Effect of the "Ribonucleic Acid Control" Locus in *Escherichia coli* on T4 Bacteriophage-Specific Ribonucleic Acid Synthesis

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Amino acid control of ribonucleic acid (RNA) synthesis in bacteria is known to be governed genetically by the *rel* locus. We investigated whether the *rel* gene of the host would also exert its effect on the regulation of phage-specific RNA synthesis in T4 phage-infected *Escherichia coli* cells. Since T-even phage infection completely shuts off host macromolecular synthesis, phage RNA synthesis could be followed specifically by the cumulative incorporation of radioactivity from labeled precursors into RNA of infected cells. Labeled uracil was shown to accumulate in phage-specific RNA for 30 to 35 min after infection, a phenomenon which probably reflects an expansion of the labile phage-RNA pool. Amino acid starvation was effected by the use of auxotrophic bacterial strains or thienylalanine. The latter substance is an amino acid analogue which induces a chemical auxotrophy by inhibiting the biosynthesis of phenylalanine, tyrosine, and tryptophan. Phage RNA synthesis was strictly dependent on the presence of amino acids, whereas phage deoxyribonucleic acid synthesis was not. By the use of several pairs of bacterial strains which were isogenic except for the *rel* gene, it was demonstrated that amino acid dependence was related to the allelic state of this gene. If the *rel* gene was mutated, amino acid starvation did not restrict phage RNA synthesis.

It has long been known that rapid ribonucleic acid (RNA) synthesis in bacteria (e.g., *Escherichia coli*) is dependent on the presence of amino acids (for review, see reference 2). Amino acid regulation of RNA synthesis has been shown to depend not merely on the presence of free amino acids, but also on a complete complement of aminoacyl-transfer RNA (tRNA; 10). There is also evidence for a link between amino acid regulation of RNA synthesis and an undisturbed peptide chain initiation, i.e., an even later step in protein synthesis (16). Transductional mapping has indicated that the *rel* locus, which governs the amino acid regulation, is situated between the *argA* and *cysC* loci of the Taylor-Trotter map (19).

Whether the *rel* gene affects a coordinate regulation of all the three types of RNA (i.e., whether messenger RNA (mRNA) as well as ribosomal RNA (rRNA) and tRNA synthesis is governed by amino acid regulation) has been the subject of many investigations, some of which have been contradictory. Because of the instability of mRNA and the marked reduction in the uptake of radioactive RNA precursors by amino acid-starved RC*str* bacteria, it has been difficult to determine mRNA regulation by RNA-labeling experiments in bacteria (3, 12). Specific measurements of tryptophan operon mRNA by hybridization assays have, however, demonstrated a non-coordinate regulation of this mRNA (4, 7). Similarly, induction of β-galactosidase mRNA was shown to occur in amino acid-starved RC*str* bacteria (9). The present investigation was undertaken with bacteriophage T4-infected *E. coli* cells. It is known that, at T-even phage infection, the host nucleic acid synthesis ceases and is replaced by RNA synthesis on the viral deoxyribonucleic acid (DNA) template (13). The RNA produced by infected cells will thus be phage-specific, i.e., mostly phage mRNA but also small amounts of phage-specific tRNA (21). All the label taken up into RNA from radioactive precursors after infection will thus represent phage-specific RNA synthesis. The aim of the investigation was to try to establish whether the synthesis of these phage-specific RNA species is governed by the *rel* gene of the host. Earlier studies have indicated that T-even phage-specific RNA synthesis is under amino acid regulation (15; Sköld, Abstr. 4th FEBS Meeting, p. 74, 1967).
MATERIALS AND METHODS

Chemicals. Amino acids, vitamins, thienylalanine, uracil, and thymidine were purchased from Nutritional Biochemicals Corp., Cleveland, Ohio. Uracil-\(^{14}\)C (30 mc/mmole) was obtained from New England Nuclear Chemicals, Dreieichenhain, Germany; thymidine-methyl-\(^{3}\)H (2,000 mc/mmole) from The Radiochemical Centre, Amersham, England; and \(^{32}\)P-phosphate from AB Atomenergi, Studsvik, Sweden.

