BIOSYNTHESIS OF PENTOSE IN *ESCHERICHIA COLI*

Synthesis of Deoxyribose in Cells Infected with Bacteriophage

ELMER M. WRIGHT,¹ HENRY Z. SABLE,² AND JOY L. BAILEY³

Department of Biochemistry, Western Reserve University, School of Medicine, Cleveland, Ohio

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The biosynthesis of ribose and deoxyribose in *Escherichia coli* involves both the oxidative pathway through 6-phosphogluconate and the nonoxidative pathway through the transketolase and transaldolase sequence (Lanning and Cohen, 1954; Bernstein, 1956; Bagatell, Wright, and Sable, 1959). The proportions of the oxidative and nonoxidative pathways depend on the nature of the principal carbon source in the medium, on the stage of growth of the culture, and on the previous history of the culture (Szykiewicz, Sable, and Pfueger, 1961). In addition evidence has been presented that ribose and deoxyribose have a common origin in this organism (Bernstein and Sweet, 1958; Bagatell et al., 1959). Two studies (Lanning and Cohen, 1955; Loeb and Cohen, 1959) suggest that in bacteriophage-infected *E. coli* the nonoxidative pathway becomes of greater importance. The studies reported here support the view that both pathways are of considerable importance in the biosynthesis of deoxyribose in the presence of bacteriophage infection.

Racker (1952) found an aldolase in *E. coli* that catalyzes the reversible reaction:

\[
\text{CH}_2\text{OPO}_4\text{H}_2^{-} + \text{CHOH}-\text{CHO} \leftrightarrow \text{CH}_2\text{OPO}_4\text{H}_2^{-} + \text{CHOH}-\text{CHO} - \text{CH}_2\text{-CHO}
\]

and it has been considered that this reaction or a similar type of aldol condensation of 3-carbon and 2-carbon precursors might be an important mechanism in the biosynthesis of deoxyribose. Evidence is presented here that the biosynthesis does not occur by such an aldol condensation.

**MATERIALS AND METHODS**

*E. coli* strain R-2 and bacteriophage strain T2H were maintained and cultured as described in detail in an earlier publication (Sable et al., 1960). Deoxyribonuclease was Dornaee (Merck, Sharpe and Dohme). Phosphodiesterase was prepared by the method of De Garile and Laskowski (1955) from rattlesnake venom (lyophilized venom of *Crotalus adamanteus*, supplied by Ross Allen's Reptile Institute). Prostatic acid phosphatase was purified by the method of Schmidt et al. (1951). Radioactive glucose was purchased from the National Bureau of Standards and from Volk Radiochemicals. Ultraviolet absorption was measured with a spectrophotometer. Turbidimetric determination was carried out in a colorimeter with a no. 66 filter.

Radioactive bacteriophage was produced as follows: *E. coli* was first adapted to grow on acetate and a large inoculum of an acetate-grown culture was transferred to S/5-0.1% glucose-0.005% casein hydrolyzate, (see Sable et al. (1960) for the composition of the medium) to give a cell concentration corresponding to 20 Klett units (2 × 10⁶ cells/ml). The cultures were then incubated aerobically at 37°C and optical density measurements taken at intervals. When the optical density had doubled a 4- to 6-fold multiplicity of phage was added, and 5 min later the radioactive glucose was added. The culture was agitated continuously during these additions. Samples were taken for counts of viable cells (Adams, 1950) just before infection and 15 min after infection. The cultures were incubated aerobically for 7.5 hr more at 37°C, and then stored at 3°C overnight. Bacteriophage was then isolated as described in the earlier publication.

