Table S1. Strains, plasmids and primers used in this study

Strain, plasmid or primer sequence	Relevant characteristics*	Source or reference
Bacterial strains		
Escherichia coli		
DH5α	F- φ80*lac*ZΔM15 Δ(*lac*ZYA-*arg*F) U169 *rec*A1 *end*A1 *hsd*R17 (r_k-, m_k+) *pho*A *sup*E44 λ- *thi*-1 *gyr*A96 *rel*A1	(Hanahan, 1983)
DH5-T1^R	F- φ80*lac*ZΔM15 Δ(*lac*ZYA-*arg*F)U169 *rec*A1 *end*A1 *hsd*R17(rk-, mk+) *pho*A *sup*E44 *thi*-1 *gyr*A96 *rel*A1 *ton*A (confers resistance to phage T1)	Invitrogen Inc.
DH5λpir	DH5α lysogenized with lambda pir	(Dunn *et al.*, 2010)
Vibrio fischeri		
AKD711	ES114*ΔnsrR*	(Dunn *et al., 2010*)
ES114	Wild-type isolate from *E. scolopes* light organ	(Boettcher & Ruby, 1990)
YLW9	ES114 carrying pVSV102, Kan^R	this study
YLW8	ES114 carrying pVSV208, Cam^R	this study
YLW62	ES114ΔnorV carrying pVSV208, Cam^R	this study
YLW61	ES114ΔnorV carrying pVSV102, Kan^R	this study
YLW60	ES114Δhmp carrying pYLW29, Cam^R	this study
YLW59	ES114Δhmp carrying pVSV105, Cam^R	this study
YLW57	ES114Δhmp carrying pVSV208, Cam^R	this study
YLW56	ES114Δhmp carrying pVSV102, Kan^R	this study
YLW55	ES114*ΔnsrR* carrying pLYW45	this study

YLW52	ES114*ΔnsrR* carrying pVSV208,Cm^R	this study
YLW51	ES114*ΔnsrR* carrying pVSV102, Kan^R	this study
YLW50	ES114*ΔnsrR* carrying pVSV209,Kan^R and promoterless Cm^R	this study
YLW48	ES114 carrying pYLW45, Kan^R and promoterless Cm^R	this study
YLW42	ES114 carrying pYLW29, Cam^R	this study
YLW40	ES114 carrying pVSV209, Kan^R and promoterless Cm^R	this study
YLW38	ES114*ΔnorV*	this study
YLW25	ES114 carrying pVSV105, Cam^R	this study
YLW20	ES114*Δhmp*	this study
YLW117	ES114*Δhmp-norV* carrying pVSV102, Kan^R	this study
YLW116	ES114*Δhmp-norV* carrying pVSV208, Cm^R	this study
YLW113	ES114*Δhmp-norV*	this study

Plasmids

pAKD711	*ΔnsrR* allele, R6Kγ and ColE1 replication origins, RP4 *oriT*, *ermR*, *kanR*	(Dunn *et. al., 2010*)
pCR-BluntII TOPO	TOPO PCR-cloning vector, Kan^R	Invitrogen Inc.
pEVS104	$oriV_{R6K\gamma}$, $oriT_{RP4,}$ RP4-derived conjugative helper plasmid, Kan^R	(Stabb, 2002)
pVSV102	$oriV_{R6K\gamma}$, $oriT_{RP4,}oriV_{pES213}$, *Dsgfp*-tagged, Kan^R,	(Dunn *et al.*, 2006)
pVSV105	$oriV_{R6K\gamma}$, $oriT_{RP4,}oriV_{pES213}$, complementation vector, Cam^R	(Dunn *et al.*, 2006)
pVSV208	$oriV_{R6K\gamma}$, $oriT_{RP4,}oriV_{pES213}$, *rfp*-tagged, Cam^R,	(Dunn *et al.*, 2006)
pVSV209	$oriV_{R6K\gamma}$, $oriT_{RP4,}oriV_{pES213}$, KanR-constitutively expressed *rfp*, transcriptional terminators-(AvrII, SalI, StuI)-promoterless CmR and *gfp*	(Dunn *et al.*, 2006)
pYLW20	*Δhmp* allele, R6Kγ and ColE1 replication origins, RP4 *oriT*, *ermR*, *kanR*	this study

pYLW27	*ΔnorV* allele, R6Kγ and ColE1 replication origins, RP4 *oriT*, *ermR*, *kanR*	this study
pYLW29	pComhmp, *hmp*+ complementing fragment cloned into pVSV105	this study
pYLW45	pPhmp::*gfp*, a 300bp *hmp* promoter region cloned into pVSV209 to drive expression of promoterless *gfp*	this study

