The authors of “Molecular Mechanisms Underlying Bacterial Persisters” that was published in *Cell* 157: 539, 2014 fail to appropriately reference our work on persister cells on four occasions, have one error, and misrepresent our work as indicated below.

1. For the first omission, the authors write on p 544, “Recently, it was reported that a subpopulation of *M. smegmatis* cells survived treatment by the bactericidal drug isoniazid (Wakamoto et al., 2013), a prodrug that requires activation by the bacterial enzyme catalase. Using microfluidics, these authors elegantly showed that expression of catalase varied stochastically in single cells. Thus, the surviving subpopulation of bacteria was able to grow in the presence of isoniazid because it did not activate the drug. We suggest that such drug escape is better viewed as an atypical case of drug avoider, rather than the multidrug tolerance phenotype exhibited by slow-growing persister cells.”

This idea that Wakamoto et al. were not studying a persister-related phenomenon was first put forth in our review that was published on-line in AEM on 13 September 2013 (79:7116–7121 December 2013,page 7119): “It has been argued that the study of Wakamoto et al. (50) with *Mycobacterium smegmatis* implies that persister cells are not dormant. This study showed that cells surviving lethal treatment with the prodrug isoniazid were metabolically active. However, isoniazid requires activation by the catalase KatG, so the cells tolerant to isoniazid were simply cells with low levels of KatG activity. In effect, these tolerant cells were never exposed to a lethal antibiotic treatment because the isoniazid remained inactive. Hence, the metabolic activity observed for cells surviving isoniazid is not indicative of metabolic activity in persister cells and this report is a special case of a prodrug requiring activation. Therefore, there is little evidence indicating that persister cells are not dormant but a wealth of evidence indicating that persister cells are dormant.”

2. For the second omission, the authors write throughout the report that persister cells are dormant. For example, on p 545 they write, “ In the persistent state, the cellular translation rate is low.” and on p 539 they write, “Thus, it was proposed early on that persisters are cells that have entered a state of low metabolic activity, here referred to as dormant or slow-growing cells”. This idea of dormancy of persister cells was suggested in the 1940s as indicated but it was proven by us in our AAC manuscript that was published in January 2013 (Antimicrobial Agents and Chemotherapy, 57: 1468–1473, 2013) but the authors fail to cite our work. We wrote in the Abstract, “Here we mimic toxins via chemical pretreatments to induce high levels of persistence (10 to 100%) from an initial population of 0.01%. Pretreatment of Escherichia coli with (i) rifampin, which halts transcription, (ii) tetracycline, which halts translation, and (iii) carbonyl cyanide m-chlorophenylhydrazone, which halts ATP synthesis, all increased persistence dramatically. Using these compounds, we demonstrate that bacterial persistence results from halted protein synthesis and from environmental cues.”

3. For the third omission, our AAC manuscript in item two (see page 1472) above showed environmental stress induces persistence and that this is more important than stochastic generation of persister cells since we wrote, “The significant increase in persistence we observe, after pretreatment with rifampin, tetracycline, and CCCP, clearly shows several ways in which bacterial persistence is induced via environmental pressure, rather than solely via a stochastic event. Hence, it is apparent that while persistence can result from stochastic fluctuations, it is also directly affected by the presence of numerous extracellular molecules.”. However, the authors fail to cite this as the basis for their conclusion in their work on page 543.

4. For the fourth omission, we indicated for the first time in our AEM persister review of item 1 above that a study by Nguyen et al. did not consider the involvement of TA loci in their persister cells as we wrote on p 7118:

“In 2011, Nguyen et al. (31) confirmed the much-earlier work of Korch et al. (16) on the necessity of ppGpp for persistence withHipA7 by demonstrating a modest decrease of 68-fold in persistence upon deleting relA and spoT in P. aeruginosa. Nguyen et al. (31) also reported results similar to those of Korch et al. (16) inregard to decreased persistence using similar E. coli relA and spot mutants. Nguyen et al. (31) argued that the necessity of ppGpp implied an active response, whereas an alternative interpretation of their results is that ppGpp is required to activate a cell response that leads to arrested growth. In P. aeruginosa, Nguyen et al. (31)found that the reduction of 4-hydroxy-2-alkylquinoline and the production of catalase and superoxide dismutase were important for the ppGpp effect. What was not considered was the effect of the simulated stringent response on TA systems in *P. aeruginosa* and their effect on dormancy.”.

The Cell authors wrote on p 544, “Interestingly, the model proposed by Nguyen and colleagues highlights an active mechanism involving the stringent response (Nguyen et al., 2011). However, it was not tested whether TAs were required for the ppGpp mediated increase in persistence.”

Hence our analysis that TA systems were not considered was the clear source of their statement but it was not attributed.

5. The incorrect statement made by the authors appears on p 543, “Lon protease degrades all known antitoxins of *E. coli* K-12 (Gerdes and Maisonneuve, 2012), including HipB (Hansen et al., 2012).”

However, we published in 2012 in Nature Chemical Biology (8:855-861, 2012) that *E. coli* antitoxin GhoS is not labile and so is not degraded by Lon (p 856).

6. The authors in the Cell paper also misrepresent our data in they claim our results are insignificant in regard to showing that deletion of the gene encoding toxin MqsR did not affect persistence. Our work (*Biochemical and Biophysical Research Communications* 391: 209–213, 2010) was the first report to show that deleting a toxin gene affects persistence; in this work, we showed that deleting *mqsR* reduced persistence 6 fold and deleting the complete *mqsRA* locus reduced persistence 7 fold. To promote their own work using multiple deletions of TA systems, the Gerdes lab misrepresents our work in the Cell paper as “These observations prompted a genetic analysis of the 11 known type II TAs in E. coli (all of which encode mRNases except hipBA): deletion of any one of these loci had minor effects on the persistence level of planktonic *E. coli* cells (Keren et al., 2004b; Kim and Wood, 2010; Maisonneuve et al.,2011).”. We argue that our work was not “minor” as it turned out to be the first direct evidence that TA systems were related to persistence (an earlier work with *hipA* was retracted due to a larger than intended DNA deletion), a fact that was built upon in the Maisonneuve et al.,2011 paper which showed our idea was correct.