**Separation of bacteriophage carbohydrates.** The glucose and deoxyribose components of the bacteriophage were isolated and degraded in each experiment. Since intact bacteriophage was known to be resistant to deoxyribonuclease, various methods were investigated which would release the deoxyribonucleic acid (DNA) from

¹ Present address: Twin Falls, Idaho.
² Markle Scholar in Medical Sciences.
³ Present address: Virus Research Laboratory, University of California, Berkeley.
the virus. In work with uninfected *E. coli*, the DNA was found to be normally sensitive to deoxyribonuclease after the cell residue was incubated in aqueous alkali (Bagatell et al., 1959). When bacteriophage was incubated in 1 N NaOH overnight at 37 C the DNA became sensitive to deoxyribonuclease; however, the alkali treatment alone converted a considerable proportion of the DNA to a form soluble in 5% HC104; the DNA, therefore, appeared to have undergone rather serious degradation. Better results were obtained by heating the virus. The opalescent concentrate of bacteriophage in 0.5% NaCl and 0.01 M tris (hydroxymethyl) aminomethane (tris) buffer, pH 8.0 was placed in a boiling water bath until a flocculent precipitate formed, leaving a clear supernatant liquid; 15 to 30 min heating usually sufficed. The precipitate was removed by centrifugation and washed once with buffer, the washings being added to the original supernatant liquid. All of the DNA was found in the solution, in a form which was still precipitated by 5% HC104. This DNA solution was then digested with deoxyribonuclease and phosphodiesterase and the degradation products separated on Dowex 1-acetate, by the methods of Bagatell et al. (1959) with the following modifications. More phosphodiesterase was used per unit of bacteriophage DNA than had been used for bacterial DNA, since preliminary experiments showed a slower rate of degradation of the oligonucleotides derived from the bacterial DNA. The ion exchange separation was also modified somewhat. In the case of nucleotide mixtures derived from DNA of uninfected cells, four major peaks were eluted, corresponding to deoxyctydylidic, thymidylidic, deoxyadenylidic, and deoxyguanylic acids. In the T-even bacteriophages, cytosine is replaced by hydroxymethyl cytosine (Wyatt and Cohen, 1953) and enzymatic digestion of the DNA liberates very little hydroxymethyl cytosine mononucleotide. Most of the latter nucleotide and about one-fourth of the other nucleotides are converted to oligonucleotides of low molecular weight (Volkin, 1954; Sinsheimer, 1954). These oligonucleotides remained on the column after the last mononucleotide was eluted with ammonium acetate. The column was then washed with water and the oligonucleotides were eluted (but not resolved into separate fractions) with 1 N HCl, and the eluates neutralized with NaOH.

The radioactive purine nucleotides were diluted with carrier nucleotide and were converted to deoxyribose 5-phosphate by hydrolysis in dilute H2SO4 as described by Wright and Sable (1959). Glucose, which occurs in the DNA of T-even bacteriophages as the glucoside of hydroxymethyl cytosine nucleotide (Volkin, 1954; Jesaitis, 1957; Loeb and Cohen, 1959) was isolated in the following manner. The radioactive oligonucleotides were first separated from inorganic salts by treatment of the acidified solution with active charcoal (Darco G-60). The nucleotides were eluted from the charcoal with ammoniacal 50% ethanol, yielding a solution containing the ammonium salts of the nucleotides and a trace of colloidal charcoal. The nucleotide concentration was estimated from the ultraviolet absorption at 260 mμ, and carrier oligonucleotide derived from pooled DNA of several nonradioactive preparations was added. The nucleotides were again adsorbed on charcoal and eluted as before. The solution was evaporated to dryness, the dry residue was dissolved in 5 ml of 1 N H2SO4, and the solution was heated for 3 hr at 100 C to hydrolyze the glucosides. The solution was then neutralized with Ba(OH)2 to remove the H2SO4, equivalent amounts of Ba(OH)2 and ZnSO4 were then added to remove some of the impurities, and the solution was filtered. Glucose was assayed spectrophotometrically by the specific enzymatic method used by Cori and Larner (1951). Carrier glucose was added and the solution was deionized with Duolite A-4 and Dowex 50 resins. Residual colored and ultraviolet absorbing impurities were removed by a final treatment with charcoal and the glucose was then purified by chromatography on a cellulose column eluted with 34-saturated aqueous butanol.

