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Article

# Lipoxygenases and CFTR Inhibitory Factors Might Share the Same Role in Host-Microbe Interactions

Running Title: Lipoxygenases and CFTR Inhibitory Factors

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## Abstract

Cystic fibrosis transmembrane conductance regulator (CFTR) is a chloride channel in humans and other vertebrates, whose mutation leads to cystic fibrosis. CFTR inhibitory factor — Cif — is a recently discovered bacterial epoxide hydrolase that downregulates CFTR protein upon the bacterial infection. However, its cleaving activity towards fatty acid epoxides — epoxygenase-derived oxylipins — has been recently characterized. We identified a list of host-associated bacteria with putative Cif proteins, identified their most prevalent ecological functions by systematic literature review, and performed similar review for the previously assembled list of host-associated bacteria carrying lipoxygenase (LOX). Both Cif and LOX showed the association with pathogenesis and symbiosis in broad host range, and similar ecological profiles of their carriers suggested that they both might target oxylipin signaling in hosts. We also described the association of Cif with plant hormone biosynthesis and plant growth promotion — which indirectly supports our previous model of bacterial LOX action in plant and vertebrate hosts.

**Keywords:** lipoxygenase; CFTR inhibitory factor; Cif; oxylipins; host-microbe interactions; nosocomial pathogens; cystic fibrosis; plants

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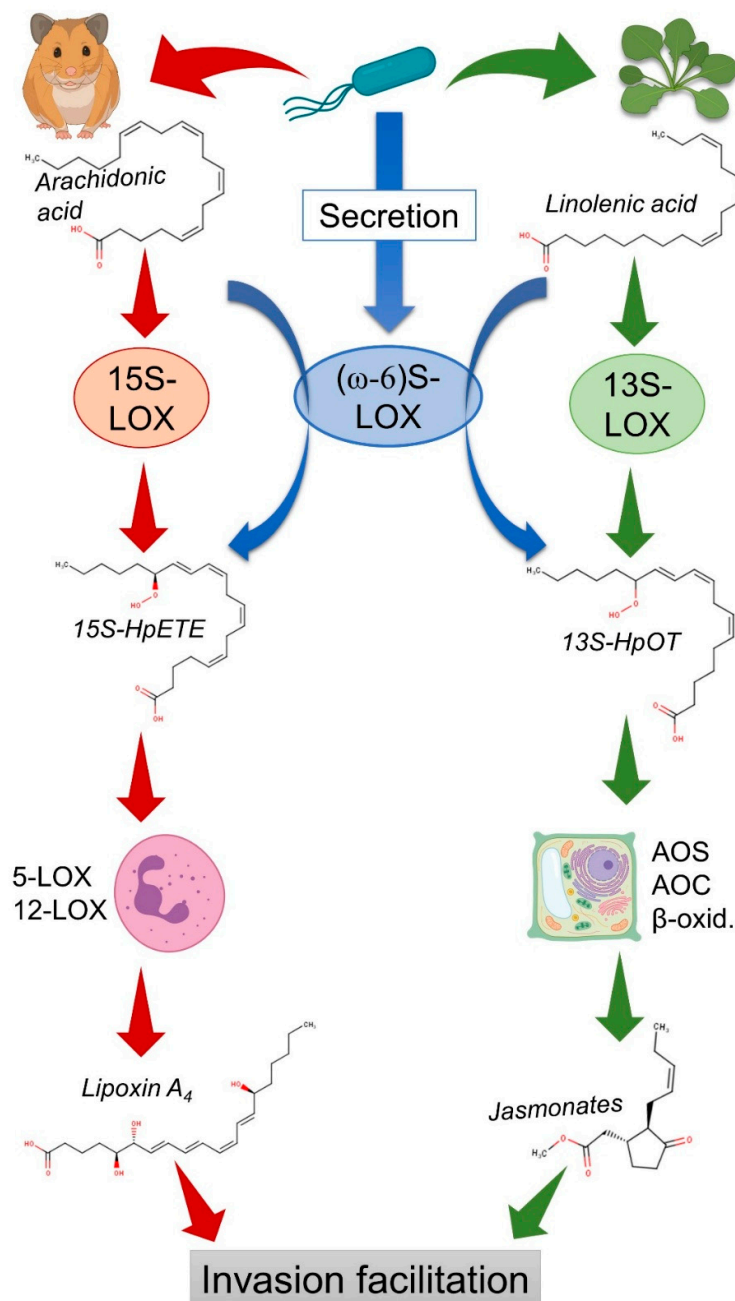
## Introduction

Lipoxygenases (LOXs) are a family of enzymes which oxidize polyunsaturated fatty acids (PUFAs) and yield fatty acid hydroperoxides which can be further transformed into a variety of oxidized derivatives — oxylipins. LOXs are ubiquitous in multicellular eukaryotes such as animals, plants, algae, and fungi; their products are involved in cell-to-cell signaling processes of all these organisms — including stress signaling, immune signaling, development and even reproduction [1–3]. LOXs are also present in some bacteria, such as cyanobacteria, myxobacteria, and a nosocomial pathogen *Pseudomonas aeruginosa* — but the experimental data on their functions are generally scarce [1,2].

*P. aeruginosa* is a notable exception — its LOX is well characterized, however still some controversies exist regarding the mechanism of its action. Mounting evidence shows that it can induce ferroptosis in host cells [4]. This mechanism, obviously, should involve direct killing of host cells and therefore could be called “invasion by destruction”. However, there are other experimental data which suggest another mechanism. Morello et al. [5] revealed that *P. aeruginosa* LOX might participate in the synthesis of anti-inflammatory oxylipins host's PUFAs, which leads to the suppression of inflammation and invasion facilitation. This mechanism could be called “invasion by deception”. It is still unclear which of these mechanisms has the highest contribution to the invasion success in *P. aeruginosa* infection.

