil and manure	Extreme desiccation resistance
Avian influenza viruses	Days to months	Cold freshwater and fecal material	Temperature–humidity coupling
SARS-related coronaviruses	Hours to weeks	Fomites and aerosols	Envelope stability, humidity sensitivity
Bacillus anthracis (spores)	Years to decades	Soil and organic waste	Endospore formation

Where T_{ij} is the transferred load, α_{ij} is the transfer coefficient between the two media, and C_i is the concentration in the source medium. Soil-to-water transfer was linked to runoff and infiltration, aerosol transfer to disturbance-driven emissions, and fomite contamination to contact frequency between contaminated materials and surfaces.

Human exposure was then estimated separately for each major environmental pathway. The general exposure dose per event was defined as

$$D = C_m \times CR \times EF$$

Where D is the pathogen dose received, C_m is the concentration in environmental medium m, CR is the contact rate or intake rate and EF is an exposure factor representing pathway-specific efficiency. For water exposure, CR represented the average water contact or ingestion volume. For soil and organic waste, it reflected incidental ingestion and dermal contact. For aerosols, it represented inhalation volume and particle deposition. For fomites, it captured hand-to-surface and hand-to-mouth transfer. This step converted environmental persistence into pathway-specific exposure.

The probability of infection from a single exposure event was estimated using dose–response functions. Where possible, pathogen-specific functions were selected from published experimental or epidemiological studies. A commonly used exponential form was applied as

$$P_{\text{inf}} = 1 - e^{-rD}$$

Where P_{inf} is the infection probability from one exposure event, D is the exposure dose and r is the pathogen-specific infectivity parameter.

When pathogen-specific human data were unavailable, conservative surrogate functions based on related organisms were used.

Because many environmentally mediated infections arise through repeated low-dose exposure rather than a single high-dose contact, cumulative infection risk was calculated over multiple exposure events. If the infection probability for one exposure is P_{inf}, then the cumulative risk over $n$repeated exposures was estimated as

$$P_{\text{cum}} = 1 - (1 - P_{\text{inf}})^n$$

Where P_{cum} is the cumulative infection probability over the chosen exposure period. This formulation captures the persistence-driven accumulation of risk and helps explain how even low environmental concentrations may generate substantial infection probability when exposure is repeated.

Uncertainty was handled using a probabilistic rather than deterministic approach. Key parameters, including decay constants, environmental concentrations, transfer coefficients, and contact rates, were represented as distributions instead of single values. Monte Carlo simulation was then applied across the full persistence–exposure–risk chain. For each simulation run, parameter values were sampled from their assigned distributions and used to compute persistence time, exposure dose, and infection probability. Final outputs were summarized as median values and uncertainty intervals rather than as single point estimates.

Sensitivity analysis was used to identify the dominant drivers of risk in different environmental media. Parameters related to decay, transfer, and exposure were varied systematically to determine their effect on cumulative infection risk. This analysis made it possible to distinguish settings in which persistence time was the main driver from settings in which human contact frequency or transfer efficiency played a larger role. In practical terms, this helps identify whether control efforts should focus more on reducing environmental survival or on reducing human exposure.

Model validation focused on qualitative and quantitative consistency rather than on predicting specific outbreaks. Simulated persistence times and relative risk rankings were compared with documented outbreak contexts and environmental detection patterns reported in the literature. Agreement between model outputs and published epidemiological patterns was taken as evidence of plausibility and internal consistency, rather than as proof of exact predictive accuracy. This validation approach was appropriate because the framework was intended to evaluate environmental risk scenarios rather than forecast exact outbreak events.

Taken together, this methodology provides a structured way to link the environmental persistence of zoonotic pathogens to public health risk. By combining empirical persistence data, mechanistic decay modeling, transfer pathways, exposure estimation, dose–response analysis, uncertainty treatment, and validation logic, the framework offers a clear and flexible basis for evaluating environmental transmission

scenarios and for supporting surveillance and intervention planning.

Results and Discussion

The results show clear variation in environmental persistence across pathogen groups and environmental media. This means that survival is not a fixed property of a pathogen, but changes with the surrounding medium and environmental conditions. The modeled outputs show that soil and organic waste have the longest persistence times, surface water occupies an intermediate position, and aerosols and fomites generally have the shortest persistence times. Figure 2 reflects this strong stratification of survival behavior across environmental media.

