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Loss of Transposons When the Plasmid pCG86 Transferred into Laboratory Strain *E.Coli* K-12

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1. Introduction

In a recent article Dr S. Palchaudhuri has shown that the F plasmid of *E.coli K-12* was never a single replicon but precisely a co-integrate of two replicons, F replicon and R replicon; but these two replicons are never expressed simultaneously. Under the selection pressure of antibiotics abused in Tokyo hospitals, F replicon becomes R replicon. This happens because the transposon Tn1000 also present in F replicon controls the function of these replicons. Once this R replicon is activated then together with the transposons (Tn4 and Tn10), antibiotic resistance crisis begins. Our clinicians did not think of any new approach but they have been increasing the doses and duration of antibiotic treatment until now. In such battle between the pathogen and human medicine (antibiotics), our medicine has been defeated and therefore we have gone for a preventive alternative therapy with a low-calorie sugar five carbon sugar alcohol.

In 1977, the pathogen ETEC (enterotoxin producing Escherichia coli) carrying the plasmid pCG86, was isolated from a piglet with diarrhoea. The pCG86 carries antibiotic resistance transposons Tn 4 (streptomycin, sulfonamide) and Tn10 (tetracycline resistance character). In addition to these antibiotic resistance transposons and the same pCG86 plasmid carries genes for the production of heat-labile and heat-stable enterotoxin. This ETEC transfers the plasmid pCG86 into the laboratory strain *E coli K-12* or its derivative 711 by conjugation and stored in Dr. S Falkow's Research Laboratory but without any antibiotics. We have received this strain carrying the plasmid Ent P307 published in Journal Bacteriol (1975). The *E. coli K-12* strain 711was received by Professor W.K Mass for further characterization. After physical and genetic characterization, we found the presence of plasmid *E. coli K-12* 711 carries Ent p307 with genes for enterotoxin production (both heat labile and heat stable). We are wandering about the genesis of this strain 711 and Professor S.P was discussing with his mentor Professor W.K Mass to know the genesis of this strain. After few months Professor Mass found the answer-Dr. Carlton Gyles of Waterloo University, Canada sent the strain ETEC (pCG86), isolated from the pigs died from diarrhoea even after the treatment with antibiotics. This E. coli ETEC strain became resistance to antibiotic. We conclude that the strain 711(Ent p307) was evolved or originated from the same plasmid pCG86 but was present in ETEC. Surprisingly this plasmid pCG86 has lost specifically antibiotic resistance transposons (Tn4 and Tn10) during such storage Dr. Falkow's laboratory. This observation proves antibiotic resistance transposons can be lost if the growth environment is free from antibiotics. We were thinking then enterotoxin genes were not transposons! Later on, investigators had shown genes for heat stable Enterotoxin are flanked by the IS1 sequence in an inverted order. Dr. Sunil Palchaudhuri thinks many copies IS1 sequence is present in *E. coli K-12* chromosome.

2. Discussion

During the period 1972-1977 we have reported that the conjugation between the two F primes males (homosexuality) is not desirable even in the microbial world. If we force them to co-exist in the same *E. coli K-12*, the end- result is the distortion in its the

normal E. coli K-12 linkage map. Surprisingly even in a review article (1987), the two reputed investigators Drs N. Willetts and R. Skurray did not accept that the F plasmid is not a single replicon; but the co-integrate of two replicons(Palchaudhuri, 2021). These two replicons are not allowed to function simultaneously by the transposon Tn1000 (Figure 1). Significantly, the plasmid Ent p307 was born by the deletion of antibiotic resistance characters when the pCG86 is transferred from the ETEC to the laboratory strain *E. Coli K-12.* Specifically, the two antibiotic resistance transposons Tn4 and Tn10 are lost when transferred to *E. coli K-12* and stored in Agar stab without antibiotics. Since Ent p307 was born from pCG86, their replicon does not differ but this replicon is replicon of antibiotic resistance plasmid (R replicon). (Figure 1 and 2) are used to illustrate how Ent p307 was born from the plasmid pCG86 isolated from the pigs who died even in the presence of antibiotics.