**Determination of isotope distribution.** The
glucose was degraded by fermentation with *Leuconostoc mesenteroides* according to Bernstein and Wood (1957), except that the acetic acid was degraded by the method of Phares (1951); labeling of deoxyribose was determined by degradation of deoxyribose 5-phosphate according to Wright and Sable (1959). The radioactivity was measured in two ways. The CO₂ samples from degradation of deoxyribose were plated as BaCO₃ on filter paper discs and counted in a Tracerlab windowless gas flow counter. All counts were converted to "infinite thinness" by a self-absorption correction factor (Kamen, 1957). The CO₂ derived from glucose was counted in a gas-phase proportional counter (Bernstein and Ballentine, 1950). All specific activities have been expressed as cecuries per mmole, to permit direct comparison of the radioactivities. The contributions of the biosynthetic pathways were calculated as described by Szynkiewicz et al. (1961) by comparing the distribution of C₁⁴ found in the deoxyribose with that found in the glucose isolated from the phage DNA.

### RESULTS

**Test for direct C-2 and C-3 condensation to yield deoxyribose.** An experiment was designed to detect direct condensation of 3-carbon and 2-carbon compounds by the mechanism studied by Racker (1952). The tracer substance used was glucose-6-C₁⁴. It was assumed that the 3-carbon compound would be derived from C-1,2,3 and C-4,5,6 of the hexose in such a way that the labeled carbon (C-6) would become the hydroxymethyl carbon (C-3) of the triose. The 2-carbon substance should be derived from C-1,2 and C-5,6 of the hexose. Depending on the pathways of formation of the 2-carbon substance, either or both of its carbon atoms might contain the tracer (Bagatell et al., 1959) and the deoxyribose would be labeled in C-5 and C-1; C-5 and C-2; or C-5, C-2, and C-1. The results are shown in Table 1. Both the glucose and deoxyribose were labeled almost exclusively in C-6 and C-5, respectively. Unless one formulates some hitherto unknown pathway for formation of an unlabeled 2-carbon compound from glucose-6-C₁⁴, he must conclude that none of the deoxyribose was formed by the direct condensation reaction as described above. In view of the evidence (Bernstein and Sweet, 1958; Bagatell et al., 1959; Szynkiewicz et al., 1961) that deoxyribose is derived from ribose in uninfected cells, it seems reasonable to conclude that this is also the case in the infected cells.

The experiment with glucose-6-C₁⁴ gives little information about the synthesis of pentose from hexose, since the labeling of C-1 and C-2 of the hexose is much too low to permit conclusions to be drawn from these carbons about the participation of the different pathways. It is seen that C-5 of pentose has only about half the specific activity of C-6 of hexose, indicating a considerable contribution of an unlabeled precursor to C-5 of the pentose. Some of this dilution may be due to the incorporation of preexisting nonradioactive cell DNA into the phage, but much of it probably arises from the contribution of C-1,2,3 of the hexose to C-3,4,5 of the pentose by aldolase plus the transketolase-transaldolase pathway. It should also be noted that the absence of any significant incorporation of radioactivity into C-1

### Table 1

**Biosynthesis of bacteriophage carbohydrates from glucose-6-C₁⁴**

<table>
<thead>
<tr>
<th>Carbon Atom</th>
<th>Relative Specific Activity*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>DNA glucose</td>
</tr>
<tr>
<td>1</td>
<td>4.7</td>
</tr>
<tr>
<td>2</td>
<td>4.2</td>
</tr>
<tr>
<td>3</td>
<td>0.3</td>
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<tr>
<td>4</td>
<td>0.2</td>
</tr>
<tr>
<td>5</td>
<td>0.6</td>
</tr>
<tr>
<td>6</td>
<td>100†</td>
</tr>
</tbody>
</table>

* For each sugar the carbon atom with the highest specific activity is arbitrarily assigned a value of 100.
† Observed specific activity: C-6 of glucose = 2.58 μc per mmole carbon; C-5 of deoxyribose = 1.37 μc per mmole carbon.

*Escherichia coli* was washed from a slant and transferred twice in S/2-0.5% sodium acetate. Samples of the final culture were transferred to three 1,800-ml portions of S/5-0.1% glucose-0.005% casein hydrolysate (Klett 24) and the cultures incubated at 37 °C for 2½ hr (Klett 41). Each of the three cultures was infected with 4.2 × 10⁸ phage (phage:cells = 5.3:1). Five minutes later, a total of 50 ml glucose containing 140 μc of glucose-6-C₁⁴ was distributed among the three flasks and incubation continued as usual.