Primers[†]

hmpcomp-for	5'-AGT CGT AC*T CTA GA*C TCG AAT GAC ATC ACC AA-3'	this study
hmpcomp-rev	5'-GCT AGA TC*G AGC TC*T GAG TTT CAT CAA CTC GAA-3'	this study
hmppro-for	5'-TGA CGC TA*C CTA GG*C GAA AGA GGT GCT TTC AT-3'	this study
hmppro-rev	5'-CAT GGT AC*A GGC CT*C CCA AAT TTA TCT TCA ACT AAC TC-3'	this study
VF0074qrt-for	5'- CGG TAT GAG TGC GTT TGG TCT TGC -3'	this study
VF0074qrt-rev	5'- CTG CAT CAC TCC TGG GTA ACG CTC -3'	this study
hmpqrt-for	5'- TGA AGT GCT ATT ACC AGC GGT TG -3'	this study
hmpqrt-rev	5'- AAG AGT GGC AAG TAA GTG ACC AC -3'	this study
norVqrt-for	5'- TCT TAC GCC ATT CAG TGC TTT GG -3'	this study
norVqrt-rev	5'- CTC GCC ATA CAC AAC CGT GAG -3'	this study

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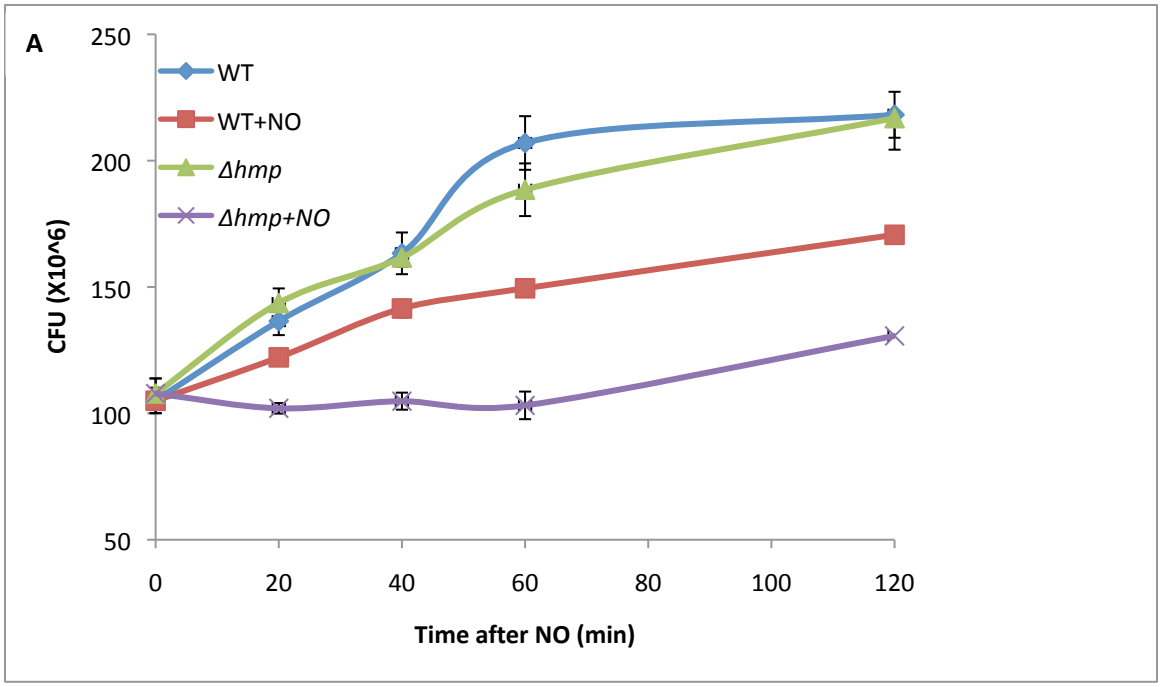
*Abbreviations used: Cam^R, chloramphenicol resistance; Erm^R, erythromycin resistance; Kan^R, kanamycin resistance; kb, kilobase; *gfp*, green fluorescent protein gene; *rfp*, red fluorescent protein gene.