Bacteriophage and bacterial strains. Wild-type bacteriophage T4D and a T4 mutant am 122 were provided by J. S. Wiberg. *E. coli* B-40 was used as a stringent, arg- bacteriophage, which was provided by O. Karlström. The K-12 strains Cp78, Cp83, Cp97, Cp99, and Cp100 were provided by Niels Fiil. Cp78, Cp83, and Cp97 were derived from *E. coli* K-12 W677, are F\(^{-}\), and require threonine, leucine, histidine, arginine, and thiamine. All three are isogenic except for the rel gene; Cp83 and Cp97 have RC\(^{rel}\) phenotypes, whereas that of Cp78 is RC\(^{estr}\) (6). Cp99 and Cp100 were derived from *E. coli* K-12 AB 325 and are F\(^{-}\) strains requiring histidine, arginine, serine, and thiamine. They are isogenic except for the rel gene; Cp99 is RC\(^{estr}\) and Cp100 RC\(^{rel}\) (6). Phage T4 was grown in *E. coli* B and the am 122 mutant in CR63 (obtained from N. Fiil); they were harvested as described earlier (17).

Media. All experiments were carried out in a mineral salts medium of the following composition per liter: 0.5 g of NH\(_4\)Cl, 0.5 g of (NH\(_4\))\(_2\)SO\(_4\), 5.0 g of NaCl, 0.5 g of KCl, 0.203 g of MgCl\(_2\)\(\cdot\)6H\(_2\)O, 14.7 mg of CaCl\(_2\)\(\cdot\)2H\(_2\)O, 0.178 g of NaHPO\(_4\)\(\cdot\)2H\(_2\)O, and 6.25 g of tris(hydroxymethyl)aminomethane (Tris); the pH level was adjusted to 8.0 with 1 M HCl, and after autoclaving the medium was supplemented with 5 g of glucose and FeCl\(_3\) to a final concentration of \(10^{-5}\) M. Supplements were amino acids (50 \(\mu\)g/ml) and thiamine (2 \(\mu\)g/ml).

Infected cells. Bacteria were grown to about 5 \(\times\) 10\(^8\) cells/ml in the low phosphate, mineral salts medium supplemented as necessary. The generation times were about 55 min for B, 50 min for B-40, and 60 min for the K strains. After cooling and harvesting by centrifugation, the cells were resuspended in 1/20 the volume of the same medium supplemented with glucose and L-tryptophan (10 \(\mu\)g/ml) and infected with 10 to 12 phages per bacterium. After 3 min of adsorption, the infected cells were diluted into aerated mineral salts medium (30 C, containing radioisotope). Phage adsorption, bacterial survival, number of infective centers, and burst size were routinely determined by standard techniques. Adsorption was usually better than 99\% at the end of the 3-min adsorption period, and bacterial survival was less than 1\%.

Measurement of RNA and DNA synthesis. The synthesis of RNA and DNA was determined by the cumulative incorporation of \(^{14}\)C-uracil, \(^{3}\)H-thymidine, or \(^{32}\)P-phosphate from the medium. When uracil or thymidine was used as a radioactive precursor, 1.5-ml samples were withdrawn at different times from the aerated, phage-infected cultures and precipitated in 7.5 ml of cold 6\% trichloroacetic acid for at least 1 hr. The samples were homogenized and divided into two halves. The precipitate of one half was immediately collected on a membrane filter (Millipore Corp. (HA) or Gelman (Metrical GA-6)); the other half was centrifuged, and the precipitate was dissolved in 1.5 ml of 1 N NaOH. RNA was then hydrolyzed at 37 C for 18 hr. After neutralization, DNA was reprecipitated with trichloroacetic acid and collected on a filter. Radioactivity was measured in a scintillation counter after immersing the dried filters in naphtalene-dioxane scintillation fluid. The amount of RNA radioactivity in each sample was calculated as the difference between the nonhydrolyzed and the hydrolyzed halves. The accuracy of the alkaline hydrolysis method is demonstrated in Fig. 1, where initial RNA