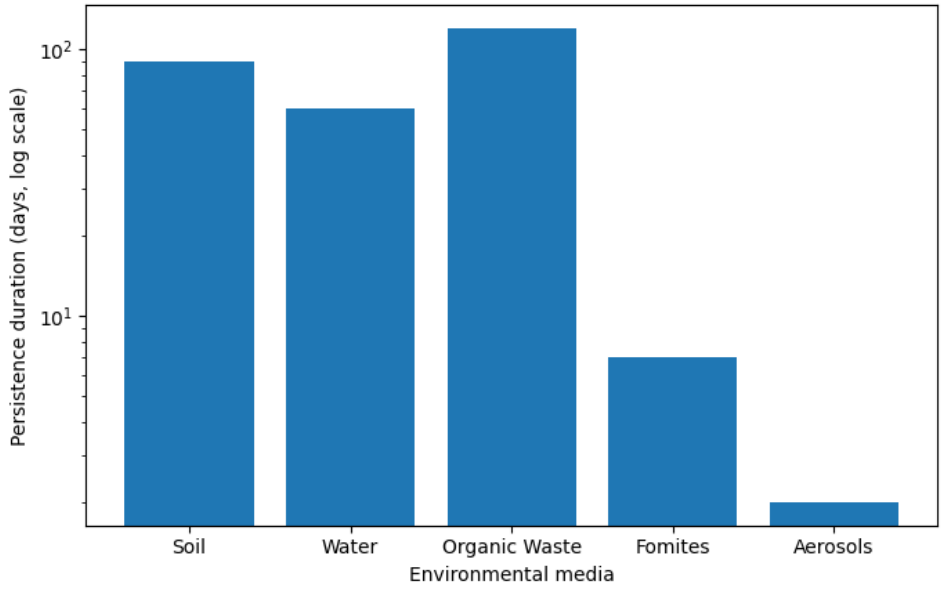

Figure 2: Comparative Persistence Duration of Zoonotic Pathogens Across Environmental Media

The longest modeled persistence occurs in matrices with high moisture and high organic content. In practical terms, this places soil, manure, slurry, and other organic waste reservoirs at the upper end of the persistence distribution, while more exposed media such as air and dry surfaces remain at the lower end. The quantitative contrast is important because it shows that the difference between environmental media is not small. In the present framework, persistence spans from short survival in hours or days for some aerosol- and fomite-based pathways to survival over weeks, months, or longer in protected reservoirs. This confirms that environmental medium is one of the strongest determinants of persistence-based public health risk.

Persistence outcomes across the different reservoirs are strongly influenced by climatic forcing. Lower temperatures and higher moisture extend survival in soil, water, and organic waste, while aerosols and surfaces show their greatest persistence under moderate temperature with low to intermediate humidity. These climate-linked shifts are quantitatively important because even modest changes in temperature and moisture can move a pathogen from short-term survival into a duration that spans multiple exposure

opportunities. To examine this climate sensitivity more clearly across the modeled reservoirs, figure 3 compares the persistence response of the major environmental media under contrasting temperature and moisture conditions.