[†]Sequences in underlined italics indicate the restriction-enzyme recognition sites introduced into primers (XbaI, TCTAGA; SacI, GAGCTC; AvrII, CCTAGG; StuI, AGGCCT).


A
250
200
150
100
50
CFU (X10^6)
WT
WT+NO
Δhmp
Δhmp+NO
0
20
40
60
80
100
120
Time after NO (min)

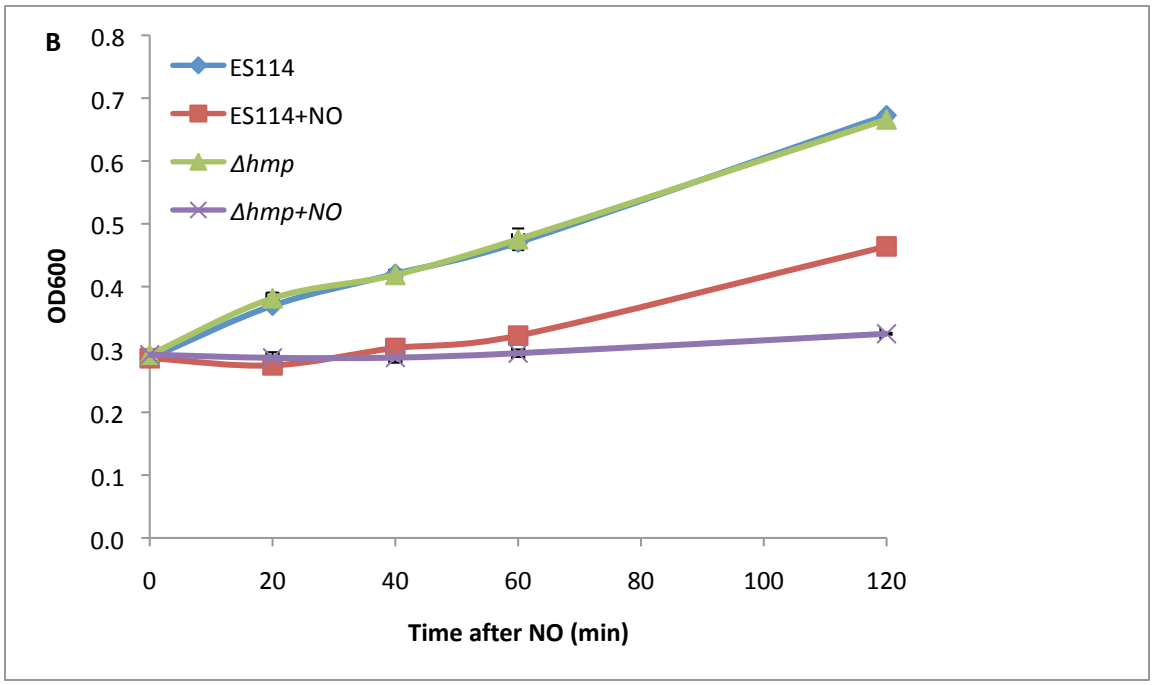

B
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
OD600
ES114
ES114+NO
Δhmp
Δhmp+NO
0
20
40
60
80
100
120
Time after NO (min)

Fig. S1.

