

Commentary

Commentary on: Biofilms in human and veterinary infections: a central link in One Health frameworks

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Article history: Received 1 September 2026; Accepted 22 September 2026; Available online 24 September 2026

Article information: Elbediwi M, 2026. *One Health Microbiol. Infect.* Vol. 2, No. 1: 253-255. <https://doi.org/10.66585/ohmi.2026.2.0033>



Abstract

Not applicable

Keywords: AMR, Biofilm formation, Human medicine, One Health, Veterinary medicine

Antimicrobial resistance (AMR) within biofilm communities is a growing yet insufficiently characterized dimension of the One Health paradigm. The finding reported by Schweizer [1] warrants further investigation. One Health approaches to AMR have focused on the transmission of resistant organisms among humans, animals, and the environment. However, the biological context in which much of this resistance develops and persists, the biofilm, has received considerably less attention. Connecting the One Biofilm concept of Jacques and Malouin [2] to parallel clinical examples across human and veterinary medicine is a genuinely useful step, and the comments offered here are intended to carry that line of thinking further toward the experimental and genomic work needed to test the framework, not merely to describe it. The integrated framework is summarized schematically in Figure 1.

Where the comparative logic holds up well

Anchoring the human–animal comparison in shared molecular machinery rather than clinical appearance alone is the right choice. The parallel between *Staphylococcus aureus* and *Staphylococcus pseudintermedius*, and between *Porphyromonas gingivalis* and *Porphyromonas gulae*, works precisely because it rests on conserved mechanisms, including adhesins, the *ica* operon, polysaccharide intercellular adhesin (PIA), coagulase activity, gingipains, and outer membrane vesicles, rather than on the observation that infections in different hosts can sometimes look alike. Two diseases can resemble each other in a ward and a veterinary clinic without sharing a single underlying mechanism; two diseases can share a mechanism while presenting quite differently in practice. Grounding the comparison in conserved virulence and matrix biology is what gives the "study one for two" principle its scientific credibility.

An evolutionary dimension worth incorporating

The article's account of biofilm-associated resistance focuses on a well-established sequence: physical protection by the matrix, metabolic dormancy, efflux upregulation, and the eventual selection of classical resistance mutations under chronic antibiotic exposure. This picture is accurate, but it omits something directly relevant to the cross-sector argument. Resistance mutations are rarely without cost. A substantial body of work shows that mutations conferring antibiotic resistance typically reduce bacterial growth rate and competitive fitness when the selecting pressure is removed [3]. Importantly, this sheltering effect persists even after antibiotic pressure is removed. Experimental evidence from *E. coli* biofilms showed that resistant mutants remained at high frequencies within the biofilm for more than 45 days after treatment, whereas equivalent planktonic populations rapidly lost resistance through competition with sensitive clones [4]. This distinction is directly relevant to the cross-sector argument: biofilms maintain resistant subpopulations long after the selective pressure that generated them has passed. The fitness landscape of resistance mutations may differ within biofilms from that of planktonic populations [5]. The structured microenvironment of a biofilm, characterized by steep nutrient and oxygen gradients, altered metabolic activity, and dense intercellular signaling, can reduce or negate the growth disadvantage typically associated with resistance mutations under laboratory planktonic conditions [6, 7]. This buffering function of the biofilm is a second, mechanistically distinct reason why biofilms serve as long-term AMR reservoirs, alongside the tolerance-to-mutation pathway the article already describes. Examining these trade-offs in parallel, using human- and animal-derived isolates from the same species pairs the article discusses, would provide the One

