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Kinetic Analysis of the Synthesis and Assembly of Type 1 Fimbriae of *Escherichia coli*

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The adhesive organelles (type 1 fimbriae) of K-12 and other isolates of *Escherichia coli* are composed of identical 17,000-dalton subunits. We examined the assembly of these subunits into fimbrial organelles. After synthesis, the nascent subunits were first processed and then assembled into the organelles; the assembly step took almost 3 min in log-phase cultures at 37°C. Even during blockage of protein synthesis, the free subunits continued to assemble until the pool was depleted. This pool was small in comparison with the amount of total fimbrial protein already assembled into surface organelles and was not sufficient to regenerate new detectable organelles after the removal of preexistent ones by blending. Assembly appeared to slow when the metabolic rate of the bacterial cells slowed, since subunits took longer to appear in the organelles at lower than optimal temperatures or as a culture entered the stationary phase. The synthetic rate of subunits slowed sooner than that of total cellular proteins as a culture approached the stationary phase and ceased completely as the culture entered the stationary phase. The amount of fimbrial antigen expressed on the surface of the cells remained relatively constant during growth of a culture.

Type 1 fimbriae (or pili) are proteinaceous appendages 7 nm in width projecting from the surface of many gram-negative bacteria (4). They consist of aggregates of helically arranged identical 17,000-dalton polypeptides with 3.1 subunits per turn of the helix (1). These structures are highly stable; they are resistant to disaggregation by sodium dodecyl sulfate (SDS) or urea (1, 2, 11, 13) but can be broken into individual subunits by saturated guanidine (1, 8) or hot acid (1, 2).

Studies have shown that for many bacterial diseases the adherence of bacteria to the mucosa is a prerequisite for subsequent infection (for reviews see references 4 and 17). Type 1 fimbriae, which are the most commonly found adhesin among *Escherichia coli* isolates (4), are termed mannose sensitive since the adherence mediated by these organelles can be inhibited or reversed by the addition of mannose or closely related compounds (19). These organelles also mediate pellicle formation (17), which occurs in nonshaking rich media when fimbria-bearing cells form a dense mat (or pellicle) at the surface of the broth. Individual *E. coli* cells undergo phase variation, whereby fimbriae expression can oscillate between on-and-off at a rate of ca. 10^{-3} per generation (1, 5). Since pellicle formation allows fimbriate-phase bacteria to outgrow non-fimbriate-phase bacteria in rich nonaerated broth (7, 17), bacteria in older cultures are more fimbriate than those in younger cultures. It is not known how the level of fimbriation varies in a culture not undergoing a selection for one phase or the other during bacterial growth.

We have recently shown that fimbrial subunits are initially synthesized as signal sequence-containing precursors (2a), which are rapidly processed into mature subunits before assembly into organelles. It has been suggested that there is a sufficient number of these nonassembled subunits to regenerate fimbriae after their removal by blending (1). In this communication we describe the kinetics of assembly of these mature subunits.

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MATERIALS AND METHODS

Strains and culture conditions. Strain CSH50 is a Cold Spring Harbor *E. coli* K-12-derived strain with the genotype F⁻ *ara* Δ(*lac-pro*) *rpsL thi* (14). VL386 is a nonfimbriate strain derived from CSH50 after insertion of the bacteriophage Mu d(Ap^r *lac*) into a gene necessary for the production of fimbriae and subsequent replacement of Mu *d* by lambda p209 (9). For all experiments, cells grown overnight in minimal medium A (14) supplemented with 0.5% glucose (or 0.4% glycerol and 0.4% maltose in experiments in which the maltose-binding protein was immunoprecipitated) were inoculated in dilutions between 1:150 and 1:10 into the same broth and grown aerated at 37°C. The cell number was determined from a standard curve of viable counts versus optical density at 550 nm measured in a Spectronic 21 spectrophotometer (Bausch & Lomb, Inc., Rochester, N.Y.)

