

Dear Bill:

4/27/79

I have sent to you under separate cover some 20 mutant isolates that have been described in a manuscript by Pittenger and West and accepted by Genetics (but still on my desk for a few final revisions). Two of the mutants described in the paper, 289-67 and 299-9, are not included in the group I sent since a graduate student of Alan Lambowitz is doing a thesis on them. I have told Alan that he can keep them "protected" until the student finishes his research, but I have sent a description of them to you along with the other mutants. I only mention this because they are very exciting mutants (see Manella, et al PNAS 1979--in press) involving tailoring of mRNA sequences. You may have requests for these mutants and I have told Alan that he can send them to you when ~~you are~~ finished, or you can request them when you see fit.

All of the mutants were uv induced either in 83201t, in 83201t,Y30539, or in 74-OR8-1a and all but two (289-63, a double mutant, one of which is ts and the other is KCN resistant, and 295-20 which behaves as a cold sensitive mutant) are temperature sensitive at 38° in their growth in either liquid or in growth tubes. However their leaky nature makes the ts phenotype difficult to determine by simply growing them in test tubes of liquid medium. All isolates were selected first as ts mutants and then the respiratory defectives among them were selected by looking for KCN resistant respiration. That is, we assumed any mutation in the respiratory chain would induce the alternate pathway and make them KCN resistant. Thus most of the mutants are pleiotrophic in terms of growth at 25° and 38°, in terms of their abnormal cytochrome spectra, and their respiratory behavior in the presence of cyanide. And there are other defects (mainly enzymatic) in some of the mutants and I simply have not gone into detail about the complex phenotypes of ~~xxxx~~ each mutants. The details are in the manuscript soon to be published.

I think most can properly be described as ts-respiratory defective mutants. Since over half of them probably effect mitochondrial ribosome assembly or mitRNA, and thus indirectly mitochondrial protein synthesis, they are going to ~~effect~~ effect those phenotypes that depend on normal mitochondrial protein synthesis. Obviously the pleiotrophic nature of the mutants creates a problem in genotype designation. In my judgement Bertrand made a very serious mistake recently in designating a lot of low cytochrome isolates as cytochrome mutants when this is most certainly not true. We have taken the approach not to give them individual mutant genotype designations until the pleiotrophic nature is cleared up. I will be glad to discuss this with you when the right time comes.

( If you have any suggestions about this  
I would appreciate your opinion )