Figure S1. Production of PVD and PCH by different strains. (A) PVD and (**B**) PCH production has been monitored after 8 h of growth in CAA for different strains used in this study. PVD production has directly been estimated from the absorbance at 400 nm of culture supernatants, while PCH monitoring required first an extraction with ethyl acetate before monitoring its absorbance at 320 nm.

Figure S2. Expression of genes from PCH pathway in WT PAO1 and in the Δ*pvd*FΔ*pch*AΔ*pch*R isogenic mutant. Analysis of changes in transcription of different genes from the PCH pathway (*pch*E, *fpt*X, *fpt*A, and *pch*R). Reverse transcription qPCR was performed on *P. aeruginosa* WT PAO1 and its isogenic mutants Δ*pvd*FΔ*pch*AΔ*pch*R (a *pchR* mutant unable to produce both siderophores), grown in CAA medium. Results are given as a ratio between the values obtained for the mutant over those obtained for the WT PAO1 strain, in logarythmic scale.

Figure S3. Fur binding to the *fpt*A promoter. Analysis of the binding of Fur on a PCR fragment encompassing the PchR and Fur boxes (-145 to -1) of the *fpt*A promoter

Figure S4. ^{55}Fe transport by iron chelators. (**A**) Δ*pvd*FΔ*pch*A cells were incubated for 15 min in 50 mM Tris-HCl pH 8 before the ^{55}Fe uptake assays. 100 nM iron chelator-^{55}Fe complex were then added. Aliquots of 100 µl were removed after 15 min, centrifuged and the radioactivity contained in the pellet measured (plain bars). The results are expressed as pmol of iron chelator-^{55}Fe transported per millilitre per OD_{600nm}. The experiment has been repeated with CCCP (a protonophore that inhibits any siderophore dependent iron uptake (slashed

bars)[32]. (**B**) Effect of desferrioxamine and deferasirox on *P. aeruginosa* growth. At 10 mM, deferasirox inhibits (but not completely) *P. aeruginosa* growth. (**C**) Results of CAS assay showing the competition between CAS and different iron chelators. The competition capacity is measured as the ratio between the OD_{630nm} of the competition sample over that of the control sample (only CAS). Low values means strong competition. (**D**) The transcriptional reporter plasmid p(*fpt*AFurPchR) has been inserted into the PAO1 mutants Δ*fpt*A and Δ*pvd*SΔ*fpt*A. mCherry fluorescence has been monitored after 20 h of growth, in the presence of different concentrations of desferrioxamine in the two backgrounds. PchE-mCherry expression is followed at 610 nm (excitation at 570 nm). Results are normalized against the expression of mCherry/OD_{600nm} in the absence of desferrioxamine, and given in percent. Since deferasirox become toxic more quickly in the double mutant Δ*pvd*SΔ*fpt*A, results are not shown.

Figure S5. Model of regulation of the PCH pathway expression. The scheme present a model of regulation of the PCH pathway expression when iron chelators are added, and compete for iron with PCH especially –PVD being a better binder-. When no iron chelators are added, whatever the strain (PVD+ or PVD-), the cell does not face difficulties in iron import. In that case, PCH is produced, PCH-Fe imported, and there is a balance between repression by Fur-Fe and activation by PchR-PCH-Fe of the PCH pathway. Nevertheless, PchR-PCH-Fe overcomes Fur repression. In a PVD+ strain, the addition of iron chelators hinders the formation of PCH-Fe, and thus the activation of PchR by PCH-Fe. In that condition, there is a balance between activation by PCH alone, much less efficient, and repression by Fur-Fe. In that condition, the PCH pathway is repressed at high iron chelator concentration. In a PVD- strain, the cell faces difficulties in iron import when iron chelators are added. In that condition, there is still a balance between activation by PCH alone, less efficient, and repression by Fur-Fe. But as iron may be less concentrated inside the cell, Fur will be less activated, the repression of the PCH pathway less efficient, which can thus still be expressed. Further experiments should be conducted to confirm these conclusions, like measuring the amount of Fur or PchR bound to such PCH

promoter construction, through ChIP, at different concentration of exogenous iron chelators and in different backgrounds.

