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AbrB Modulates Expression and Catabolite Repression of a *Bacillus subtilis* Ribose Transport Operon†

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A *Bacillus subtilis* ribose transport operon (*rbs*) was shown to be subject to AbrB-mediated control through direct AbrB-DNA binding interactions in the vicinity of the promoter. Overproduction of AbrB was shown to relieve catabolite repression of *rbs* during growth in the presence of poorer carbon sources such as arabinose but had much less effect when cells were grown in the presence of glucose, a rapidly metabolizable carbon source. A *ccpA* mutation relieved catabolite repression of *rbs* under all conditions tested. One of the AbrB-binding sites on the *rbs* promoter contains the putative site of action for the *B. subtilis* catabolite repressor protein CcpA, suggesting that competition for binding to this site could be at least partly responsible for modulating *rbs* expression during carbon-limited growth.

At the onset of stationary phase and sporulation of *Bacillus subtilis*, a number of genes normally silent during active growth commence expression. Most of these genes are controlled by the Spo0A-AbrB regulatory circuit (19–21). Spo0A can act as either an activator or repressor of transcription (8). Activation of transcription by Spo0A depends upon its being converted into a phosphorylated form through a cascade of phosphotransfer reactions known as the phosphorelay (4, 8). A number of genes required for the later stages of sporulation have been shown to be directly activated by Spo0A~P (2, 16, 24, 30). In contrast, repression of transcription mediated via direct Spo0A-DNA binding has been proven for only one gene, *abrB* (7, 18).

The primary function of the DNA-binding AbrB protein seems to be to prevent inappropriate expression during active growth of many genes whose functions are required only under growth-limiting conditions. The Spo0A~P repression of *abrB* transcription that begins during the transition from vegetative growth to stationary phase serves to lower the intracellular AbrB concentration and so relieves *abrB*-dependent negative regulation. Thus, any positive effects attributed to Spo0A can be due either to direct transcriptional activation or to negative regulation of *abrB* or both.

Recent studies (27) indicated that Spo0A activates expression of a ribose transport operon (*rbs*) independently of *abrB*. I have examined *rbs* expression in a number of genetic backgrounds and, contrary to the previous reports, have found a consistent effect of AbrB on *rbs* expression both in the presence and in the absence of a functional *spo0A* gene. DNase I footprinting experiments showed that the effect is most likely due to direct AbrB-DNA binding in the *rbs* promoter region. Furthermore, overexpression of *abrB* (due to a *spo0A* mutation) relieved catabolite repression of *rbs* during carbon-limited growth. AbrB modulation of catabolite repression has been observed previously for numerous degradative enzymes (6), and in at least one case, the effect was interpreted as the result of competition between AbrB and the putative catabolite repressor protein CcpA for a binding site on the DNA (6). Since one of the AbrB binding sites overlaps a sequence resembling a CcpA-binding site, a similar mechanism may be involved in modulating *rbs* expression.

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MATERIALS AND METHODS

Bacterial strains and growth media. The strains used in this study are listed in Table 1. KD891 (27) was generously supplied by K. Devine and served as the source of the *rbs-lacZ* fusion in constructing other strains. Transformations with chromosomal DNA were performed by standard procedures (1). Schaeffer's nutrient broth medium (SM) and morpholinopropanesulfonic acid (MOPS) minimal medium have been described (14, 17). Carbon sources (0.5% final), glutamate (0.2% final), and citrate (0.2% final) were added to MOPS minimal medium.

Assays. β-Galactosidase assays were performed as previously described (22). DNase I footprinting assays were done with end-labeled DNA fragments from a clone containing a portion of the *rbs* gene (−221 to +125 relative to the transcriptional start point) obtained via PCR and by procedures described previously (23).

