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Cell Division Gene *ftsQ* Is Required for Efficient Sporulation but Not Growth and Viability in *Streptomyces coelicolor* A3(2)

JOSEPH R. MCCORMICK AND RICHARD LOSICK*

Department of Molecular and Cellular Biology, The Biological Laboratories, Harvard University, Cambridge, Massachusetts 02138

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We show that the cell division gene *ftsQ* of *Streptomyces coelicolor* A3(2) is dispensable for growth and viability but is needed during development for the efficient conversion of aerial filaments into spores. Combined with our previous demonstration that *ftsZ* of *S. coelicolor* is not needed for viability, these findings suggest that cell division has been largely co-opted for development in this filamentous bacterium. This makes *S. coelicolor* an advantageous system for the study of cell division genes.

Streptomycetes are gram-positive filamentous soil bacteria that grow as a mycelial fungus-like branching network of multinucleoid hyphae. As streptomycete colonies age they undergo an elaborate progression of morphological differentiation, which involves the formation of an aerial mycelium consisting of multinucleoid hyphae that grow away from the surface of the colony. At a subsequent stage of development, the aerial hyphae undergo extensive septation into chains of uninucleoid cells that further metamorphose into spores. In *Streptomyces coelicolor* A3(2), the species for which morphological differentiation has been most extensively characterized, two principal classes of genes that govern spore formation, *bld* and *whi*, have been identified (8). The *bld* genes are required for the formation of the aerial mycelium, whereas the *whi* genes are needed for the subsequent conversion of aerial filaments into chains of spores. *bld* mutants have smooth, bald colony surfaces, and *whi* mutant colonies produce fuzzy white aerial mycelia and do not acquire the gray pigment associated with spore formation.

Recently, we initiated a study of the role of cell division in growth and morphological differentiation in *S. coelicolor* (25). A centrally important cell division gene in *Escherichia coli* and *Bacillus subtilis* is *ftsZ*, whose product is a tubulin-like protein (4, 11, 15, 26, 27, 30) that acts at an early stage of cell division, polymerizing into a ring at the site of septum formation (3, 21). The ring presumably serves as a scaffold for the assembly of the cell division apparatus. In several kinds of bacteria, *ftsZ* is essential for cell division (1, 9) and is therefore required for viability. Strikingly, in *S. coelicolor*, however, *ftsZ* is dispensable for growth even though a null mutant lacks all cross walls, including those normally associated with vegetative growth (25). Presumably, the mycelial lifestyle of this filamentous organism, which normally has only occasional cross walls, does not require cell division. However, *ftsZ* is required during sporulation. A *ftsZ*-null mutant forms an aerial mycelium but is unable to convert aerial hyphal filaments into uninucleoid compartments prerequisite for spore formation. Thus, *ftsZ* is an additional developmentally important gene in *S. coelicolor*.

To further study the role of cell division in growth and morphological differentiation of *S. coelicolor*, we initiated a genetic analysis of the homolog of the *E. coli ftsQ* gene (2). FtsQ is a cytoplasmic membrane protein containing a single membrane-spanning segment near the N terminus and a large C-terminal domain oriented in the periplasm (6). In *E. coli*, FtsQ appears to be required throughout cell division (6), but in *B. subtilis*, the FtsQ homolog (DivIB) is thought to regulate the frequency of division initiation (18). We report here that *ftsQ* is dispensable for growth of *S. coelicolor* but is required for the efficient subdivision of aerial filaments into uninucleoid spores. From these and our previous results with *ftsZ*, we infer that cell division is dispensable in *S. coelicolor* and that cytokinesis has been mainly co-opted for development. This suggests that *S. coelicolor* is an unusually favorable organism to use for genetic studies of cell division.

MATERIALS AND METHODS

Bacterial strains, plasmids, and media. The prototrophic, SCP1$^-$ SCP2$^-$ *S. coelicolor* A3(2) strain M145 was used for all experiments (19). HU133 (Δ*ftsZ*::*aphI*) was derived from M145 (25). Strains HU151 and HU153 (both Δ*ftsQ*::*aadA*) were made by transforming M145 to spectinomycin resistance with pJR122. Standard procedures for transformation and mating were used (19). *Streptomyces* media have been described previously (19, 25). *Streptomyces* strains were grown at 30°C. Concentrations of antibiotics used for selection were thiostrepton at 50 μg ml^{-1}, spectinomycin at 200 μg ml^{-1}, and lincomycin at 200 μg ml^{-1}.

pJRM9 is a bifunctional replicon which was made by fusing *Streptomyces* low-copy-number vector pIJ922 (19) and a pBluescript II SK(+) (Stratagene) derivative containing a *S. coelicolor* DNA insert with *ftsQ* and *ftsZ* (25). pES7, pJR89, and pJR92 are pBluescript II SK(+) derivatives with DNA inserts from the *ftsZ* region of the chromosome (25). pIJ2925 is pUC18 with *Bgl*II sites flanking the multiple cloning site (20). pHP45Ω was the source of *aadA* as the omega fragment (29). pIJ4026 was the source of *ermE* (5). C69 (provided by M. Redenbach, H. M. Kieser, and D. A. Hopwood) is cosmid SuperCos I (Stratagene) containing an insert DNA from the *ftsZ* region of the *S. coelicolor* chromosome.