In the first article of our project [6], we found bioinformatic evidence that *P. aeruginosa* could be just the tip of the iceberg — only one of the plenty of bacteria using LOXs to establish host-microbe interactions. This enzyme might be also associated with a broad range of hosts and ecological functions — from plant symbiosis to human pathogenesis. This finding increased the possibility of the “invasion by deception” hypothesis — immune system suppression is more likely in symbiosis than direct destruction and killing of a host's cells and tissues.

We further elaborated this finding in our preprint [7], where we presented additional computational evidence for the involvement of LOXs in cross-kingdom host jumps and outlined a possible biochemical explanation for it (Figure 1). It is a generalization of a model by Morello et al. [5] where *P. aeruginosa* LOX oxidizes arachidonic acid to its normal products (such as 15-HETE) which are, in turn, transformed to lipoxin A<sub>4</sub> and other pro-resolving mediators in human leukocytes. We tried to infer the mechanism of action of LOXs with the same regio- and stereospecificity in plants and assumed that the similar mechanism, involving the bacterial LOX and the downstream enzymes in the host's cells, would lead to the production of jasmonates — which are known to undermine the host's immune defense in the case of their overproduction and imbalance between jasmonates and salicylates [8–11].



**Figure 1.** A possible common mechanism of immune response evasion by LOX-carrying bacteria in plants and vertebrates.  $(\omega-6)S$ -LOXs transform arachidonic acid into 15S-HpETE, which is normal precursor of lipoxin A<sub>4</sub> in humans and could be transformed into it by human downstream enzymes. In plants, bacterial lipoxygenase

with the same specificity converts linolenic acid into 13S-HpOT, which is normal precursor of jasmonates. The both classes of compounds could suppress the particular defense responses in the corresponding organisms being overproduced. *Image credit: Georgy Kurakin, bioRxiv, 2022-06 [7], CC-BY-NC 4.0. Created with BioRender.com.*

This preprint was not published due to multiple rejections without review and negative reviews in the remaining journals/peer review services. We analyzed the major criticisms in our blog post on *Springer Nature Research Communities* [12]. Of them, the most pressing criticism is the lack of a negative or a positive control in our bioinformatic analysis. It is almost impossible to address this criticism fully because the grounds and conditions of the assignment of such control are unclear. However, we could compare the bioinformatic results for LOX and any other marker for which the involvement in host-microbe interactions is suggested.

CFTR inhibitory factor (Cif) had been earlier characterized as an epoxide hydrolase disrupting CFTR (cystic fibrosis transmembrane conductance regulator) trafficking by interfering into ubiquitination mechanisms. However, a recent article shows that it also cleaves epoxy-fatty acids — oxylipins formed by epoxygenase pathway in humans — and thus might interfere with immune signaling [13]. Moreover, it was also experimentally characterized in *P. aeruginosa* [14] and in *Acinetobacter nosocomialis* [15] — another nosocomial pathogen. An oxylipin-cleaving virulence factor is of particular interest for the comparison with LOX in a bioinformatic study.

Thus, we decided to identify the list of potential host-associated bacteria harboring Cif — like we did for LOX previously — and compare their ecological characteristics with those of bacteria harboring LOXs.

## Materials and Methods

For LOX, we reused list of bacteria from our preprint [7]. In turn, this list was obtained by repetitive BLAST searches using LOXs identified in our paper in *Biochemistry (Moscow)* [6] as search queries. For these searches, we used UniProt BLAST (against UniProt KB) and NCBI BLAST (against Non-redundant protein sequences in the NCBI database) with E-value threshold of 0.01 in all cases and taxonomical restriction of the searches to bacteria.

We performed targeted selection of search results where LOX belonging to host-associated bacteria were appeared in groups in the top of hits list, and downloaded the top part of the hit set to include these LOX of host-associated bacteria and the nearest group of cyanobacterial or myxobacterial LOXs as an outgroup. Thus, we obtained four FASTA files containing sequences of LOXs of host-associated bacteria. These sequences were used for phylogenetic analysis and for additional statistical analysis, while their names were used for assembling the list of bacteria having LOXs (hereinafter referred to as “LOX list”). This list has been reused for the current paper and used for the further network text analysis in the preprint [7].

For the current research, we used the extended version of the LOX list, which includes the bacteria with exotic hosts which had been used only for phylogenetic/binding site analysis in the preprint itself and had not been included in the main list for linguistic analysis within the preprint. The resulting extended LOX list comprised 40 bacterial species and is shown in the **Table 1**. This list was subjected to systematic literature review to obtain ecological characteristics of bacteria and perform statistical analysis of them. For the LOX list, the literature review was mostly based on the PubMed-indexed sources identified in the preprint [7] with the exclusion of additional sources mentioned and cited below.

**Table 1.** Bacterial species of the LOX list by orders, reused from our previous preprint [7]. Order names corrected as per current taxonomy.

