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T4 Bacteriophage-Coded Polynucleotide Kinase and RNA Ligase Are Involved in Host tRNA Alteration or Repair

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T4-induced polynucleotide kinase, RNA ligase, and a tRNA-specific endonuclease were previously implicated in host tRNA breakage and reunion. To examine this hypothesis we followed the fate of host tRNAs in T4-infected *Escherichia coli* CTr5x, a strain restricting phage mutants lacking polynucleotide kinase (pnk^-) or RNA ligase (rli^-). Two host tRNA species which were cleaved in the anticodon loops were further processed in the wild type but not in the mutant infections, indicating that polynucleotide kinase and RNA ligase are involved in the alteration or repair of these *E. coli* CTr5× tRNA species.

T4-coded polynucleotide kinase (*1*) and RNA ligase (*2*) have been thoroughly characterized *in vitro* (*3–5*) but their roles in phage physiology still need to be determined. We have previously proposed that both enzymes participate in reactions of host tRNA breakage and reunion, required for phage development in certain host strains (*6, 7*). By following the fate of host tRNA molecules in *Escherichia coli* CTr5× (*8*), a host strain restrictive to T4 mutants deficient in 3′-phosphatase- polynucleotide kinase (*8–10*) or RNA ligase (*11*), we demonstrate here the involvement of these T4 functions in the alteration or repair of host tRNA species.

We have recently shown that *E. coli* CTr5× differs from other *E. coli* strains which are permissive to T4 polynucleotide kinase (pnk^-) or RNA ligase deficient (rli^-) mutants in the cleavage pattern of host tRNAs following infection (*12*). Thus, while only leucine $tRNA_1$ is cleaved in T4-infected *E. coli* B (*13, 14*) and in other permissive hosts, two additional tRNA species are cleaved during infection of *E. coli* CTr5×. Contrary to leucine tRNA, which is cleaved in the extra-arm (*14*), the CTr5×-specific cleavages occur next to the anticodon and are mediated by a separate T4-induced tRNA-specific endonuclease (*12*). In the present experiment we compared host tRNA cleavage patterns during infection of *E. coli* CTr5× with either wild-type, pnk^- (*8, 9*), or rli^- (*15*) strains of T4. Figure 1 depicts the kinetics of host tRNA fragment appearance and disappearance during these infections. Both the wildtype and the mutant infections gave rise to six tRNA fragment bands. Bands I and IV originated from leucine $tRNA_1$ and are common to all T4-infected *E. coli* strains tested (*12–14*). Fragments (II and VI) and (III and V) correspond, respectively, to the 3′ and 5′ fragments of two CTr5×-specific tRNA species which are cleaved in their anticodon loop (*12*). In all these infections, leucine $tRNA_1$ fragments appeared and disappeared with a similar time course. However, a difference was noted between the wild-type and the mutant infections with respect to the behavior of the CTr5×-specific fragments. Thus, during wild-type infection the CTr5×-specific fragment bands reached maximal intensity between 4 and 6 min postinfection and then began to fade, having almost completely disappeared at 22 min postinfection. By contrast, in the pnk^- and rli^- infections these fragments appeared somewhat later than in the wild-type infection and persisted (II + VI) or seemed to have been only

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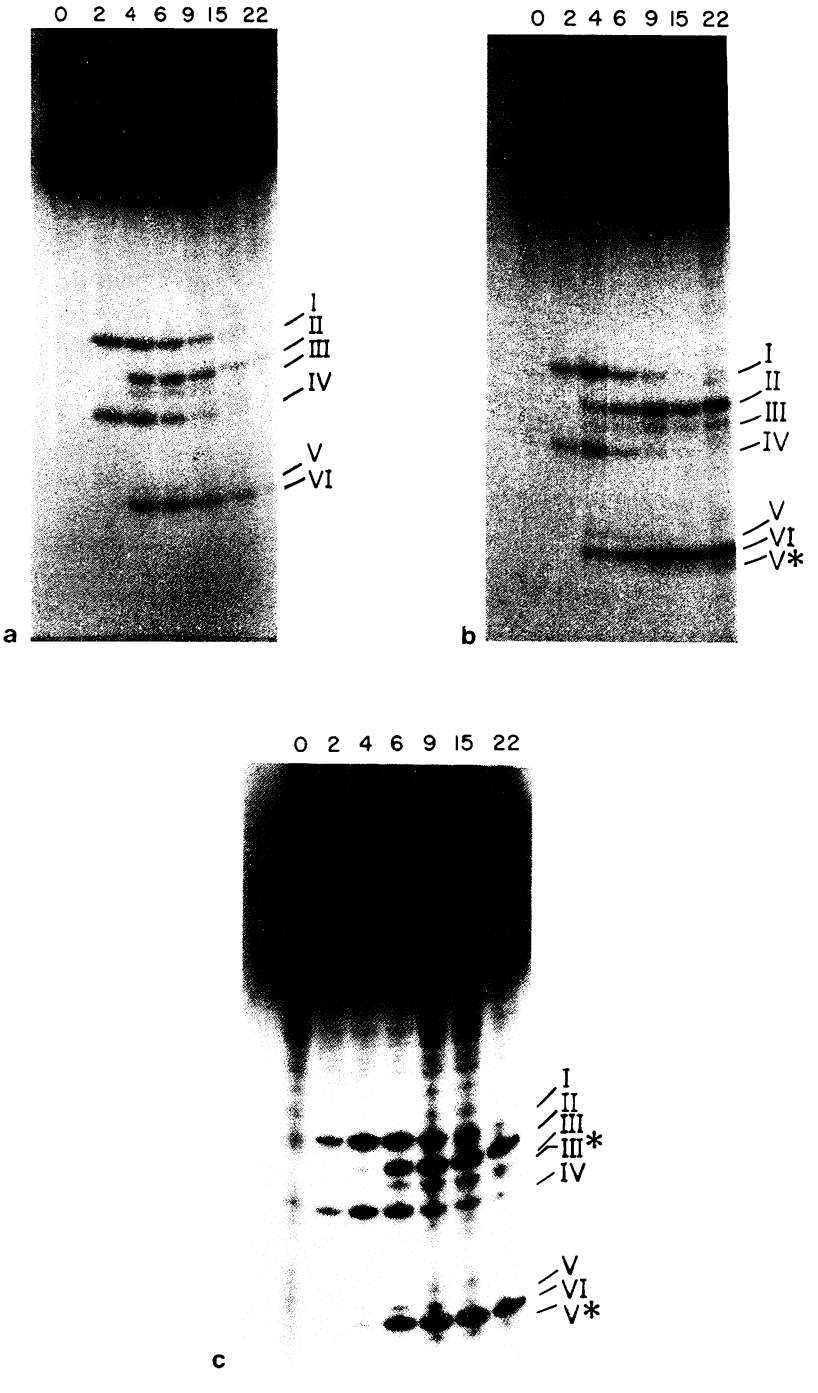