E. coli LL308 was used for standard plasmid manipulation (34). MJC129 is *ftsQ1*(Ts) strain MJC127 (*recA*::*cat* [6]) and was obtained from L. Guzman. Helper phage R408 (31) or M13K07 (33) was used for single-stranded plasmid production. Unmodified plasmid DNA used for *Streptomyces* transformation was prepared from the *dam dcm E. coli* strain GM2163 (28). *E. coli* strains were propagated in Luria-Bertani medium. Ampicillin was used at 100 μg ml^{-1}.

DNA manipulation and nucleotide sequence analysis. Restriction and modifying enzymes were used according to the manufacturer's recommendations. Plasmid DNA was prepared by alkaline lysis. DNA fragments were purified from low-melting-point agarose by using Elutip-d columns (Schleicher & Schuell). Single-stranded DNA was sequenced with modified T7 DNA polymerase (Sequenase; U.S. Biochemicals). dITP was substituted for dGTP to resolve ambiguities and compressions. A set of nested deletions of pJR89 (25) was used to sequence one DNA strand. The second strand sequence was completed by using custom oligonucleotides with appropriate templates. The Genetics Computer Group DNA-protein programs were used for sequence assembly and analysis (12). Pairwise comparisons of FtsQ amino acid sequences were done by using the GAP program with a Gap Weight of 5.0 and a Gap Length Weight of 0.3. Amino acid sequences were aligned by using the PILEUP program with a Gap Weight of 5.0 and a Gap Length Weight of 0.3. The species and GenBank accession numbers for the *ftsQ* sequences used are as follows: *E. coli*, X02821; *B. subtilis*,

* Corresponding author. Phone: (617) 495-4905. Fax: (617) 496-4642. Electronic mail address: losick@biosun.harvard.edu.

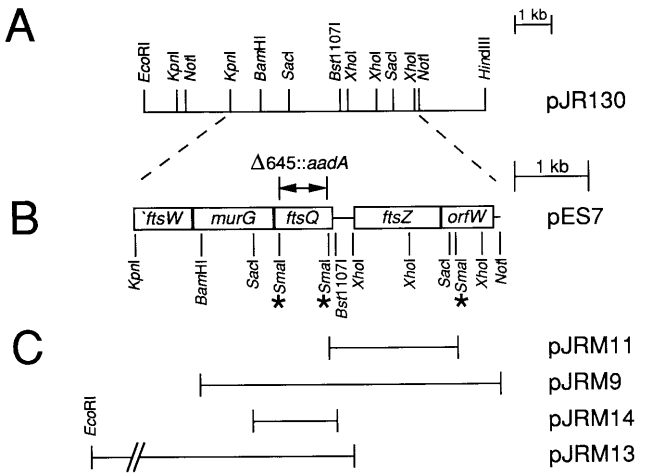

FIG. 1. Physical map of cloned DNA containing $ftsQ_{SC}$. Restriction maps determined for the 9.4-kb *Eco*RI-*Hin*dIII insert in pJR130 (A) and for the 5.1-kb *Kpn*I-*Not*I insert in pES7 (B). The asterisks indicate that only a subset of *Sma*I sites are shown. Gene positions are indicated by the labeled open boxes. All reading frames are oriented from left to right. The location of the region of $ftsQ_{SC}$ that was replaced by the spectinomycin resistance gene *aadA* in the construction of a deletion-mutated copy of $ftsQ_{SC}$ contained in pJR122 is indicated by the double-headed arrow above the gene. (C) DNA inserts in plasmids used for complementation experiments. The ends of the DNA inserts are aligned with the appropriate sites on the restriction map in panel B except that the DNA insert in pJRM13 extends upstream of the *Kpn*I site to the *Eco*RI site shown in panel A.

L25649; *Bacillus licheniformis*, U01958; *Haemophilus influenzae*, L45777; *Borrelia burgdorferi*, U43739; *Streptomyces griseus*, U07344 and unpublished sequence information (14). The translation start codon of *ftsQ* from *S. griseus* is unknown, and the predicted product may extend 16 amino acids more than is shown (14).

