Differential Sensitivities of Portions of the mRNA for Ribosomal Protein S20 to 3'-Exonucleases Dependent on Oligoadenylation and RNA Secondary Structure*

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The 3'-exonucleolytic decay of the mRNA for ribosomal protein S20 has been reconstituted in vitro using purified RNase II and crude extracts enriched for polynucleotide phosphorylase (PNPase) activity. We show that RNase II can catalyze the degradation of the 5'-two-thirds of the S20 mRNA and that prior oligoadenylation of the 3' termini of truncated S20 mRNA substrates can significantly stimulate the initiation of degradation by RNase II. The intact S20 mRNA is, however, insensitive to attack by RNase II and polyadenylation of its 3'-end cannot overcome the natural resistance of the S20 mRNA to RNase II. Complete degradation of either the entire S20 mRNA without prior endonucleolytic cleavage or the 3'-terminal 147-residue fragment is dependent on both oligoadenylation and PNPase activity. Moreover, this process can take place in the absence of RNase E activity. Our data point to the importance of oligoadenylation in facilitating 3'-exonucleolytic activity and indicate that there are alternative degradative pathways. The implications for mRNA decay are discussed.

Current models for the turnover of mRNAs in Escherichia coli postulate that degradation is initiated by an endonucleolytic cleavage usually catalyzed by RNase E (1-3). Subsequent steps involve the attack on newly created 3'-ends by one or both of two exonucleases, RNase II or polynucleotide phosphorylase (PNPase) (4-6). All three enzymes attack single-stranded RNAs (4, 5, 7). This raises the question of how RNAs containing regions of extensive secondary structure can be degraded, since stem-loop structures can occlude potential RNase E sites (8). This work has implicated the products of the pncB and pnp genes encoding poly(A) polymerase (9, 10) and PNPase, respectively, in the degradation of RNase II, a highly structured, small (108 nucleotides), untranslated, antisense inhibitor of the replication of colE1 replicons (11, 12). Polyadenylation also destabilizes the mRNA encoding ribosomal protein S15 (13) and appears to regulate the decay of mRNAs generally (14). These results have prompted Cohen to propose that polyadenylation facilitates the exonucleolytic degradation of RNAs, particularly by PNPase, and that degradation of RNAs mediated by RNase E is functionally coordinated with the 3'-exonucleolytic activity of PNPase (15). The latter seems plausible in view of the association between these enzymes (16, 17).

The mRNA for ribosomal protein S20 is a well characterized substrate for RNase E both in vivo and in vitro (18-20). Both crude and purified RNase E cleave a synthetic S20 transcript (372 residues) to generate a number of products, the most prominent of which spans 147 residues coterminally with the substrate's 3'-end and is identical to a degradative intermediate found in vivo (18, 20). Further degradation of the 3'-terminal 147-residue product of RNase E-mediated cleavage in vivo requires PNPase activity, since this product is stabilized and accumulates in pnp mutants (18). The 147-residue product is, however, essentially stable in vitro, even in relatively crude extracts (8, 20). The S20 mRNA has served as a model to search for conditions that would permit its total degradation in vitro and to test some of the predictions of Cohen's model (15). We show here that RNase II can participate in the degradation in vitro of the 5'-two-thirds of the S20 mRNA and that oligoadenylation can significantly stimulate the activity of RNase II. Furthermore, oligoadenylation is a prerequisite for the complete degradation of the S20 mRNA, a process also dependent on PNPase, but both steps can be completely uncoupled from endonucleolytic decay by RNase E.

EXPERIMENTAL PROCEDURES

Bacterial Strains and Plasmids—Strains RD100 (lacZ, met, pnp-7, rpsA1, rna, trpD9778; Ref. 21), N3431 (HfrH, lacZ43, relA1, rne3071ts), spot1, thi-1; 22), and CF881 (recB, xthA, Arna; 23) were obtained from Drs. M. Pearson, B. Bachman, and M. Cashel, respectively. Plasmid pGM87, containing the P2 leader, coding sequences and Rho-independent terminator of the gene for ribosomal protein S20 in the vector pT718U, has been described previously (8). Plasmid pG194 encompasses residues 258-447 of the S20 mRNA preceded by a T7 RNA polymerase promoter.2

Preparation of S20 mRNA Substrates—Transcription of linear DNA templates with T7 RNA polymerase in the presence of [α-32P]CTP was performed as described (8, 20). 5'-End-labeled RNAs were prepared by transcription of linear DNA templates in the presence of 50 μCi of [α-32P]GTP to a final concentration of 50 μM, while ATP, CTP, and UTP were present at 0.5 μM. Labeled RNA transcripts were purified by extraction with phenol/chloroform/isomyl alcohol (25:24:1) and two cycles of ethanol precipitation. The 365-nucleotide full-length S20 substrate (t87D) was generated from pGM87, which had been linearized previously with Dral. Two additional substrates, t87H and t87S, lacking 32- and 144-nucleotide residues from the 3'-end of the S20 transcript, were synthesized from pG194, which had been linearized with restriction enzymes HindIII and Sau3AI, respectively. The S20 deletion substrate t194D was transcribed from the plasmid pG194, which had been linearized previously with Dral (8).