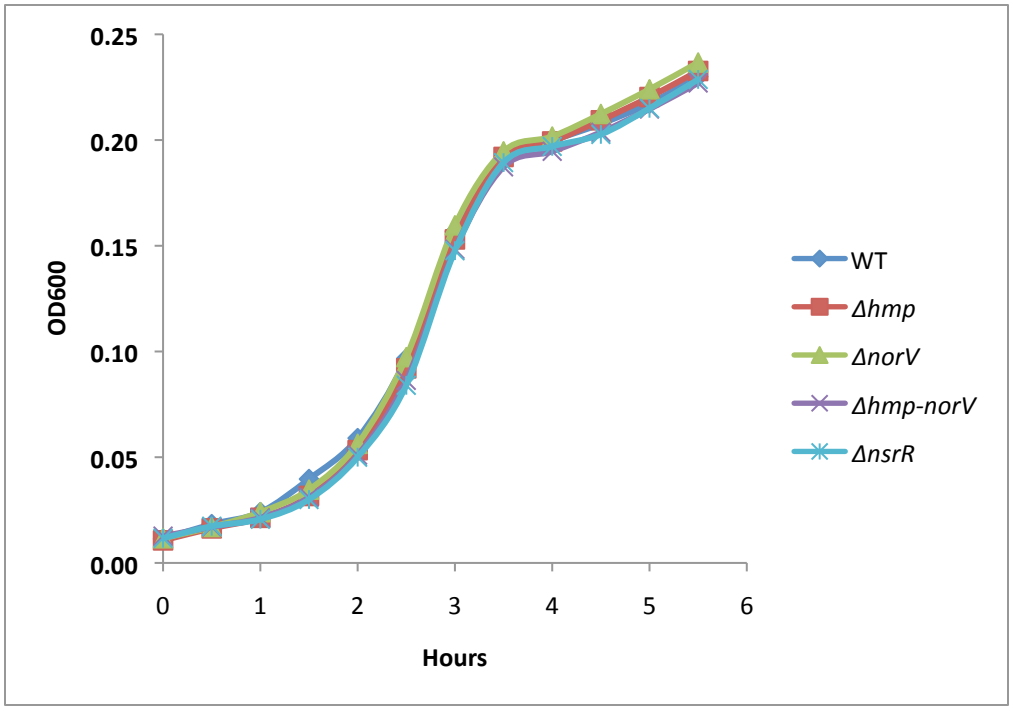

0.25
0.20
0.15
0.10
0.05
0.00
OD600
0
1
2
3
4
5
6
Hours
WT
Δhmp
ΔnorV
Δhmp-norV
ΔnsrR

Fig. S2.