Viable count before infection: 4.2 × 10⁸ cells per ml (average); viable count after infection: 1 × 10⁸ cells per ml (average).
of hexose means that very little of the hexose monophosphate pool arises by resynthesis via aldolase and fructose 1,6-diphosphatase.

**Determination of proportion of deoxyribose formed via oxidative and transketolase-transaldolase pathways.** The calculation of the relative amounts of pentose synthesized by the two pathways, according to the procedure of Szynkiewicz et al. (1961), requires information on the distribution of C\textsubscript{14} in a hexose derivative representative of the hexose phosphate pools. It seemed probable that the glucose bound to the phage DNA would be most representative of the pools present inside the cells during synthesis of this DNA. Table 2 shows an experiment in which glucose-2-C\textsubscript{14} was used as the tracer and the contributions of the two pathways were calculated as described by Szynkiewicz et al. (1961). Fifty-eight per cent of the pentose was formed by the oxidative pathway and the remainder by the nonoxidative pathway. The incorporation of label into C-4 of the pentose confirms the participation of the nonoxidative pathway. C-4 of pentose formed by the oxidative pathway would be derived from C-5 of the hexose monophosphate, and judging from the glucose of the DNA this carbon contained little C\textsubscript{14}. Thus it is clear that the oxidative pathway could not have been the source of the C-4 labeled pentose. The relatively small movement of label into C-1, 3 of hexose shows that very little of the hexose monophosphate arises from resynthesis by a complete pentose cycle (Katz and Wood, 1960; Wood and Katz, 1958). The low activity of C-5 of hexose confirms the conclusion drawn from the experiment with glucose-6-C\textsubscript{14} that only a small amount of resynthesis of hexose monophosphate occurred by the aldolase and fructose 1,6-diphosphatase pathway.

In another experiment the glucose was equally labeled in C-2 and C-6. When the sugar is labeled simultaneously in the two positions it is possible to detect transaldolase- or aldolase-catalyzed exchange with unlabeled triose phosphate which might arise from metabolites formed prior to the addition of the labeled

**TABLE 2** 

*Labeling of bacteriophage carbohydrates by glucose-2-C\textsubscript{14}*

<table>
<thead>
<tr>
<th>Carbon Atom</th>
<th>Relative Specific Activity</th>
<th>DNA glucose</th>
<th>DNA deoxyribose</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>9.2</td>
<td>100*</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>100*</td>
<td>60.0</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>12.7</td>
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<td></td>
</tr>
<tr>
<td>4</td>
<td>1.1</td>
<td>41.0</td>
<td></td>
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<tr>
<td>5</td>
<td>9.5</td>
<td>8.1</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>1.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Observed specific activity: C-2 of glucose = 2.91 μc per mmole carbon; C-1 of deoxyribose = 2.35 μc per mmole carbon.

**TABLE 3** 

*Labeling of bacteriophage carbohydrates by glucose-2,6-C\textsubscript{14}*

<table>
<thead>
<tr>
<th>Carbon Atom</th>
<th>Relative Specific Activity</th>
<th>DNA glucose</th>
<th>DNA deoxyribose</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12.2</td>
<td>100*</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>100*</td>
<td>48.5</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>5.6</td>
<td>5.6</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>1.1</td>
<td>45.6</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>10.2</td>
<td>87.9</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>76.3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Observed specific activity: C-2 of glucose = 2.0 μc per mmole of carbon; C-1 of deoxyribose = 1.12 μc per mmole carbon.

*Escherichia coli* from a slant was transferred twice on S/2-0.5% sodium acetate-0.02% glucose. Samples of the final culture were transferred to three 1,800-ml portions of S/5-0.1% glucose-0.005% casein hydrolyzate (Klett 19) which were then incubated for 2 hr (Klett 41). Each of these three cultures was infected with 4 × 10\textsuperscript{12} phage (phage:cells = 5.1:1). Five minutes later, a total of 46.8 mg of glucose containing 60.5 μc of glucose-2-C\textsubscript{14} and 61 μc of glucose-6-C\textsubscript{14} was distributed among the three flasks.