Polyadenylated S20 transcripts, including 187D-oligo(A), 187H-oligo(A), and 187S-oligo(A) (30–40 pmol) were prepared in a 60-μl reaction volume containing 25 μl HEPES-NaOH, pH 7.5, 5% glycerol, 5 μM...

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1 The abbreviation used is: PNPase, polynucleotide phosphorylase.

2 G. A. Mackie, J. G. Genereaux, and S. K. Masterman, unpublished results.
MgCl₂, 100 mM NH₄Cl, 60 mM KCl, 1 mM dithiothreitol, 0.02 mM EDTA, and 2 mM ATP. Purified E. coli poly(A) polymerase (0.3 unit) (Pharmacia Biotech Inc.) was added last, and incubations were performed for 45 min at 37°C. Oligoadenylated transcripts were purified by extraction with phenol/chloroform/isoamyl alcohol (25:24:1) and collected by ethanol precipitation. The S20 transcripts were extended by approximately 25–45 nucleotides, as determined by sizing on a sequencing gel.

Preparation of Extracts, Enzymes, and Ribonuclease Assays—AS-26 extracts were prepared as described (20). Extracts from strains N3431 and N3433 were heated for 10 min at 43°C at a concentration of 1 mg/ml in 50 mM Tris-HCl, 60 mM NH₄Cl, 10 mM MgCl₂, 0.5 mM EDTA, 0.2 mM dithiothreitol, 5% glycerol, pH 7.8, prior to assay. Incubations were performed at 30°C and contained 20 nM RNA substrate and 50–100 μg/ml protein (20). Additions of 3 mM ATP and 10 mM sodium phosphate, pH 7.0, were made as indicated in the legends. The effective concentration of ATP in the extract may be lower, depending on the extent of ATPase activity. Products of digestion were analyzed by electrophoresis on 6% polyacrylamide gels containing 8 M urea in Tris-borate-EDTA buffer. Gels were fixed and dried before analysis by autoradiography or with a Molecular Dynamics PhosphorImager system.

Recombinant RNase II (Rnb) was purified to approximately 95% of homogeneity as described previously (24). RNase II assays were assembled as described previously (24) in a 35-μl reaction volume containing 60 nM RNA substrate. Purified RNase II (6 milliunits) was added to a final concentration of 28.5 nM and incubations were performed at 37°C. Samples were withdrawn at each time point and quenched in 3 volumes of loading buffer containing 90% formamide and tracking dyes. The products were resolved on 5 or 6% polyacrylamide gels containing 8 M urea and visualized by autoradiography or with a Molecular Dynamics PhosphorImager system (see above).

