**Suppl. Table: PA14 mutants** (previously published: Sass, G. et al., J. Bacteriol. 200(1):e00345-17, 2018.)

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| --- | --- | --- | --- |
| **Mutant number** | **Mutant name** | **Mutation result** | **Ref.** |
| **1** | *pvdD-pchE-* | Pyoverdine-pyochelin double siderophore mutant | ([[1]](#endnote-1)) |
| **2** | *pqsE-* | Gene mediates the regulatory activity of the MvfR system (see *mvfR-*); HAQs similar to wild type but defective in pyocyanin, HCN | ([[2]](#endnote-2)) |
| **3** | *mvfR-* | Regulates the transcription of *pqsABCDE* operon; under-production of various QS-regulated factors related to loss of HAQs, phenazines, proteases, HCN, lectins and others ([[3]](#endnote-3)) | ([[4]](#endnote-4)) |
| **4** | *pqsA-* | First step in HAQ synthesis; gene for anthranilate-CoA ligase lost; loss of extracellular quinolones, HAQ biosynthesis, including HHQ, PQS, DHQ; also decreased activity of MvfR ([[5]](#endnote-5)) | (1) |
| **5** | *pqsH-* | Product converts HHQ into PQS; loss of 2-heptyl-3-hydroxy-4(1H)-quinolone synthase; loss of PQS biosynthesis, thus decreased activity of MvfR; MvfR not as negatively affected than for pqsA-, since pqsH- still produces HHQ and other HAQs that can act as ligands of MvfR | ([[6]](#endnote-6)) |
| **6** | *lasR-rhlR-* | Double mutant defective for most QS-regulated metabolites, including phenazines, rhamnolipids, AHL, HAQs, proteases, HCN, chitinase, elastase and others | ([[7]](#endnote-7)) |
| **7** | *lasR-* | Lacks several QS-regulated factors, including proteases, oxo-C12-HSL; delayed activation of *pqsABCDE* and of RhlR QS pathway | (1) |
| **8** | *rsmA-* | Global post-transcriptional regulator mutant; various effects including less rhamnolipids, more phenazines and HCN **(**[[8]](#endnote-8)) | ([[9]](#endnote-9)) |
| **9** | *pqsA-pqsH- NOT polar* | Same predicted outcome as pqsA-. In both, the *pqsA-* mutation is nonpolar on downstream genes in the operon, thus not preventing their transcription. | (5) |
| **10** | *pvdD-* | Loss of pyoverdine (siderophore) | (8) |
| **11** | *rhlR-* | Lacks several QS-regulated factors. Loss of rhamnolipids, phenazines, HCN, lectins, C4-HSL | (2) |
| **12** | *HSI-1/2-* | Double deletion mutant defective in 2 of 3 type VI secretion systems | ([[10]](#endnote-10)) |
| **13** | *pvcA-* | Loss of paerucumarin and pseudoverdin | (8) |
| **14** | *rhlA-* | Loss of rhamnolipids (viii) | ([[11]](#endnote-11)) |
| **15** | *phzC1-*  *phzC2-* | Double phenazine mutant (completely abrogated), no pyocyanin | (1) |
| **16** | *pchE-* | Loss of pyochelin (siderophore) | (8) |
| **17** | *exoU-* | Loss of exotoxin U, via type III secretion | (8) |
| **18** | *rsmY-,rsmZ-* | Loss of genes for coding small regulatory RNAs, antagonistic to RsmA;, decreased production of C4-HSL, phenazines, chitinase (vii) | (1) |
| **19** | *HSI-2/3-* | Double deletion mutant defective in 2 of 3 type VI secretion systems | (9) |
| **20** | *HSI-1/3-* | Double deletion mutant defective in 2 of 3 type VI secretion systems | (9) |
| **21** | *pqsA-pqsH- polar* | pqsA::TnPhoA, pqsH::Gm, the mutation in pqsA is polar (thus theoretically preventing transcription of downstream genes in the operon), Kan and Gm resistant | (1) |
| **22** | *chiC-* | Chitinase C mutant | (8) |
| **23** | *lecA-* | Lectin A mutant | (8) |
| **24** | *hcnA-* | Loss of hydrogen cyanide | (8) |
| **25** | *lasI-* | Impaired phenazine pathway, no oxo-C12-HSL release |  |
| **26** | *pscC-* | Defective in all 3 secretion systems |  |

Abbreviations: QS: quorum-sensing; HAQ: 4-hydroxy-2-alkylquinolones; HCN: hydrogen cyanide; AHL: acylhomoserine lactones; HSL: homo serine lactone; HHQ: 4-hydroxy-2-heptylquinoline; PQS: 3,4-dihydroxy-2-heptylquinoline ([[12]](#endnote-12)); HQNO:   
4-hydroxy-2-heptylquinoline *N*-oxide. Kan: kanamycin; Gm: gentamicin.

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