E319 Effect of Divalent Cations on the Intracellularly Uptake of Cadmium and Change of Fatty Acids by Cadmium Stress in *Azomonas agilis* PY101

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Cd^{2+} transport by *Azomonas agilis* PY101 was reduced about 40~50% in the presence of Mn^{2+} or Zn^{2+} and was not almost effected in the presence of Co^{2+} or Ni^{2+}. That is to say, when the cells were suspended in the presence of Mn^{2+} or Zn^{2+}, there was decreased Cd^{2+} transport by these cations. Early growth of *A. agilis* PY101 is inhibited by the addition of Cd^{2+}. However, although *A. agilis* PY101 was not spared from the growth inhibition effect of Cd^{2+} by the addition of Co^{2+} or Ni^{2+}, the strain was spared from the inhibitory effect of Cd^{2+} by the addition of Mn^{2+} or Zn^{2+}. The fatty acid composition and content of *A. agilis* PY101 cells grown with and without 1000 ppm of $CdCl_2$ was analyzed by gas chromatography. The increase or decrease of cellular fatty acids prepared from the cells cultured with and without cadmium was observed in the profiles of fatty acids. In saturated fatty acids, 10:0, 12:0, 14:0, 17:0, and 18:0 were increased by cadmium stress, but 15:0 and 16:0 were decreased by cadmium stress. In unsaturated fatty acids, 18:1 w9c was increased by cadmium stress, but *anteiso* 17:1 w9c was not detected by cadmium stress. All the hydroxy fatty acids (10:0 3OH, 12:0 2OH, 12:1 3OH, 12:0 3OH, and 16:0 2OH) were increased by cadmium stress, but all the cyclopropane fatty acids (17:0 cyclo and 19:0 w8c) were decreased by cadmium stress. In these results, it is showed that the increased the rigidity of cell membrane resulting from changes in fatty acid composition and content, exclusion of cadmium from the cell membrane contributes to the cadmium tolerance of *A. agilis* PY101.

E320 Isolation and Characterization of Pigment Biosynthesis Defective Mutants from *Azomonas agilis* PY101, a Cadmium-Resistant Strain

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That the fluorescent pigment of the cadmium-resistant *Azomonas agilis* PY101 is shown tc interact with cadmium and consequently affect its toxicity, we carried out Tn5 mutagenesis and isolated four pigment biosynthesis defective mutants. None of the mutants grew in the presence of 1500 ppm of cadmium chloride in growth medium but exhibited differential sensitivity to cadmium. From comparison with VITEK system using GNI card, Pbg1, Pbg2, and Pbg3, the pigment-defective mutants, were lacked the oxidation activity of xylose and therefore could not utilize xylose as a carbon sourse. It is reasonable to think that the lack of xylose oxidation activity would lower the extracellular level of fluorescent pigment, which is responsible for cadmium tolerance in *A. agilis* PY101. However, although Pbg4 mutant was not produced the fluorescent pigment at all, biochemical characteristics of Pbg4 was similar to that of the wild type. Thus, it is supposed that Pbg4 is lack of gene encoding the fluorescent pigment biosynthesis by insertion of Tn5. Isolation and characterization of these mutants suggested that the production of the fluorescent pigment and the damage of xylose oxidation activity were important in cadmium resistance mechanism.