RESULTS

Sensitivity of the S20 mRNA (t87D) to Digestion by RNase II—To determine whether the full-length S20 mRNA, t87D (see Fig. 1), is a substrate for RNase II, it was incubated with the purified enzyme. After 15 min of incubation t87D is still intact and is resistant to digestion by RNase II (Fig. 2A) under conditions where other single-stranded substrates would be rapidly attacked and degraded (24). A second substrate, t87H, lacking the Rho-independent terminator stem-loop structure (stem VII in Fig. 1), was also incubated with purified RNase II. Less than 10% of the substrate was degraded to a somewhat shorter, but stable, product denoted as "D" during a 15-min digestion (Fig. 2B). A third substrate, t87S, terminating at residue C303, mimics the 218-residue intermediate generated by a single RNase E cleavage at residues 300/301/302 (see Fig. 1). This "precleaved" substrate was incubated with purified RNase II, and approximately 75% of the substrate was degraded after 15 min of digestion (Fig. 2C). Three discrete intermediates (A–C) could be detected in this assay. Using 5'-end-labeled substrates, the sizes and 3'-ends of these intermediates were subsequently determined on a high resolution (sequencing) gel (Fig. 3). Intermediates A–C could be detected in this assay. Using 5'-end-labeled substrates, the sizes and 3'-ends of these intermediates were subsequently determined on a high resolution (sequencing) gel (Fig. 3). Intermediates A–C could be detected in this assay. Using 5'-end-labeled substrates, the sizes and 3'-ends of these intermediates were subsequently determined on a high resolution (sequencing) gel (Fig. 3). Intermediates A–C could be detected in this assay. Using 5'-end-labeled substrates, the sizes and 3'-ends of these intermediates were subsequently determined on a high resolution (sequencing) gel (Fig. 3). Intermediates A–C could be detected in this assay. Using 5'-end-labeled substrates, the sizes and 3'-ends of these intermediates were subsequently determined on a high resolution (sequencing) gel (Fig. 3). Intermediates A–C could be detected in this assay. Using 5'-end-labeled substrates, the sizes and 3'-ends of these intermediates were subsequently determined on a high resolution (sequencing) gel (Fig. 3). Intermediates A–C could be detected in this assay. Using 5'-end-labeled substrates, the sizes and 3'-ends of these intermediates were subsequently determined on a high resolution (sequencing) gel (Fig. 3).
ation of RNA I, the antisense regulator of coIE1-type plasmid replication, by poly(A) polymerase stimulates the activity of PNPase against RNA I in vitro (12). We have tested whether prior polyadenylation would also stimulate the activity of RNase II against various substrates derived from the S20 mRNA. Purified RNase II was incubated with equimolar amounts of t87S and t87S-oligo(A), substrates which encompass the 5′ two-thirds of the S20 mRNA (refer to “Experimental Procedures”). During a 15-min digestion, t87S-oligo(A) is degraded by RNase II almost 2-fold faster than the nonadenylated t87S substrate (Fig. 4A). Degradative intermediates A, B, and C accumulate as before. Addition of a 3′ single-stranded poly(A) extension to the otherwise resistant substrate t87H results in a 4–5-fold stimulation in the rate of disappearance of t87H-oligo(A) compared with nonadenylated t87H (Fig. 4B). Degradative intermediate D accumulates linearly to ~4-fold greater levels than in the experiment of Fig. 2B. Finally, t87D-oligo(A) was also prepared and incubated with purified RNase II. The oligo(A) extension is gradually removed to regenerate the 365-nucleotide S20 transcript which, unlike the others, is not significantly shortened further by RNase II (Fig. 4C).

Oligoadenylation of t87D Leads to Its Destabilization in AS-26 Fractions—AS-26 fractions faithfully catalyze endonucleolytic cleavages dependent on RNase E in a number of substrates, including the S20 mRNA (t87D), to yield a stable 147-residue fragment coterminal with the 3′-end of the substrate resulting from cleavages at residues 300/301/302 (Fig. 1; Refs. 8 and 20). We examined whether the 147-residue product could be degraded in an AS-26 fraction supplemented with cofactors that might activate other enzymes such as poly(A) polymerase and/or PNPase. The t87D substrate was incubated for 10 min in an AS-26 extract prepared from CF881 (pnp1), after which aliquots were supplemented with buffer, ATP, or ATP and sodium phosphate. The addition of ATP causes the otherwise discrete 147-residue product of the first stage of digestion to form a smear extending over approximately 150–180 residues, but does not promote significant further breakdown of these products (data not shown). In the presence of both ATP and sodium phosphate, the smear is less pronounced and the 147-residue product disappears completely by 20 min of incubation (i.e. within 10 min of addition of cofactors) as shown in Fig. 5. The same experiment was repeated with an AS-26 extract prepared from strain RD100, which largely lacks PNPase activity. In this case, the 147-residue product of RNase E-mediated cleavage is lengthened substantially in the presence of cofactors (Fig. 5).

![Fig. 2. Digestion of 3′-truncated S20 mRNAs by RNase II.](http://www.jbc.org/)

Substrates t87D (A), t87H (B), and t87S (C) were incubated with purified RNase II for 15 min at 37 °C. Aliquots were removed at the indicated times (in minutes) as described under “Experimental Procedures.” The products were resolved by electrophoresis through a 6% polyacrylamide gel under denaturing conditions, then visualized by autoradiography and quantified using a PhosphorImager. The letters A–D in the margins represent clusters of intermediates generated by RNase II digestion (see text).