Construction of a plasmid used to create the *ftsQ*-null mutant. We constructed a deletion-insertion mutated version of *ftsQ* in *E. coli*. The resulting plasmid, pJR122, contains *aadA* as part of the omega fragment inserted in place of a 645-bp *Sma*I fragment internal to the *ftsQ* coding sequence (see Fig. 1). First, the omega fragment of pHP45Ω was cloned as a 2-kb *Hin*dIII fragment in pBluescript II SK(+) in the same orientation as the *lac* promoter, creating pJR115. A 330-bp *Sac*I-*Sma*I fragment (*'murG-ftsQ'*) from pJR89 was cloned into pIJ2925 digested with *Sac*I and *Eco*RI (filled in), creating pJR117. A 1.7-kb *Kpn*I-*Sac*I fragment (*'ftsW-murG'*) from pES7 was cloned into pJR117, creating pJR118. A 2-kb *Kpn*I-*Bgl*II (filled in) fragment (*'ftsW-murG-ftsQ'*) of pJR118 was cloned into pJR115 digested with *Kpn*I and *Xho*I (filled in), creating pJR119. A 1.7-kb *Eco*RI-*Xba*I fragment (*'ftsQ-ftsZ-orfW'*) of pJR92 was cloned into pJR119, creating pJR120. Finally, *ermE* was cloned (for use as an additional selectable marker in *S. coelicolor*) as a 1.7-kb *Acc*65I fragment from pIJ4026 into pJR120, creating pJR122.

Southern blot hybridization. Southern blot hybridization was done as previously described (25). Images were copied from an autoradiograph with a UMAX UC840 scanner and processed by using Adobe Photoshop on a Macintosh PowerPC 9500.

Construction of plasmids for complementation experiments. We constructed a bifunctional replicon, pJRM10, and its derivatives with various insert DNAs (Fig. 1C) in *E. coli* and then introduced them into *S. coelicolor*. The resulting plasmids were used to transform M145 to thiostrepton resistance and subsequently mated into the *ftsQ*-null mutant HU151. A 23.4-kb *Eco*RI-*Bam*HI fragment (the *Streptomyces* replicon) of pJRM9 was cloned into pBluescript II SK(+), creating the bifunctional replicon pJRM10. The 1.7-kb *Sma*I fragment of pJR92 was cloned into the *Hin*cII site of pBluescript II SK(+), creating pJR124. Then, a 23.4-kb *Eco*RI-*Bam*HI fragment of pJRM9 was cloned into pJR124, creating pJRM11. A 9.4-kb *Eco*RI-*Hin*dIII fragment of C69 was cloned into pBluescript II SK(+) creating pJR130. A 5.4-kb *Eco*RI-*Xho*I (filled in) fragment of pJR130 was cloned into pBluescript II SK(+) digested with *Eco*RI and *Hin*dIII (filled in), creating pJR135. Then, a 23.4-kb *Eco*RI-*Spe*I fragment of pJRM10 was cloned into pJR135, creating pJRM13. pJR136, containing a 1.1-kb *Sac*I-*Bst*1107I fragment cloned into pBluescript II SK(+) digested with *Sac*I and *Spe*I (filled in), was created directly from pJR89 by deleting a *Bst*1107I-*Spe*I (filled in) fragment. A 23.4-kb *Eco*RI-*Bam*HI fragment of pJRM10 was cloned into pJR136, creating pJRM14.

Microscopic examination methods. Impression slides were made by lightly touching the surface of a sporulating colony to a coverslip, and the coverslips were mounted in 50% glycerol for viewing. A glycerol solution was used to slow the desiccation of prepared slides and to improve the wetting of the surface of hydrophobic spores and aerial filaments. Coverslip lifts were made by inoculating the surface of the agar medium adjacent to the inserted coverslips. Following a period of growth, the coverslips were removed and mounted in 50% glycerol. Microscopy and photomicrography were performed as described previously (16). Images were copied from 35-mm black-and-white negative film with a Kodak RFS 2035 Plus film scanner and processed by using Adobe Photoshop on a Macintosh PowerPC 9500.

Nucleotide sequence accession number. The nucleotide sequence of *ftsQ* has been submitted to GenBank under the accession number U10879.