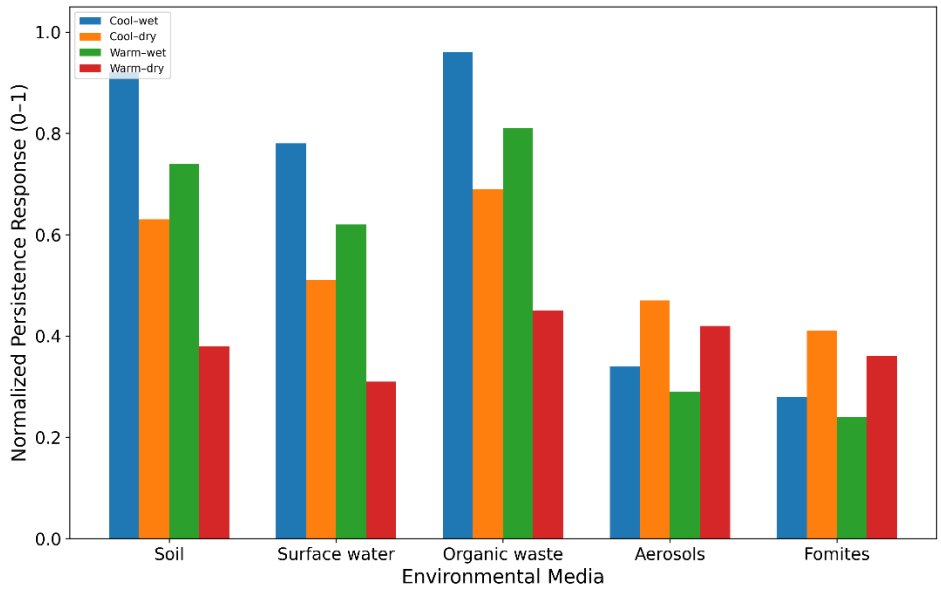

Figure 3: Climate-Adjusted Persistence Response Across Environmental Media Under Contrasting Temperature and Moisture Conditions

Figure 3 illustrates how climate impacts environmental media surrounding soil and organic waste more than streams, aerosols, and fomites. Streams have a moderate sensitivity, while soil and organic waste have a greater impact of persistence extension under cooler and moist conditions. This comparison shows us that climate may overall not simply increase or decrease persistence, but influences which reservoirs become most critical under varying environmental conditions. When these conditions exist long reservoirs become more persistent which means that contamination can survive longer, increasing the chances of overlapping with multiple human contact. In contrast, shorter reservoirs do not become dominant simply because they have the longest survival time, but because climatic conditions prolong their survival enough to increase the chances of meaningful exposure opportunities. This means the risk is dependent on the interaction between persistence duration and pathway efficiency more than survival time alone.

The results also show that persistence time alone does not fully determine epidemiological importance. Aerosols and fomites generally have shorter survival durations than soil or water, but their high contact frequency and efficient transfer routes can still produce substantial cumulative exposure. In repeated-contact environments such as workplaces, transport settings, indoor public areas, and shared built spaces, even short-lived reservoirs can support meaningful transmission risk when exposure occurs often enough. This means that a medium with shorter persistence may still remain epidemiologically important if human contact is frequent.

When the persistence outputs are translated into public health terms, a strongly non-linear relationship appears between environmental survival time and relative infection risk. Figure 4 shows that risk does not increase in a simple linear way with increasing persistence. Instead, once persistence extends beyond a threshold that allows contamination to remain across repeated exposure cycles, the infection risk rises much more steeply. In other words, a modest increase in survival time may produce a disproportionately large increase in cumulative infection probability.

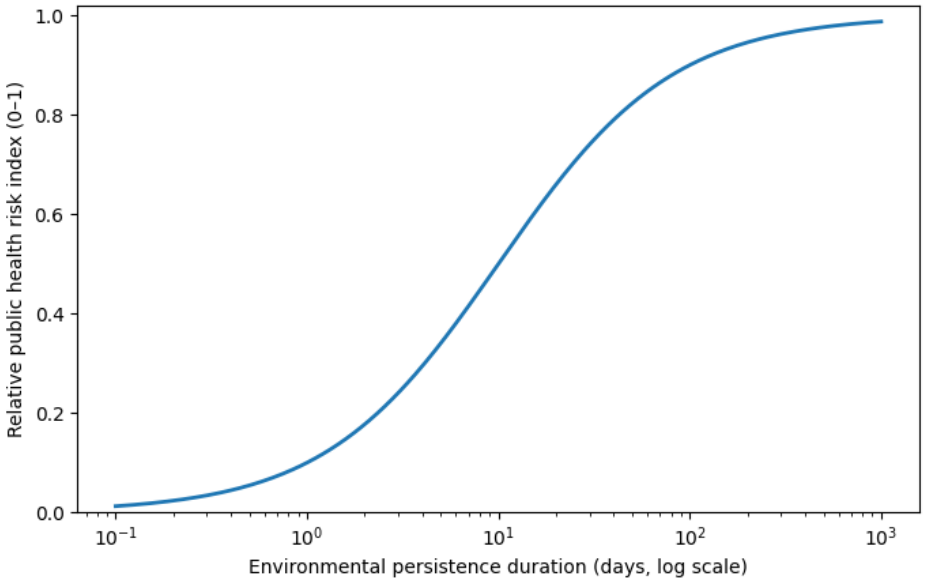

Figure 4: Modeled Relationship Between Environmental Persistence and Relative Public Health Risk Index