RESULTS

AbrB affects *rbs* expression in rich medium. I first examined *rbs* expression in cells growing in a rich medium (SM) which would allow the bacteria to enter the sporulation cycle upon depletion of nutrients. In overall agreement with previously reported results (27), the level of *rbs* expression (Fig. 1A) showed a steep increase ~1.5 h prior to the onset of stationary phase (t_0), and the maximum level attained was higher in KD892 (*spo0A12*) than in KD891 (wild type). After the steep increase, both levels began to fall off, but in the *spo0A12* mutant, β-galactosidase activity then remained at a higher plateau level than in the wild type. With a *lacZ* fusion to a promoter (*veg*) not controlled by *abrB* (11), no evidence for stability differences of β-galactosidase activity in various *abrB*, *spo0A*, *abrB spo0A*, and wild-type strains had been noted (29), and thus the observed differences in *rbs-lacZ* expression most likely reflect altered regulation of *rbs* transcription.

It had been reported that a *spo0A12 abrB23* strain showed the same pattern of *rbs* expression as the *spo0A12 abrB*$^+$ strain (27). However, when a *spo0A12 abrB23* strain (SWV222) was assayed, I observed that its pattern of *rbs* expression (Fig. 1A) was essentially identical to that of the wild type, indicating that

TABLE 1. Strains used in this study

Strain	Relevant genotype[a]	Derivation, source, or reference
168	*trpC2*	J. Hoch
GM1225	Δ(*bgaX*) *amyE*::ϕ(*gntRK'-lacZ*) *ccpA*::Tn*917* Δ(*lacZ-erm*)*spec*^{r}	5
JH642	*trpC2 pheA1*	J. Hoch
JH703	*spo0AΔ204*	J. Hoch
JH703*abrB2*	*spo0AΔ204 abrB2*	25
JH703*abrB4*	*spo0AΔ204 abrB4*	25
JH703*abrB7*	*spo0AΔ204 abrB7*	25
R2-11	*spo0A12 abrB19*	25
R15-8	*spo0A12 abrB20*	25
R15-13	*spo0A12 abrB23*	25
KD891	Δ*amyE*::ϕ(*rbs'-lacZ*)*cat*	27
KD892	*spo0A12* Δ*amyE*::ϕ(*rbs'-lacZ*)*cat*	27
SWV119	*abrB*::*tet*	Laboratory stock
SWV201	Δ*amyE*::ϕ(*rbs'-lacZ*)	JH642 × KD891 DNA
SWV202	*spo0AΔ204* Δ*amyE*::ϕ(*rbs'-lacZ*)	JH703 × KD891 DNA
SWV203	*spo0AΔ204 abrB4* Δ*amyE*::ϕ(*rbs'-lacZ*)	JH703abrB4 × KD891 DNA
SWV205	*abrB*::*tet* Δ*amyE*::ϕ(*rbs'-lacZ*)	SWV119 × KD891 DNA
SWV217	*spo0AΔ204 abrB*::*tet*	JH703 × SWV119 DNA
SWV218	*spo0AΔ204 abrB2* Δ*amyE*::ϕ(*rbs'-lacZ*)	JH703abrB2 × KD891 DNA
SWV219	*spo0AΔ204 abrB7* Δ*amyE*::ϕ(*rbs'-lacZ*)	JH703abrB7 × KD891 DNA
SWV220	*spo0A12 abrB19* Δ*amyE*::ϕ(*rbs'-lacZ*)	R2-11 × KD891 DNA
SWV221	*spo0A12 abrB20* Δ*amyE*::ϕ(*rbs'-lacZ*)	R15-8 × KD891 DNA
SWV222	*spo0A12 abrB23* Δ*amyE*::ϕ(*rbs'-lacZ*)	R15-13 × KD891 DNA
SWV234	*spo0A12 abrB*::*tet* Δ*amyE*::ϕ(*rbs'-lacZ*)	KD892 × SWV119 DNA
SWV235	*spo0AΔ204 abrB*::*tet* Δ*amyE*::ϕ(*rbs'-lacZ*)	SWV217 × KD891 DNA
SWV236	*abrB*::*tet* Δ*amyE*::ϕ(*rbs'-lacZ*)	KD891 × SWV119 DNA
SWV247	*ccpA*::*spec*	168 × GM1225 DNA
SWV254	*spo0AΔ204 ccpA*::*spec* Δ*amyE*::ϕ(*rbs'-lacZ*)	SWV202 × SWV247 DNA
SWV256	*spo0AΔ204 abrB*::*tet ccpA*::*spec* Δ*amyE*::ϕ(*rbs'-lacZ*)	SWV235 × SWV247 DNA
SWV259	*ccpA*::*spec* ΔamyE::ϕ(*rbs'-lacZ*)	SWV201 × SWV247 DNA
SWV261	*abrB*::*tet ccpA*::*spec* Δ*amyE*::ϕ(*rbs'-lacZ*)	SWV205 × SWV247 DNA