Order	Species
Burkholderiales	<i>Variovorax paradoxus</i> , <i>Variovorax guangxiensis</i> , <i>Variovorax gossypii</i> , <i>Burkholderia gladioli</i> , <i>Burkholderia singularis</i> , <i>Burkholderia thailandensis</i> , <i>Burkholderia stagnalis</i>
Mycobacteriales	<i>Nocardia seriolae</i> , <i>Nocardia pseudobrasiliensis</i> , <i>Nocardia brasiliensis</i> , <i>Mycobacteroides abscessus</i> , <i>Rhodococcus erythropolis</i> , <i>Rhodococcus sp. 66b</i>
Enterobacterales	<i>Pluralibacter gergoviae</i> , <i>Kosakonia sp. AG348</i> , <i>Kosakonia sacchari</i> , <i>Enterobacter hormaechei</i> , <i>Enterococcus faecium</i> , <i>Cedecea lapagei</i> , <i>Pantoea sp. OXWO6B1</i> , <i>Pantoea ananas</i> , <i>Moellerella wisconsensis</i> , <i>Dickeya zeae</i>
Alteromonadales	<i>Shewanella waksmanii</i> , <i>Colwellia echini</i> , <i>Algibacillus agarilyticus</i>
Holosporales	<i>Candidatus Finniella inopinata</i>
Nitrospinae/ Tectomicrobia group	<i>Candidatus Entotheonella palauensis</i>
Oceanospirillales	<i>Gynuella sunshinyii</i> , <i>Endozoicomonas numazuensis</i> , <i>Aliikangiella coralliicola</i>
Oligoflexales	<i>Pseudobacteriovorax antillogorgiicola</i>
Pseudomonadales	<i>Pseudomonas aeruginosa</i>
Pseudonocardiales	<i>Kutzneria sp. 744</i> , <i>Pseudonocardia acaciae</i> , <i>Lentzea kentuckyensis</i>
Vibrionales	<i>Vibrio penaeicida</i> , <i>Enterovibrio norvegicus</i> , <i>Enterovibrio coralii</i> , <i>Enterovibrio calviensis</i> , <i>Enterovibrio nigricans</i>

For Cif, we collected an extra dataset. We used BLAST search on the UniProt platform starting from *Pseudomonas aeruginosa* Cif (accession ID: Q9HZR3\_PSEAE) as a query. The search was performed against the whole UniProt KB without taxonomical restrictions with the E-value threshold of 0.01. 1000 resulting sequences were aligned using MAFFT-online (<https://mafft.cbrc.jp/alignment/server/index.html>) [16] with concomitant run of MaxAlign [17] to increase the number of gap-free sites. The resulting multiple sequence alignment was subjected to phylogenetic analysis using MAFFT-online itself (for neighbor joining method) and MEGA X [18] (for

minimum evolution method). For both methods, bootstrap support calculations were used with 500 replications.

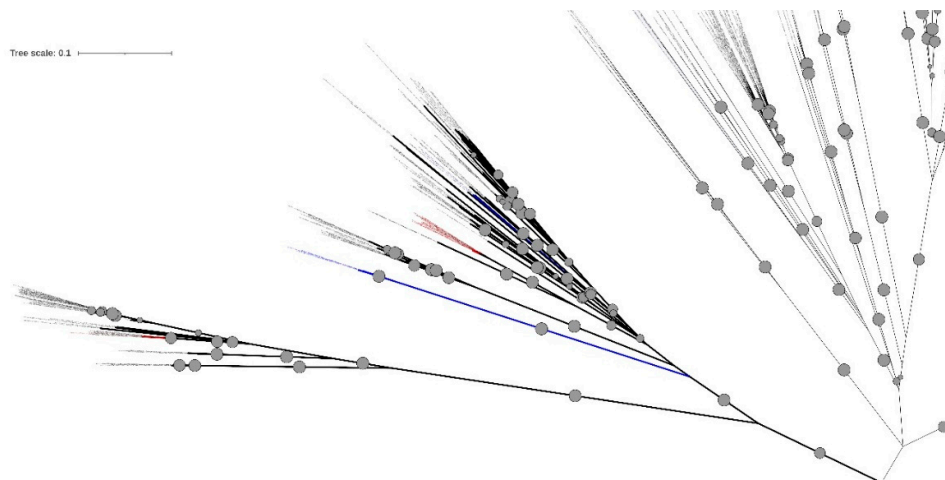
Construction of two trees was a check for the correctness of the phylogeny and also a chance to map different kinds of data onto them for visualization. We visualized phylogenetic trees with iTOL [19].

The list of bacteria obtained from phylogenetics (see Results and Discussion section below) was also subjected for systematic literature review, as well as the LOX list.

The ecological traits of bacteria carrying LOX and Cif have been compared by the means of statistical analysis in MS Excel 2019, PAST [20], and Google Colab using Python with *statsmodels* library.

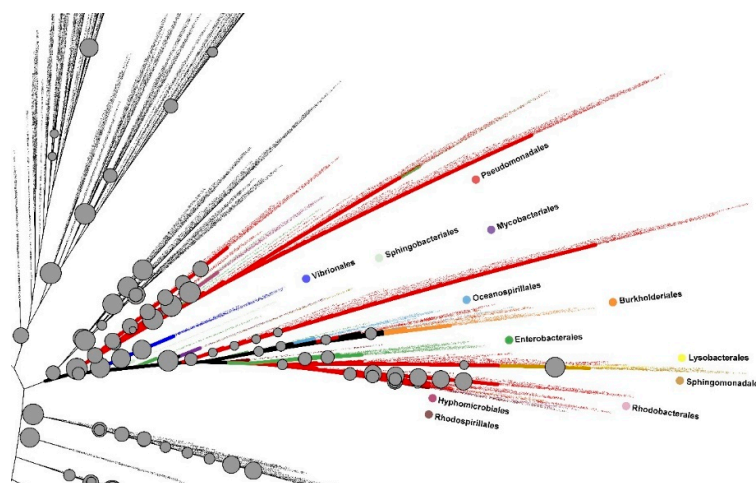
## Results and Discussion

The both obtained trees revealed a cluster comprising 135 leaves where both *Pseudomonas aeruginosa* Cif and *Acinetobacter baumannii* Cif were located (Figure 2). The last common ancestor of their Cif's was also the last common ancestor of all this cluster, and this fact led to the assumption that this common ancestor was already a functional Cif, and all protein in this cluster (hereinafter referred to as "putative Cif cluster") are functional Cif's. The alternative scenario is that some biological functions characteristic for Cif's could be acquired by *Pseudomonas aeruginosa* and *Acinetobacter baumannii* convergently. However, convergent acquisition of epoxide hydrolase activity is still highly improbable in these settings — thus, even in this best-case scenario, "putative Cif cluster" could be considered to be a cluster of closely related epoxide hydrolases, some of which exert Cif inhibiting properties.