At shorter persistence times, the increase in relative risk remains limited because the pathogen disappears before many repeated exposures can occur. Once persistence becomes long enough to overlap with multiple contact events, the curve rises sharply. This means that the public health effect of persistence is threshold-dependent rather than gradual. Such a pattern explains why environments with moderate contamination may still generate substantial risk if the pathogen remains viable long enough to cross several exposure cycles.

This persistence-driven accumulation of risk also helps explain why early transmission can remain difficult to detect. In soil, sediment, and organic debris, contamination may remain at low levels for extended periods without immediately producing a large number of recognized clinical cases. However, because environmental reservoirs retain viable pathogens over time, they can support slow and repeated exposure that later becomes epidemiologically important. This delays outbreak recognition and makes source attribution more difficult, especially when transmission does not occur immediately after shedding.

The results further show the importance of short-lived but frequently contacted reservoirs in dense urban environments. Aerosols and fomites in built settings may have lower survival times

than soil or organic waste, but they can still generate high cumulative risk because of repeated human contact and high population density. Under such conditions, even small reductions in ventilation quality, humidity control, or surface hygiene may increase repeated exposure and shift the risk curve upward. This helps explain why urban outbreaks can change quickly in response to environmental control measures, even when environmental persistence is relatively short.

Agricultural systems show a different persistence–risk pattern because they combine several long-lived reservoirs at the same time. Soil, manure, slurry, waste storage systems, and contaminated water can all maintain pathogen loads for extended periods and support multiple exposure routes simultaneously. In these settings, occupational contact, seasonal labor, and rainfall-driven redistribution further increase exposure opportunities. Persistence therefore acts as a structural amplifier in agricultural environments by maintaining transmission potential even when animal shedding varies over time.

At wildlife–human interfaces, environmental persistence acts as a bridge between intermittent wildlife shedding and human infection. When pathogens survive for long periods in soil or surface water, even infrequent contamination by wildlife may create an extended window of human exposure. This means that direct contact with wildlife is not always necessary for infection to occur. The results therefore help explain why zoonotic outbreaks may appear in places where wildlife contact is limited, irregular, or not clearly documented. Because these persistence effects operate differently across urban, agricultural, and interface settings, a direct comparison of cumulative risk across the main environmental contexts is presented in figure 5.

Figure 5 shows that different combinations between persistence and exposure across environments shape cumulative infection risk. Agricultural systems have the overall greatest risk by combining repeated occupational exposure and long-lived reservoirs, while the wildlife–human interface remains significant because persistence increases the exposure window after irregular contaminations. In dense urban environments, the shorter-lived reservoirs create a meaningful risk due to high contact and population density, which creates a different pattern. This comparison strengthens the overall interpretation that public health risk depends on the survival time of pathogens and the structure of exposure and persistence of the environment.

The broader implication is that the dominant mechanism of risk is not the same in every environmental setting. In agricultural systems and wildlife-linked environments, persistence mainly enlarges the time window of exposure and supports delayed or concealed transmission. In dense urban settings, shorter-lived reservoirs may still remain important because repeated human contact can compensate for reduced survival time. This shows that public health risk cannot be ranked by persistence duration alone, but must instead be interpreted together with exposure structure, contact frequency, and setting-specific transmission opportunities.