RESULTS AND DISCUSSION

Nucleotide sequence of *ftsQ*. By extending our previous sequence analysis of the *ftsZ* region of the *S. coelicolor* chromosome upstream (Fig. 2), we identified an open reading frame of 264 codons whose inferred product (28.6 kDa) is similar to FtsQ of *E. coli* (Fig. 3). Residues at 17% of the positions of FtsQ from *S. coelicolor* ($FtsQ_{SC}$) are identical to FtsQ of *E. coli* ($FtsQ_{EC}$), and residues at an additional 23% of the protein are similar. Dharmatilake and Kendrick (13) have independently noted a *ftsQ*-like gene upstream of *ftsZ* in *S. griseus*. The deduced products of the two *Streptomyces ftsQ* genes are very similar (63% identical, 74% similar) but much less similar than the *ftsZ* gene products of the two species (91% identical, 93% similar). A somewhat lower level of similarity was observed for *S. coelicolor* FtsQ and the putative FtsQ protein from the spirochete *B. burgdorferi* (14% identical, 40% similar) and the putative FtsQ proteins (DivIB) from gram-positive bacteria *B. subtilis* (13% identical, 34% similar) and *B. licheniformis* (13% identical, 33% similar). Each of the FtsQ proteins contains a stretch of hydrophobic residues near the N terminus, which represents a membrane-spanning segment as previously shown for FtsQ of *E. coli* (Fig. 3). Interestingly, only 2 amino acids are identical in all members of the FtsQ family of proteins (Fig. 3). A similar observation that few amino acids are conserved in the FtsQ-like proteins was made for the analysis of FtsQ homologs of *Rhizobium meliloti* and *Agrobacterium tumefaciens* (22). A block of sequence with the highest density of similar and identical residues occurs at amino acid positions 62 to 171 in the alignment shown in Fig. 3. This block of similarity is in the C-terminal domain which was previously shown (6) to be located in the periplasm for FtsQ of *E. coli*, where it could act as part of a link between cell division and cell wall synthesis machinery. The N-terminal domain of FtsQ-like proteins lack conserved residues (Fig. 3), suggesting that its sequence may not be important for function. Consistent with this interpretation, the N-terminal domain of $FtsQ_{EC}$ can be replaced by an analogous segment from an integral membrane protein without loss of function (10). Although the predicted products are similar, $ftsQ_{SC}$ borne on a high-copy-number plasmid expressed from the *E. coli lac* promoter (pJR136) is unable to complement an *E. coli ftsQ* mutant (MJC129) for growth or viability at 42°C.

Construction of a *ftsQ*-null mutant. To construct a null mutant of $ftsQ_{SC}$, we deleted a 645-bp segment of DNA internal to the predicted $ftsQ_{SC}$ reading frame, using *Sma*I sites at bases 107 and 752 in our sequence (Fig. 1 and 2). This deletion includes the single predicted membrane-spanning segment of the deduced product. The deleted sequences in $ftsQ_{SC}$ were replaced by a spectinomycin resistance gene (*aadA* on the omega fragment). Then, we introduced the deletion-mutated version of the $ftsQ_{SC}$ gene into the chromosome by a marker replacement (double) recombination strategy similar to that used to create a $ftsZ_{SC}$-null mutant (25). Spectinomycin-resistant transformants of wild-type strain M145 were selected. The plasmid vector used for marker replacement, pJR122, does not replicate in *S. coelicolor* and was designed to also contain the