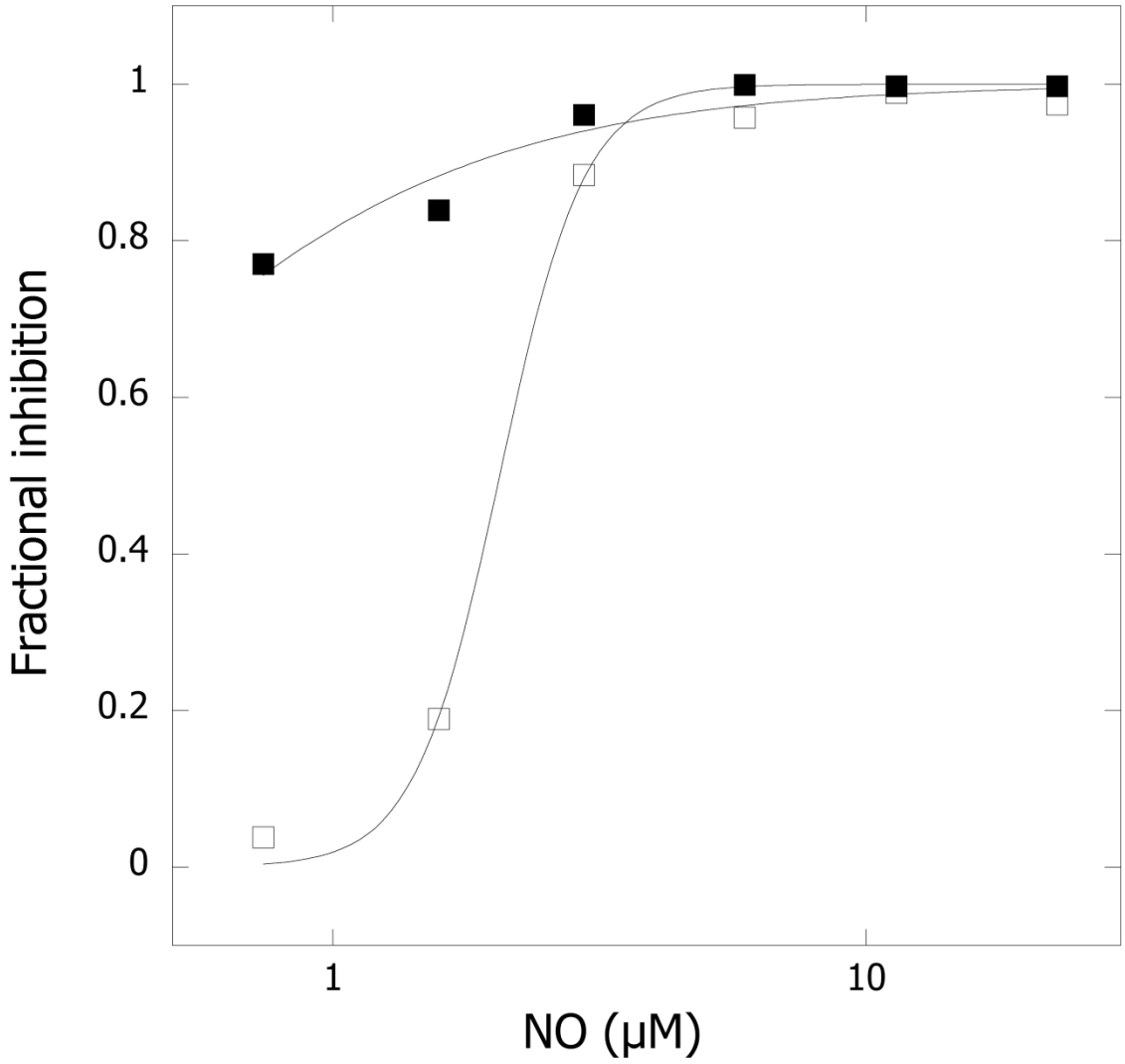


Fig. S3.

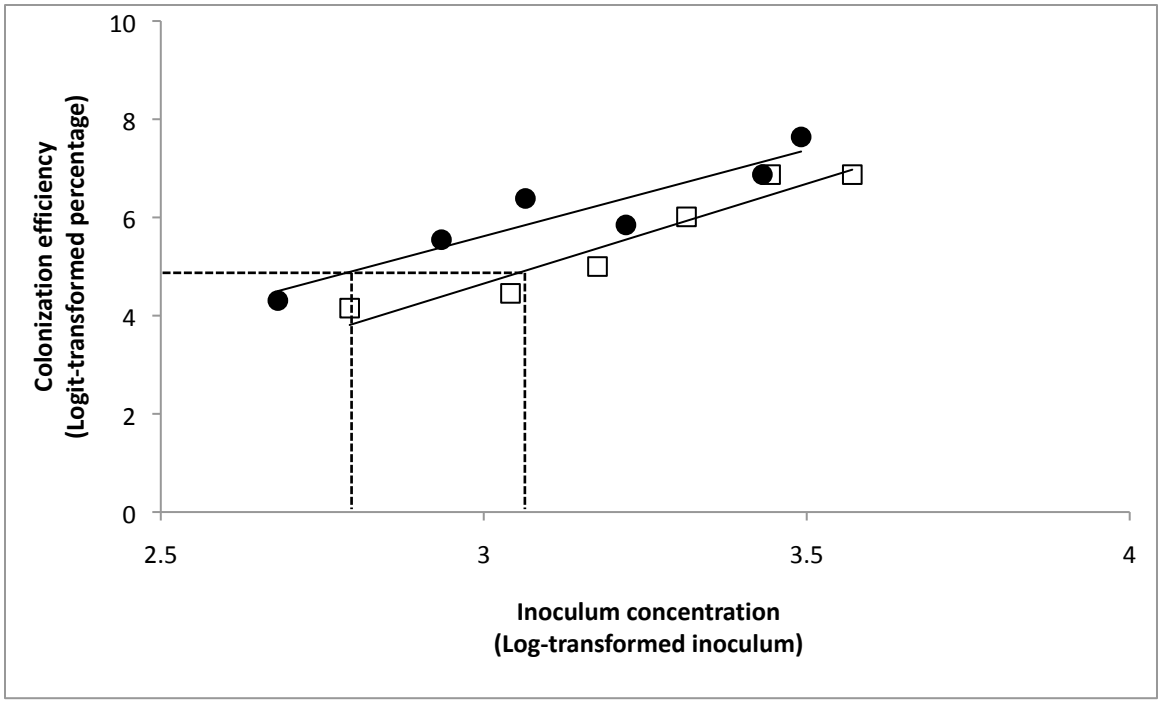

10
8
6
4
2
0
2.5
3
3.5
4
Colonization efficiency
(Logit-transformed percentage)
Inoculum concentration
(Log-transformed inoculum)

Fig. S4.

