Control of Expression of the L-Arabinose Operon in Temperature-Sensitive Mutants of Gene *araC* in *Escherichia coli* B/r

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Expression of the L-arabinose operon in *Escherichia coli* B/r is dependent on the temperature of growth of the *araC* mutants reported in this paper. Analysis of these temperature-sensitive regulatory mutants indicates that both repressor and activator activities are thermolabile. The simplest model to explain the manner in which the operon is controlled is one suggesting that the regulatory gene, *araC*, codes for a protein which upon synthesis acts as a repressor molecule and prevents operon function. When inducer is added, the repressor undergoes a conformational shift and becomes an activator which switches on enzyme synthesis, provided the repressor concentration is reduced to a sufficiently low level in the cell. These data lend strong support to the model that both activities are the result of the same gene product.

Induction of the enzymes specified by the structural genes of the L-arabinose operon is subject to the action of the product coded by the gene *araC*. The *araC* gene product, on the basis of genetic evidence, is thought to be a protein (8). It has been suggested that the *araC* gene product exists in two discrete conformational states, both of which participate in the control of the expression of the operon. First, it acts as a repressor molecule to prevent production of the enzymes. Second, it acts as an activator to "switch" on enzyme synthesis. The model further suggests that the addition of inducer, L-arabinose, causes the conformational shift from the repressor configuration to that of the activator (4).

Adjacent to the three structural genes of the operon exist two control elements, *araI* and *araO*. *AraI* is the site for the activator to turn on the operon and is termed the initiator (5). The other site, *araO*, is called the operator and is the region which interacts with the *araC* gene protein when it exists as a repressor (6). The action of the operator site supercedes the action of the initiator site. Hence, when a cell contains both activator molecules and repressor molecules, there is little or no production of the enzymes of the operon (4, 11).

Three allelic states of the *araC* gene have been previously described. First, the wild type, designated *araC*+, has the properties described above. Second is the pleiotropic negative allele, designated *araC*-. The *araC*+ mutants fail to yield any appreciable levels of the enzymes of the pathway in response to inducer and may be either nonsense mutants (8) or deletions (11). They are recessive to the wild-type and *araC*+ alleles which are described below. Third, the constitutive alleles are designated *araC*-. Mutants of this type produce the enzymes of the pathway in a coordinate fashion in the absence of inducer and are dominant to *araC*+ mutants. However, the *araC*− allele is recessive to the wild type when inducer is not present (4, 11). This latter observation provided the initial proof that the *araC* gene protein acts as a repressor molecule, whereas the former observation indicated its action as an activator. Presumably, then, the *araC*− mutants are producing an *araC* gene protein, the configuration of which is only of the activator type.

In this communication we describe two additional classes of mutants with sites in the *araC* gene. Both types are temperature sensitive but they differ in one unique physiological property. One group is inducible at 28 C and pleiotropic negative at 42 C, whereas the other group produces the enzymes of the pathway even in the absence of inducer at 28 C and are pleiotropic negative at 42 C. For convenience the former group will be designated *araC*+ and the latter *araC*−.
The analysis of these mutants lends further support to the positive control model and strengthens considerably the argument that the araC gene protein acts both as a repressor and an activator.

MATERIALS AND METHODS

Organisms. All bacterial strains used in this study were derived from *Escherichia coli* B/r. Their genotypes are listed in Table 1.

Growth of cultures. All media employed have been previously described (2, 7). The following abbreviations will be used for media designations: M, mineral base; A, l-arabinose; G, glucose; L, L-leucine; EMB, eosin methylene blue agar lacking lactose but supplemented with 1% l-arabinose; CAA, mineral base supplemented with 1% Casamino Acids. When cultures were grown for enzyme assays CAA was always utilized. When the culture was to be induced by l-arabinose, it was added to CAA to a final concentration of 0.4%.

Cultures prepared for enzyme assays were grown to late log phase in 50 ml of CAA in 250-ml Erlenmeyer flasks on gyrotary water bath shakers (New Brunswick) equilibrated to the proper temperature. Extracts were prepared by the methods of Cribbs and Englesberg (2). Protein was measured by the method of Lowry et al. (10). Cell density was estimated turbidimetrically at 425 nm in a Klett-Summerson colorimeter.

Enzyme assays. L-Arabinose isomerase (EC 5.3.1.4) was assayed by the methods of Cribbs and Englesberg (2). The reaction mixture contained: 75 μmoles of L-arabinose, 0.5 μmole of MgCl₂, water, and cell free extract in a total volume of 1.0 ml. One unit of enzyme activity equals 1 μmole of L-ribulose formed per hr.