FIG. 1. Characteristics of the assay system for RNA synthesis in phage-infected bacteria. *E. coli* B was grown and infected by phage as described in the Methods section. After adsorption for 3 min at 20 C the infected complexes were diluted 10-fold into mineral salts medium containing \(^{14}\)C-uracil (10 \(\mu\)g/ml, 0.05 \(\mu\)c/ml). Uninfected bacteria were incubated at 20 C for 5 min and then diluted 10-fold into medium containing isotope. Samples (1.5 ml) were withdrawn at different times and treated as described in the text; however, in the case of the am 122-infected bacteria, the alkaline hydrolysis step was omitted. The values along the ordinate are expressed as counts per min of RNA or DNA per 1.5-ml sample. Phage was added at time zero. (\(\triangle\)) T4\(^{+}\) infection, RNA; (\(\times\)) am 122 infection, RNA; (\(\oslash\)) uninfected bacteria, RNA; (\(\odot\)) T4\(^{+}\) infection, DNA.
synthesis after T4+ infection is shown to be identical with that after am 122 infection (no DNA synthesis, no alkaline hydrolysis). When 32P-phosphate was used as a radioactive precursor, 2.5-ml samples were withdrawn and precipitated in 2.5 ml of 10% trichloroacetic acid. The precipitates were collected and washed three times by centrifugation with 5 ml of 5% trichloroacetic acid. Nucleic acids were extracted and purified by the procedure of Davidson and Smellie (18). The recrystallized samples were finally dissolved in 1 ml of 0.1 N NaOH, and RNA was hydrolyzed for 22 hr at 37°C. DNA was then precipitated by the addition of 0.2 ml of 1 N HCl (30 min at 0°C) and centrifuged off. A 0.6-ml sample from the supernatant fraction containing the free ribonucleotides was transferred to a scintillation vial, evaporated to dryness, dissolved in 1 ml of 0.1 N Tris, and counted in the scintillation counter after the addition of 10 ml of dioxane-naphthalene scintillation fluid.

RESULTS

Characteristics of the assay system for RNA synthesis in phage-infected bacteria. The cumulative incorporation of [14C]-uracil into E. coli B with and without phage is demonstrated in Fig. 1. In the case of wild-type T4 infection, there is a continuous increase in RNA label until about 30 to 35 min after infection, at which time a plateau level is reached, which then remains constant for another 15 to 20 min. One experiment was performed with the T4 mutant am 122, which is deficient in gene 42 and will not induce the formation of deoxyctydylate hydroxymethylase and, thus, of no DNA in E. coli B. Since there is no DNA synthesis in am 122-infected E. coli B, there is no formation of late mRNA (Fig. 1; reference 1). It can be seen that [14C]-uracil incorporation into RNA closely follows that of T4+-infected cells for 20 min, at which time the radioactivity content in RNA becomes constant for a short time and then gradually decreases. The cumulative incorporation of [14C]-uracil into uninfected cells is also demonstrated in Fig. 1. The increase in label is linear throughout the experimental period of 40 min, at which time it reaches a level which is more than eight times higher than that of T4+-infected cells at 40 min. This reflects the massive incorporation of [14C]-uracil into stable RNA. Figure 1 also shows the DNA synthesis of T4+-infected cells as measured by [14C]-uracil incorporation into acid-precipitable, alkali-resistant material. Phage DNA synthesis is observed to start 12 to 19 min after infection and then to proceed at a high rate through the 50-min experimental period.

A separate one-step growth control of the T4- E. coli B system in mineral salts medium (30°C) showed the eclipse period to be about 25 min and the burst size about 170 per infective center.

Phage RNA synthesis in E. coli B starved for amino acids by thienylalanine. The phenylalanine analogue thienylalanine is known to inhibit the biosynthesis of phenylalanine, tyrosine, and tryptophan (5). Turnock and Wild demonstrated that thienylalanine inhibited protein synthesis to the same extent in RCstr and RCrel bacteria, but inhibited RNA synthesis much more in an RCstr than in an RCrel bacterial strain (20). The analogue is thus useful in the study of amino acid regulation of RNA synthesis in bacteria. In the present investigation, phage-infected E. coli B was made chemically auxotrophic for the aromatic amino acids with thienylalanine to study the amino acid regulation of T4 RNA.

The inhibitory effect of different concentrations of thienylalanine on phage production in E. coli B is demonstrated in Table 1. It can be seen that more than 30 μg of inhibitor per ml reduced phage production to an insignificant level. The thienylalanine inhibition of phage production at 50 μg/ml could be reversed completely by the addition of tyrosine, tryptophan, and phenylalanine, each to a final concentration of 50 μg/ml. This inhibition reversal could be observed as late as 40 min after the addition of thienylalanine.