SDS-PAGE. SDS-polyacrylamide gel electrophoresis (PAGE) was performed in a 1.5-mm slab gel apparatus (Hoefer Scientific, San Francisco, Calif.) by the method of Laemmli (12) as previously described (2).

Chemicals and buffers. All reagents were as previously described (2, 6) except that [^{3}H]leucine (55 Ci/mmol) was from Schwarz/Mann (Spring Valley, N.Y.), chloramphenicol was from Sigma Chemical Co. (St. Louis, Mo.), and glycerol and formaldehyde were from Fisher Scientific (Pittsburgh, Pa.).

Antibodies. For the enzyme-linked immunosorbent assay (ELISA) inhibition assay, we used rabbit serum specific for type 1 fimbriae as previously described (2). For immunoprecipitations, we used this serum, a monoclonal antibody (MAb) previously described (6), and monospecific rabbit anti-maltose-binding protein serum, kindly provided by Phil Bassford, Jr. (10). Rabbit antimouse serum was kindly provided by Judy Teale (6).

RIP. For radioimmunoprecipitation (RIP) we used the method of Ito et al. (10) as follows. Bacteria in 1 ml of minimal medium were labeled for 20 to 30 s with 15 μCi of [^{3}H]leucine. A chase was performed by the addition of 200 μg of L-leucine per ml and 50 μg of L-isoleucine per ml.

Reactions were stopped by the addition of an equal volume of ice-cold 10% trichloracetic acid (TCA). We recovered the precipitate by centrifuging the solution for 3 min at 4°C in a Beckman Microfuge model B (Fullerton, Calif.). After washing the precipitate twice with ice-cold acetone, we solubilized it in 0.1 ml of a 1% SDS–50 mM Tris-hydrochloride (pH 8)–1 mM EDTA solution and performed the immunoprecipitation as previously described (2, 6). The TCA-acetone treatment did not affect the efficiency of immunoprecipitation or cause any detectable disaggregation of organelles into subunits.

ELISA inhibition assay. We performed the ELISA for quantitative determination of fimbrial antigen on the surface of *E. coli* as previously described (2, 6). To prevent any change in the amount of fimbriae present in a culture until completion of the assay, we fixed samples in 1% formaldehyde at the time of removal from a culture. The formaldehyde fixation did not affect the assay.

Temperature studies. To determine the effect of various temperatures on assembly, we placed samples of a culture growing at 37°C in shaking water baths at the appropriate temperature 30 min before labeling. We checked the temperature of the samples by immersing a thermometer (Fisher) directly into the culture tube.

RESULTS

RIP of *E. coli* cells with organelle-specific MAb. When fimbrial subunits assemble into the organelle, they form stable structures capable of resisting disaggregation by SDS. As a result, subunits that have assembled into an organelle will not enter the separation gel of an SDS-polyacrylamide gel (2, 13). We have recently described an MAb that recognizes only subunits that have already assembled into organelles (6). To perform the RIP, we radiolabeled a culture of *E. coli* with [^{3}H]leucine for 30 s followed by a chase of 30 s with excess unlabeled leucine. We found that, similar to our results with immunoblotting (6), cultures labeled in this fashion and then immunoprecipitated with rabbit antifimbria serum display the autoradiographic pattern of both nondisaggregated fimbriae, which are caught in the stacking gel, and fimbrial subunits (data not shown). We have previously shown that if the fimbriae are first disaggregated with hot acid and then subjected to SDS-PAGE, rather than getting caught in the stacking gel, they comigrate with the free subunits (2, 6). The MAb only immunoprecipitated material that does not enter the separating gel (i.e., nondisaggregated fimbriae) (Fig. 1A). When the MAb-cleared supernatant was then immunoprecipitated with monospecific rabbit antifimbria serum, a small amount of whole fimbriae was seen at the stacking gel-separating gel interface (Fig. 1, arrow). Such fimbriae are presumably small enough to migrate through the entire stacking gel. The remainder of the label migrated at apparent molecular sizes of 17,000 and 18,000 daltons. (The two bands were probably due to subunits existing in two conformations during electrophoresis [13] rather than due to a mature subunit and an unprocessed precursor, which should migrate at 19,000 daltons [2a].) We were unable to detect any precursor-product relationship between the 17,000- and 18,000-dalton bands; the ratio of intensity of the two bands remained constant for all labeling times in a particular gel. Since the MAb immunoprecipitated only intact fimbrial organelles, apparently because it recognizes an epitope formed when subunits assemble (6), we were able to use this MAb as a probe for fimbrial assembly in our subsequent kinetic studies.