FIG. 1. *E. coli* CTr5× tRNA cleavage patterns during infection with T4 wild type (a), pnk^- (b), or rli^- (c) phage strains. *E. coli* CTr5× cells were pulse labeled with ^{32}P, chased with unlabeled P_i and then infected with the indicated phage strain. Culture aliquots were withdrawn before infection or at the indicated infection times, and RNA was extracted from them and separated by denaturing gel electrophoresis as previously described (*12*). The mutant strains employed were: *pse*T2 (polynucleotide kinase deficient, Ref. (*9*)) and *am* M69 (RNA ligase deficient, Refs. (*15, 16*).

slightly modified (III + V) at later infection times. Fragments II and VI remained virtually unchanged, both in quantity and electrophoretic mobility until 22 min in the *pnk*$^-$ and *rli*$^-$ infections. Fragment III did not change in the *pnk*$^-$ infection while in the *rli*$^-$ system it appeared to have been partially converted into a slightly faster migrating species designated III* (15- and 22-min lanes) which at 22 min constituted about one-half of the original band III. With both mutants, Band V completely disappeared within 15 min postinfection with a parallel appearance of a new band migrating somewhat faster than band VI and designated V*.

The failure of *pnk*$^-$ and *rli*$^-$ mutants to process further the CTr5×-specific tRNA fragments which disappeared late in the wild-type infection indicates that polynucleotide kinase and RNA ligase are normally involved in the alteration or repair of the corresponding tRNA species. Although the reactions which cause the "disappearance" of the CTr5×-specific tRNA fragments are not known presently, it seems reasonable to assume, based on the *in vitro* properties of polynucleotide kinase (*3*) and RNA ligase (*1, 5*), that these fragments undergo phosphoryl rearrangement and perhaps additional modifications prior to their ligation. The slight changes which seemed to occur with fragments III and V during the mutant infections (Fig. 1) may reflect such additional steps, presumably leading to an altered tRNA structure following the ligation. Alternatively, these changes could be due to abnormal degradation of the fragments whose processing was arrested by the mutation. The results also indicate that polynucleotide kinase and RNA ligase are not related to the removal of the leucine $tRNA_1$ fragments since this process was not affected by *pnk*$^-$ and *rli*$^-$ mutations.

Given the existence of a pathway which includes the T4-induced anticodon nuclease (*12*), polynucleotide kinase, RNA ligase, and perhaps additional processing enzymes, what do these host tRNA alterations, or breakage and subsequent repair, contribute to phage development and why are they needed in *E. coli* CTr5× and not in other host strains permissive to *pnk*$^-$ and *rli*$^-$ mutants? One explanation is that this putative pathway provides a suppressor tRNA species required later in T4 gene expression. Such a suppressor tRNA species may already exist in most T4 host strains but probably not in *E. coli* CTr5× (*7, 9, 10*) in which this function could be provided by the putative T4-induced alteration pathway. Supporting this view is the fact that *E. coli* CTr5× can be converted into a host permissive to a *pnk*$^-$ deletion mutant by transformation with an amber suppressor function (*10, 11*). Alternatively, polynucleotide kinase and RNA ligase may restore the tRNAs cleaved earlier by the anticodon nuclease without further change. We have recently isolated, among pseudorevertants of *pnk*$^-$ mutants able to grow on *E. coli* CTr5×, a phage lacking the anticodon nuclease activity. This double mutant forms small plaques on *E. coli* CTr5× but wild-type sized plaques on *E. coli* B, providing further evidence for a functional connection between the anticodon nuclease, polynucleotide kinase, and RNA ligase and suggesting that the alteration of host tRNAs, if it occurs, is not an "essential" function for T4.

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