[a] All strains carry the *trpC2* and *pheA1* alleles except 168 and SWV247, which have only *trpC2*.

the *spo0A* effect was mediated through *abrB*. Additionally, while the pattern of *rbs* expression in a *spo0A*$^+$ *abrB23* strain was similar to that of the wild type, the *abrB23* mutant exhibited a lower peak level of expression during the increase, initiating at $t_{-1.5}$. Since it was possible that the observed *abrB*-mediated effects might somehow be allele specific (due to either the *spo0A* or *abrB* allele), I tested a number of different combinations of *spo0A* and *abrB* alleles. Strains SWV221 (*spo0A12 abrB20*) and SWV234 (*spo0A12 abrB*::*tet*) gave results identical to those for SWV222 (*spo0A12 abrB23*) (data not shown). Representative results for strains with the *spo0AΔ204* allele, with and without different *abrB* mutations, are shown in Fig. 1B. The results were generally consistent with the interpretation that the observed *spo0A* effects were mediated through *abrB*. Since *spo0A* mutations result in higher levels of *abrB* transcription (15, 22), the higher levels of *rbs* expression seen in *spo0A* mutants can be attributed to some type of positive regulatory effect of AbrB on *rbs*. Additionally, the lower peak of *rbs* expression seen in *abrB* mutants than in the wild type can be explained by assuming a positive effect of

