Supporting Information

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SI Text

Detailed Theoretical Model Presentation and Analysis. Our model tracks the dynamics of three distinct patch types characterized by their occupants, empty (with prevalence E), cooperator only (with prevalence C), and defector only (with prevalence D), building on the metapopulation models by Levins (1, 2) describing the dynamics of subpopulations within a metapopulation. A schematic diagram of our model is presented in Fig. S1. We assume that defectors always displace cooperators if they co-occur within a patch (i.e., the patches are well-mixed and the cooperative provision of the public good entails a direct cost, ensuring a local tragedy of the commons) (3, 4) and that the dynamics of strain replacement are sufficiently fast that we can reduce within-patch dynamics to simple transition rates among the single-strain states. Our model tracks three classes of transition events: colonization of empty patches (by cooperators), extinction of occupied patches (at a higher rate for defector patches), and replacement of cooperators by defectors (because of both transmission of defectors and de novo mutation).

These transition events define the following system of ordinary differential equations controlled by cooperator and defector transmission rates c and d, patch extinction rate e, cooperator to defector mutation and replacement rate m, and public good decay rate u (Eqs. **S1–S4**):

$$\mathrm{d}E/\mathrm{d}t = e(C+D) - cCE + uD \qquad \textbf{[S1]}$$

$$\mathrm{d}C/\mathrm{d}t = cCE - eC - (dD+m)C \qquad \textbf{[S2]}$$

$$\mathrm{d}D/\mathrm{d}t = (dD+m)C - (u+e)D \qquad \textbf{[S3]}$$

$$E + C + D = 1. \qquad \textbf{[S4]}$$

When $d = m = u = D = 0$, we recover the classic metapopulation model by Levins (2), with the prevalence of C dictated by a colonization–extinction balance, tending to $\{E^* = e/c, C^* = 1 - e/c\}$ if $c > e$. Defector patches D can be generated by two processes, either through the de novo generation and spread of defector mutants in a cooperator patch (at rate mC) or through the transmission of defectors to a cooperator patch from existing defector patches (at rate dDC), being able to supplant cooperators because of the within-patch advantage of noncontribution to the public good. Defector patches are, in contrast, unable to effectively colonize empty patches because of their lack of public goods construction and are also more vulnerable to extinction, because they must rely on public goods inherited from their cooperator predecessors within a patch. The additional extinction rate per defector patch is $e + u$, where $1/u$ can be viewed as a measure of the half-life of the essential public good created by cooperators; if u is large, the public good rapidly degenerates after loss of cooperators, and defectors have only a brief tenancy before they find their patch inhospitable and the patch returns to state E. In contrast, if u is small, the public good is durable, and defectors can persist effectively as long as cooperators themselves (if $u << e$, where e can be viewed as a baseline patch disturbance rate). Maintenance of cooperation in this system can be understood as the result of high relatedness combined with global competition (5–7), with high relatedness ensured by the monomorphic patch assumption and global competition ensured by complete dispersal of individuals among patches.

A stability analysis (8) of Eqs. S1–S4 reveals that coexistence of all three patch types is inevitable when $c > e + m$ [i.e., when the rate of cooperator colonization outweighs the rate of cooperator loss (to empty or defector patches)]. Note that the coexistence of cooperators and defectors can be understood as the result of strong selection on the social trait (9). If the condition $c > e + m$ fails, then the only stable state is pure empty patches. In the limit of no within-patch mutation ($m -> 0$), coexistence of all three patch types is assured if $c > e$ and $d(1 - e/c) > u + e$, with the proportion of cooperative patches at equilibrium being $C^* = (e + u)/d$. From this expression, it is clear that greater durability (lower u) results in a loss of market share for cooperators (i.e., $dC^*/du > 0$, illustrated by solid line in Fig. 1*A*), implying that selection will favor more fragile public goods up to a lower limit for fragility given by constraints on immediate functionality of the public good.