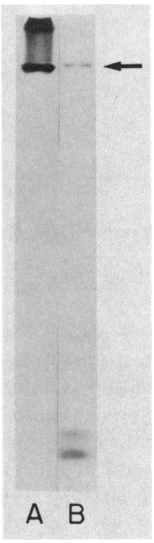

FIG. 1. Specificity of an Mab for fimbrial organelles and of antifimbria serum for both organelles and fimbrial subunits, as shown by RIP. A culture of strain CSH50 was pulse-labeled with [^{3}H]leucine, chased for 30 s with unlabeled leucine, and precipitated with TCA to prevent further metabolic processes. Lane A: Immunoprecipitation with an Mab previously shown to be organelle specific (6). After immunoprecipitation, the immunoprecipitates were analyzed by SDS-PAGE (13% separating gel) and fluorography. Only SDS-stable material (intact organelles) was immunoprecipitated. Lane B: Immunoprecipitation with rabbit antifimbria serum after the removal of intact fimbriae by the MAb. A small amount of material was caught at the stacking gel-separating gel interface (arrow), but most of the immunoprecipitated material migrated as subunits.

Kinetics of subunit assembly into organelles. We used the technique of pulse-chase to analyze the kinetics of subunit assembly. After labeling the bacteria with [^{3}H]leucine for 30 s and then chasing with excess unlabeled leucine for various times, we immunoprecipitated the detergent-solubilized bacteria with either anti-maltose-binding protein Ab or the organelle-specific MAb (Fig. 2). Although the amounts of both total TCA-precipitable label (data not shown) and labeled maltose-binding protein remained constant during the chase, the amount of labeled fimbrial organelles increased to a plateau after 3 min. When the MAb-cleared supernatants were then immunoprecipitated with the rabbit antifimbria serum, we could recover labeled subunits during, but not after, the first 3 min (data not shown).

Since free subunits could be detected at least within the first 3 min after labeling, we reasoned that fimbriae most likely exist in a free subunit pool before their assembly. Nevertheless, some of the observed delay in organelle assembly might actually have been due to a slow rate of subunit synthesis. If, for instance, peptide chain elongation was arrested until specific export sites were located (20), then most of the labeled subunits would not be completely synthesized at the end of the short labeling period. To distinguish between these two possibilities (i.e., unassembled subunit pool or arrested translation), we repeated the pulse-chase analysis under conditions of total translation inhibition. If the delay in label uptake into mature organelles was due to arrested translation, the addition of chloramphenicol at the beginning of the chase period should prevent completion of translation of most of the already labeled elongating polypeptides and therefore prevent their assembly. We found that when the chase was performed with 100