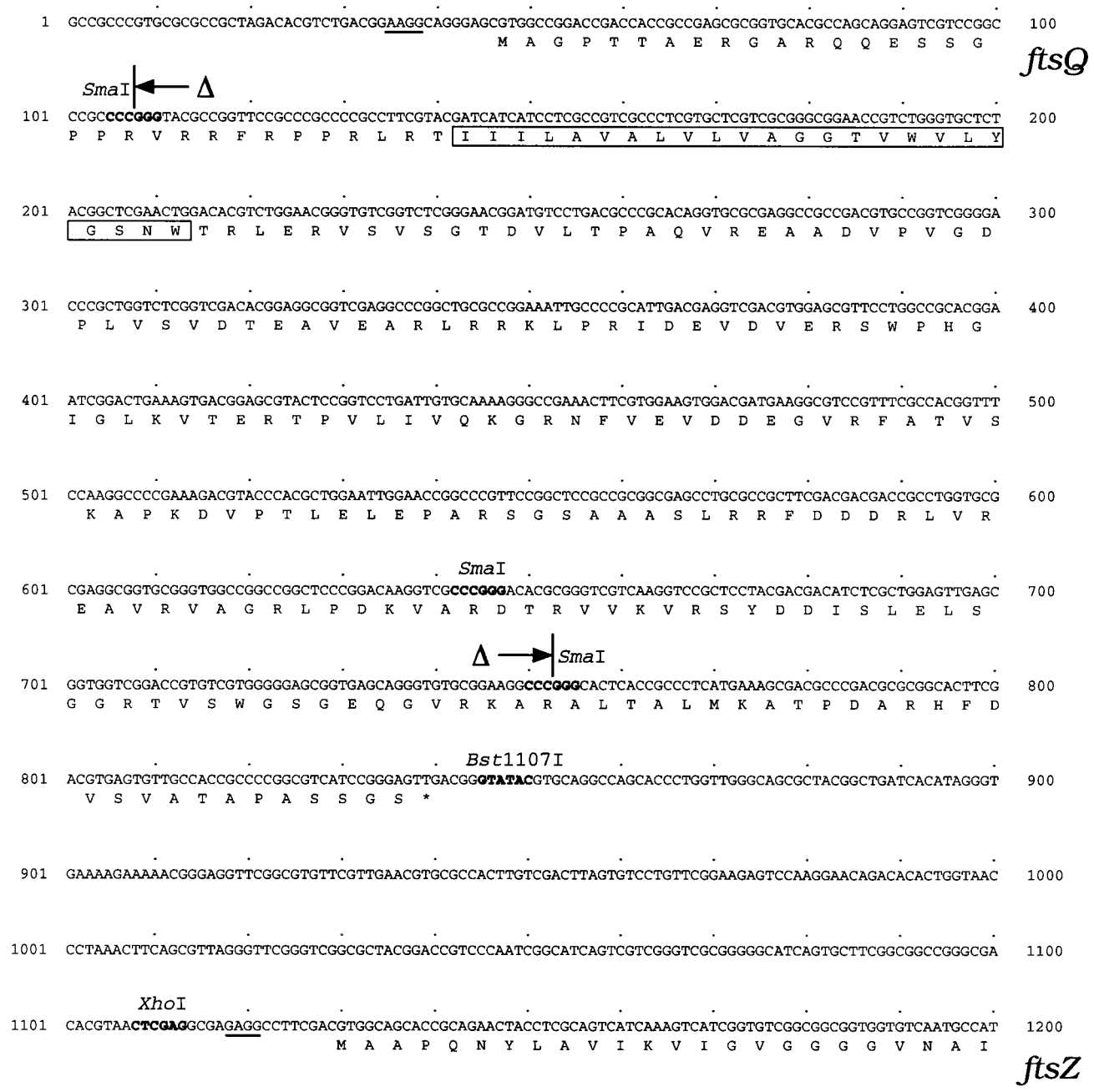

FIG. 2. Nucleotide sequence of the $ftsQ_{SC}$ region of the chromosome and deduced amino acid sequence. The DNA sequence of the nontemplate strand is shown with the deduced amino acid sequence given below in single-letter code. The stop codon is indicated by an asterisk. Putative Shine-Dalgarno sequences are underlined. The amino acid sequence of an inferred membrane-spanning segment is boxed. Restriction enzyme recognition sequences are indicated in boldface type. The *Sma*I sites used for the construction of the deletion mutation are indicated by arrows.

erythromycin resistance gene *ermE*, an additional antibiotic resistance gene selectable in *S. coelicolor* (Fig. 1 and Materials and Methods). This allowed spectinomycin-resistant transformants arising from single reciprocal recombination (Campbell-like) events to be distinguished from marker replacement (double) recombination. Putative $ftsQ_{SC}$-null mutant strains were identified as transformants resistant to spectinomycin but sensitive to erythromycin. The predicted deletion-insertion mutation was verified by Southern blot hybridization experiments (Fig. 4) (24). Two such identified and verified strains, HU151 and HU153, were chosen for further work. That it was possible to obtain deletion mutants indicates that $ftsQ_{SC}$ is not essential for viability. Coupling this result with a similar previous result for $ftsZ_{SC}$ (25), we infer that dispensability is a general feature of cell division genes in *S. coelicolor*.

Phenotype of the *ftsQ*-null mutant. Because of their mycelial nature, streptomycetes are routinely subcultured by mechanical maceration, which generates fragments that can give rise to new colonies. When mechanical maceration was used to subculture the $ftsQ_{SC}$ mutants, CFU were readily obtained but at a slightly lower efficiency than for the wild type. Whereas minimal medium-grown wild-type colonies produced little pig-

FIG. 3. Amino acid sequence alignment of FtsQ proteins. Amino acid sequences, shown in single-letter code, were aligned by using the PILEUP program, and amino acid similarities are enclosed by blocks. The amino acid sequences are from *S. coelicolor* (Sc), *S. griseus* (Sg), *E. coli* (Ec), *H. influenzae* (Hi), *B. subtilis* (Bs), *B. licheniformis* (Bl), and *B. burgdorferi* (Bb) (see Materials and Methods for GenBank accession numbers). Solid boxes indicate identical residues in at least six of seven sequences. Open boxes indicate identical residues in four or five of seven sequences. Gray boxes indicate positions where similar residues occur in all sequences. The asterisk indicates the residue that is changed in the missense mutation of *E. coli ftsQ1*(Ts) (32).

ment, $ftsQ_{SC}$ mutant colonies produced copious amounts of a blue pigment presumably related to the antibiotic actinorhodin, a phenotype similar to that observed for $ftsZ_{SC}$ mutant colonies (25).

Streptomycetes grow vegetatively as branching hyphal filaments with only occasional cross walls and with multiple nucleoids per syncytial compartment. Previously, we showed by electron microscopic analysis of thin sections of cells that a $ftsZ_{SC}$-null mutation blocks cross wall formation (25). A similar analysis demonstrated that the $ftsQ_{SC}$-null mutation substantially but not completely prevented cross wall formation (23). Thin sections prepared from $ftsQ_{SC}$-null mutant cells contain 5 to 10% of the level of cross walls observed for the wild-type strain. Thus, $ftsQ_{SC}$ is needed for efficient initiation of cross wall formation but is evidently not absolutely required for cytokinesis. However, the possibility that $ftsQ_{SC}$ is required for cell division but that transformants incorporating a null mutation in the gene readily acquire a suppressor mutation(s) that partially compensates for the absence of $FtsQ_{SC}$, resulting in the observed level of cross wall formation, is not excluded.