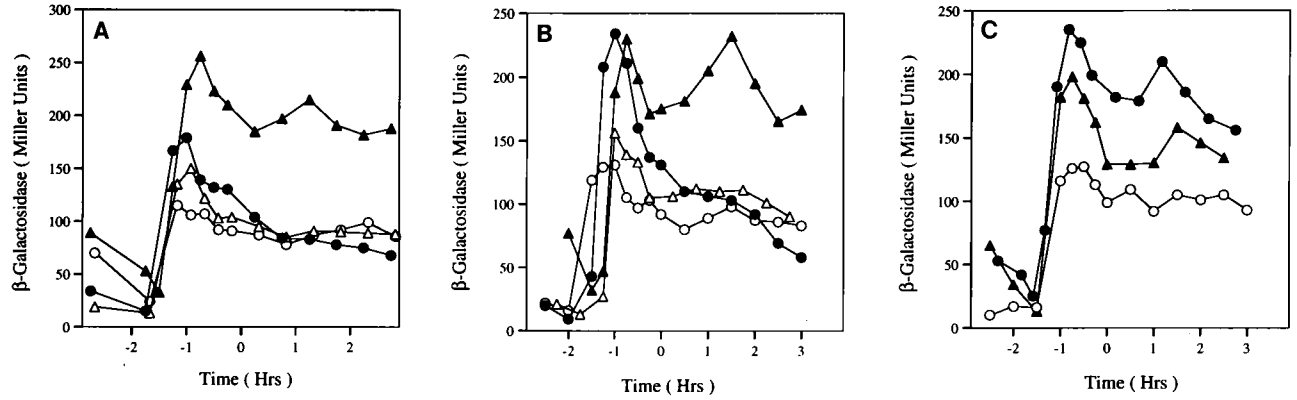

FIG. 1. Time course of *rbs-lacZ* expression. (A) ●, KD891 (wild type); ▲, KD892 (*spo0A12*); ○, SWV236 (*abrB*::*tet*); △, SWV222 (*spo0A12 abrB23*). SWV221 (*spo0A12 abrB20*) and SWV234 (*spo0A12 abrB*::*tet*) gave patterns and levels that were the same as those of SWV222. (B) ●, SWV201 (wild type); ▲, SWV202 (*spo0AΔ204*); ○, SWV205 (*abrB*::*tet*); △, SWV203 (*spo0AΔ204 abrB4*). SWV235 (*spo0AΔ204 abrB*::*tet*) gave the same pattern and levels as SWV203. (C) ●, SWV202 (*spo0AΔ204*); ▲, SWV219 (*spo0AΔ204 abrB7*); ○, SWV218 (*spo0AΔ204 abrB2*).

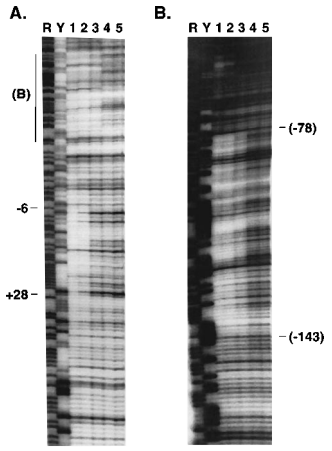

FIG. 2. DNase I footprints of AbrB binding to the *rbs* promoter. (A) Results obtained when the DNA fragment was end labeled on the nontemplate strand. Lanes: 1, 24 μM AbrB; 2, 16 μM AbrB; 3, 8 μM AbrB; 4 and 5, no AbrB. Numbers to the left denote base positions relative to the start point (+1) of transcription (see Fig. 3). The protected region indicated (B) corresponds to the DNase I footprint region shown in panel B. Maxam-Gilbert purine (R) and pyrimidine (Y) sequencing ladders are shown for reference. (B) Results obtained when the template strand was end labeled. Lanes 1 to 5 contained the same AbrB concentrations as indicated for panel A.

AbrB. As further confirmation of this positive role of AbrB, I examined *rbs* expression in a *spo0A* strain containing the *abrB7* mutation. This mutation is located in the promoter and results in lower transcription of *abrB* (29). However, some functional AbrB protein is present intracellularly, and these lower levels can (at least partially) fulfill the regulatory role of AbrB for some genes (6, 15, 25). As shown in Fig. 1C, the level of *rbs* expression in a *spo0AΔ204 abrB7* strain was intermediate between the levels seen in *spo0AΔ204 abrB*$^+$ and *spo0AΔ204 abrB2* (null mutation in *abrB*) strains.

AbrB binds to the *rbs* promoter region. To determine if the observed positive effect of AbrB on *rbs* expression was due to a direct DNA-binding interaction at the promoter, I performed DNase I footprinting analysis. As shown in Fig. 2, AbrB binds to two separate regions on the *rbs* promoter. These regions essentially bracket the −35 and −10 elements (Fig. 3). The regulatory role of the more extensive upstream binding site is not readily apparent, but a clue to the function of the shorter downstream site was suggested by the fact that it covers a sequence (−1 to +13) that is probably involved in catabolite repression of *rbs* transcription (26, 27).

AbrB modulation of catabolite repression and ribose induction of *rbs*. While it cannot overcome the repressive effects of glucose on *hut* expression, overexpression of AbrB can partially restore *hut* transcription when cells are grown on a more slowly metabolized but catabolite-repressing carbon source (i.e., arabinose) (6). The latter effect was correlated with competition between AbrB and the putative *B. subtilis* catabolite repressor protein (CcpA) for a binding site in the *hut* operon (6). Since the presence of 1% glucose in a rich medium has been shown to result in catabolite repression of *rbs* transcription (27), I sought to ascertain if a similar mechanism was responsible for modulating catabolite repression of *rbs*.