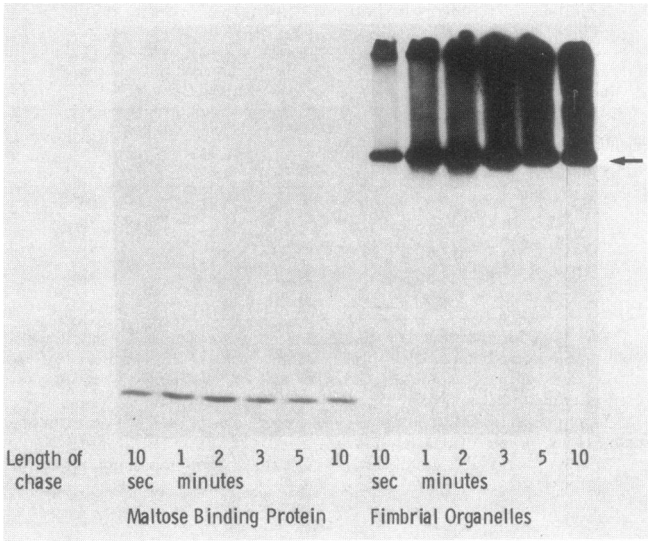

FIG. 2. SDS-PAGE analysis of the time course of assembly of subunits into a form recognizable by the organelle-specific MAb. A culture of strain CSH50 was pulse-labeled with [^{3}H]leucine for 30 s followed by a chase with unlabeled leucine for the indicated times. The first six lanes result from immunoprecipitations of the maltose-binding protein done at the designated time points. Note that the intensity of the band remains constant over the chase period. The last six lanes result from immunoprecipitations done with the organelle-specific MAb at the designated time points. Note that the total amount of immunoprecipitable material (fimbrial organelles) increases for the first few time points and that the amount of smaller fimbriae (those that migrate to the separating gel [arrow]) decreases at the later times relative to the larger fimbriae (those that do not migrate through the stacking gel).

μg of chloramphenicol per ml (a concentration that inhibits both new TCA-precipitable label and new fimbrial subunits by at least 99% within 10 s), the kinetics of fimbrial assembly was virtually unaffected (Fig. 3). Moreover, the inhibition of subunit incorporation into organelles (39%, relative to the sample without chloramphenicol) was not much different from that of the rapidly synthesized maltose-binding protein (40%) or from that of total TCA-precipitable material (31%). Taken together, these results suggest that the delay in labeled subunits appearing in the organelles is almost entirely due to the time required for assembly and that assembly can occur in the absence of protein synthesis.

Effect of temperature on assembly. In contrast to many mannose-resistant fimbriae (3) and F-pili (16), type 1 fimbriae can be expressed at low temperatures such as 18°C (3, 4). It is not known, though, what the effect of temperature is on the rates of synthesis and of assembly of type 1 fimbriae. Therefore, we next examined the kinetics of fimbrial expression of cultures grown at 18, 24, 30, and 37°C. The proportion of TCA-precipitable label that was finally incorporated into the organelles was not affected significantly by temperature (Fig. 4), although the rate at which the subunits were incorporated into the organelles slowed somewhat with lower temperatures. Since the amount of TCA-precipitable label remained constant during the chase period for each temperature, it seems likely that this slowing of incorporation of labeled subunits into fimbriae is due to a decrease in assembly rate. The culture growing at 18°C is growing much slower than the 37°C culture, taking up less than 10% of the [^{3}H]leucine into TCA-precipitable material compared with the 37° culture during the 30-s labeling time.

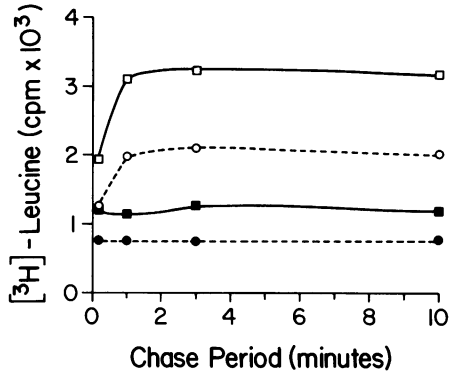

FIG. 3. Effect of chloramphenicol on the synthesis and assembly of fimbriae and on the synthesis of the maltose-binding protein. Immunoprecipitations were performed after different chase periods with either anti-maltose-binding protein serum or antifimbriae MAb, described in the legend to Fig. 2, except that, in addition, the chase was performed either in the presence or in the absence of 100 μg of chloramphenicol per ml. Shown are total counts immunoprecipitated by the MAb in the absence (□) or presence (○) of chloramphenicol or by anti-maltose-binding protein in the absence (■) or presence (●) of chloramphenicol.