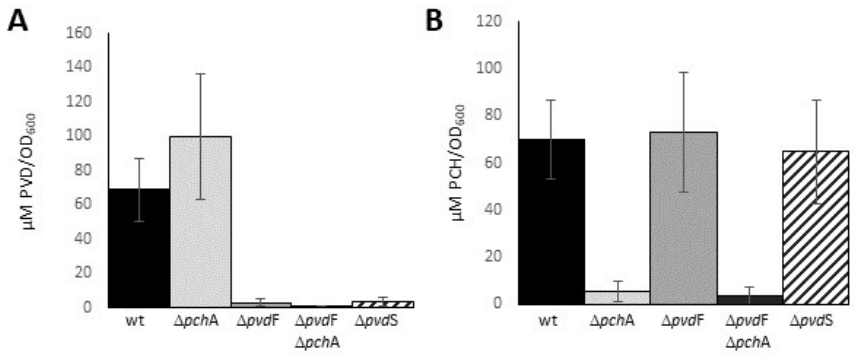
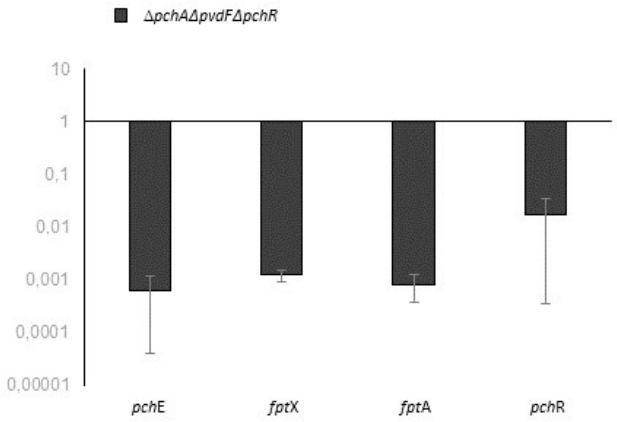

A
160
140
120
100
80
60
40
20
0
µM PVD/OD600
wt
ΔpchA
ΔpvdF
ΔpvdF ΔpchA
ΔpvdS
B
120
100
80
60
40
20
0
µM PCH/OD600
wt
ΔpchA
ΔpvdF
ΔpvdF ΔpchA
ΔpvdS

Figure S1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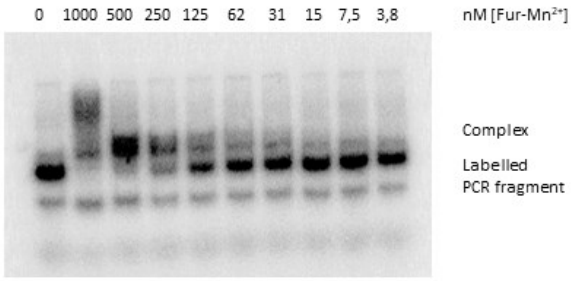
0,001
0,0001
0,00001
pchE
fptX
fptA
pchR

Figure S2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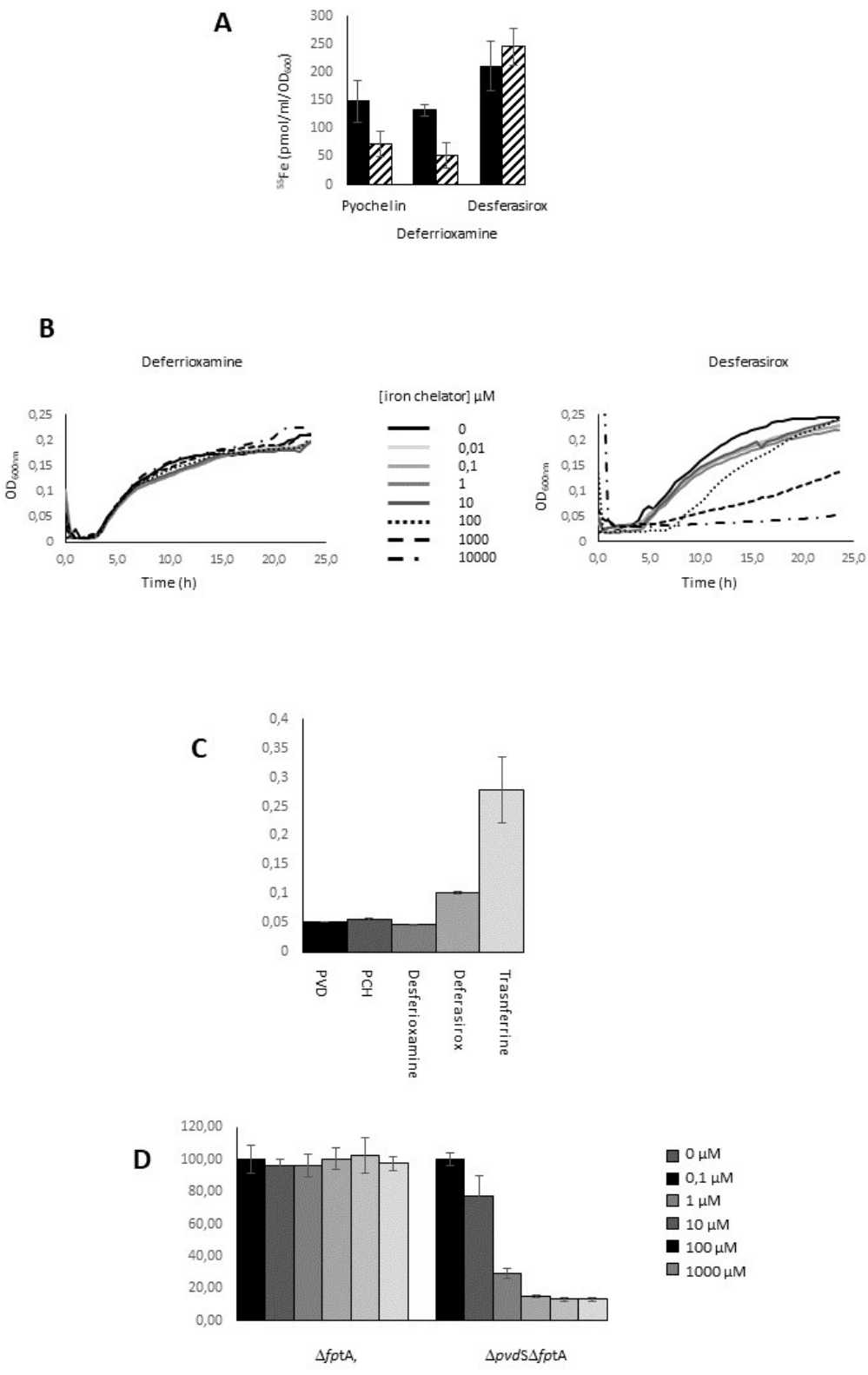
nM [Fur-Mn2+]
Complex
Labelled
PCR fragment