L-Ribulokinase (EC 2.7.1.16) was assayed by the methods of Lee and Englesberg (9) with the following modifications. The reaction mixture was reduced to 0.1 ml total volume, and the entire mixture was assayed for L-ribulose-5-P. This modified reaction mixture contained: 8 μmoles of tris(hydroxymethyl)aminomethane (Tris)-hydrochloride at pH 8.6, 0.8 μmoles of reduced glutathione, 0.4 μmoles of ethylenediaminetetraacetic acid (EDTA); 4 μmoles of MgCl₂; 4 μmoles of NaF; 1.6 μmoles of adenosine triphosphate (ATP); and 2 μmoles of L-ribulose-14C (specific activity, 10⁶ counts per min per μmole). One unit of enzyme activity equals 1 μmole of L-ribulose-5-P formed per hr.

Glucose-6-PO₄ dehydrogenase (EC 1.1.1.49) was assayed by the method of DeMoss (3) with the following modifications. In a total volume of 0.3 ml, there were 15 μmoles of Tris-hydrochloride at pH 7.8, 0.008 μmole of nicotinamide adenine dinucleotide phosphate (NADP), 1.0 μmole of MgCl₂, 4.5 μmoles of glucose-6-PO₄, water, and cell-free extract. Absorbance at 340 nm was measured at 37 C in a Gilford recording spectrophotometer. A unit of enzyme activity equals a change of 1.0 unit of optical density per min.

Merodiploid construction. F' ara strains were used for merodiploid formation. The usual technique was to select for Leu + strains which were fertile and Ara - at the proper temperature. Several merodiploids were selected as Ara + exconjugates when it was already known that such a response was possible. Genotypes of the endog- enote and exogenote were confirmed by isolating Ara - segregants from the complementing diploids, and then confirming their mutant sites by progeny testing with suitably marked F' homogenotes (see reference 11). Pure isolates of the complementing diploids were used for the enzyme assays. An estimate of the frequency of segregation of Ara + clones was made for every culture assayed for enzyme activity. If the frequency of segregation approached or exceeded 5% of the total population, the enzymatic data was discarded. In most cases, however, the frequency of segregation was 1% or less.

RESULTS

Temperature-sensitive araC mutants. Ten independent isolates characterized previously as araC- mutants by map position, complementation, and enzymatically when grown at 37 C were tested for temperature sensitivity by streaking them on MA agar and incubating them at both 28 and 42 C. Three of the mutants, *araC42*, -47, and -51 grew on the MA agar at 28 C but not at 42 C. The other mutants failed to grow at either temperature (8).

The three temperature-sensitive mutants, with *araC5* (a nonsense mutant) and the wild type as controls, were grown in CAA plus minus L-arabinose at both 28 and 42 C and were subsequently assayed for L-arabinose isomerase and glucose-6-PO₄ dehydrogenase activities. None of the cultures produced significant levels of isomerase when grown without L-arabinose at either temperature, and the data are not reported. Induced cultures of *araC42*, -47, and -51 grown at

<table>
<thead>
<tr>
<th>Strain designation</th>
<th>Source or reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>ara + (wild type)</td>
<td>Gross and Englesberg (7)</td>
</tr>
<tr>
<td>araA2</td>
<td>leuB</td>
</tr>
<tr>
<td>araC5</td>
<td>leuB</td>
</tr>
<tr>
<td>araC42</td>
<td>leuB</td>
</tr>
<tr>
<td>araC47</td>
<td>leuB</td>
</tr>
<tr>
<td>araC51</td>
<td>leuB</td>
</tr>
<tr>
<td>araC67</td>
<td>leuB</td>
</tr>
<tr>
<td>araA2, C67</td>
<td>T6'</td>
</tr>
<tr>
<td>araC5-10</td>
<td>leuB</td>
</tr>
<tr>
<td>araC5-11</td>
<td>leuB</td>
</tr>
<tr>
<td>araC5-12</td>
<td>leuB</td>
</tr>
<tr>
<td>araC5-13</td>
<td>leuB</td>
</tr>
<tr>
<td>araC5-14</td>
<td>leuB</td>
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<tr>
<td>araC5-15</td>
<td>leuB</td>
</tr>
<tr>
<td>araC5-16</td>
<td>leuB</td>
</tr>
<tr>
<td>araC5-17</td>
<td>leuB</td>
</tr>
<tr>
<td>araC5-18</td>
<td>leuB</td>
</tr>
<tr>
<td>F' ara +/araA2, araC19</td>
<td>T6' trp</td>
</tr>
</tbody>
</table>