**Figure 2.** The fragment of the minimum evolution tree of Cif homologs. Thickened branches indicate the putative Cif cluster, rounds indicate bootstrap values more than 0.7. Red branches show experimentally characterized Cif's of *Pseudomonas aeruginosa* and *Acinetobacter baumannii*, blue branches — groups of bacteria associated with aquatic invertebrates. This colour coding graphically shows that all this cluster consist of putative Cif's — and the horizontal Cif gene transfers to aquatic-associated bacteria are relatively rare. Created in iTOL.

The most basal branches of this cluster belonged to Pseudomonadales order which suggests that all this cluster had pseudomonadal origin. However, the cluster also contained multiple intercalations of Cif's belonging to bacteria from other orders, such as Enterobacterales, Vibrionales, Hyphomicrobiales, Sphingomonadales, Burkholderiales (Figure 3, Supplementary Figure). Different representatives of these orders occupied different places in the cluster, which clearly points to multiple horizontal gene transfers. This resembles the case of lipoxygenases we described in our preprint [7]



**Figure 3.** The fragment of the neighbor joining tree of Cif homologs. Thickened branches indicate the putative Cif cluster, rounds indicate bootstrap values more than 0.7. Each order is colored in their own color, each color code explanation label is located near the biggest cluster formed by the enzymes of bacteria in the respective order. The uneven coloring of this cluster indicates multiple inter-order horizontal Cif gene transfers. The full colored high resolution image of this tree is available as **Supplementary Figure**. Created in iTOL.

We analyzed the full list of bacterial species whose Cif's belonged to this cluster. More than a half of these bacteria were uncharacterized from the ecological point of view. From characterized bacteria, we selected host-associated bacteria (to balance the methodology between this research and the LOX dataset from our preprint [7] which was also restricted to host-associated bacteria. We obtained the resulting list of 32 species (hereinafter referred to as "Cif list"), whose taxonomical breakdown largely overlapped with the taxonomical composition of the LOX list (**Table 2**). However, the species-level overlap between the LOX list and Cif list was relatively subtle and only amounted to two species — *Pseudomonas aeruginosa* and *Burkholderia gladioli*.

We then performed systematic literature review on them by performing search of articles in peer-reviewed journals that mentioned ecophysiological traits of the respective bacteria. We used PubMed and Google search with the names of the respective bacteria as the search queries. In total, we obtained 99 literature sources for the bacteria from the Cif list [21–117]. We also used additional references for some LOX-carrying bacteria to address the criticisms for our previous analysis obtained from colleagues in reviews and personal communications [118–123]. Methodology of the search in this case was the same as for the bacteria from the Cif list.

**Table 2.** Bacterial species of the Cif list by orders. *Pseudomonas amygdali* and *Pseudomonas syringae* are counted as the strains of the same species because technically they are the same species (like *Escherichia coli* and *Shigella dysenteriae*) and have the same ecological traits in our dataset. Counting them as separate species could lead to overestimation of some statistics in favor of our hypothesis.

Order	Species
Pseudomonadales	<i>Acinetobacter lactucae</i> , <i>Acinetobacter oleivorans</i> , <i>Acinetobacter baumannii</i> , <i>Acinetobacter seifertii</i> , <i>Acinetobacter nosocomialis</i> , <i>Acinetobacter cumulans</i> , <i>Pseudomonas fluorescens</i> , <i>Pseudomonas aeruginosa</i> , <i>Pseudomonas</i> sp. FDAARGOS, <i>Pseudomonas putida</i> , <i>Pseudomonas daroniae</i> , <i>Pseudomonas frederiksbergensis</i> , <i>Pseudomonas amygdali</i> – <i>Pseudomonas syringae</i> complex (counted as 1 species), <i>Pseudomonas poae</i> , <i>Pseudomonas taetrolens</i> , <i>Pseudomonas brassicacearum</i>
Sphingomonadales	<i>Novosphingobium</i> sp. P6W, <i>Sphingomonas</i> sp. RIT328
Vibrionales	<i>Vibrio</i> sp. F13, <i>Vibrio crassostreae</i> , <i>Vibrio gigantis</i>
Enterobacterales	<i>Klebsiella oxytoca</i> , <i>Sodalis praecaptivus</i> , <i>Erwinia typographi</i> , <i>Pantoea dispersa</i>

Burkholderiales	<i>Burkholderia gladioli</i> , <i>Paraburkholderia terricola</i>
Hyphomicrobiales	<i>Ochrobactrum</i> sp. LCB8 ( <i>Ochrobactrum teleogrylli</i> ), <i>Phyllobacterium phragmitis</i>
Oceanospirillales	<i>Alcanivorax dieselolei</i>
Mycobacteriales	<i>Mycolicibacterium iranicum</i>
Rhodobacterales	<i>Paracoccus liaowanqingii</i>

We studied the literature sources on these bacteria listed above and manually created the list of their characteristic ecological traits. For each bacterial species, we chose the traits to cover the following aspects of the bacterial ecophysiology:

- clinical forms (for affecting humans),
- host's taxonomic group and habitat,
- effect on the host,
- localization within the host's organism,
- environmental effect,
- public health effect.