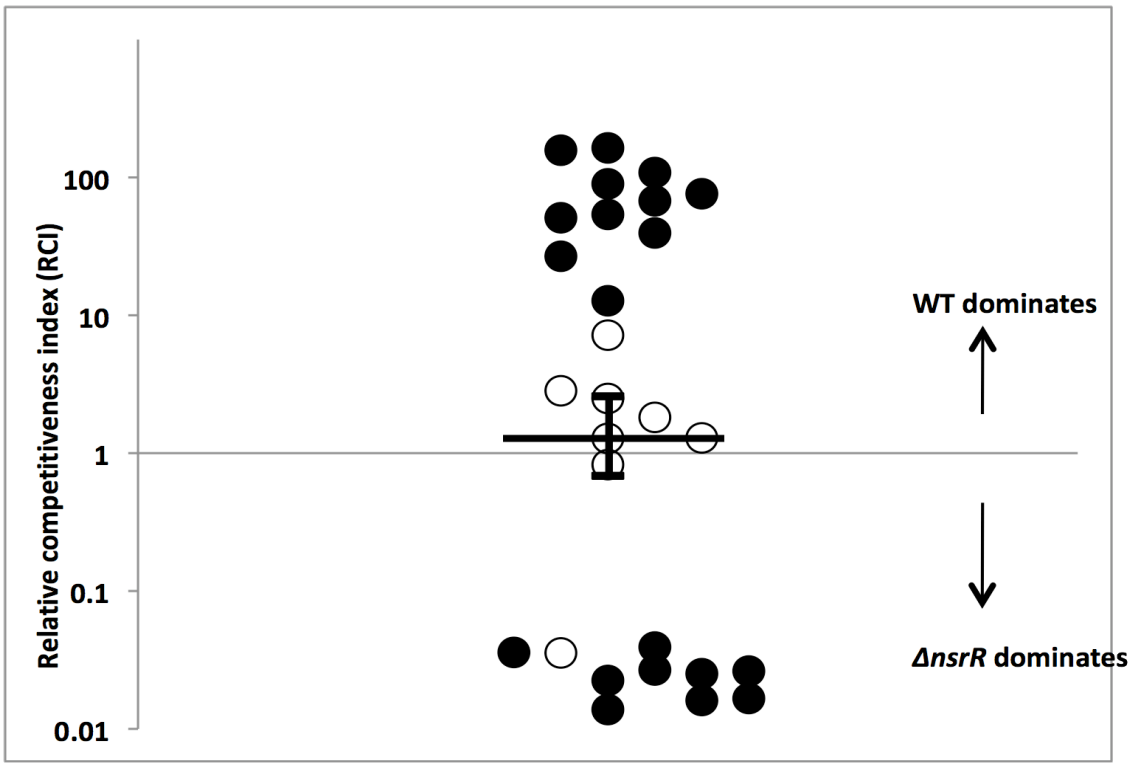

Relative competitiveness index (RCI)
100
10
1
0.1
0.01
WT dominates
ΔnsrR dominates

Fig. S5.