* Nine revertants of *araC7*, designated *araC7-10*, etc., are listed. Four of the strains are intragenic revertants: *araC5-10*, *araC5-11*, *araC5-12*, and *araC5-14*. The other five strains carry independent isolates of nonsense suppressor mutations and are designated Su +. Various heterogenotes and homogenotes were isolated after infecting some of the haploid strains with F' ara and are reported in the text.

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28 C had considerable isomerase activity, whereas the cultures grown at 42 C had negligible isomerase levels. The two control activities (araC5 and wild type) were as expected. Glucose-6-PO4 dehydrogenase activities did not vary significantly under the two conditions of growth (Table 2). Hence, the enzymatic data support the conclusion that the three araC mutants, -42, -47, and -51, respond to inducer at 28 C to synthesize the enzymes needed for L-arabinose catabolism but are lacking in this function at 42 C. For convenience, these three mutants will be termed araCmutants.

**Temperature-sensitive revertants of araC5.**

Nine revertants of the nonsense mutant araC5 were selected for their temperature sensitivity (growth on MA at 28 C but not at 42 C). Four of the revertants are the result of secondary mutations in the araC gene and the other five are independently isolated nonsense suppressors (8). Each of the nine along with araC5 and the wild type were grown at 28 C and 42 C in CAA plus L-arabinose; crude extracts were prepared and assayed for L-arabinose isomerase, L-ribulokinase, and glucose-6-PO4 dehydrogenase. The cultures grown at 42 C produced low or undetectable levels of isomerase and kinase similar to their parent araC5 (isomerase, 0.4; kinase, 0.01). The cultures of the revertants grown at 28 C had levels of 18 to 44 for isomerase and 1 to 5 for the kinase, whereas araC5 was the same as at 42 C. The wild-type levels were as expected. Glucose-6-PO4 dehydrogenase activities did not vary significantly (Table 3). Although the levels of isomerase and kinase found in the revertants when grown at 28 C varied from strain to strain, the ratios of the two activities remained rather constant, thus indicating a coordinate effect upon the expression of the structural genes coding for these two enzymes.

When the same strains described above were grown at 28 C in CAA without inducer, it was determined that all nine of the temperature-sensitive revertants of araC5 were producing significant levels of isomerase and kinase (Table 4). These levels are not as high as found in the induced cultures, but the ratios of enzyme activities is rather constant. Thus the nine temperature-sensitive revertants of araC5 produce coordinate levels of L-arabinose isomerase and L-ribulokinase constitutively but only at low temperatures. Those four revertants which are the result of secondary mutations within the araC gene will be designated as araCmutants for convenience. They are araC5-10, araC5-11, araC5-12, and araC5-14.

### Table 2. Enzyme assays of araCmutants

<table>
<thead>
<tr>
<th>Strain</th>
<th>Temp of growth</th>
<th>Isomerase</th>
<th>G6PDH</th>
<th>Isomerase</th>
<th>G6PDH</th>
</tr>
</thead>
<tbody>
<tr>
<td>araC42</td>
<td>28 C</td>
<td>33.9</td>
<td>0.99</td>
<td>0.8</td>
<td>1.03</td>
</tr>
<tr>
<td>araC47</td>
<td>42 C</td>
<td>9.6</td>
<td>0.93</td>
<td>0.9</td>
<td>1.10</td>
</tr>
<tr>
<td>araC51</td>
<td>28 C</td>
<td>36.2</td>
<td>1.24</td>
<td>0.6</td>
<td>1.31</td>
</tr>
<tr>
<td>araC5</td>
<td>42 C</td>
<td>0.4</td>
<td>1.19</td>
<td>0.3</td>
<td>1.33</td>
</tr>
<tr>
<td>ara* (wild type)</td>
<td></td>
<td>50.4</td>
<td>1.25</td>
<td>55.8</td>
<td>1.17</td>
</tr>
</tbody>
</table>

* L-Arabinose isomerase (isomerase) and glucose-6-PO4 dehydrogenase (G6PDH) activities are reported as units of enzyme per milligram of protein. Assay procedures are described in the text. Cultures were grown at the indicated temperatures in 1% mineral base plus Casamino Acids (CAA) plus 0.4% L-arabinose for at least three generations. Inocula for the cultures were grown overnight at the desired temperature in CAA without inducer. Crude cell-free extracts were used as the source of the enzymes.