Production of fimbriae during growth. To determine the effect of growth phase on fimbrial assembly, we examined incorporation of subunits into organelles at various times during the growth of the culture. Figure 5 shows the points (▲) at which samples were taken and examined for subunit assembly and fimbrial expression. Cultures were pulsed for 30 s and then chased for either 90 s (early assembly phase) or 20 min (total assembly). After the chase periods, the samples were analyzed to determine the fraction of the label that had been incorporated into fimbrial organelles. Figure 6 shows a fluorograph of the stacking gel of an SDS-polyacrylamide gel after immunoprecipitation with the organelle-specific MAb. At all time points, free subunits could no longer be detected at 20 min, and further chasing resulted in no additional subunit-to-organelle incorporation. During the log phase of growth, the amount of subunits incorporated at 90 s was

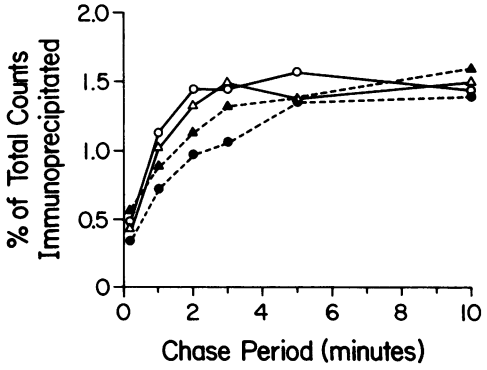

FIG. 4. Kinetics of fimbrial assembly as a function of temperature, as determined by immunoprecipitation with the organelle-specific MAb. Shown are the relative amounts of total TCA-precipitable counts that were specifically immunoprecipitated (in percentages) after various chase periods. The temperatures examined were 37°C (△), 30°C (○), 22°C (▲), and 18°C (●).

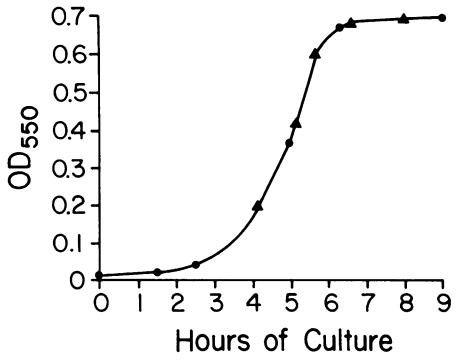

FIG. 5. Growth curve of strain CSH50 in glucose-supplemented minimal medium at 37°C. Optical density readings (at 550 nm) (OD_{550}) were taken at each point indicated (●). Samples were taken at points marked (▲) for examination of fimbrial synthesis, assembly, and expression.

TABLE 1. Kinetics of fimbrial expressions during bacterial growth in broth

Start of labeling[a]		Immunoprecipitable label in fimbriae organelles	
Time in growth curve (min)	OD_{550}	% Relative to total protein synthesis[b]	% of new subunits rapidly assembled[c]
95	0.197	0.98	95
155	0.415	0.67	120
185	0.600	0.37	12
240	0.680	0.25	10
280	0.690	0.20	7.5

[a] See Fig. 5 for growth curve. Samples (2 ml) were removed from culture at the times indicated and labeled for 30 s. Half of each sample underwent a 90-s chase before TCA precipitation; the other half was chased for 20 min. OD_{550}, Optical density at 550 nm.

[b] Immunoprecipitable counts after 20 min of chase/total TCA-precipitable counts after 20 min of chase × 100.