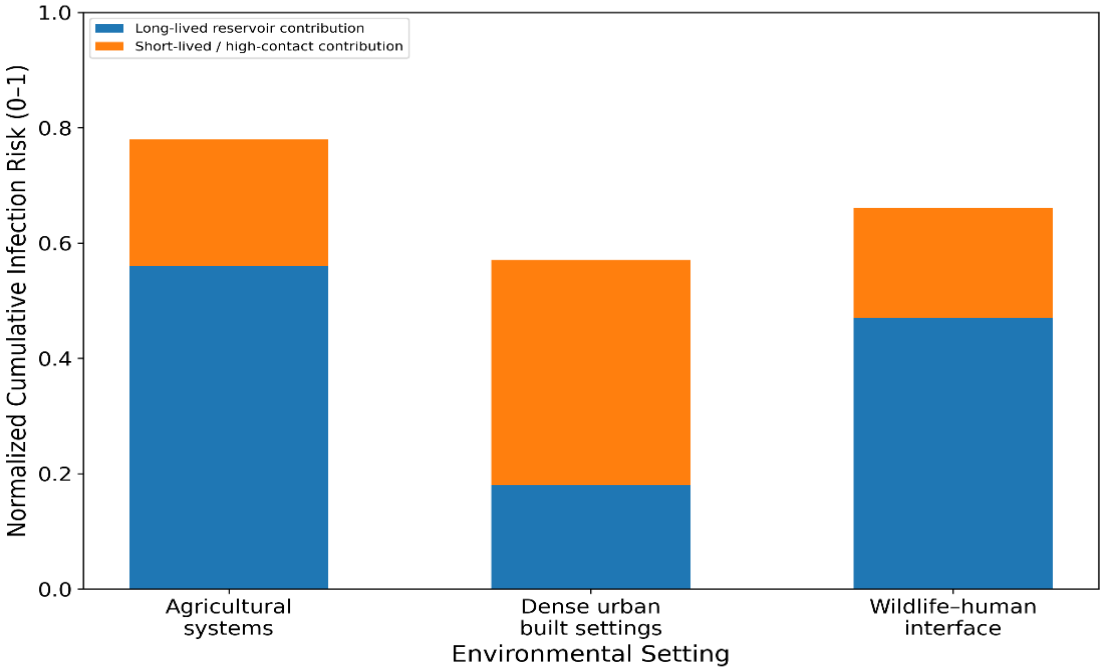

Figure 5: Comparative Cumulative Infection Risk Across Agricultural, Urban, and Wildlife–Human Interface Environments

Conclusion

This study shows that one should not look at environmental persistence as a minor feature of zoonotic pathogens. It is a critical factor that contributes to transmission, increases exposure, and alters the dynamics of the outbreak. The analysis across soil, water, organic waste, aerosols, and fomites shows that these environmental media act as connected reservoirs that separate human exposure from the original animal source. This helps explain why zoonotic outbreaks may continue or reappear even after animal-source control measures have been introduced, and why environmental contamination may exist before human cases become clearly visible.

The results also show that the effect of persistence on public health risk is not linear. When pathogens remain viable in the environment for longer periods, the cumulative probability of infection rises more sharply once contamination persists across repeated exposure cycles. Even small increases in survival time, due to supportive environmental matrices, or climate conditions, increases overall risk. The climate-adjusted analysis further shows that this persistence behavior is strongly medium-specific, with soil, water, and organic waste becoming especially important under cool and moist conditions, while shorter-lived reservoirs can still remain epidemiologically relevant when climatic conditions extend survival just enough to support repeated exposure. Environmental persistence is an important risk enhancer in the transmission of zoonotic disease.

The findings also imply that the environmental persistence of zoonotic pathogens may hinder surveillance and outbreak detection. In agricultural systems and wildlife–human interface settings, long-lived reservoirs may

support low-level but sustained transmission that is not easily captured by routine clinical surveillance. In urban settings, even short-lived reservoirs may still create substantial risk when contact frequency is high and population density is large. The comparative risk analysis across environmental settings further shows that persistence-driven public health risk does not arise through a single mechanism. In some settings, risk is dominated by long survival in protected reservoirs, while in others it is driven by frequent human contact with shorter-lived but highly accessible pathways. For this reason, surveillance systems that focus only on infected hosts or reported clinical cases may fail to capture the full extent of zoonotic transmission when environmental pathways are involved.

In summary, the study supports the need for mitigation strategies that include environmental monitoring together with host-centered control measures. Active surveillance of persistent reservoirs, better understanding of environmental decay processes, and interventions that reduce environmental survival can strengthen outbreak prevention and early detection. A public health approach that includes environmental persistence is therefore likely to improve preparedness and reduce dependence on reactive responses after clinical cases have already appeared.

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