The curves of Fig. 2 demonstrate the effect of thienylalanine on RNA synthesis in E. coli B infected with phage T4. The formation of RNA was measured as incorporation of 32P-phosphate into RNA nucleotides. It can be seen that the presence of thienylalanine inhibited 32P-phosphate incorporation almost completely and that the inhibition was reversed by the addition of phenylalanine, tyrosine, and tryptophan. A similar inhibition of [14C]-uracil incorporation into T4 phage RNA was observed to take place in

| TABLE 1. Inhibitory effect of thienylalanine on phage production in Escherichia coli B* |
|-----------------------------------------|-------------|
| Thienvlalanine | Burst size |
| μg/ml | %    |
| 0    | 100.0 |
| 1    | 54.4  |
| 3    | 13.9  |
| 10   | 7.0   |
| 15   | 1.8   |
| 30   | 0.4   |
| 40   | 0.4   |
| 50   | 0.2   |

* After phage adsorption for 3 min the infected complexes were diluted 10-fold into medium containing the different concentrations of thienylalanine. The suspensions (5 ml) were aerated at 30°C for 90 min and then induced to lyse with chloroform. The burst size without inhibitor was 158 per infective center.
the presence of thienylalanine. At the addition of aromatic amino acids, uracil incorporation into RNA started with a rate similar to that of the control (data not shown).

Effect of amino acid starvation on phage nucleic acid synthesis in an E. coli B strain auxotrophic for arginine. The synthesis of nucleic acids in E. coli B-40 infected with phage T4 and starved for arginine is demonstrated in Fig. 3. It is shown that the incorporation of 14C-uracil into phage nucleic acids was reduced dramatically during arginine starvation. Addition of arginine at 20 min after infection was accompanied by an immediate increase in uracil uptake which reached a rate similar to that of the control. A longer time is allowed for the complete commitment of bacteria to phage-specific RNA synthesis in the experiment shown in Fig. 4, where the initial 10 min of phage infection of E. coli B-40 took place in the presence of arginine, which was then removed by filtration and finally added back at 29.5 min. A few minutes after the initiation of amino acid starvation, the incorporation of 14C-uracil into RNA stopped completely and resumed slowly at
the readdition of arginine. The uracil incorporation into phage DNA did, however, continue during amino acid starvation, obviously supported by early enzymes formed during the initial 10 min in the presence of amino acid. For comparison, the same experiment without phage infection is shown in Fig. 4. After readdition of arginine to uninfected bacteria, the uracil incorporation into RNA resumed at an intensive rate, which did not change during the last 20 min of the experiment (data not shown). This massive incorporation represents the formation of stable bacterial RNA.

Effect of amino acid starvation on phage nucleic acid synthesis in E. coli K strains which are isogenic except for the rel-gene. To investigate the relation between the function of the rel gene and the regulation of phage-specific RNA synthesis, several bacterial strains, which were isogenic except for the rel gene, were tested for their ability to support T4 RNA synthesis during amino acid starvation. In Fig. 5, the $^{14}$C-uracil incorporation during amino acid starvation into isolated RNA was compared in three T4-infected K strains, two of which are RC$^{rel}$ (Cp83, Cp97) and one RC$^{str}$ (Cp78). Phage adsorption in this case took place in the absence of the required amino acids. It can be seen that the $^{14}$C-uracil uptake in RNA during amino acid starvation was quite small in the RC$^{str}$ strain compared to that of the two RC$^{rel}$ strains. When starvation was ended at 29.5 min by addition of the required amino acids, the uptake of radioactive uracil in phage RNA of the RC$^{str}$ cells resumed without any observable delay. No DNA synthesis could be observed during amino acid starvation in any of the three bacterial strains; when the amino acid starvation period was ended, DNA synthesis started with a delay of about 8 min, with the same rate in both the RC$^{str}$ and the RC$^{rel}$ strains (data not shown). For comparison, a curve describing $^{14}$C-uracil incorporation into RNA of T4-infected Cp83 (RC$^{rel}$) in the presence of all required amino acids is included in Fig. 5. The two curves for

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**FIG. 4.** Synthesis of RNA and DNA during amino acid starvation of phage-infected and uninfected E. coli B-40. The experimental procedure was similar to that described in the legend to Fig. 3; however, after adsorption the infected cells were suspended in medium containing 50 $\mu$g of arginine per ml. Amino acid starvation was initiated by filtration 10 min after infection and 50 $\mu$g of arginine per ml was added back at 29.5 min (arrow). Uninfected cells were treated similarly. Ordinate: radioactivity per 1.5-ml sample of RNA and DNA, respectively. (●) RNA, infected cells; (X) RNA + DNA, infected cells; (△) RNA, uninfected cells; (○) RNA + DNA, uninfected cells.