![Fig. 3. Mapping of intermediates generated by RNase II digestion.](http://www.jbc.org/)

5′-End-labeled RNAs t87H (lanes 1–4) and t87S (lanes 5–8), prepared from pGM87 that had been linearized by HindII and Sau3AI, respectively (refer to “Experimental Procedures”) were treated as follows: lanes 1 and 5, partial alkaline digestion, pH 9.0, for 5 min at 100 °C; lanes 2 and 6, partial digestion with 1.2 units of RNase T1 in 8 M urea, pH 5.0, for 6 min at 50 °C; lanes 3 and 7, no treatment. End-labeled t87H (lane 4) and t87S (lane 8) at a concentration of 100–200 mM were digested with 41 and 16.4 milliunits of purified RNase II, respectively, at 37 °C. After 10 min of incubation, portions of the incubation mixture were removed and diluted into 10–20 volumes of 0.25 M sodium acetate, containing 25 μg/ml of yeast RNA, 10 mM EDTA, extracted with phenol/chloroform/isooamyl alcohol (25:24:1), and precipitated with ethanol. The digestion products were separated electrophoretically through a 6% polyacrylamide sequencing gel containing 8 M urea, then visualized by autoradiography. Selected G residues are indicated in the left margin, while degradation intermediates generated by RNase II are indicated in the right margin as groups A–D.
ence of ATP and sodium phosphate (data not shown), but remains stable after 45 min of incubation (Fig. 5).

Aprimer extension experiment showed that identically sized cDNAs can be synthesized from RNA templates previously incubated in an AS-26 fraction in the absence or presence of ATP (data not shown). The most prominent 5'-ends in both sets of cDNAs map to residues 301 and 302, a known site of RNase E cleavage (20). Thus, ATP and/or polyadenylation are not altering the specificity of RNase E.

RNase E Cleavage and Oligoadenylation Occur Independently—A second set of experiments was designed to determine whether prior cleavage by RNase E was an obligatory requirement for subsequent steps in the degradation of the S20 mRNA. The 365-residue substrate was incubated in a heat-treated AS-26 fraction prepared from strain N3431, which carries a ts mutation in the rne gene encoding the catalytic subunit of RNase E (25). ATP and sodium phosphate were added to the incubation at the same time as the AS-26 or 10 min later. In the absence of any added cofactors, the 365 residue substrate disappears very slowly (Fig. 6a) and the rate of appearance of the 147-residue product (Fig. 6b) is greatly reduced to <5% of the wild type rate, as anticipated. Addition of ATP and sodium phosphate at either T = 0 or T = 10 min leads to the rapid disappearance of the 365-residue substrate (Fig. 6a) with no detectable intermediates (data not shown). Likewise, any accumulated 147-residue product of RNase E cleavage also disappears very rapidly upon the addition of cofactors (Fig. 6b).

Unlike t87D, t194D is cleaved only slowly by crude or purified RNase E, and 20% conversion of t194D to the 147-residue product requires 180 min (Fig. 7). As in the case of t87D, supplementing the AS-26 with ATP and sodium phosphate results in a 4–5-fold acceleration of the disappearance of the substrate (Fig. 7). In the latter case the anticipated 147-residue product of RNase E digestion could not be detected, perhaps due to its rapid exonucleolytic decay once it is formed and oligoadenylated. Together, this and the preceding experiment show that 3'-oligo(A) and PNPase-dependent degradation of the S20 mRNA in vitro can occur in the absence of significant levels of RNase E activity. An attempt was made to increase the rate of cleavage of t194D in an AS-26 fraction or with the purified Rne protein by adding Rho factor, an RNA helicase (26), and ATP. No increase in the rate of disappearance was observed; rather, the substrate was elongated (oligoadenylated) in the AS-26 fraction whether Rho was absent or present (data not shown).

**DISCUSSION**

The Role of Exonucleases in mRNA Decay—Degradation of most bacterial mRNAs is usually believed to be initiated by one or more endonucleolytic cleavages catalyzed by RNase E (1–3, 8, 25). A number of RNase E cleavage sites have been mapped in the S20 mRNA, most in the 5' quarter, but others are scattered between residues 130–300 (7). In the most extreme case, a single RNase E cleavage would generate two fragments, one encompassing the 5'-end to residue 300 (218 residues), the other encompassing the 3' 147 residues. Our data show that subsequent 3'-exonucleolytic degradation of residues 5' to the most distal RNase E cleavage site at position 300/301/302 can be effected by RNase II alone, albeit inefficiently. This en-
zyme's action can be facilitated, however, by oligoadenylation of the 3' termini of endonucleolytic fragments by poly(A) polymerase. This modification of the RNA enhances the initiation of RNase II action at permissive 3' termini, presumably by providing a single-stranded extension that favors binding of RNase II and that protects it from inactivation (24). Polyadenylation does not affect subsequent steps in RNase II-mediated decay as the enzyme still stalls at the same internal sites, independent of prior oligoadenylation. Were stalling and dissociation to occur at the same internal sites, independent of prior oligoadenylation. Were stalling and dissociation to occur in vivo, we presume that a second round of oligoadenylation of the resultant 3' termini would reinitiate the action of RNase II. Although we have not tested it directly, PNPase should catalyze the phosphorolysis of endonucleolytic fragments 5' to residue 300, likely without their prior oligoadenylation. In contrast to its behavior on the 5'-218-residue fragment, RNase II cannot degrade the tightly folded 3'-end of the S20 mRNA. Rather, the sequential action of poly(A) polymerase and PNPase is required for complete degradation of the 3'-147 residues of the molecule, in agreement with data obtained in vitro (18).