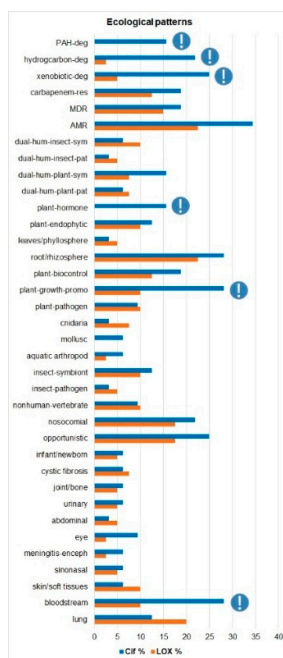
We listed the traits based on the availability of their descriptions in literature, which resulted in different sets of traits for different host groups (for example, for insects and plants). The resulting list of the traits (abbreviations in our data analysis and meaning) is provided in the **Table 3**.

**Table 3.** Key ecological traits of bacteria in LOX and Cif groups: their abbreviation in our data analysis and meaning with the particular emphasis on the selection criteria upon systematic literature review.

Trait abbreviation	Meaning and criteria
<i>lung</i>	Affecting lungs in humans (e.g. causing pneumonia)
<i>bloodstream</i>	Bloodstream infections in humans, including sepsis
<i>skin/soft tissues</i>	Infections of skins and soft tissues in humans
<i>sinonasal</i>	Sinonasal infections in humans
<i>meningitis-enceph</i>	Affecting brain or brain membranes in humans
<i>eye</i>	Eye infections in humans
<i>abdominal</i>	Infections of abdominal cavity (e.g. peritonitis) or digestive system in humans
<i>urinary</i>	Urinary tract infections in humans
<i>joint/bone</i>	Joint or bone infections in humans
<i>cystic fibrosis</i>	Affecting patients with cystic fibrosis
<i>infant/newborn</i>	Infections in human infants/newborns
<i>opportunistic</i>	Opportunistic infections in humans, including affecting immunocompromised patients
<i>nosocomial</i>	Infections or presence in nosocomial environment
<i>nonhuman-vertebrate</i>	Association with non-human vertebrates
<i>insect-pathogen</i>	Insect pathogen
<i>insect-symbiont</i>	Insect symbiont
<i>aquatic arthropod</i>	Association with aquatic arthropods
<i>mollusc</i>	Association with molluscs
<i>cnidaria</i>	Association with cnidarians
<i>plant-pathogen</i>	Plant pathogen
<i>plant-growth-promo</i>	Plant growth promotion

<i>plant-biocontrol</i>	Biocontrol functions in plants, plant pathogen suppression
<i>root/rhizosphere</i>	Localization in plant roots/rhizosphere
<i>leaves/phyllosphere</i>	Localization in plant leaves/phyllosphere
<i>plant-endophytic</i>	Endophytic growth in plants
<i>plant-hormone</i>	Synthesis or degradation of plant hormones which affects hormonal signaling of a plant host
<i>dual-hum-plant-pat</i>	Dual function: affecting humans and plant pathogenesis
<i>dual-hum-plant-sym</i>	Dual function: affecting humans and plant symbiosis
<i>dual-hum-insect-pat</i>	Dual function: affecting plants and insect pathogenesis
<i>dual-hum-insect-sym</i>	Dual function: affecting humans and insect symbiosis
AMR	Antimicrobial resistance
MDR	Multi-drug resistance
<i>carbapenem-res</i>	Carbapenem resistance
<i>xenobiotic-deg</i>	Xenobiotic degradation
<i>hydrocarbon-deg</i>	Hydrocarbon degradation
<i>PAH-deg</i>	Polycyclic aromatic hydrocarbon (PAH) degradation

Both LOX carriers and Cif carriers were associated with a wide range of hosts, so a virtual average LOX- or Cif-carrying bacterium should be a generalist. Regarding humans, the ability to cause nosocomial and opportunistic infections was prevalent among them, with lung, bloodstream, and skin/soft tissue infections being the most prevalent forms (Figure 4). However, bloodstream infections were notably more frequent in Cif carriers than in LOX carriers (28,125% vs. 10%, correspondingly). Antimicrobial resistance (AMR) and multiple drug resistance (MDR) were frequent in both groups, including carbapenem resistance, which could raise public health concerns. Neither LOX list nor Cif list contained human symbionts.



**Figure 4.** Bar plot of per cent prevalences of key ecological trait of bacteria in LOX and Cif groups, compared. Exclamation mark stamps designate the traits with the highest difference between the LOX list and the Cif list (p-value < 0.05).

Affecting cystic fibrosis patients was not the most prevalent feature in the both groups (3 in LOX group, 2 in Cif group). Moreover, this trait prevalence was mostly contributed to by the same bacteria (*P. aeruginosa* and *Burkholderia gladioli*) which were present in the both groups. Cif is a virulence factor in cystic fibrosis is biologically plausible due to its ability to downregulate CFTR; LOX might potentiate its action given its association with lung infections in a wider range of pathogens. Thus, LOX could be not selective towards cystic fibrosis patients (this selectivity could be assumed from our preprint [7]). Instead, Cif could be responsible for this selectivity (which has a good explanation in terms of its molecular functions), while LOX could just have more general association with opportunistic lung infections.