The above model is consistent with a constitutive expression of a public goods trait. Under this model, any variation in public goods design (u) will result in a change in the equilibrium density of the public good in a patch of cooperators but no change in the per capita effort of cooperators (yielding a constant vulnerability to cheat replacement, represented by d and m). Conversely, given an appropriate regulation of public goods production, changing u would yield a constant public goods density and changing costs of production (i.e., a switch from effort constancy to outcome constancy). To introduce facultative production of a public good, we consider that the rates of within-patch cooperator replacement by defectors (whether arising from spontaneous mutation or colonization) will decrease for more durable public goods, because the costs of production (driving cheat replacement) will only be paid intermittently. Specifically, we assume that d and m are positive functions of u, and therefore, fragile public goods ensure a high (i.e., constant) production cost and subsequently, high rates of replacement by cheats. In Fig. 1*B*, we specify that $d = d_0 u$ and $m = m_0 u$, ensuring coexistence whenever $c > e + m_0 u$. Under this model of perfect outcome constancy (doubling the durability allows a halving of expenditure to ensure the same equilibrium density of public good), $C^* = (e + u)/du$ (again, in the $m- > 0$ limit). Now, we see that $dC^*/du < 0$ (i.e., greater durability results in a gain in market share for cooperators) (solid line in Fig. 1*B*).

Finally, we consider a model of intermediate regulation, where $d = (1-f)\,d_0 + f\,d_0\,u$ and $m = (1-f)\,m_0 + f\,m_0\,u$. When $f = 0$, we have pure effort constancy and selection for fragility (Fig. 1*A*); when $f = 1$, we have pure outcome constancy and selection for durability (Fig. 1*B*). Further illustration of this model is provided in Fig. S3, where Fig. 1*A* is recovered in Fig. S3*D* and Fig. 1*B* in Fig. S3*F*. The proportion of cooperative patches in the coexistence equilibrium of this complete model (in $m- > 0$ limit) becomes $C^* = (e + u)/(d(1- f)+ fdu)$, which suggests that selection will favor more durable public goods (i.e., $dC^*/du < 0$) whenever $f > 1/(1 + e)$. Note that the shift to selection for durability occurs at an intermediate regulatory threshold capacity that, in turn, depends on the risk of environmental perturbation e. Consequently, for selection to favor durability, only a low regulatory capacity is needed as disturbance e increases to the limit of defector persistence [$e < (d - u)/(1 + d/c)$]. In contrast, regulatory capacity must be highly developed (high f) when e is low (and the density of cheats D^* is correspondingly high) to favor the evolution of more durable public goods. The importance of the disturbance parameter e in shaping the joint evolution of regulation and public goods durability can be understood in the light of ex-

perimental and theoretical work, highlighting that the density of cooperators peaks at intermediate levels of environmental disturbance (10). In the context of bacterial infections, the rate of disturbance or perturbation e can be related to the time course of infection (with mean duration of a cooperative infection in absence of cheats equal to $1/e$), implying, in turn, that precisely regulated and durable public goods are most likely to be favored in more persistent bacterial infections (lower e).

Across-Strain Comparison of Pyoverdin Durability. We found high levels of pyoverdin being maintained after 48 h for all three pyoverdin types (type I: 90.4% ± 0.8%; type II: 90.7% ± 1.1%; type III: 81.5% ± 1.8%) (Fig. S2), suggesting high durability of pyoverdin. Despite this overall slow decay, durability was significantly lower in strains with pyoverdin type III than in strains with pyoverdin type I [combined analysis for measures after 6, 24, and 48 h, linear mixed model (LMM): $t_7 = -4.81$, $P = 0.0019$] and type II (LMM: $t_7 = -5.13$, $P = 0.0014$), whereas there was no significant difference between strains with pyoverdin type I and II (LMM: $t_7 = 0.32$, $P = 0.76$).