Although there are excellent data to show that RNase E and PNPase associate tightly during purification (16, 17), our experiments show that the two activities can be functionally uncoupled in vitro, unlike the situation for RNA I in vivo (11, 15). Moreover, with the exception of the highly structured 147-residue product, virtually all the other initial products of RNase E digestion of the S20 mRNA disappear during incuba-

\[ \Delta G = -12.9 \text{ kcal/mol; Ref. 7} \]

3 K. H. Niguma and G. A. Mackie, unpublished results.
There is clearly a hierarchy in the susceptibility of 3′ termini to attack by RNase II. Unpaired ends preceded by weak secondary structure (e.g. residue 303 and stem IV) are directly available to RNase II without modification. More stable stems (e.g. V, III) reduce the rate of attack and/or lead to stalling and dissociation. These results parallel those observed for RNA-OUT, the antisense regulator of IS10/Tn10 transposition, which demonstrated a correlation between the thermodynamic stability of a stem-loop and its ability to act as a barrier to degradation by RNase II (28). A novel finding of our work is that these barriers to RNase II action can be overcome by oligoadenylation. Finally, very stable structures, such as stem VII, are completely resistant to RNase II, even after oligoadenylation.

Like RNase II, PNPase favors single-stranded substrates and pauses at regions of secondary structure (6). It is, however, capable of digesting through some stem-loop structures (6, 12). Our data show that the 147-residue 3′-terminal fragment of the S20 mRNA, which encompasses a highly folded domain, can be degraded efficiently only after it has been modified by addition of a 3′-oligo(A) extension. This extension facilitates subsequent exonucleolytic attack by PNPase, in agreement with previous work on RNA I (11, 12). Our results explain earlier work, which showed that the S20 mRNAs are stabilized 2–3-fold in strains carrying a pnp mutation and that a 147-residue fragment accumulates under such conditions (18). The present work also shows that even a full-length S20 mRNA can be degraded in an ATP and sodium phosphate-dependent manner in the absence of significant RNase E activity. In this context it is relevant that the pnp gene can determine the rate-limiting step in the decay of the S20 mRNAs in vivo (18). Together these experiments provide strong evidence for an alternative exonucleolytic pathway of mRNA decay, which could be important for small highly structured RNAs.

What are the properties of PNPase that necessitate the modification of substrates with a poly(A) tail? PNPase undergoes a transition from nonprocessive to processive phosphorolysis accompanied by tight binding to an RNA substrate when the single-stranded region is extended from <12 residues to >20 (reviewed in Ref. 29). Neither PNPase nor RNase II can bind efficiently to substrates possessing fewer than 6–10 unpaired 3′ residues (24). A poly(A) tail would, therefore, permit the binding of PNPase to an otherwise inaccessible substrate. Even if PNPase were to stall at the base of a stem (6, 30), “breathing” of the stem or unwinding promoted by other enzymes or factors may ultimately permit PNPase to digest through many stem-loop structures and thence through the rest of the molecule. Alternatively, a stalled PNPase may dissociate from its substrate or be displaced by poly(A) polymerase. Such behavior may explain the observed heterogeneity of polyadenylated 3′-ends on RNA I (11).

In view of our results, it seems probable that the poly(A)-dependent degradation of RNAs in E. coli exists largely to facilitate exonucleolytic removal of highly folded RNA fragments. Such RNA fragments would otherwise be resistant to the three major nucleases of mRNA decay, RNase E, RNase II, and PNPase, and would not necessarily be susceptible to RNase III, whose specificity is relatively stringent and whose gene is, in any event, dispensable (31, 32). Such a function clearly distinguishes the purpose of poly(A) tails in E. coli from their role in eukaryotic organisms where they promote mRNA stability, translational initiation, and nuclear export (33).

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