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International Journal of Radiology & Radiation Therapy

Short Communication

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X irradiation induces *E. coli C* from its semi-lysogenic state to lytic state

Abstract

Two strains of Gram-negative bacteria: *E. coli* K-12 and the *E. coli* C have been used in developing microbial genetics. These two bacteria have single chromosome of length 4736 Kb (double stranded DNA) that prevails in *E. coli* cytoplasm in the CCC form (Form I) but needs to be converted into its replicative form (Form II) to initiate replication. Significantly, their DNA sequences don't differ except the XYLITOL operon of *E. coli* C at the 46.5 min location when their chromosomes are measured in time unit. Not only their morphological look but also their growth pattern differs. Unlike *E. coli* K-12 the *E. coli* C grows in three phases- pre competent, competent and post competent.

Keywords: Xylitol, adhesion zone

Volume 11 Issue 5 - 2024

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Received: September 07, 2024 | **Published:** September 24, 2024

Short Communication

The two strains of Gram-negative *E. coli*: *E. coli* K-12 and *E. coli* C have been used in developing bacterial genetics. They have chromosomes almost of the same length (4637Kb) except the *E. coli* C carries Xylitol operon. In order to appreciate the difference between these two Gram-negative bacterial strains, I have drawn a diagram to make it clear that the chromosomes of same length (4637 Kb) except the *E. coli* C has xylitol operon chromosomes of these two *E. coli* accommodate in the cytoplasm but remaining attached to their cell walls (Figure 1). Due to presence of xylitol operon in *E. coli* C their morphology differs considerably.

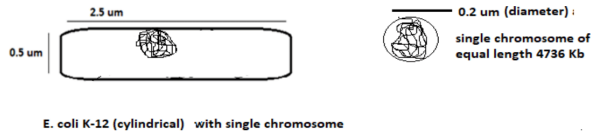

Figure 1 Shape and size difference between *E. coli* K-12 *and E. coli C.*

Gram negative *E. coli* C and the Gram-positive Streptococci pneumoniae grow in three phases: pre-competent, competent and post competent. It is already established that this pathogen with low G-C content is responsible for several respiratory diseases bacterial pneumonia, otitis media, meningitis etc. Interestingly another pathogen S.mutans (low GC content) not belonging to the Mitis group but causes dental caries in children.[1]

In 1960 Dr. Aurthur Kornberg shared Nobel prize with Dr. Severo Ochoa by specifying the importance of molecular biochemistry. Dr. Arthur Kornberg used the bacteriophage ΦX174 and its host *E. coli* C to study DNA replication at a molecular level without the knowledge of molecular genetics.[2,3] In 1963, I therefore decided to develop molecular genetics in my pre-doctoral project. After completion of M.Sc in physics (Calcutta University) I was looking for a mentor and met Dr. RK Poddar who just returned from the USA after his post- doctoral experience in the subject area. (Figure 2)

E. coli C is sensitive to phage phiX174 but the *E. coli* K-12 is not. Only difference is the presence of XYLITOL operon in *E. coli* C at 46.5 min but absent in *E. coli* K-12. We must not forget that the presence of XYLIYOL (operon) in *E. coli* C chromosome confers on it an ability to grow in three phases: pre-competent, competent with an ability to communicate by its bio-signalling and the post -competent (mother with progeny).[2]

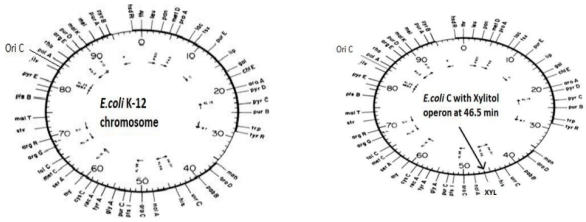

Figure 2 *E. coli* K-12 chromosome (100 min) with operons (e.g. leucine, threonine).

Briefly after the adsorption of ΦX174, delivery of its SS DNA genome takes place via its spike into the adhesion zone.[4,5] Evidently such adhesion zone is absent in *E. coli* K-12. What is this adhesion zone? Both membranes (outer and inner) and the cell wall (peptidoglycan) are fused where the SS DNA becomes double stranded using the enzyme DNA polA1 to convert into its replicative form (RF DNA) before its multiplication. Our article published in 1965 in BBA has demonstrated how the X-irradiation could distinguish the phage genome from its host *E. coli* C genome of length 4736 Kb(double stranded DNA, covalently closed circle) and ΦX174 genome of length 5.3 Kb (single stranded DNA, covalently closed circle). At the same dose of irradiation, the replication from the origin of *E. coli* C genome stops but the phage ΦX174 genome continues.[6,7] Dr. Arthur Kornberg used the polA1 of its host *E. coli* C in the multiplication of phage ΦX174. This phage phiX174 is semi- lysogenic. How does it differ from the lysogenic phage lambda? *E. coli* K-12 does not have adhesion zone.