### Table 3. Enzyme activities of revertants and suppressors of araC5

<table>
<thead>
<tr>
<th>Strain</th>
<th>Temp of growth</th>
<th>Isomerase (units/mg protein)</th>
<th>G6PDH (units/mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>araC5-10</td>
<td>28 C</td>
<td>35.9</td>
<td>2.89</td>
</tr>
<tr>
<td>araC5-11</td>
<td>42 C</td>
<td>4.36</td>
<td>4.14</td>
</tr>
<tr>
<td>araC5-12</td>
<td>28 C</td>
<td>42.7</td>
<td>5.00</td>
</tr>
<tr>
<td>araC5-13Su+</td>
<td>42 C</td>
<td>43.8</td>
<td>3.82</td>
</tr>
<tr>
<td>araC5-14</td>
<td>28 C</td>
<td>30.5</td>
<td>2.05</td>
</tr>
<tr>
<td>araC5-15Su+</td>
<td>42 C</td>
<td>14.3</td>
<td>0.80</td>
</tr>
<tr>
<td>araC5-16Su+</td>
<td>28 C</td>
<td>22.3</td>
<td>1.72</td>
</tr>
<tr>
<td>araC5-17Su+</td>
<td>42 C</td>
<td>23.4</td>
<td>1.50</td>
</tr>
<tr>
<td>araC5-18Su+</td>
<td>28 C</td>
<td>17.8</td>
<td>1.33</td>
</tr>
<tr>
<td>araC5-19</td>
<td>42 C</td>
<td>0.4</td>
<td>0.01</td>
</tr>
<tr>
<td>ara* (wild type)</td>
<td>28 C</td>
<td>50.4</td>
<td>7.50</td>
</tr>
<tr>
<td>ara* (wild type)</td>
<td>42 C</td>
<td>54.6</td>
<td>8.70</td>
</tr>
</tbody>
</table>

* L-Arabinose isomerase (isomerase), L-ribulokinase (kinase), and glucose-6-PO4 dehydrogenase (G6PDH) activities are reported in units of enzyme per milligram of protein. Kinase levels of 0.01 are minimum values obtained within the limits of the assay. Details are in Table 2 and the text.

### Table 4. Constitutive synthesis of L-arabinose isomerase and L-ribulokinase in revertants and suppressors of araC5 grown at 28 C

<table>
<thead>
<tr>
<th>Strain</th>
<th>Isomerase (units/mg protein)</th>
<th>Kinase (units/mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>araC5-10</td>
<td>17.9</td>
<td>0.71</td>
</tr>
<tr>
<td>araC5-11</td>
<td>20.6</td>
<td>0.85</td>
</tr>
<tr>
<td>araC5-12</td>
<td>14.5</td>
<td>0.58</td>
</tr>
<tr>
<td>araC5-13Su+</td>
<td>8.1</td>
<td>0.28</td>
</tr>
<tr>
<td>araC5-14</td>
<td>20.3</td>
<td>0.89</td>
</tr>
<tr>
<td>araC5-15Su+</td>
<td>17.2</td>
<td>0.64</td>
</tr>
<tr>
<td>araC5-16Su+</td>
<td>10.6</td>
<td>0.36</td>
</tr>
<tr>
<td>araC5-17Su+</td>
<td>12.1</td>
<td>0.35</td>
</tr>
<tr>
<td>araC5-18Su+</td>
<td>8.6</td>
<td>0.41</td>
</tr>
<tr>
<td>araC5</td>
<td>0.3</td>
<td>0.01</td>
</tr>
<tr>
<td>ara* (wild type)</td>
<td>0.5</td>
<td>0.01</td>
</tr>
</tbody>
</table>

* Units of enzyme per mg protein. Cultures were grown in 1% CAA without the addition of inducer.
Dominance relationships between araC+ and the araC" and araC' alleles. To characterize the temperature-sensitive mutants further, their patterns of complementation with respect to the wild-type allele of the araC gene were studied. Heterogenotes of the type araC"/araC" were constructed by crosses between the F- leu- araC" strains and F' ara+/araA, araC47 trp+. Trp+ Leu" exconjugants were selected on MG agar. These heterodiploids were then purified and tested for L-arabinose fermentation on EMB at 28 and 42 C, and positive responses were observed at both temperatures. Sected colonies were noted on the EMB plates incubated at 42 C. Isolates obtained from the white sectors all proved to be Ara+ at 28 C and Ara- at 42 C. Hence, it was concluded that the only ara- sites present in the heterogenotes were the araC" mutations present in the original F- strains used in the crosses. Heterogenote araC"/araC47 was unstable and was not studied further.