In plants, both LOX and Cif carriers predominantly colonized roots/rhizosphere and were beneficial for plants (biocontrol or growth promoting) rather than plant pathogens. However, Cif carriers were growth promoters more frequently than LOX carriers (28.125% vs. 10%, correspondingly). Only Cif carriers were often characterized in literature as plant hormone producers or degraders (15,625%), none of LOX carriers exerted this function. 2 of 5 Cif-carrying plant hormone hijackers (*A. baumannii* and *Pantoea dispersa*) produced indole acetic acid (IAA, auxin) [45,72], one species complex (*Pseudomonas amygdali* – *Pseudomonas syringae* complex) produces a jasmonate mimic coronatine [87,91,92], one species (*Novosphingobium* sp. P6W) degrades abscisic acid [96,97]. One species (*Acinetobacter oleivorans*) produced cytokinin zeatin, jasmonate, and abscisic acid [25].

Coincidence of root/rhizosphere localization and growth promotion and high occurrences of the both traits in the bacteria of both LOX and Cif lists suggests that one of the normal ecological functions of LOX/Cif carrier in plants is to be a plant growth promoting rhizobacterium.

Additional ecological function characteristic for plant-associated LOX-or Cif-carrying bacteria is plant pathogenesis. In the both groups, some plant symbionts and plant pathogens showed the ability to affect humans. The same is true for insect symbionts and insect pathogens. Like in the case of LOXs, Cif is associated with pathogenic and symbiotic functions in plants and invertebrates, but only with pathogenesis in humans. This fact suggests that they could share the common target in the hosts' organisms.

One more important fact is the absence of coincidence of association with aquatic invertebrates and affecting humans in both LOX and Cif groups. In the preprint on lipoxygenase [7], we suggested the presence of a biochemical barrier between plants and vertebrates, on one side, and water invertebrates, on the other side. It could be represented by different metrics of the ligand binding site of an enzyme. An alternative explanation is possible: maybe this pattern is caused only by the physical barrier between terrestrial and aquatic environments. However, epidemiology of *Vibrio* infections in humans shows that this physical barrier is not reliable enough to prevent contact and infection. In contrast, in both LOX and Cif lists, no human-affecting vibrios are found, despite the presence of vibrios in the both sets. This fact does not allow to rule out the possibility that LOX and Cif most have peculiar structure to fit the aquatic invertebrate hosts – probably cnidarians or aquatic arthropods which can be colonized by bacteria from the both groups.

In the Cif list, bacteria were more frequently prone to degrade environmental xenobiotics (25%), predominantly polycyclic aromatic hydrocarbons (PAH) (15,63%). In contrast, LOX list had only 5% of xenobiotic degraders with 0% of confirmed degraders.

Six aforementioned traits (bloodstream infections, plant growth promotion, plant hormone production, xenobiotic degradation, hydrocarbon degradation, PAH degradation) differ from other 30 traits in terms of statistics: two proportions Z-test shows  $p$ -value  $< 0.05$ , while other traits have  $p$ -value  $> 0.05$ . This underscores that these traits reflect the largest differences between LOX list and Cif list in numerical terms. However,  $p$ -value  $< 0.05$  is not sufficient for a statistical conclusion due to multiple comparison of 36 traits.

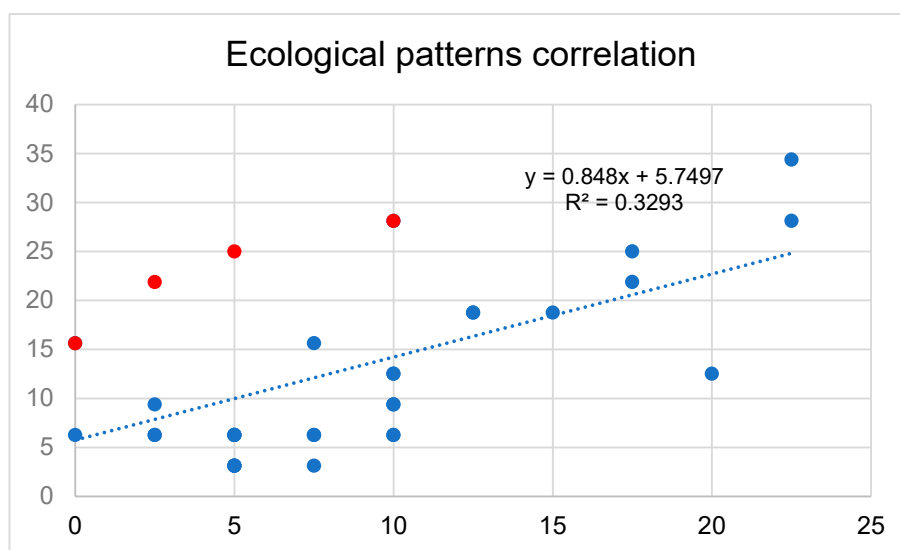
The lowest  $p$ -value of 0.009 corresponded to PAH degradation trait, while the  $p$ -value threshold for significant difference is calculated as  $0.05 / 36 = 0.0014$ . Thus, two proportions Z-test is scantily conclusive in this case; it allows only to flag the traits with the most extent of difference without drawing any conclusion by this method only.

We decided to explore the trait statistics by linear regression analysis, which allowed us to avoid multiple comparisons problem. Here, the whole set of traits is treated by a single test iteration; any trait is treated as a dot on a plane with coordinates  $(x,y)$ , where  $x$  = per cent occurrence of a trait in the LOX list,  $y$  = per cent occurrence of a trait in the Cif list.