I first examined *rbs* expression in a rich medium (SM) containing either glucose or arabinose (Table 2). Glucose severely repressed *rbs* in wild-type, *spo0A*, *abrB*, and *spo0A abrB* cells. Arabinose decreased *rbs* expression only about twofold in wild-type, *abrB*, and *spo0A abrB* cells, but the *spo0A* mutant SWV202, which overexpresses *abrB*, had a derepressed level. The presence of a *ccpA* mutation relieved both glucose and arabinose repression in all genetic backgrounds tested, and in fact, the *ccpA* derivatives gave about twofold-higher levels in the absence of added sugars than their *ccpA*$^+$ counterparts. These results indicated that overexpression of AbrB could at least partially mimic the effect of a *ccpA* mutation on *rbs* expression in the presence of arabinose but not glucose.

I next examined *rbs* expression in a defined minimal medium containing various added sugars. Each medium also contained citrate and glutamate, since *B. subtilis ccpA* mutants are known to have growth defects in minimal medium containing a variety of carbon sources unless tricarboxylic acid cycle intermediates or their precursors are also present (9, 28). Relative to the level in the absence of added sugars, *rbs* expression in the wild-type background (SWV201) was subject to catabolite repression (to varying degrees) by all three sugars and combinations tested (Table 2). A *ccpA* mutation relieved these repressive effects. A *spo0A* mutation increased *rbs* expression (relative to that in the wild type) in each medium, with the two highest levels being observed in arabinose-grown cells and in cells grown without added sugars. These levels were about three times higher than those seen for the *ccpA* mutant in identical media. The significantly lower *rbs* levels in the *spo0A abrB* strain indicated that these highest levels were mainly due to the *spo0A* effect on *abrB* expression. A *ccpA spo0A* strain gave consistently higher *rbs* expression than the *ccpA* mutant, implying that the AbrB effect cannot be completely attributable to antagonism between AbrB and CcpA. An additional regulatory factor may also be controlled by *spo0A*, since *spo0A abrB* derivatives gave slightly higher levels than did *spo0A*$^+$ *abrB*$^+$ strains in some media. Finally, although AbrB bound regions near the promoter in vitro (Fig. 3), AbrB did not appear to significantly repress *rbs* transcription in vivo (Table 2, *ccpA* versus *ccpA abrB*) (see Discussion).

Overproduction of AbrB can suppress growth defects caused by *ccpA* mutations. Surprisingly, a *ccpA abrB* strain could not grow in the presence of arabinose even if supplemented with citrate, glutamate, and either glucose or ribose (Table 2). As mentioned above, *ccpA* strains are known to have various growth defects. It has also been observed that *spo0A* strains generally have faster growth rates that can be attributed to overproduction of AbrB (6). To examine the relationship between *ccpA*, *abrB*, and growth rate, the approximate mean doubling times in MOPS minimal medium (glutamate and citrate) supplemented with glucose, arabinose, or glucose plus arabinose were determined (Table 3). The poor growth of *ccpA* mutants was overcome by a *spo0A* mutation in all three media. For glucose-grown cells, the *spo0A* suppressor effect could be wholly correlated to overproduction of AbrB (*spo0A*

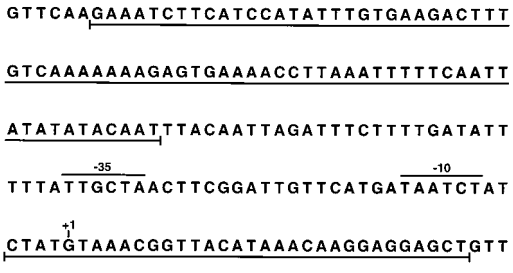

FIG. 3. AbrB-binding regions in the *rbs* promoter. For clarity, the sequence of only the nontemplate strand is shown. The brackets mark the areas of *abrB* afforded protection from DNase I cleavage. The −35, −10, and +1 elements of the promoter are indicated. The putative site of action of the catabolite repressor function is from −1 to +13 (TGTAAACGGTTACA) (26, 27).