Detailed Description of Strains Used. We used 11 different *Pseudomonas aeruginosa* strains originating from different environmental and clinical backgrounds (Table S1). Six of these strains produce pyoverdin type I, whereas three and two strains produce pyoverdin type II and type III, respectively. Pyoverdin consists of a conserved fluorescent chromophore linked to a short peptide, with the pyoverdin types I, II, and III differing in the amino composition of their peptide chain (11). For each of these 11 strains, we were in possession of a cheating mutant that produced no or reduced amounts of pyoverdin (12). As the reference wild type–mutant (i.e., cooperator–cheat) pair, we used strain PAO1 (pyoverdin type I; ATCC 15692; ATCC) and the knockout mutant (PAO1Δ*pvdD*), which was directly derived from PAO1; it is unable to produce pyoverdin, because the peptide synthetase (*pvdD*) is knocked out (13). Moreover, we used the strain pair PAO6049-PAO9(PAO6609), where PAO6049 is a methione auxotrophe derivate from PAO1 but a wild-type pyoverdin producer and PAO9 is pyoverdin-deficient mutant derived by UV mutagenesis from PAO6049 (14). For the other nine wild types, spontaneous pyoverdin-defective mutants have been isolated and characterized by Jiricny et al. (12).

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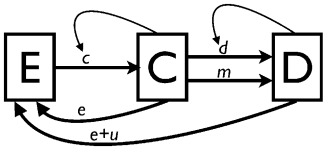

Fig. S1. Schematic diagram of the metapopulation model. Boxes represent patch states (E, empty; C, cooperators; D, defectors), and thick arrows represent patch transitions, both labeled with associated rate constants. Thin arrows highlight transmission terms.

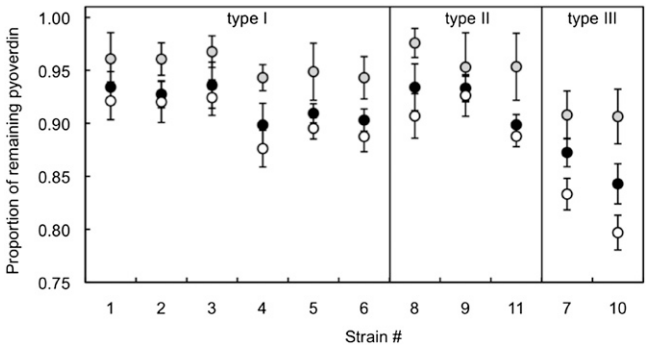

Fig. S2. Pyoverdin molecules are durable. Durability of pyoverdin of 11 different strains measured after 6 (gray circles), 24 (black circles), and 48 h (white circles). The durability (±95% confidence interval) is measured on the basis of relative fluorescence of pyoverdin and represents the proportion of remaining fluorescence over a given time interval for pyoverdin types I–III.