We treated each ecological trait as an enumeration unit and calculated:

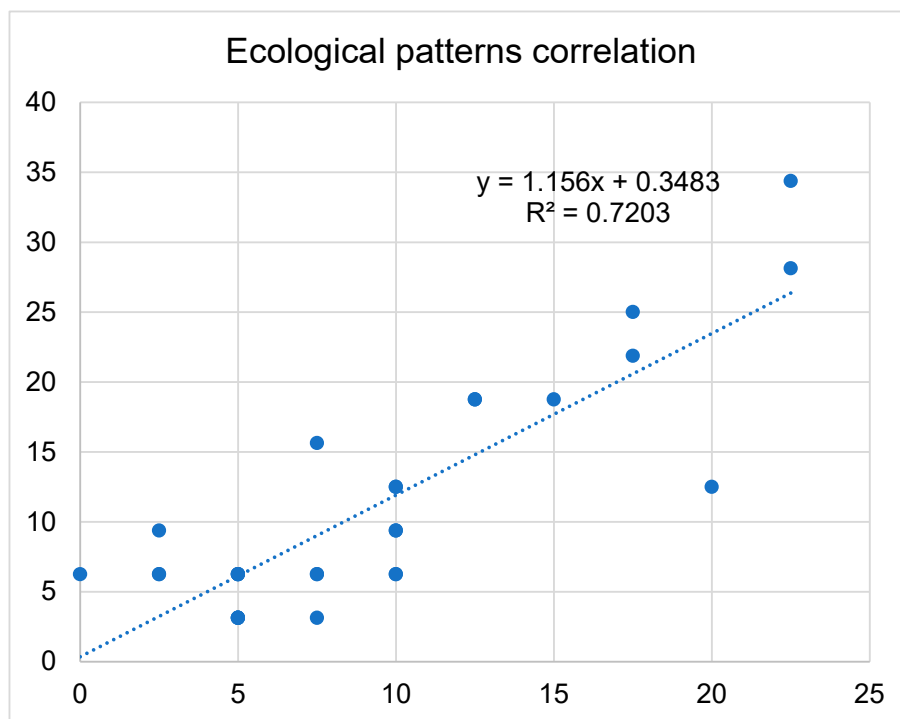
- correlation between trait prevalences in Cif and LOX groups,
- linear regression of trait prevalences in Cif group on LOX trait prevalences in LOX group.

With the whole set of traits, correlation was only moderate (correlation coefficient = 0.57), and approximation was relatively poor with  $R^2 = 0.33$  (Figure 5). Graphically, we noted a group of traits that formed a “humpback” on the scatterplot and worsened the correlation — despite not being obvious outliers (highlighted on Figure 5). They corresponded to aforementioned traits with high difference flagged by two proportions Z-test: plant hormone hijacking, plant growth promotion, bloodstream infections, and the group of traits related to xenobiotic degradation.



**Figure 5.** Scatter plot of the correlation of per cent prevalences of key ecological trait of bacteria in LOX and Cif groups, with linear regression.  $y$  axis — prevalences in the Cif group,  $x$  axis — prevalences in the LOX group. Off-trend dots (upper left) are highlighted.

After the removal of these 6 traits, the correlation improved drastically (correlation coefficient = 0.85) and became strong positive (Figure 6). The slope and the intercept of the regression equation were close to 1 and 0, correspondingly, with 95% bootstrapped confidence interval for slope covering 1 (0.83, 1.6) and 95% confidence interval for intercept covering 0 (-2.57, 3.6).



**Figure 6.** Scatter plot of the correlation of per cent prevalences of key ecological trait of bacteria in LOX and Cif groups, with linear regression.  $y$  axis — prevalences in the Cif group,  $x$  axis — prevalences in the LOX group. Off-trend dots are removed (and the corresponding traits in the dataset), which drastically improved the regression.

This allows to round slope to 1 and intercept to 0, thus obtaining correlation formula  $y = x$ , or “*trait prevalence in the Cif group*” = “*trait prevalence in the LOX group*”. Upon this rounding, the determination coefficient  $R^2$  decreases down to 0.66, which is equitable to 0.72 and still represents a moderately strong regression. The rounded formula, in turn, indicates that LOX-carrying bacteria and Cif-carrying bacteria have very similar ecological profiles — with the exception of six traits we removed before.

Such similarity increases the probability of the common mechanism in LOX and Cif interactions with the hosts. These two enzymes are quite different from the enzymological perspective: LOX is a fatty acid dioxygenase, while Cif is an epoxide hydrolase. However, their action on oxylipins could explain the similarity in the ecological profiles of LOX carriers and Cif carriers. The off-trend ecological traits could provide additional insights into this common mechanism.

Only three of these six removed traits reflect aspects of host-microbe interactions, and two of them are linked to plants: (1) plant hormone biosynthesis and (2) plant growth promotion. Moreover, these two traits are connected: the ability to synthesize plant hormones might make bacteria better plant growth promoters — given the fact that the most of hormone hijackers in the Cif group produce auxins or cytokinins — plant hormones with powerful growth-promoting action. Thus, all detected differences between LOX carriers and Cif carriers regarding plant-microbe interactions could be reduced to the ability to produce plant hormones.

This ability has not been documented for LOX carriers, but is relatively prevalent in Cif carriers. This could be one more indirect confirmation of our model of lipoxygenase action in cross-kingdom host jumps shown on the Figure 1.

LOX-carrying bacteria cannot produce plant hormones themselves — they have only LOX and even don't have the source of fatty acids which are its substrates. But within a plant hosts, they act as host-dependent jasmonate producers, obtaining fatty acids from the host and exploiting its downstream enzymes to complement their LOX activity. Thus, LOX-carrying bacteria are jasmonate producers “by design”. Jasmonates are potent plant hormones themselves and could suppress plant's defense responses in overproduction [8–11]. This explains why LOX-carrying bacteria did not acquire

the ability to synthesize other hormones, such as IAA or zeatin — jasmonates are potent enough to facilitate the plant colonization, and LOX-carrying bacteria have no biological grounds to be altruists with good growth-promoting properties.

