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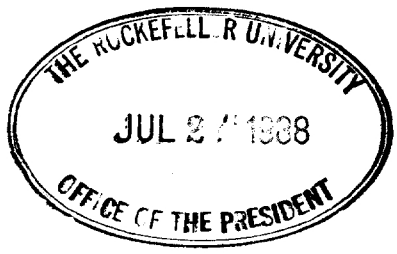
July 25, 1988



Dr. Joshua Lederberg, President
Rockefeller University
1230 York Avenue
New York, New York 10021

Dear Josh:

Persons working on E. coli genetics and molecular biology have been very good about sharing their strains, plasmids, and DNA fragments. We have had very few refusals when we requested strains for distribution from the Stock Center. At this moment, I can think of only one person who would not send out his unique mutant for, I think, around 5 years. By then, other labs had isolated mutants at the same locus so he was largely ignored.

Some labs don't want to distribute a mutant as soon as it is published because they want to give a graduate student a chance to complete his work - or a post-doc. A student can spend a year or two isolating a new type of mutation, with patience, skill and ingenuity, in order to do a biochemical or molecular study. There are big labs which would love to take the mutant and quickly do the experiment toward which he has been working. (Labs that could not have isolated the mutant in the first place.) I am entirely in favor of protecting the student (or post-doc) in cases like this, and I think that almost all people in the field share this attitude.

I am trying to revise the linkage map and so am ploughing through the literature. I see in paper after paper thanks to other labs who sent mutants, plasmids, DNA fragments and unpublished information to the authors of the papers. I don't think that there is a problem in E. coli genetics. I hear that the situation in the "biomedical sciences" is another matter, but I don't have any idea as to what can be done about that. It may be related to other problems of the field of medicine which will need to be solved first.

Regarding the mtl mutants, I'm not sure that I understand exactly what it is you want to know.

You isolated two Mtl$^-$ (mannitol non-utilizing) mutants very early:

mtl-1 is in strain W945 and mtl-2 in strain W595.

mtl-2 is now thought to be a mutation in the mtlA locus (Lengeler and Lin 1972 J. Bacteriol. 111:566).

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This mutation is polar so the strain carrying this mutation lacks both the mannitol specific Enzyme II of the phosphotransferase system (PTS), coded by the mtlA gene, and the mannitol-1-phosphate dehydrogenase, coded for by the mtlD gene.

mtl-1 is also probably a mutation in mtlA. Strains carrying this mutation lack the mtlA product but have "unusual" levels of the mtlD product and do not appear to be regulated normally. For this reason we still just list this mutation as mtl-1 rather than as mtlA1.

I hope that this answers your questions.

Sincerely,

Barbara

Barbara J. Bachmann
Curator, E. coli Genetic Stock Center