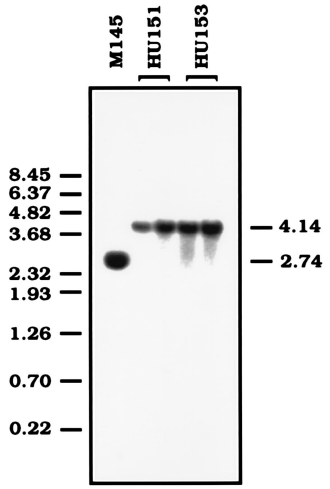

FIG. 4. Southern blot hybridization analysis of DNA from wild-type and spectinomycin-resistant transformant cells. *Sac*I-digested chromosomal DNA samples from a wild-type strain (M145) and from two $ftsQ_{SC}$ deletion-mutant strains (HU151 and HU153) were fractionated and blotted onto nylon membrane. Two independently prepared chromosomal DNA samples were used for each mutant. The blot was hybridized with a radiolabeled 2.75-kb *Sac*I DNA fragment (containing *'murG-ftsQ-ftsZ-orfW'*) (Fig. 1B). The sizes given are in kilobases. The expected sizes of *Sac*I-digested DNA fragments of the wild-type and $ftsQ_{SC}$ deletion-insertion mutants are indicated to the right of the blot. *Bst*EII-digested lambda DNA was used as a molecular weight standard, and the migration positions of a subset of fragments are indicated to the left of the blot.

***ftsQ* is needed for efficient production of sporulation septa.** *S. coelicolor* undergoes an elaborate cycle of morphological differentiation involving the formation of an aerial mycelium composed of aerial hyphae that differentiate into chains of gray-pigmented spores. During development, the multinucleoid aerial hyphal filaments are coiled and then are subdivided into uninucleoid compartments by the formation of sporulation septa. The resulting compartments further metamorphose into mature oval-shaped spores (Fig. 5A). Colonies of the $ftsQ_{SC}$ mutant HU151 form an aerial mycelium both on minimal and on nutrient-rich R2YE media (19). The $ftsQ_{SC}$ mutant was however largely blocked in the conversion of the aerial hyphae into spores, as judged by phase-contrast microscopy of impression preparations of the colonies. The majority of filaments observed in the aerial mycelium are undifferentiated (Fig. 5B). A wide range of aberrant phenotypes were less frequently observed, including straight noncoiled aerial filaments that were undulated in appearance (Fig. 5C) and coiled aerial filaments similar in appearance to known developmental mutants (7) blocked in the early stages of the conversion of aerial filaments into chains of spores (Fig. 5D). Some aerial filaments contained lysed compartments interspersed with undulated regions and/or spores (Fig. 5E). These spores were viable on the basis of the fact that some had germinated (Fig. 5E). The proportion of a single aerial filament that was observed as lysed compartments varied from a small fraction of the filament (Fig. 5E) to the entire filament (Fig. 5F), and the lysed regions of aerial filaments contained remnants of sporu-