TABLE 2. Expression of an *rbs-lacZ* fusion in wild-type and mutant cells[a]

Medium[b] and sugar added	β-Galactosidase activity (Miller units)							
	SWV201 (wild type)	SWV202 (*spo0A*)	SWV205 (*abrB*)	SWV235 (*spo0A abrB*)	SWV259 (*ccpA*)	SWV254 (*ccpA spo0A*)	SWV261 (*ccpA abrB*)	SWV256 (*ccpA abrB spo0A*)
Rich medium								
None	200	300	140	150	370	530	310	310
Glucose	10	12	15	27	310	250	260	390
Arabinose	110	290	70	77	370	510	300	300
Minimal medium								
Glucose	5	28	10	13	180	450	200	300
Arabinose	30	440	20	44	140	550	DNG[c]	240
Ribose	49	120	43	53	160	440	160	240
Glucose + arabinose	10	26	4	12	140	450	DNG	230
Glucose + ribose	5	21	4	17	170	380	110	200
Arabinose + ribose	26	170	12	31	90	320	DNG	130
None	84	530	110	170	160	510	230	310

[a] Cells grown in minimal medium were sampled during mid-logarithmic growth. Samples of cells grown in Schaeffer's were from 1 h prior to the onset of stationary phase (t_{-1}). The values are averages of at least two separate determinations. The standard errors of these values did not exceed 20% in any instance.
[b] Rich medium was Schaeffer's medium containing the indicated sugar at 1%. Minimal medium was MOPS minimal medium containing 0.2% NH_4Cl, 0.2% glutamate, and 0.2% citrate and the indicated sugars at 0.5%.
[c] DNG, did not grow in this medium.

abrB ccpA mutant equivalent to *ccpA* mutant). In the presence of arabinose (with or without glucose), overproduction of AbrB can be only partially responsible for the *spo0A* effect. An *abrB ccpA* strain grew very poorly on glucose and not at all if arabinose was present. The nature of this arabinose-repressive effect on *ccpA abrB* mutant growth is unclear, but since *spo0A* could partially overcome the phenomenon, some unknown function controlled by Spo0A appears to be involved.

DISCUSSION

The results demonstrate that AbrB plays a role in regulating expression of a ribose transport operon of *B. subtilis*. While AbrB does not appear to affect the temporal pattern of *rbs* transcription in a rich medium, the absence of AbrB does result in a lower peak level of expression seen at ~1 h prior to the onset of stationary phase, and overproduction of AbrB (due to a *spo0A* mutation) causes a higher level of *rbs* expression to be maintained during the transition from active growth and into the early stages of stationary phase and sporulation (Fig. 1). The footprinting results strongly suggest that these effects are due to a direct binding interaction of AbrB with the *rbs* promoter (Fig. 2 and 3).

The experiments examining *rbs* expression in cells grown in the presence of different carbon sources (Table 2) indicate that AbrB modulates catabolite repression of *rbs*. These results, in conjunction with the location of one of the AbrB-binding sites, suggest a mechanism involved in this modulation. The downstream AbrB-binding site contains a sequence believed to be necessary for catabolite repression (26, 27). Presumably, this catabolite repression sequence is a binding site for the *B. subtilis* catabolite repressor protein (CcpA). Since overproduction of AbrB (due to a *spo0A* mutation) relieves catabolite repression of *rbs*, a situation involving binding competition between AbrB and CcpA for the catabolite repression sequence region can be envisioned. Such a model has already been invoked to account for AbrB modulation of *hut* expression under partial catabolite-repressing conditions (6). AbrB-CcpA competition for shared binding sites may be a general regulatory mechanism for many but not all (6) *B. subtilis* genes subject to catabolite repression.

Binding competition between AbrB and CcpA is probably not the only regulatory mechanism controlling the level of *rbs* expression. A *spo0A ccpA* strain gave higher levels of expression than either the *ccpA* or *spo0A abrB ccpA* mutant, suggesting that overproduction of AbrB might also inhibit a negative regulator other than CcpA. This unidentified negative regulator may also be directly controlled to some extent by Spo0A; alternatively, the slight differences in *rbs* expression between the *spo0A abrB ccpA* and *ccpA* (with or without *abrB*) mutants in minimal medium may reflect Spo0A control of a different regulatory factor.