Viable count before infection: 3.5 × 10\textsuperscript{8} cells per ml (average); viable count after infection: 1 × 10\textsuperscript{4} cells per ml (average).
glucose. Such exchange would become evident by a greater dilution of C\(^{14}\) in C-6 than in C-2. The results given in Table 3 show that such dilution does occur in the glucose derived from the phage DNA. The dilution of C-6 was not accompanied by an equivalent introduction of C\(^{14}\) into C-5, so the exchange appears to have occurred with triose phosphate formed from sources other than the glucose-2,6-C\(^{14}\). The distribution of C\(^{14}\) in the deoxyribose derived from the DNA in this experiment confirms the results of the two previous experiments, and it is calculated that 60\% of the pentose was formed by the oxidative pathway and 40\% by transketolase and transaldolase.

An experiment also was done with glucose-1-C\(^{14}\) as tracer and is shown in Table 4. The usual calculation of pathways cannot be made with the results obtained with this labeled sugar, since synthesis by the oxidative pathway gives pentose which is unlabeled in C-1, and only transketolase and transaldolase produce C-1 labeled pentose. The labeling pattern of the glucose and deoxyribose is entirely in accord with the conclusion drawn from the other experiments. The labeling of the pentose gives no indication that any unsuspected pathway operates. Again, the high labeling of C-5 of pentose indicates a considerable role of the nonoxidative reactions. However, there is a discrepancy between this experiment and the previous experiments. It is noted that C-1 of the hexose monophosphate is not introduced into the pentose by the oxidative pathway, whereas transketolase and transaldolase yield pentose whose C-1 is derived from C-1 and C-3 of the hexose monophosphate in a proportion of 2 to 1 (Szynekiewicz et al., 1961). Therefore the maximal specific activity of the pentose should be \(\frac{2}{3}\) that of C-1 of the hexose. The observed specific activity is 77\% of this \(\frac{2}{3}\) value, and on this basis one is led to conclude that 77\% of the pentose arose by transketolase-transaldolase. This estimate appears too high in the light of the results obtained in the experiments shown in Tables 2 and 3. Some possible explanations for this discrepancy are presented in the Discussion.

**DISCUSSION**

All calculations of biosynthetic pathways on the basis of distribution of label in the ultimate products depend on an accurate knowledge of the labeling of the precursors. It was shown in the earlier studies in this series that it is not valid to assume that the intracellular hexoses retain the unique labeling of the radioactive tracers which were added to the cultures. In the studies with uninfected cells, glycogen was assumed to be a valid indicator of the hexose monophosphates (Bagatell et al., 1959). It was also assumed that the hexose monophosphate pool from which the pentose was synthesized was either equilibrated with or at least was labeled similarly to the hexose monophosphate pool from which the glycogen received its radioactive glucose units. The question of separation of pools, whether in space (due to possible subcellular structure) or in time (due to asynchronous synthesis of various substances) has been discussed by Szynekiewicz et al. (1961). The same assumptions and limitations which applied to the glucose units of glycogen in the earlier studies apply to the DNA-bound glucose in the present work.

The calculation of the contributions of the two

**TABLE 4**

<table>
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<tr>
<td></td>
<td>DNA glucose</td>
</tr>
<tr>
<td>1</td>
<td>100*</td>
</tr>
<tr>
<td>2</td>
<td>1.2</td>
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<td>1.0</td>
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<tr>
<td>6</td>
<td>13.2</td>
</tr>
</tbody>
</table>

* Observed specific activity: C-1 of glucose = 4.15 \(\mu\)c per mmole carbon; C-1 of deoxyribose = 2.12 \(\mu\)c per mmole carbon.

*Escherichia coli* was transferred twice on S/2-0.5\% sodium acetate-0.02\% glucose. Samples of this culture were added to three 1,800-ml portions of S/5-0.1\% glucose-0.005\% casein hydrolysate (Klett 20) which were then incubated at 37 C for 2 hr (Klett 47). Each culture was infected with \(4 \times 10^{12}\) phage (phage:cells = 4.5:1). Five minutes later, a total of 54.8 mg of glucose containing 232 \(\mu\)c of glucose-1-C\(^{14}\) was distributed among the three flasks.