In contrast, Cif predominantly interact with epoxygenase-derived oxylipins both in plants and in humans. Here, we come on shaky grounds because plant epoxygenases have been described only recently [124,125], and we know almost nothing about downstream enzymes. However, we could suggest that epoxygenase pathway in plants does not produce potent hormones and has rather local action. This creates selective pressure for Cif-carrying bacteria to acquire additional hormone producing ability to communicate with the plant hosts and manipulate them.

In this regard, it is important fact that the only bacterial species complex that produces a jasmonate mimic in the Cif group (*P. amygdali* – *P. syringae* complex) is a plant pathogen, not a growth promoting bacterium. If we revisited the Cif group and mark *P. aeruginosa* and *B. gladioli* — dual Cif/LOX producers — as jasmonate producers, then the association would be clearly visible: jasmonate producers are predominantly plant pathogens, IAA producers tend to be growth-promoting bacteria.

This indirectly confirms that LOX in plant-associated bacteria might be used for jasmonate signaling hijack. This, in turn, indirectly confirms our model of LOX-powered cross-kingdom host jumps which we suggested earlier in our preprint [7] and showed here on Figure 11. The data from the current study show that Cif could be also involved in these cross-kingdom host jumps by interfering with oxylipin signaling both in plants and humans. In plants and insects, which do not have full-fledged adaptive immunity, this could lead both to pathogenesis and symbiosis. In humans with their adaptive immunity, spoofing LOX signaling would not help bacteria to evade antibody and T-cell response for a long time — thus LOX- and Cif- carriers are predominantly opportunistic pathogens.

Only one discrepancy which does not fit in this explanation so far is the larger prevalence of bloodstream infections caused by Cif carriers. On one hand, this could be an accidental difference without any significance — in multiple comparison such differences are almost inevitable. While the other off-trend features could be analyzed *en bloc* and were connected — which decreases the probability that they are accidental — we cannot draw robust conclusions on one outlier.

On the other hand, clinically relevant bloodstream infections are almost always associated with systemic inflammatory response syndrome (SIRS). Its magnitude and incidence might be directly linked to the peculiarities in suppressing immune response by pathogens — however, given scarce data on the role of epoxygenase pathway, pathophysiological explanation is still pending.

A recent article in *Science* [126] provides additional evidence for the association between LOX and Cif on the strain level in *Pseudomonas aeruginosa* and corroborates our findings outlined above. The differential expressions of LOX and Cif (found in the supplementary data) have comparable direction and magnitude: in strains which are prone to affect cystic fibrosis patients, expressions are about 1.5-2 folds lower than in non-cystic fibrosis strains, but with a poor statistical support. Taken in complex with our data, these differential expressions show that Cif and LOX can act together in host-microbe relationship and exploit a common pathway in a host's organism.

The discrepancy in xenobiotic degradation between Cif and LOX groups shows that xenobiotic oxidation is at least not characteristic for bacterial lipoxygenases in real ecosystems. Human lipoxygenases were earlier characterized as enzymes oxidizing some xenobiotics as co-substrates [127] — and there was a possibility that bacteria could use lipoxygenases this way. However, now we can state that at this moment, we have no data for detoxicating functions of bacterial lipoxygenases — and the available data point at cell-to-cell signaling functions of lipoxygenase and oxylipins even in bacteria, like in our previous papers [6,7]. The causes and nature of association between Cif and xenobiotic degradations are expected to become clear later — upon accumulation of experimental data on Cif enzymology.

## Conclusions

Putative Cif proteins — being epoxide hydrolases — are present in host-associated bacteria with an ecological profile extremely similar to those of LOX carriers. This suggests that the both enzymes could be used by bacteria to expand their host ranges by interfering a host's oxylipin signaling. This

underscores the importance of oxylipin signaling as a universal immune signaling system across all multicellular eukaryotes, from plants to insects and vertebrates. This could be a universal “backdoor” for immune evasion by pathogens — and further evolutionary studies are needed to explore the origins of this universal vulnerability.

By comparing the LOX data and the Cif data, we found additional evidence in support of our hypothesis on the common mechanism of immune response suppression by oxylipin-spoofing pathogens in plants and humans. Hydroperoxides produced by bacterial lipoxygenases (from a host’s fatty acids) are likely to be further transformed into jasmonates in plants and into specialized pro-resolving mediators, including lipoxins, in humans. This biochemical effect might create a common mechanism of affecting plants and humans.

This host-dependent mechanism of producing bioactive compounds to evade the host’s immune responses requires precise and elaborate experiments to be confirmed for a wide range of bacteria proposed by us and for a wide range of hosts. Until we don’t have an experimental pipeline to detect excessive oxylipin production in a wide range of host-pathogen systems, bioinformatic data are especially valuable, despite their limited reliability.

New insights could be also provided by clinical data. High prevalence of opportunistic and nosocomial infections in both Cif carriers and LOX carriers raise the concerns for their importance for public health and necessity of molecular epidemiology surveillance for them — which could provide not only additional safety for inpatients worldwide, but also new data on the association of LOX and Cif with various clinical conditions.

**Supplementary Materials:** The following supporting information can be downloaded at the website of this paper posted on Preprints.org. The full image of our neighbor joining phylogenetic tree in ultra-high definition (600 dpi) is available as the **Supplementary Figure**. The interactive versions of the phylogenetic trees of Cif homologues used in this paper can be found on the public iTOL profile of the author: <https://itol.embl.de/shared/GeorgyKurakin>. The trees are located in the project titled “Cif project”: • the neighbor joining tree: “cif\_pseai1000\_NHX.nh”; • the minimum evolution tree: “cif\_pseai1000\_MinEv.nwk”.

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## Abbreviations

CFTR — cystic fibrosis transmembrane conductance regulator, Cif — CFTR inhibitory factor, LOX — lipoxygenase, IAA — indole acetic acid

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