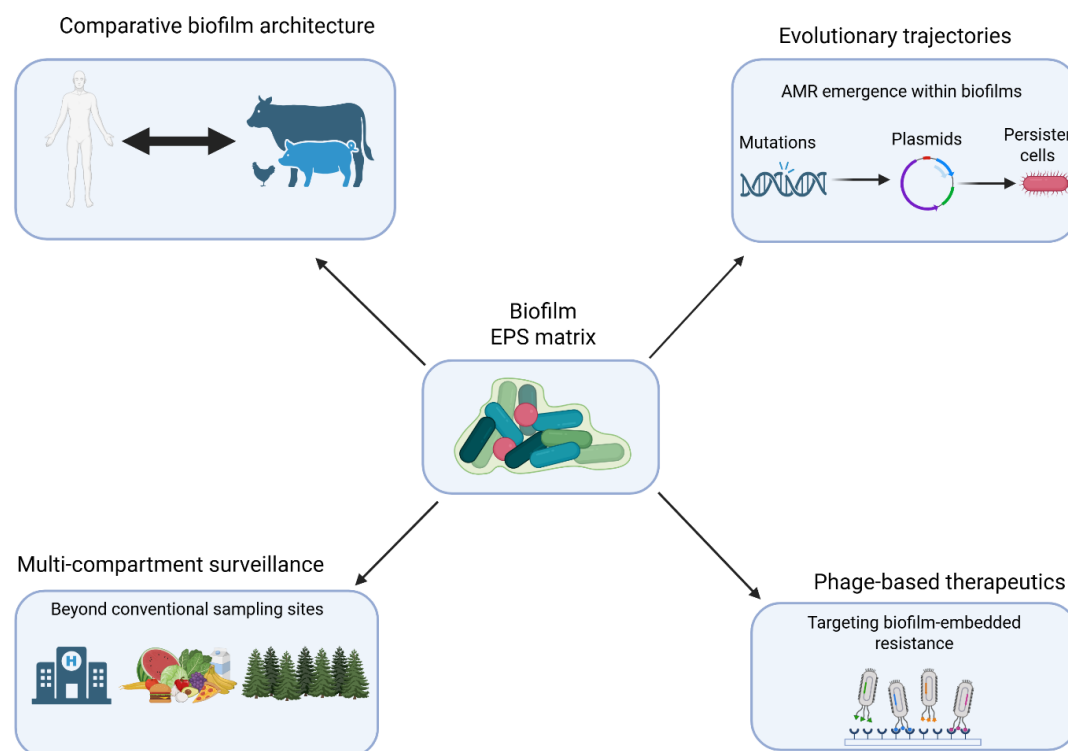


Figure 1: The One Biofilm framework across One Health interfaces. The central panel shows a biofilm community within an extracellular polymeric substance (EPS) matrix, the shared niche in which AMR develops and persists. Four dimensions radiate outward: (top left) Comparative biofilm architecture, conserved resistance mechanisms are shared between human and animal hosts; (top right) Evolutionary trajectories, AMR escalates within biofilms via mutation, plasmid-mediated horizontal gene transfer, and persister cell selection; (bottom left) Multi-compartment surveillance, monitoring must extend beyond clinical settings to hospital environments, agricultural systems, and natural ecosystems; (bottom right) Phage-based therapeutics, bacteriophages penetrate the EPS matrix to target biofilm-embedded, antibiotic-tolerant populations. Arrows indicate AMR dissemination from the biofilm outward across One Health compartments.

Biofilm framework with its first layer of comparative experimental evidence.

Surveillance requires more than additional sampling sites

Extending biofilm-focused sampling to implanted devices, chronic wounds, and oral surfaces is a sound recommendation. The harder problem is what to do with the material once it is collected. Standard culture methods for recovering isolates from biofilms still favor the subpopulation that grows most readily under laboratory conditions, reintroducing the bias that the article rightly criticizes in existing surveillance approaches. Direct metagenomic and metatranscriptomic profiling of biofilm communities, characterizing them where they live rather than after selective recovery, has been proposed as a more faithful route to the resistome and to the mobile genetic elements, plasmids, integrons, and resident phages that drive resistance exchange within these communities [8]. The methodological gap here is practical rather than conceptual, and it is also where collaboration between human and veterinary laboratories could be made concrete: a shared, sector-agnostic pipeline for biofilm metagenomics would do considerably more to realize the

"study one for two" ambition than parallel narrative reviews carried out independently in each field. It should be emphasized that culture-independent and culture-based approaches are complementary rather than competing [9]. While metagenomic and metatranscriptomic profiling can identify the full microbial composition of a biofilm community, including viable-but-difficult-to-culture organisms capable of sustaining infection, culture-based methods such as sonication of explanted implants retain a distinct and practical role [10]. Sonication-based recovery allows targeted identification of the organisms most likely to be driving clinical disease, along with their individual antibiotic resistance profiles, information that remains essential for guiding treatment decisions, managing the host inflammatory response, and preventing extensive tissue damage and associated morbidity [11]. A mature cross-sector biofilm surveillance framework will need to integrate both strategies rather than pit them against each other.

The therapeutic case for phage-based strategies is stronger than stated

The article reviews key anti-biofilm strategies, including surface modification, quorum-sensing interference, enzymatic matrix disruption, and localized drug delivery. However, the article mentions bacteriophage therapy only briefly, missing an opportunity given its potential within the article's framework. Phage and CRISPR-guided phage constructs are particularly well-suited for biofilm applications, as they can be engineered to penetrate the matrix and directly target the biological trade-offs discussed earlier. In some systems, phage resistance has been linked to a co-located resistance determinant, and CRISPR-Cas delivery platforms have been used experimentally to reverse antibiotic resistance by targeting specific resistance genes in bacterial populations [12]. If biofilm biology is sufficiently conserved across sectors to be treated as a single problem, the article's central premise holds, and phage or CRISPR-phage constructs validated in one clinical setting, with companion-animal wound isolates as the obvious starting point, become probable candidates for direct translation. This connection between the article's evolutionary framing and its therapeutic recommendations could be made more explicit. More broadly, the therapeutic case for any single anti-biofilm modality should be made with caution. Combining direct matrix-targeted approaches, such as enzymatic degradation or antibody-mediated disruption of biofilm structure, with antibacterial agents, whether antibiotics, phages, or small molecules, addresses two related problems simultaneously [13]. Physical disruption of the matrix increases the penetration and efficacy of antibacterial measures, while the accompanying bactericidal activity reduces the selective pressure that would otherwise favor resistance to the matrix-targeting component alone. Evidence from *in vitro* and early clinical studies suggests that such combination strategies outperform single-modality approaches in biofilm eradication and in limiting resistance emergence [13]. As the One Biofilm framework moves from concept toward experimental application across human and veterinary settings, evaluation of rationally designed combination regimens, rather than individual agents in isolation, should be a design principle from the outset.

Declarations and supplementary information

Acknowledgments: Not applicable.

Author contributions: Not applicable.

Funding source: Not applicable.

Conflict of interest statement: The author declares no conflict of interest.

Ethics statement: Not applicable.

Data availability: Not applicable.

Supplementary materials: Not applicable.

Declaration regarding the use of generative AI and AI-assisted technologies in the writing process: The author declares that the illustrative figure was created using BioRender web tools (<https://www.biorender.com>).

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