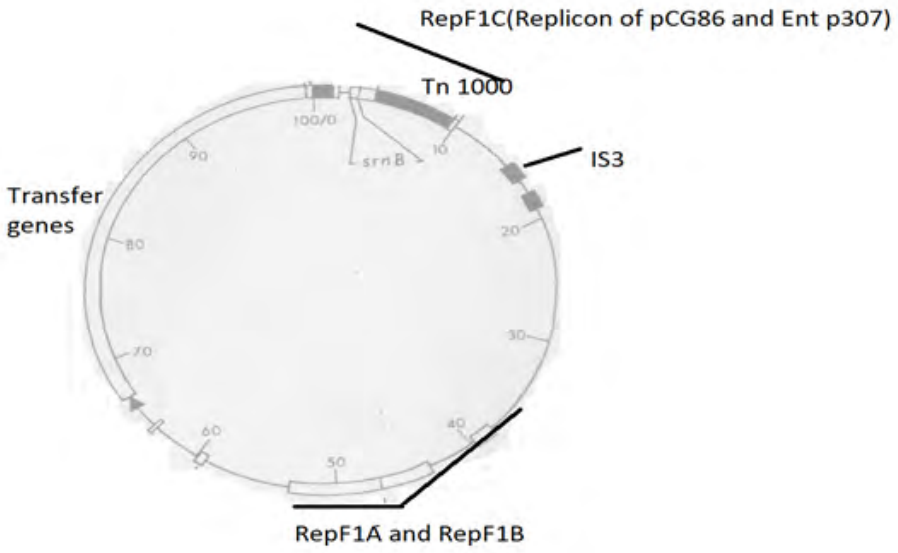

Figure 1: Map of F plasmid as co-integrate of two replicons {F1C and F1A/F1B} pCG86 and Ent p307(RepF1C). Transposon Tn1000 inserted into the replicon F1C.

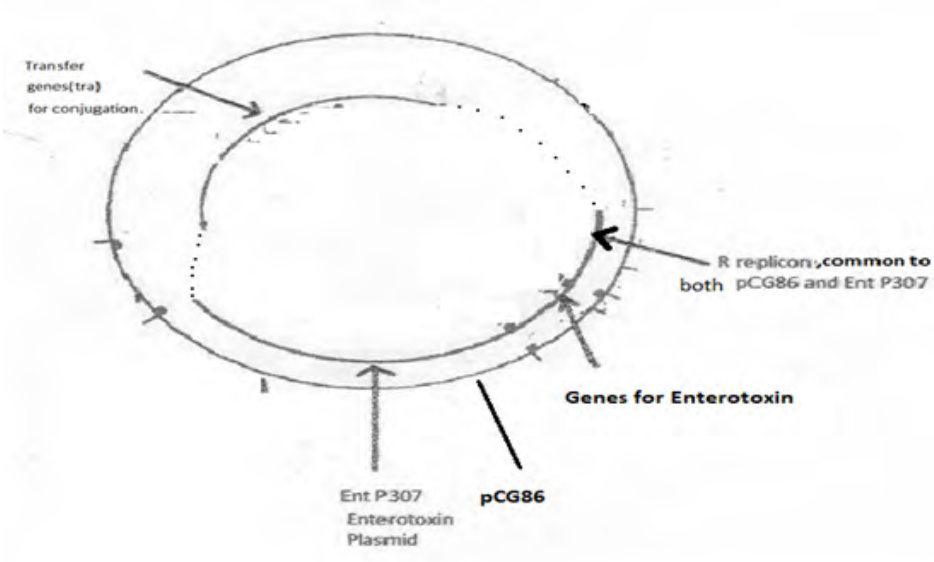

Figure 2: Plasmids pCG86 and Ent P307 has the same antibiotic resistance replicon(Rep F1C).