in the nucleoid. HipA is then no longer able to phosphorylate glutamyl-tRNA synthetase, preventing initiation of the forthcoming stringent response (Wen et al. 2016). Overexpression of toxins that inhibit essential functions, such as translation, may then contribute to persister formation and tolerance to some antibiotics. Maeda et al. (2017) also described a new TA system, YjjJ, which is encoded by a single gene and is a homologue of the toxin HipA. YjjJ seems to have different cellular targets than HipA, but HipB, the cognate antitoxin of HipA, also acts as an antitoxin for YjjJ.

The RelE and MazF toxins cause dormancy by cleaving mRNA (Lewis 2012). During amino acid starvation, RelE cleaves mRNA in the ribosomal A-site, inhibiting protein translation. RelE is structurally similar to bacterial RNases that employ general acid–base catalysis to facilitate RNA cleavage (Dunican et al. 2015).

The TisB toxin forms a membrane pore, leading to a decrease in the proton motive force and ATP consistent with its role in forming dormant cells (Lewis 2012). Dörr et al. (2010) showed the induction of persisted formation by ciprofloxacin and tolerance to multiple antibiotics by cells producing TisB toxin. Ciprofloxacin kills cells primarily by converting its target proteins, which are DNA topoisomerases, into DNA endonucleases. A decrease in ATP will prevent topoisomerases from damaging the DNA, indicating a link between fluoroquinolones and TisB induction.

The induction of Doc toxin of the Phd-Doc TA system mimics the effects of treatment with the aminoglycoside hygromycin B; both interact with 30S ribosomal subunits, stabilize polysomes, and significantly increase mRNA half-life. The antibiotic also competes with ribosome-bound Doc, whereas hygromycin B-resistant mutants suppress Doc toxicity, suggesting that the Doc-binding site includes that of aminoglycosides (Liu et al. 2008).

As in *Mycobacteria*, multiple *vapBC* modules have been described in *E. coli*, contributing to enhanced survival within the host. VapCs are endoribonucleases that can inhibit translation by site-specific cleavage of initiator tRNA or the universally conserved region in 23S rRNA (Winther et al. 2016), and we can easily imagine that they interfere with macrolides or aminoglycosides, which have sites of action on the ribosome.

Van Acker et al. (2014) investigated whether TA modules contribute to persistence toward antibiotics in *Burkholderia cenocepacia*, a well-known resistant pathogen that colonizes cystic fibrosis patients. The overexpression of toxins results in growth inhibition, often increasing the number of surviving persisters, especially in untreated sessile cells. Nine toxin-encoding genes are upregulated after treatment with tobramycin, but none after treatment with ciprofloxacin.

TA systems are constantly being discovered in genetic analysis and bioinformatics searches (Budde et al. 2007; Harrison et al. 2009; Chan et al. 2012; Gil et al. 2015; Jaiswal et al. 2016; Thakur et al. 2018). Some are associated with