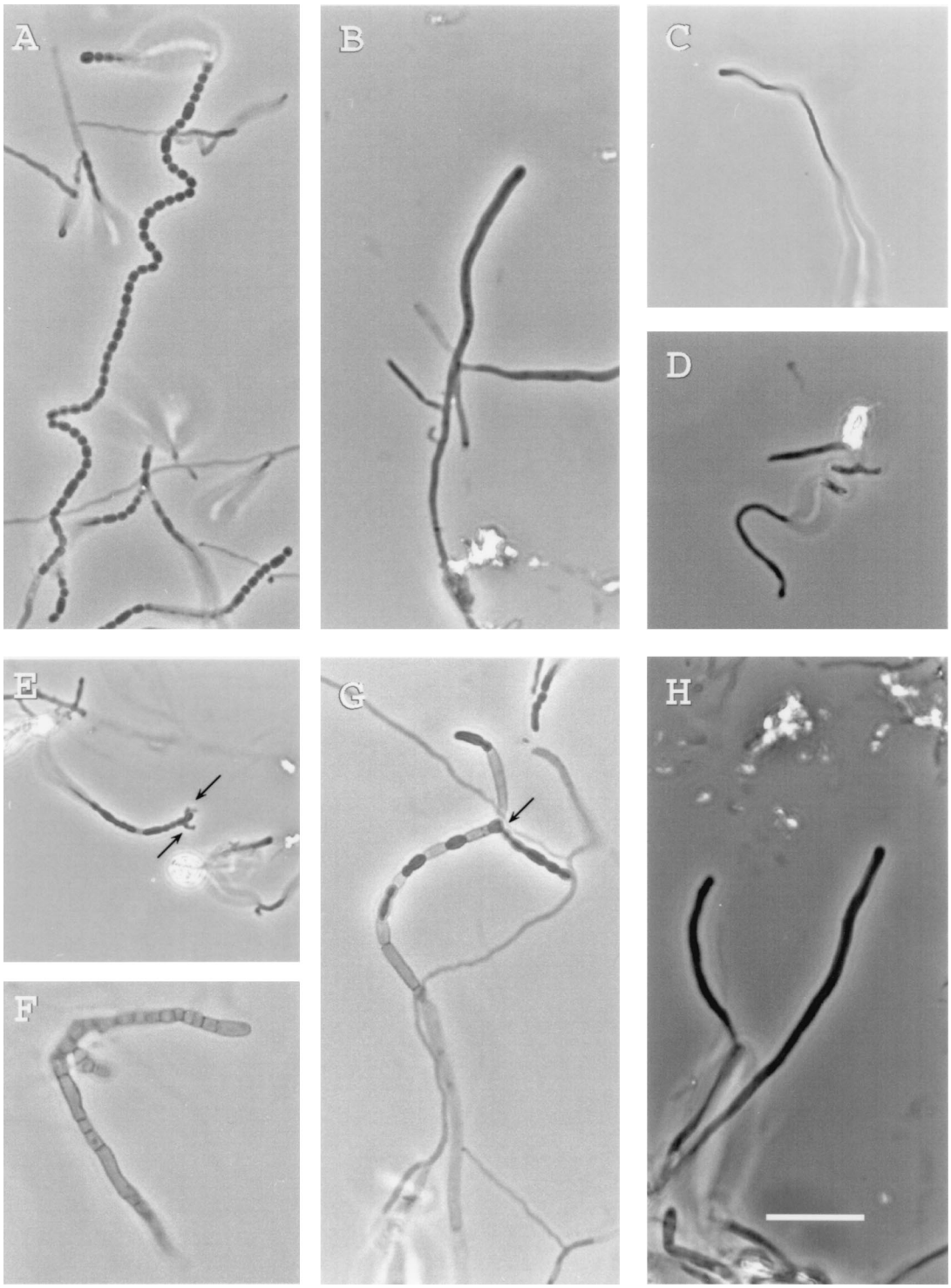

FIG. 5. Phase-contrast microscopy of wild-type, *ftsQ*$_{SC}$-null mutant, and *ftsZ*$_{SC}$-null mutant aerial hyphae. The panels are representative phase-contrast micrographs showing morphological differentiation of aerial hyphal filaments of wild-type M145 (A), *ftsQ*$_{SC}$-null mutant HU151 (B to G), and *ftsZ*$_{SC}$-null mutant HU133 (H). The predominant phenotype of *ftsQ*$_{SC}$-null mutant HU151 is shown in panel B. Germinated spores of the *ftsQ*$_{SC}$-null mutant are indicated by arrows (E). A branched spore in HU151 is indicated by an arrow (G). Panels B and H are impression slides, and panels A and C to G are coverslip lifts. The strains were grown for 5 days on minimal glucose agar. Scale bar = 10 μm.

lation septa (Fig. 5F and G). Most surprisingly, branched spores were also observed (Fig. 5G). Rarely, completed chains of spores were observed (not shown but similar to those in Fig. 5A). Completed chains of spores were also observed for the *ftsQ*$_{SC}$ mutant by electron microscopic analysis (23). This result is in contrast to that for the *ftsZ*$_{SC}$ mutant, which is completely blocked in spore formation (Fig. 5H).

