Understanding bacterial survivability in the environment is an important subject that has both ecological and health implications. We have examined bacterial survivability under stress conditions using *Escherichia coli* as a model. Previous papers found that the presence of the amino acid methionine, an essential amino acid, reduces survivability under various stress conditions (such as heat stress, acid stress, or pH stress). It was also found that the enzyme MetE, the last in the pathway of the biosynthesis of methionine, is linked to stress resistance. This protein constitutes approximately 3-5% of the bacteria’s total amount of cellular proteins; it is sensitive to heat, is directly regulated by methionine, and is suppressed in the presence of vitamin B12, which is found in the medium.

Studies examining the effect of various stress conditions on the growth rate of bacteria have shown that during exposure to stress, the protein MetE undergoes structural change preventing it from carrying out the last reaction in the process of biosynthesis, leading to the cessation of bacterial growth. I examined the effect of the presence of MetE on survivability as a reaction to environmental stress. The results show that when bacterial cells are exposed to environmental stress (such as heat, acidity, or pH), the ability for survival depends on the amount of MetE found in the cell. That is, as the amount of this protein in the cell increases, so does the ability to cope with environmental stress. Over-expression of MetE conferred even higher resistance to environmental stress. Since MetE is a protein that has a tendency to undergo aggregation as a result of exposure to environmental stress, I examined whether the high survivability of the bacteria was a result of this characteristic. I compared over-expression of MetE to over-expression of the protein LacZ, which also has a tendency to undergo aggregation. The results showed that stress resistance is directly linked to the protein MetE and not to its tendency to undergo aggregation. Over-expression of MetE having the structural mutation H641N, which impairs the action of MetE by decreasing the binding affinity of the substrate, homocysteine, to the active site, showed an increase in bacterial survivability. In a similar manner, the mutation C643S, which impairs the activity of MetE due to the increase of the binding affinity of the substrate (homocysteine) to the zinc ion, showed an increase in bacterial survival. All of these results hint at a new role for MetE, as a shield against environmental stress on the cell, a role which is not linked to its known activity. These results and the understanding of the mechanism by which the protein MetE confers stability against environmental stress are of paramount importance, since it can also be utilized to make the production of methionine more efficient as well as developing bacteria and plants that are resilient under stress conditions.