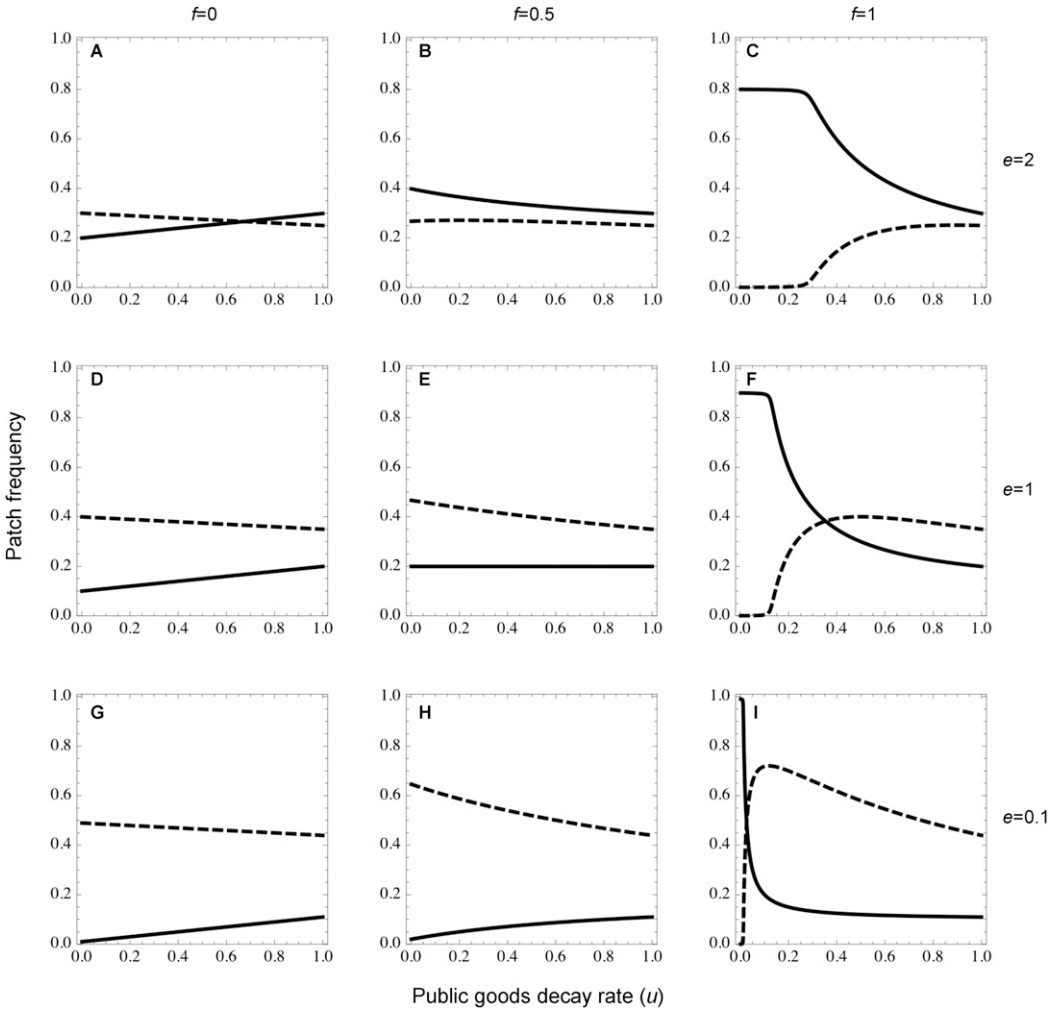

Fig. S3. Increased durability favors cooperation in a structured population if production is sufficiently facultative. Frequency of cooperator patches (solid lines) and defector patches (dashed lines) as a function of the rate of public goods decay (u) for differing conditions of regulation (f) and disturbance (e). (*Left*) Constitutive production by cooperators ($f = 0$). (*Center*) Partially regulated production ($f = 0.5$). (*Right*) Fully regulated production ($f = 1$). (*Top*) High disturbance ($e = 2$). (*Middle*) Intermediate disturbance ($e = 1$). (*Bottom*) Low disturbance ($e = 0.1$). Parameters are $c = d = d_0 = 10$ and $m = m_0 = 0.01$.

Table S1. Pairs of cooperator and cheat strains used in this study

Wild type no.	Wild-type strain name	Description	Pyoverdin type	Pyoverdin-negative mutant description	Ref.
1	PAO1 (ATCC15692)	European laboratory isolate	I	PAO1Δ*pvdD* knockout mutant	(1, 2)
2	PA6094	Methionine auxotroph from PAO1	I	PAO9 UV mutagenesis	(3, 4)
3	PAO1 (P3)	Laboratory isolate United Kingdom	I	3a-spontaneous mutation	(5, 6)
4	PAO1 (P76)	Laboratory isolate United States	I	4a-spontaneous mutation	(5, 6)
5	PAO1 (P93)	Laboratory isolate Italy	I	5b-spontaneous mutation	(5, 6)
6	PA14	Human clinical isolate United States (UCBPP-PA14)	I	6b-spontaneous mutation	(5, 7)
7	ATCC 013	Laboratory isolate United States	III	7a-spontaneous mutation	(5, 8)
8	1-60	CF isolate United States	II	8b-spontaneous mutation	(5, 8)
9	2-164	CF isolate United States	II	9a-spontaneous mutation	(5, 8)
10	206-12	CF isolate United States	III	10c-spontaneous mutation	(5, 8)
11	MSH	Environmental isolate United States	II	11a-spontaneous mutation	(5, 8)

CF, cystic fibrosis; MSH, Mount St. Helens; PA, *Pseudomonas aeruginosa*.

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