Because *ftsQ*$_{SC}$ mutant colonies grown on minimal medium

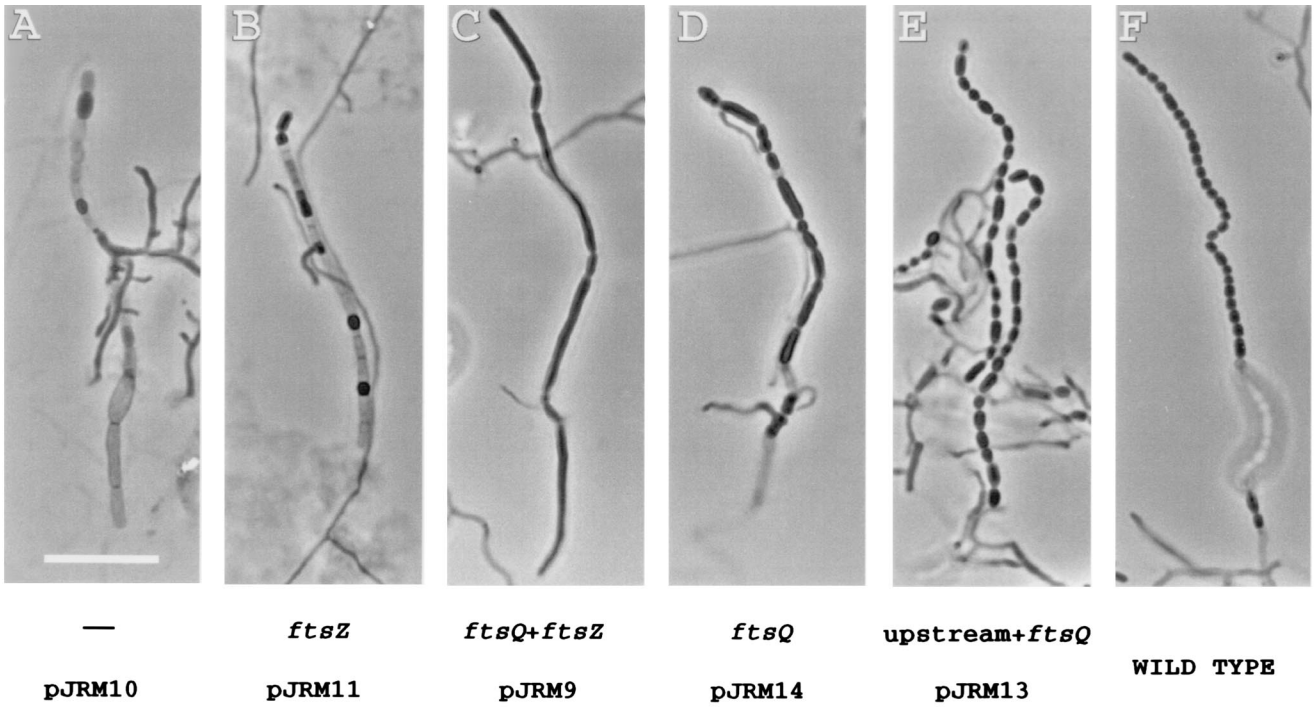

FIG. 6. Phase-contrast microscopy of complementation experiments of the $ftsQ_{SC}$-null mutation by a wild-type copy of the gene. (A to E) The panels are representative phase-contrast micrographs of aerial hyphae of the $ftsQ_{SC}$-null mutant HU151 containing the indicated plasmids (lower line) bearing the listed genes (upper line) on their DNA inserts. A representative micrograph of wild-type strain M145 is included for comparison (F). The strains were grown for 5 days on minimal glucose agar containing thiostrepton (10 μg ml^{-1}). Scale bar = 10 μm.

were similar in appearance to $ftsZ_{SC}$ colonies, it was possible that the $ftsQ_{SC}$ phenotype was entirely the result of polarity on the expression of $ftsZ_{SC}$ rather than being due to the absence of $ftsQ_{SC}$. Complementation experiments to investigate this possibility were conducted. Complementation efficiency was ascertained by viewing and categorizing 100 random examples of aerial filaments. When introduced into HU151, the low-copy-number plasmid pJRM10, which lacks a DNA insert, did not complement the developmental defect of HU151 (Fig. 6A). Complementation was also not observed when pJRM11 was introduced into HU151 (Fig. 6B). pJRM11 contains a complete copy of $ftsZ_{SC}$ and is capable of complementing the defect of a $ftsZ_{SC}$-null mutation (24). In contrast, when pJRM9, which contains $ftsQ_{SC}$ and $ftsZ_{SC}$, or pJRM14, which contains $ftsQ_{SC}$ alone, was introduced into HU151, spore formation was partially restored (Fig. 6C and D). Moreover, when pJRM13, which contains $ftsQ_{SC}$ and 4 kb of sequence upstream of $ftsQ_{SC}$ but not $ftsZ_{SC}$, was introduced into HU151, spore formation was substantially restored (Fig. 6E). We conclude that the phenotype of the $ftsQ_{SC}$ mutant is at least in part a consequence of the absence of $ftsQ_{SC}$ itself and that efficient expression of $ftsQ_{SC}$ evidently depends on promoter sequences located upstream of the *Bam*HI site in $murG_{SC}$, the endpoint of the DNA insert in pJRM9 (Fig. 1). Transcription of the putative *B. subtilis* homolog (*divIB*) of *ftsQ* similarly originates from one or more promoters located several kilobases upstream (17).

In summary, $ftsQ_{SC}$ is dispensable for the growth and viability of *S. coelicolor* but is needed for the efficient conversion of multinucleoid aerial hyphae into uninucleoid spores. Combining these results with our findings with $ftsZ_{SC}$, we conclude that cell division has been largely co-opted for morphological development in this filamentous bacterium, making *S. coelicolor* an advantageous system for the study of cell division genes.

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