[c] Immunoprecipitable counts after 90 s of chase/immunoprecipitable counts after 20 min of chase × 100.

roughly equal to that incorporated at 20 min, indicating that, during the log phase, most of the subunits assembled within 90 s. In contrast, as the culture approached the stationary phase of growth, subunit assembly slowed appreciably, as shown by the fact that a lower percentage of incorporated [^{3}H]leucine was incorporated into fimbriae relative to total cellular synthesis (Table 1). (We used the ratio of counts incorporated after 90 s of chase to counts incorporated after 20 min of chase as a measure of rapidly assembling subunits.) We found that the percentage of rapidly assembling subunits decreased dramatically as the culture entered the stationary phase (Table 1), which could be due to slowing of polypeptide chain elongation, assembly, or both. In addition, a sample of the culture at each time point was assayed for total amount of fimbriae present on the surface of the bacteria by the ELISA inhibition assay. The amount of fimbriae measured from the time of passage to 24 h of culture varied between 4 and 5 ng/10^6 cells. After assaying samples from several growth curves, we found that this small variation is unrelated to the period of growth. Thus, in the absence of selection for fimbriation (fimbrate bacteria outgrow nonfimbriate bacteria when grown without aeration in rich broth [7]), a culture remains relatively constant in its degree of fimbriation during the growth cycle.

Size of the free subunit pool. We have demonstrated that fimbrial subunits are first synthesized and processed (2a) and then, within 3 min of synthesis, assembled into quarternary-structured organelles (see above). Since these data indicate that there is a preassembled pool of mature subunits within the cell, we next determined an upper bound of the size of this pool. We reasoned that if the pool was large enough, even total interruption of protein synthesis (by chloramphenicol) should still allow measurable elongation of the organelles given the observation that assembly is independent of protein synthesis (see above). In the absence of new subunit synthesis, the degree of elongation of fimbriae should be proportional to the size of the preexistent subunit pool.

We measured organelle elongation by two independent techniques. In the first, we performed a standard pulse-chase of growing bacteria and then determined the length of the labeled (new) organelles by RIP, SDS-PAGE in a long stacking gel, and fluorography (Fig. 7). When bacteria were harvested after a very short chase (10 s), we found that the labeled fimbriae were distributed in a broad size range (Fig. 7A). In contrast, a long chase (20 min) allowed all of the short, labeled fimbriae enough time to grow to large size (Fig. 7B). The addition of chloramphenicol at the beginning of the chase period had a pronounced effect on elongation; despite a chase of 20 min, there was no appreciable growth of labeled fimbriae (Fig. 7C). Thus, the size of the subunit pool must be small enough to prevent detectable further growth of the organelles.

To confirm these findings, we measured new organelle formation after defimbriation of the bacteria. Cultures were harvested in either the stationary or the mid-log phase of

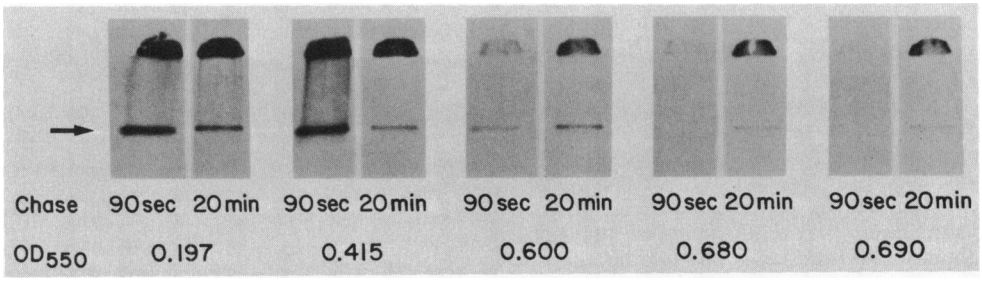

FIG. 6. Kinetics of new organelle expression at various times in the bacterial growth cycle. Bacteria at times indicated in Fig. 5 were pulse-labeled for 30 s with [^{3}H]leucine and chased for either 90 s or 20 min with unlabeled leucine before immunoprecipitation with the organelle-specific MAb. The optical density at 550 nm (OD_{550}) of the culture at the time of initial harvesting is indicated below each pair of lanes, which were prepared as described in the legend to Fig. 1. The arrow indicates the stacking gel-separating gel interface.