Both araC"/araC42 and araC+/araC51 along with the appropriate haploid controls were grown at 42 C in CAA plus inducer; extracts were prepared and assayed for isomerase activity. Both heterogenotes had elevated levels of isomerase, whereas the haploid araC" strains produced little enzyme activity. The elevated levels of about twice the wild-type control seen in the heterogenotes are due to the presence of the additional structural genes for isomerase in the strains (Table 5). It is concluded that the three temperature-sensitive mutants, araC42, -47, and -51, are all recessive to the wild-type araC" allele.

Heterogenotes of the type araC"/araC" were also constructed in a similar fashion described above for the araC"/araC" strains. Three stable merodiploids were obtained: araC+/araC5-10, araC+/araC5-11, and araC+/araC5-14. The three stable heterogenotes and the fourth unstable one, araC+/araC5-12, were Ara+ on EMB at both 28 and 42 C. Each of the three stable strains along with the proper haploid controls were grown at 28 C in CAA minus inducer and at 42 C plus inducer. At 28 C the isomerase levels produced by the heterogenotes were 10-fold lower than the araC" haploid controls, whereas at 42 C the heterogenotes produced elevated levels of isomerase, and the haploid strains had negligible activity (Table 5). Hence, the wild-type araC+ allele exerts dominance over the araC" mutants under both conditions. It appears that at 28 C the protein coded by the wild-type araC gene neutralizes the activator coded by the temperature-sensitive mutants; at 42 C the wild-type protein is converted to repressor, whereas the protein coded by the araC" mutants remains completely inactive both as an activator and repressor.

Table 5. Dominance relationships between the wild-type and temperature-sensitive alleles of the araC gene

<table>
<thead>
<tr>
<th>Alleles</th>
<th>Conditions of growth</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>28 C (no inducer)</td>
</tr>
<tr>
<td><strong>Merodiploids</strong></td>
<td></td>
</tr>
<tr>
<td>F' ara+ araC42</td>
<td>-c</td>
</tr>
<tr>
<td>F' ara+ araC5</td>
<td>-</td>
</tr>
<tr>
<td>F' ara+ araC5-10</td>
<td>1.3e</td>
</tr>
<tr>
<td>F' ara+ araC5-11</td>
<td>1.2</td>
</tr>
<tr>
<td>F' ara+ araC5-14</td>
<td>1.5</td>
</tr>
<tr>
<td><strong>Haploid Controls</strong></td>
<td></td>
</tr>
<tr>
<td>araC42</td>
<td>-</td>
</tr>
<tr>
<td>araC5</td>
<td>-</td>
</tr>
<tr>
<td>araC5-10</td>
<td>15.5</td>
</tr>
<tr>
<td>araC5-11</td>
<td>16.9</td>
</tr>
<tr>
<td>araC5-14</td>
<td>17.4</td>
</tr>
<tr>
<td>ara+ (wild type)</td>
<td>0.2</td>
</tr>
</tbody>
</table>

Exogenote.  
Endogenote.  
Not tested.

*a Units of L-arabinose isomerase per milligram of protein.

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were constructed. Each was Ara+ on EMB agar when incubated at 42 C. As a control, F′ araC5/araA2, araC′67 heterogenote was constructed and assayed for isomerase activity when grown with the araC′67 heterogenotes and the appropriate haploid strains at 42 C with and without inducer. The haploid controls, except for araC′67, had isomerase levels of 1.0 or less, and araC′57 had 80 units of isomerase. The heterogenotes all produced about 100 units or more of isomerase under the same growth conditions (Table 6). Therefore, the araC′57 mutant alleles are recessive to araC′67 when grown at 42 C. It seems likely that the araC′57 mutants not only lack activator activity at the elevated temperature but also lack repressor activity.