**FIG. 5.** Effect of amino acid starvation on the synthesis of phage RNA in Cp78, Cp83, and Cp97. The experimental procedure was similar to that described in the legend to Fig. 3. Phage adsorption took place in the absence of the required amino acids, and starvation was continued after dilution of the infected complexes and finally ended at 29.5 min (arrow) by the addition of amino acids, each to a final concentration of 50 $\mu$g/ml. One experiment with Cp83 was carried out with all required amino acids present from infection throughout the entire experimental period. Ordinate: total radioactivity in RNA per 1.5-ml sample. (X) Cp78 (RC$^{str}$); (○) Cp97 (RC$^{rel}$); (●) Cp83 (RC$^{rel}$); (●) Cp83, with amino acids.
Cp83, with and without amino acid starvation, are almost identical, demonstrating that the phage infection with subsequent shut-off of host RNA synthesis was also well-established when amino acid starvation was initiated during the adsorption period.

The observation that the phage was also able to induce shut-off of host RNA synthesis when infection took place in the absence of the required amino acids indicates a small phage protein synthesis, at least initially. This could be supported by residual pools of amino acids after filtering bacteria or by host protein turnover. This is also illustrated in Fig. 6, which describes experiments in which DNA synthesis during amino acid starvation was compared in T4-infected and uninfected Cp78 and Cp83, respectively. Amino acid starvation was initiated at time zero, i.e., during adsorption in the phage-infected cultures. In spite of adsorption in the absence of the required amino acids, phage infection almost completely inhibits the DNA synthesis which occurs in uninfected bacteria during amino acid starvation. When amino acids were added at 29.5 min, phage DNA synthesis started after a latency of 6 to 8 min.

A different pair of strains, isogenic except for the rel gene, were also tested for their ability to support phage-RNA synthesis during amino acid starvation. Figure 7 illustrates 14C-uracil incorporation into phage RNA in T4-infected Cp99 (RC*tr) and Cp100 (RC*rel), respectively. Amino acid starvation was initiated by filtration 10 min after the addition of phage and was ended 19.5 min later. It is shown that 14C-uracil incorporation into phage RNA in the RC*tr strain diminished and finally stopped during the amino acid starvation period (Fig. 7A). It resumed without observable delay at the re-addition of amino acids. The same observations could be made when the same strain was starved for the required amino acids already during adsorption (Fig. 7A). In the RC*rel strain, there was no decrease in 14C-uracil incorporation into phage RNA during amino acid starvation (Fig. 7B).

**Fig. 6.** Effect of amino acid starvation on DNA synthesis in T4-infected and uninfected Cp78 and Cp83. Phage adsorption was performed as described for Fig. 5. The infected complexes or uninfected bacteria were diluted 10-fold into medium containing 3H-thymidine (20 μg/ml, 0.5 μCi/ml). At 29.5 min (arrow), amino acids were added, each to a final concentration of 30 μg/ml. Ordinate: total activity (counts/min) per 1.5-ml sample after alkaline hydrolysis and reprecipitation. (X) Cp83 (RC*rel), uninfected; (Δ) Cp83, T4-infected; (○) Cp78 (RC*tr), uninfected; (●) Cp78 (RC*tr), T4-infected.

**Fig. 7.** Effect of amino acid starvation on phage RNA synthesis in Cp99 and Cp100. The experimental procedure was similar to that described in the legend to Fig. 4. The first 10 min of the experiment (including adsorption, 3 min) took place in the presence of the required amino acids. Amino acid starvation was then initiated by filtration. It was ended at 29.5 min (arrow) after infection by the addition of the required amino acids, each to a final concentration of 50 μg/ml. The experiment belonging to the lower curve of part A was performed as described in the legend of Fig. 5, i.e., amino acid starvation was initiated at injection. At 29.5 min (arrow), amino acids were added, each to a final concentration of 50 μg/ml. Ordinates: total activities (counts/min) in RNA per 1.5-ml sample. A: ○, Cp99 (RC*tr); X, Cp99 (RC*tr), amino acid starvation from time zero. B: ○, Cp100 (RC*rel).
DISCUSSION

The aim of the experiments was to determine whether T4 phage-specific RNA synthesis is dependent on the presence of amino acids, i.e., whether phage RNA synthesis is governed by the rel gene of the host. Phage RNA synthesis was mostly determined by measuring the cumulative incorporation of 14C-uracil into RNA, and it is thus important to consider how this reflects true phage-specific RNA synthesis.