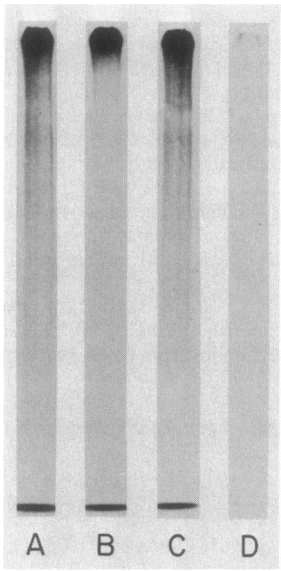

FIG. 7. Effect of chloramphenicol on the elongation of fimbriae. Fimbriae were separated by size on a 5% acrylamide gel after immunoprecipitation of solubilized *E. coli* cells that had been pulse-labeled for 20 s and chased for either 10 s or 20 min in the absence or presence of chloramphenicol. Lane A: 10-s chase, no chloramphenicol added. Lane B; 20-min chase, no chloramphenicol added. Note the disappearance of the label in the smaller sized fimbriae. Lane C: 20-min chase, chloramphenicol added after 10 s of chase. Note the persistence of label, as in lane A, in the smaller-sized fimbriae. Lane D: 20-min chase, chloramphenicol, at the same concentration as in lane C, added 1 min before pulse-labeling. Note inhibition of label uptake, demonstrating efficacy of chloramphenicol at this concentration.

growth, fimbriae were removed by blending, and bacteria, after being washed, were allowed to continue growing in either the absence or the presence of chloramphenicol. We quantitated the amount of fimbriae on the bacterial cells by the ELISA inhibition assay (2) at different times of growth before and after blending (Fig. 8). We found that even without inhibition of protein synthesis, stationary-phase bacteria did not regenerate fimbriae. In contrast, actively growing bacteria regenerated their fimbriae to a full extent by 90 min in the absence of antibiotic, although they assembled little in the way of new organelles when antibiotic was added. Thus, as opposed to the preliminary findings of Brinton (1), we conclude that there is an unmeasurably small pool of free fimbrial subunits in *E. coli*.

DISCUSSION

Except for work performed by Brinton and co-workers almost 20 years ago (1, 16), little has been published concerning the kinetics of the assembly of type 1 fimbriae. In experiments that were based on the association of a greater negative charge on the surface of fimbriate, as compared with nonfimbriate, bacteria, Brinton showed that bacteria could regenerate their negative charge through several cycles of blending. Of note, streptomycin, but not chloramphenicol, when added to the bacteria prevented the fimbrial regeneration. (Unfortunately, experimental details concerning the antibiotic use were not given.) In a later study, Novotny et al. (16) examined by electron microscopy the regeneration of F-pili and type 1 fimbriae after blending. They found that F-pili have a substantial pilin pool (later localized by Moore et al. (15) to the inner membrane). The only results reported with type 1 fimbriae showed that, after blending, the fimbriae could grow from 30% of their original unblended length to 90% of that length (as measured by electron microscopy) after 30 min of additional bacterial growth in rich broth.