A strong candidate for an additional regulator of *rbs* is the protein encoded by the first gene (*rbsR*) of the operon (27). This protein has significant homology to the *Escherichia coli* RbsR repressor protein (13). Ribose induction of *E. coli rbs* (12) is believed to be the result of ribose binding to RbsR, an event that reduces the affinity of the repressor protein for its DNA target (13). However, a similar mechanism may not occur in *B. subtilis*, as suggested by the results presented in Table

TABLE 3. Doubling times in minimal medium[a]

Sugar(s) added	Doubling time (min)							
	SWV201 (wild type)	SWV202 (*spo0A*)	SWV205 (*abrB*)	SWV235 (*spo0A abrB*)	SWV259 (*ccpA*)	SWV254 (*spo0A ccpA*)	SWV261 (*abrB ccpA*)	SWV256 (*spo0A abrB ccpA*)
Glucose	80	50	75	80	180	70	>300	180
Arabinose	105	60	105	85	>300	60	NG[b]	120
Glucose + arabinose	80	60	65	80	>300	60	NG	120

[a] Cells were grown in MOPS minimal medium containing 0.2% NH_4Cl, 0.2% glutamate, 0.2% citrate, and the indicated sugar(s) at 0.5% (each).
[b] NG, no growth.

2. The *rbs-lacZ* levels of *ccpA* strains grown in the presence of ribose did not exceed the levels when the cells were grown in the absence of ribose, implying that ribose inactivation of a repressor protein that acts in the immediate vicinity of the *rbs* promoter did not occur. (The possibility that the two copies of the *rbs* promoter present in these strains—one at the native chromosomal locale, and the other at *amyE*—titrated the effective concentration of a repressor seems unlikely but cannot be excluded.) Additionally, *rbs-lacZ* levels in *ccpA*$^+$ strains were noticeably lower during growth on ribose than during growth in the absence of added sugars. These lower levels probably reflected catabolite-repressive effects and were subject to elevation when AbrB was overproduced (Table 2).

The DNase I footprint indicates that AbrB binds to two separate regions flanking the −35 and −10 elements of the promoter, but there is no evidence that AbrB inhibits transcription of *rbs* (see Results). The location of AbrB bound to the upstream region would not appear to present a significant barrier to RNA polymerase access. In contrast, AbrB binding at the downstream position, which gives protection from DNase I cleavage from −4 to +29, might be expected to hinder RNA polymerase interaction to some extent. But protected bases near the ends of DNase I footprints are usually the result of steric exclusion of the enzyme (3) and do not necessarily reflect points of strong interaction between the binding protein and the DNA. Therefore, it is not unreasonable to assume that the large region between bound AbrB molecules would be sufficient to allow RNA polymerase access, with subsequent displacement of the AbrB bound at the downstream location. However, assuming that CcpA binding is centered around a catabolite-responsive element (CRE) sequence as it is at the *amyO* site (10), the CcpA bound to the *rbs* promoter (CRE located at −1 to +13) might mask RNA polymerase recognition elements more effectively than bound AbrB. Additionally, the relative in vivo association and dissociation constants of binding would be determining factors.

Besides modulating CcpA-dependent catabolite repression of individual operons (6; this study), overproduction of AbrB can also alleviate some general growth defects caused by *ccpA* mutations (Table 3). Since the nature of these *ccpA* defects is unknown, speculations about the role played by AbrB cannot be made at this time.

Although the primary function of AbrB is believed to be in preventing inappropriate stationary-phase gene expression from occurring during active growth (20, 21), evidence is accumulating that AbrB functions to modulate gene expression during slow exponential growth. This regulatory role is not surprising, considering that the onset of stationary phase is the quintessential slow-growth period.

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