**DISCUSSION**

The enzymatic data reported here for the haploid temperature-sensitive araC mutants clearly indicate that the expression of L-arabinose operon in these particular strains is dependent on the temperature of incubation during growth. Thus, it appears some component of the system required for operon expression has been rendered thermolabile as a result of the mutations. Since the mutant sites all are definitively located within the confines of the araC gene (8), the regulatory gene of the operon, it is likely that this gene product is thermolabile. Because the operon is not expressed at 42 C but significant levels of the enzymes are found at lower temperatures, the thermolabile component of the system must act in a positive fashion to allow enzyme synthesis. Such a finding is entirely compatible with the positive control model which implies that the araC gene codes for an activator molecule that "switches on" protein synthesis.

The complementation tests performed with the merodiploids bearing the temperature-sensitive mutant alleles lend further support to the idea that the araC gene protein is thermolabile as a result of the mutations. When tested either qualitatively by the fermentation test on EMB agar or by the enzymatic data obtained from the stable merodiploids, the temperature-sensitive alleles were recessive to both wild-type, araC+, and constitutive, araC+, alleles when grown at 42 C. Hence, the thermolabile protein produced by the mutants does not prevent expression of the operon, but merely fails to participate in induction at 42 C.

The simplest explanation of the patterns of operon control expressed by the araC gene is that its protein product, as it is first synthesized in the cell, has the ability to repress enzyme synthesis and upon the addition of inducer the repressor is converted to the activator. When deletions extend through the araC gene both repressor and activator activities are lost (11). Nonsense mutants likewise have the same effect (8). However, the deletions could extend into another adjacent cistron which codes for the repressor, and the nonsense mutants could exert a polar effect upon the other cistron. Therefore, it is conceivable that there could be two distinct but adjacent cistrons involved in the regulation of the operon. Nevertheless, this alternative explanation seems unlikely since, in the case of the temperature-sensitive mutants which are not deletions or nonsense mutants, repressor activity and activator activity is lost to the cell. This effect can be clearly seen by comparing the levels of isomerase produced in the partial diploids of the type araC+′/araC′5. Growth at 28 C without inducer yields low isomerase activity, but growth at 42 C without inducer produces high isomerase activity. These data indicate that the temperature-sensitive alleles form an araC′ gene protein that has repressor activity at low temperature but that this activity is missing when the partial diploids are grown at 42 C. Therefore, even the effect of conditional mutants is to remove both repressor and activator activity. Thus, the simplest interpretation of the model is the one most likely to be valid.

At the present, little is known of the molecular details by which the araC′ gene protein regulates expression of the operon. A priori it has always been assumed that control was at the level of transcription; no definitive proofs, however, exist at the present. Furthermore, there is no indication as to the way an activator molecule would

**Table 6. Dominance relationships between constitutive and temperature-sensitive alleles of araC**

<table>
<thead>
<tr>
<th>Alleles</th>
<th>Conditions of growth</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>28 C (no inducer)</td>
</tr>
<tr>
<td>Merodiploids</td>
<td></td>
</tr>
<tr>
<td>F′ araC′47 araA2, araC′67</td>
<td>8.2</td>
</tr>
<tr>
<td>F′ araC′51 araA2, araC′67</td>
<td>5.1</td>
</tr>
<tr>
<td>F′ araC′5-11 araA2, araC′67</td>
<td>—</td>
</tr>
<tr>
<td>F′ araC′5-14 araA2, araC′67</td>
<td>—</td>
</tr>
<tr>
<td>F′ araC′5 araA2, araC′67</td>
<td>—</td>
</tr>
<tr>
<td>Haploid controls</td>
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<tr>
<td>araC47</td>
<td>0.3</td>
</tr>
<tr>
<td>araC51</td>
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</tr>
<tr>
<td>araC5-11</td>
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</tr>
<tr>
<td>araC5-14</td>
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<tr>
<td>araC′5</td>
<td>—</td>
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<tr>
<td>araA2, araC′67</td>
<td>0.1</td>
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<tr>
<td>araC′67</td>
<td>65.0</td>
</tr>
<tr>
<td>araA2</td>
<td>0.1</td>
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</table>

* Exogenote.

† Endogenote.

‡ Units of L-arabinose isomerase per milligram of protein.

* Not tested.
function to turn on the operon under its control. It is tempting to suggest that such an activator is a ribonucleic acid polymerase sigma factor (12) which upon activation by the inducer directs messenger ribonucleic acid synthesis specific for the arabinose operon. Before such a model can be entertained seriously, it is first necessary to demonstrate that the control lies at the level of transcription. Such experiments are now underway.

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LITERATURE CITED