We have demonstrated in the present study that nascent subunits enter a small pool of free subunits and then are rapidly assembled into organelles. We have previously demonstrated that fimbrial subunits are initially synthesized as larger signal sequence-containing precursors, which are secreted by the normal export sites in a SecA-dependent fashion (2a). Thus, the subunits go through a multistep process during their maturation into assembled organelles. First, the precursor is synthesized, processed, and translocated into or through the inner membrane. This translocation occurs predominantly cotranslationally since the signal-containing precursor can only be detected under conditions that cause precursor accumulation (e.g., the *secA* mutation, or growth in ethanol or cyanide-*m*-chlorophenyl hydrazone) (2a). When bacteria are examined during their log phase of growth at 37°C, most of the pulse-labeled subunits enter an SDS-stable, MAb-recognizable (assembled) form within 1 min of the chase period, and all seem to be assembled within 3 min. As the rate of bacterial growth slows progressively during and after log phase, so does the rate of organelle assembly relative to the rate of total protein synthesis; a less-pronounced slowing occurs in relative subunit synthesis. The effect of lowered growth temperature on fimbrial assembly, relative to total protein synthesis, is minimal, in marked contrast to the block in assembly of mannose-resistant fimbriae (3, 4) and F-pili (16) at lowered temperatures of bacterial growth.

We also found, by assaying the regeneration of fimbriae in the absence of protein synthesis, that the size of the free subunit pool is small relative to the number of subunits assembled into organelles. We utilized two complementary methods. The first consisted of pulse-labeling a culture with [^{3}H]leucine, followed by further cultivation in the absence or presence of chloramphenicol. After either a 10-s or a 20-min chase, the fimbriae were separated on the basis of size. The

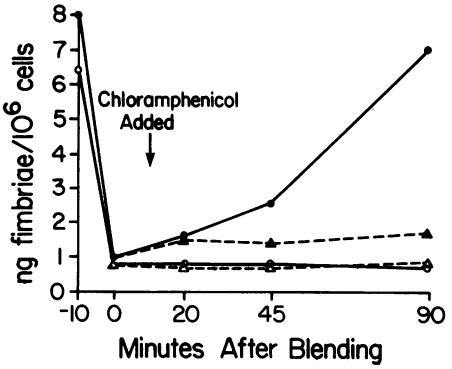

FIG. 8. Effect of protein synthesis on regeneration of fimbriae after their removal by blending, as determined by ELISA inhibition assay. Regeneration was examined during the early log phase in the absence of (●) or in the presence (▲) of 20 μg of chloramphenicol per ml and during the stationary phase in the absence (○) or in the presence (△) of chloramphenicol. Note that regeneration of fimbriae occurred only in actively growing, protein-synthesizing cells.

size distribution of label from the chloramphenicol-treated culture after 20 min of chase was nearly identical to that from the control culture after only 10 s of chase, demonstrating that fimbriae do not grow more than a few subunits in the absence of new subunit synthesis. Since chloramphenicol does not affect assembly, its affect must be due to depletion of the (small) subunit pool. These results were confirmed by the ELISA inhibition assay: fimbrial organelles that have been removed by blending will not regenerate in the absence of continuing protein synthesis. For the bacterial cell to maintain new assembly, new subunits must be made; no large preexistent pool of subunits is available.

Finally, we found that the rate of production of fimbriae slows significantly during the growth cycle such that little new synthesis of subunits or net assembly of organelles is occurring by the mid-late log phase of growth. Nevertheless, the total amount of fimbriae expressed on the bacterial surface remains fairly constant, as judged by the ELISA inhibition assay (unpublished data). Traditionally, *E. coli* cultures have been grown in rich broth without aeration for 24 to 48 h for the maximal production of fimbriae (3, 4). Because individual *E. coli* cells oscillate randomly between a fimbriate and a nonfimbriate phase (10^{-3} per generation [5]) and because bacteria in the fimbriate phase grow more rapidly than those in the nonfimbriate phase due to the ability of fimbriae to mediate pellicle formation at the broth-air interface (17), this long culture period allows time for selection of predominantly fimbriate bacteria. This selective growth advantage is abrogated by the addition of a sugar-such as glucose; moreover, the effect of glucose is unrelated to the genetic regulation of fimbriation (7). We have not ruled out, though, the possibility that other environmental factors may influence the kinetics of fimbrial expression. When bacteria are grown under the conditions of this study, fimbrial expression is influenced by the growth rate of the cells. Whether or not this effect is due to specific factors has not yet been determined.

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