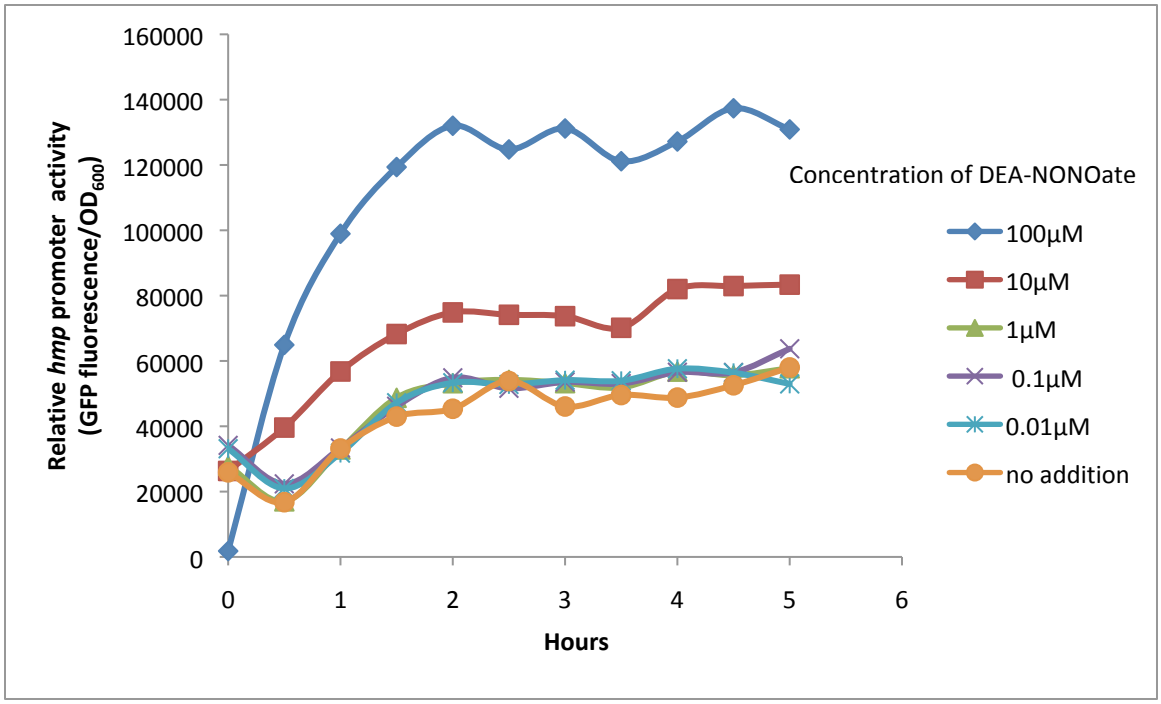

160000
140000
120000
100000
80000
60000
40000
20000
0
0
1
2
3
4
5
6
Hours
Relative hmp promoter activity (GFP fluorescence/OD600)
Concentration of DEA-NONOate
100µM
10µM
1µM
0.1µM
0.01µM
no addition

Fig. S6.

SUPPLEMENTAL DATA

Legends for supplementary figures

Fig. S1. NO exerts bacteriostatic effects on the growth of *V. fischeri*. Wild type (WT) and the *Δhmp* mutant were grown in minimal-salts medium plus GlcNAc, either in the presence or absence of an NO generator (100 μM DEA-NONOate). OD_{600} (A) and viable colony forming units (CFU) of the cultures (B) were monitored over time.

Fig. S2. Growth under anoxic conditions without NO challenge. Wild-type (WT) and four mutant strains were grown in minimal-salts medium plus GlcNAc. Growth was measured as OD_{600}.

Fig. S3. NO inhibition of oxygen consumption. The fractional inhibition of oxygen consumption of wild-type (open squares) and *Δhmp* (filled squares) strains was calculated from the steady-state oxygen consumption rate immediately after NO addition, relative to the rate immediately before addition. The NO donor, Proli-NONOate, was added at several concentrations. The data were fit to the Hill equation, from which an estimate of the NO concentration required for half-maximal inhibition was derived. The experiment was repeated three times with similar results, and one representative data set is shown here.

Fig. S4. The minimal infection dose (MID) of wild-type (WT) and *Δhmp* strains. Newly hatched juvenile squids were exposed to between approximately 500 and 3000 CFU (per ml) of either WT (circles) or *Δhmp* (squares), and the percentage of animals colonized was determined after 24 hrs. Colonization efficiency was estimated as the

inoculation concentration at which 50% of the animals became colonized (dashed lines). The R^2 values of both regressions (solid lines) were > 0.8.

Fig. S5. Colonization after a mixed inoculation with wild-type (WT) and *⊿nsrR* strains. Newly hatched juvenile squids were exposed to a mixture of WT and *⊿nsrR* at a ratio of approximately 1:1 (total dose: 3000 CFU/ml). The relative competitive index (RCI) was determined 24 hrs post-inoculation for each animal (circles). Dominance of the wild type over the mutant is indicated by an RCI value >1. The experiment was repeated three times with similar results. The mean value and 1 SEM are indicated. One representative data set is shown here. Filled circle: light organ was mono-colonized by one strain; open circle: light organ was colonized by both strains.

Fig. S6. Activation of the *V. fischeri hmp* promoter by exogenous NO. Wild-type (WT) cells carrying the *hmp* promoter driving *gfp* were exposed to different concentrations of the NO generator DEA-NONOate in FSW. Cell density (OD_{600}) and GFP fluorescence level were monitored over time. The experiment was repeated twice with similar results. One representative data set is shown here.