When exposed to low dose of X-irradiation, the population of *E. coli* C is induced showing the phage ΦX174 DNA multiplies, encapsulated and lyses but few of them still prevails in the bacteriophage plaques of *E. coli* C. Figure 3 shows the delivery of ΦX174 genome into *E. coli* C by going into its adhesion zone. Nobel Prize winner A Kornberg has shown that the single stranded DNA genome of ΦX174 becomes double stranded DNA (replicative form), ΦX174 multiplies in *E. coli* C and produces bacteriophage ΦX174 particles (plaques).[4] In 1965 with the knowledge

of Molecular Biology I used X-irradiation to distinguish between the DNA replication of host *E. coli* C chromosome (double stranded CCC DNA) and DNA replication of its bacteriophage ΦX174(single stranded CCC DNA).[7] Thus, ΦX174 multiplied using the host's enzymes, matured with coat proteins and released as mature phage particles. I counted these phage particles and surprised to see one phage produces about few hundred particles! In nutrient agar medium these phage particles were visible. I incubated for another day or two, and observed few bacterial colonies growing in the clear zone (plaques). Based on data I want to mention the phage ΦX174 is a semi temperate or semi lysogenic unlike lambda bacteriophage which is lysogenic (Figure 3). Why does this phage ΦX174 infect *E. coli* C only but not *E. coli* K-12?

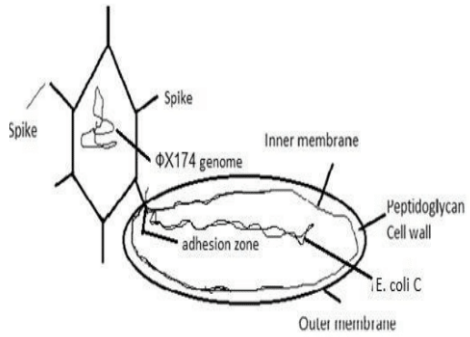

Figure 3 Entry of ΦX174 genome into the adhesion zone of *E. coli* C.

In 1967 in my Ph.D. dissertation work I reported that the size of *E. coli* C is very small but still it grows in three phases (pre competent, competent and post competent). The ΦX174 infects *E. coli* C by spiking using spikes on its surface. All the spikes are identical (unpublished data).

Morphologically these two bacteria (*E. coli* K-12 and *E. coli* C) differ considerably but *E. coli* C also differs in their sensitivity to the phage phiX174. This phage ΦX174 contains approximately 5.3 Kb genome (single stranded covalently closed circular) and is capable of making 11 proteins. Growth of Molecular Biology and bio-macromolecule helped to develop bacterial genetics. ΦX174 is a temperate phage or semi-lysogenic and adheres to enter into the adhesion zone of *E. coli* C. ΦX174 does not multiply in *E. coli* K-12 because there is no adhesion zone in *E. coli* K-12. These two bacterial chromosomes (double stranded DNA) replicate bi-directionally from the origin of replication at 84.5 min. Professor Arthur Kornberg initially showed the presence of DNA polymerase PolA1 but subsequently he discovered few more polymerases PolA2, PolA3, PolA4 and PolA5 in *E. coli* K-12 and in *E. coli* C.[4] We may think that the *E. coli* C chromosomally does not differ and may contain all the polymerases but the DNA replication of bacteriophage phiX174 as observed by him can't be generalized! Major question is how such a non- living bacteriophage becomes living by entering into the adhesion zone of *E. coli* C.

E. coli C (early log phase) is sensitive to the bacteriophage ΦX174 (single stranded covalently closed circular DNA). Is there any bio-signalling when the phage touches *E. coli* C (adsorption) by spiking (PhD thesis of Sunil Palchaudhuri, 1967 from Calcutta University). The chromosome of this phage is approximately 5.3 Kb chromosome but capable of producing 11 proteins. It seems to me that there is an overlap of operons and the growth of molecular biology. After attachment its genome (single stranded circular DNA) enters into the adhesion zone of *E. coli* C and converted into its replicative form, double stranded DNA (RF DNA)). Then ΦX174 genome, multiplies and forms complete bacteriophage particles after encapsulation (approximately 300) within few hours and released into its growth environment by the lysis of the host bacterium. Interestingly, if they are incubated for a longer period few bacterial colonies become visible even in their clear plaques. Are these bacterial colonies contaminants? Answer is "No". In order to understand, I made a comparison with the lambda bacteriophage which may prevail in the *E. coli* K-12 as a lysogen. The lysogenic bacteriophage lambda (double stranded DNA of length 46.5 Kb) prevails in two different states-lytic and lysogenic. In 1976 Palchaudhuri et al. have reported the dimerization of lambda (dsd) phage operon (23Kb to 46 Kb) appropriately to fit in its capsid.[8] This article published in J. Bacteriol confirms the mechanism (dimerization) used by lambda bacteriophage to use the same capsid for the continuity of its kind. Interestingly, the dimerization uses the homologous recombination between the two dsd operons. How is it activated? The dsd operon differs from the lac operon because it does not have any repressor gene.[9]

Acknowledgment

None

Conflicts of interest

Author declare that he is no conflicts of interest.

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