It has been known for a long time that T-even phage infection induces a rapid and complete stop in host-specific RNA synthesis (13, 14).

This would mean that all the 14C-uracil incorporation into RNA after phage infection represents phage-specific RNA synthesis. Nierlich has shown, however, that an RNA base will not enter the intracellular nucleotide pool if there is no utilization of nucleotides from this pool for net RNA synthesis (11, 12). The incorporation of 14C-uracil into RNA of T4-infected bacteria (Fig. 1) must then represent a net synthesis of phage-specific mRNA. This expansion of the labile phage mRNA pool seems to end about 30 to 35 min after infection with wild-type T4 phage and at about 20 min with the early gene mutant (am 122, gene 42). The difference could be explained by the synthesis of late mRNA, which will not take place with the mutant because of its inability to induce phage DNA synthesis (1).

Phage-specific RNA synthesis measured by 14C-uracil uptake in E. coli B was restricted by thienylalanine, which inhibits the formation of aromatic amino acids and thus creates amino acid starvation. The inhibition of RNA synthesis was reversed by the addition of the appropriate amino acids and the observed restriction is interpreted as stringent amino acid regulation of phage-RNA synthesis. The same conclusion was drawn from the inhibition by thienylalanine of 32P-phosphate incorporation into phage RNA. Amino acid starvation experiments with a T4-infected E. coli B strain, auxotrophic for arginine, also indicated stringent regulation of phage RNA synthesis (i.e., 14C-uracil uptake ceased upon withdrawal of arginine and immediately resumed upon its readdition). Phage DNA synthesis continued during starvation, ruling out the possibility of a simple effect on pyrimidine formation.

Several experiments with amino acid auxotrophic, RCstr, E. coli K strains also demonstrated the stringent amino acid dependence of 14C-uracil incorporation into phage RNA. In strains which were isogenic with these, except for carrying mutations in the rel gene, 14C-uracil incorporation into phage RNA was independent of the required amino acids. See, for example, the two curves for Cp83 (RCrel) in Fig. 5, which compare the phage-specific RNA synthesis with and without amino acid starvation. The two curves are almost identical, showing amino acid independence, in sharp contrast to the curve obtained with Cp78, which is the corresponding RCstr strain. In the latter case, a strong amino acid dependence was demonstrated. Similar observations were made with other pairs of RCstr and RCrel but otherwise isogenic strains. It thus seems that the rel gene of the host controls the RNA synthesis of the phage. It could be argued that amino acid starvation would not allow the phage to shut off stable host RNA synthesis completely (13, 14), and that these experiments only demonstrate normal amino acid regulation of host RNA synthesis. This interpretation is ruled out by three independent experimental findings.

(i) The experiment described in Fig. 6 shows that phage infection of Cp78 and Cp83 was able to shut off host-specific DNA synthesis completely under the conditions used for amino acid starvation. This finding also indicates that host-specific RNA synthesis is completely abolished, because Nomura et al. (14) could demonstrate that the phage inhibitory effect on host RNA synthesis was larger than that on host DNA synthesis in the absence of protein synthesis.

(ii) The two curves obtained for Cp83 (RCrel), with and without amino acid starvation, were almost identical (Fig. 5). If amino acid starvation were to interfere with phage-induced inhibition of host RNA synthesis, the 14C-uracil incorporation would have been much higher in the starved culture (Fig. 1) because stable RNA synthesis of uninfected Cp83 will be almost unimpaired, at least initially, by amino acid starvation (6).

(iii) In many experiments, the amino acid starvation of the phage-infected bacteria was not initiated until after 10 min of infection, thus allowing substantial phage-specific protein synthesis and phage-induced changes in host macromolecular synthesis.

In many of the experiments shown, it was seen that, at the time of readdition of amino acids, there was a decrease in phage RNA label accumulated during amino acid starvation of a phage-infected RCrel strain (see Fig. 5). This might be interpreted as an increased instability of the phage mRNA as a result of the reinitiated protein synthesis (8).

In conclusion, the results presented in this paper indicate that the T4 phage-specific RNA synthesis is under amino acid control and is effected by the rel gene of the host.
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LITERATURE CITED