Figure S3


A
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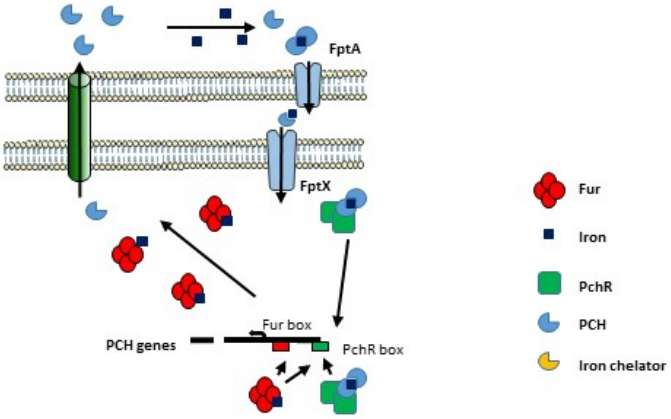
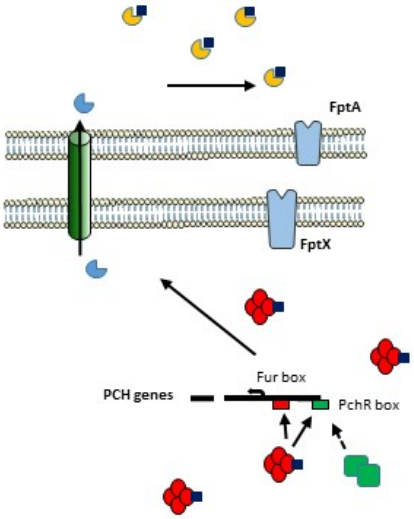
250
200
150
100
50
0
^{55}Fe (pmol/ml/OD$_{600}$)
Pyochelin
Desferasirox
Deferrioxamine
B
Deferrioxamine
[iron chelator] µM
0
0,01
0,1
1
10
100
1000
10000
Desferasirox
OD$_{600nm}$
Time (h)
C
PVD
PCH
Desferioxamine
Deferasirox
Trasnferrine
D
120,00
100,00
80,00
60,00
40,00
20,00
0,00
0 µM
0,1 µM
1 µM
10 µM
100 µM
1000 µM
ΔfptA,
ΔpvdSΔfptA

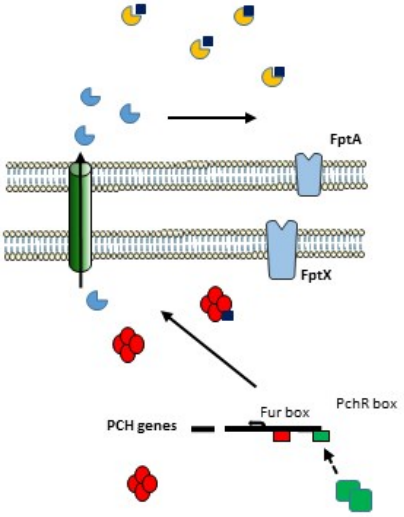
Figure S4



PVD (+/-) strains



PVD (+) strains
Iron chelators



PVD (-) strains
Iron chelators

Figure S5