Viable count before infection: \(4.7 \times 10^6\) cells per ml (average); viable count after infection: \(2.7 \times 10^4\) cells per ml (average).
pathways is subject to error, as indicated below, but probably is sufficiently reliable to give the correct order of magnitude of the two pathways. Calculations based on the experiments with glucose-2-C\textsuperscript{14} and glucose-2,4-C\textsuperscript{14} indicate that about 60% of the pentose was synthesized by the oxidative pathway. This resembles very closely the result obtained with acetate-adapted uninfected cells in the early logarithmic phase after transfer to glucose medium. In the present experiments, the cells were always acetate-adapted, and then transferred to glucose. Since infection with bacteriophage was carried out when the optical density had doubled, one is dealing with cells in the early logarithmic stage, entirely comparable to the uninfected cells. The infection with bacteriophage does not appear to have caused any increase in the transketolase-transaldolase pathway, relative to similarly grown uninfected cells. However, the cells were already synthesizing a sizable proportion of their pentose by the nonoxidative pathway and for that reason these findings cannot be taken as conclusive evidence against the conclusion of Loeb and Cohen (1959) that bacteriophage infection leads to increased participation of a nonoxidative pathway. The latter authors used glucose-grown cells which (when uninfected) synthesize pentose predominantly by the oxidative pathway. A shift from the oxidative to a nonoxidative pathway would be more apparent in such a metabolic system than in the acetate-grown cells used in the present study.

The complete transketolase-transaldolase pathway is not the only nonoxidative mechanism by which C\textsuperscript{14} from hexose may be incorporated into pentose. Transketolase alone could yield pentose and erythrose 4-phosphate from C-1,2 of fructose 6-phosphate and triose phosphate. An appreciable amount of such synthesis or exchange by transketolase apart from the complete pathway would alter the ratio of C\textsuperscript{14} in C-1 and C-2 of the pentose from the theoretical value assumed for transketolase-transaldolase synthesis, and thus would affect the accuracy of the calculations presented here. Such synthesis by transketolase would form pentose-2,4-C\textsuperscript{14} from glucose-2-C\textsuperscript{14} and pentose-1,5-C\textsuperscript{14} from glucose-1-C\textsuperscript{14}. This may indeed explain the unexpectedly high specific activity of the pentoses in the experiments reported in Tables 2 and 4. It has been suggested in an earlier publication (Sable et al., 1960) that this participation of transketolase may explain the labeling pattern of the deoxyribose of bacteriophage produced from CH\textsubscript{3}C\textsuperscript{14}OOH.

All the experiments show that there is a large contribution of C-1,2,3 of hexose to C-3,4,5 of pentose under conditions in which these positions of the hexose contribute only slightly to C-4,5,6 of the hexose. This shows that the label in C-3,4,5 of pentose arose from the participation of triose phosphate in the transketolase-transaldolase or transketolase pathways, rather than because of prior labeling of the hexose monophosphate.

In general it seems likely in studies of pathways that it is preferable to analyze the distribution of label in the various products rather than to attempt to assess the different pathways entirely by comparison of the total radioactivities in the products. The discrepancy between our findings and those of Loeb and Cohen (1959) may arise from the different methods and may not be real. It would be of interest to apply the present technique to glucose-grown cells, infected with bacteriophage in glucose medium.

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SUMMARY
The biosynthesis of deoxyribose and glucose by Escherichia coli infected with bacteriophage T\textsubscript{2}H has been studied with C\textsuperscript{14} tracer techniques. The glucose bound glycosidically to hydroxymethylcytosine of bacteriophage deoxyribonucleic acid (DNA) has been isolated and degraded, and the deoxyribose of the phage DNA has also been isolated and degraded. It has been assumed that the glucose reflects the labeling of the hexose monophosphate pool in the infected cell at the time the deoxyribose is synthesized. The distribution of C\textsuperscript{14} in the glucose and deoxyribose has been used to calculate the proportion of deoxyribose synthesized via the pathways involving the oxidative decarboxylation of 6-phosphogluconate and by transketolase and transaldolase reactions. About 60% of the pentose ap-
pears to have been synthesized via the oxidative pathway. There was no evidence that phage infection altered the proportion of the pathways since this is the same proportion as that observed in the uninfected cells. There was no evidence of condensation of 3-carbon and 2-carbon precursors to yield deoxyribose directly. In these respects the infected cells resemble uninfected cells studied with similar techniques.

REFERENCES


