The expression of some very short open reading frames (ORFs) in *Escherichia coli* results in peptidyl-tRNA accumulation that is lethal to cells defective in peptidyl-tRNA hydrolase activity. In an attempt to understand the factors that affect this phenotype, we have surveyed the toxicity of a complete set of two-codon ORFs cloned as minigenes in inducible expression vectors. The minigenes were tested in hydrolase-defective hosts and classified according to their degree of toxicity. In general, minigenes harboring codons belonging to the same box in the standard table of the genetic code mediated similar degrees of toxicity. Moreover, the levels of peptidyl-tRNA accumulation for synonymous minigenes decoded by the same tRNA were comparable. However, two exceptions were observed: (i) expression of minigenes harboring the Arg codons CGA, CGU, and CGC, resulted in the accumulation of different levels of the unique peptidyl-tRNAArg<sup>-2</sup> and (ii) the toxicity of minigenes containing CUG and UCU codons, each recognized by two different tRNAs, depended on peptidyl-tRNA accumulation of only one of them. Non-toxic, or partly toxic, minigenes prompted higher accumulation levels of peptidyl-tRNA upon deprivation of active RF1, implying that translation termination occurred efficiently. Our data indicate that the nature of the last decoding tRNA is crucial in the rate of peptidyl-tRNA release from the ribosome.

Minigenes are DNA sequences present in bacterial chromosomases that may be expressed into functionally active oligopeptides. In *Escherichia coli* for example, translation of a peptide encoded in a minigene present in the 23 S rRNA, turns cells erythromycin resistant (1); also, peptides containing five to eight amino acid residues encoded in the attenuator sequence regions is lethal to cells partly defective in peptidyl-tRNA hydrolase activity, but not to wild-type bacteria (3). Translation of bar minigene mRNAs results in premature release of peptidyl-tRNAs from ribosomes (a phenomenon called “drop-off”); under limited Pth<sup>1</sup> activity, these peptidyl-tRNAs accumulate in the cell. It has been proposed that lethality stems from the subsequent shortage in the pool of specific tRNAs for further involvement in protein synthesis (4). Recently, evidence that seems to support this inference has been obtained for a ribosome bypassing system (5), but the alternative explanation that peptidyl-tRNAs might be toxic per se has not been ruled out (6).

Translation ends at the termination codon in an mRNA, when the ribosomal peptidyl-transferase presumably hydrolyzes the ester bond between the completed polypeptide chain and the last tRNA. The termination reaction requires the concurrence of the release factors RF-1 or RF-2 (depending on the nature of the termination codon) and other factors catalyzing the release of the mature protein (7, 8). Drop-off is a normal, if relatively rare, event in protein synthesis that can occur during elongation or instead of polypeptide termination (9, 10). If the rates of peptidyl-tRNA synthesis and drop-off exceed the rates of termination and Pth hydrolysis, peptidyl-tRNA accumulates and thus critically reduces the concentration of aminoacylable tRNAs and increases that of peptidyl-tRNAs (4, 9). The up-shift to non-permissive temperatures of a thermosensitive pth mutant, *pth(Ts)*, results in peptidyl-tRNA buildup of all the tRNAs assayed. The rates of peptidyl-tRNA accumulation differ as a function of the tRNA species. Thus, families of tRNA cognate to codons for Lys, Thr, and Asn accumulate the fastest, whereas those cognate to codons for Leu, Gly, and Cys accumulate the slowest (11). These results suggest that the drop-off rates depend on the codons involved.

Toxicity and peptidyl-tRNA accumulation in the *pth(Ts)* mutant is alleviated in strains defective for the translation termination factors RF-3 and RRF (12). Drop-off during minigene mRNA translation is enhanced by these termination factors as well as the elongation factor EF-G (9). *In vitro* experiments with different synthetic minigenes have shown that the relative rates of termination and drop-off vary according to the composition of the last sense codon, the nature and context of the stop codon, and the length of the mini-ORF and that toxicity is correlated to these conditions (9, 13). In addition, the strong effect of the SD sequence affects peptidyl-tRNA accumu-
mRNA was revealed by Northern blot analysis using a 32P-labeled DNA probe (2× 106 cpm/pg; Ref. 23). The 150-bp DNA probe was synthesized by 50 cycles of PCR (95 °C, 30 s; 55 °C, 30 s and 72 °C, 1 min) using 30 fmol of pKQV4 template and 10 pmol of each sequencing oligo (defined above) in a 50-µl reaction mixture containing 40 mM Tris-HCl, pH 8.0, 5 mM MgCl2, 10 mM dithiothreitol, 50 mM NaCl, 50 µCi of [α-32P]dATP (6,000 Ci/mmol, Amersham Biosciences), 150 µM concentration each of the other three dNTPs, and 1 unit of Tag DNA polymerase (Applied Biosystems).

RESULTS

Minigenes Harboring Codons from the Same Genetic Code Box Show Similar Toxicity—Changes in the last sense codon of some minigenes determine variations in the degree of toxicity for phth mutants (9, 24, 25). To understand the nature of this effect, we used an expression library of two-codon ORF minigenes in which the second, and last, sense codon was any of the 64 possibilities in the genetic code (Fig. 1A). By maintaining the same transcriptional promoter, SD region, initiation and termination codons, and the shortest possible “ORF,” we attempted to minimize the number of variables affecting minigene expression and focus on the effect of the different sense codons on toxicity (9, 13). The minigene constructs were expressed in two phth mutants differing in the level of Pth activity (pTh[Ts] > pth[rap]; Ref. 15), as a means for ranking minigene toxicities. The results, summarized in Fig. 1B, showed a wide variation in the degree of toxicity to the two mutant strains arising from minigene expression and depending on the nature of the second codon: 27 were toxic to both pth[Ts] and pth[rap], 18 were lethal to pth[rap], and 16 had no deleterious effects on either mutant. Unlike the toxic minigenes isolated by Tenson et al. (24), none of our constructs affected wild-type cell growth under the same assay conditions (data not shown). This effect may be due to the lower strength of minigene expression in the vector we used relative to that recorded for the vector used in the previous report (24). Minigenes with any of the three termination codons in the second position do not encode peptides and are therefore expected to be harmless to Pth-defective strains. In effect, the two constructs tested, carrying TAA and TGA respectively, had no toxic effects (data not shown). No correlation was observed between codon pair bias (26) or cognate tRNA scarcity (underlined codons in Fig. 1B; Ref. 22) and minigene toxicity. In general, minigenes harboring codons grouped within the same genetic code box (Fig. 1B) showed a similar degree of toxicity (38 minigenes in 10 out of 16 boxes). Five of these boxes contain codons for two different amino acids (e.g. TTN, Phe/Leu; ATN, Ile/Met-, etc.), suggesting that the codons, rather than the decoded amino acids, determine minigene toxicity. This would not be the case for those minigenes bearing codons within each of the remaining six boxes in which mixed degrees of toxicity were observed (dashed boxes in Fig. 1B).

Non-toxic Minigenes Do Not Mediate Peptidyl-tRNA Accumulation—For a number of minigenes, a direct correlation between toxicity and peptidyl-tRNA concentration has been observed in phth mutants (4, 9, 24). To assess the breadth of this observation, we estimated the relative concentrations of peptidyl-tRNAs for selected minigenes. Initially, we determined the time length of IPTG minigene induction which generated the highest peptidyl-tRNA concentrations. The data showed that, for the toxic (AAT and AAA) and partly toxic (GAT and GAA) minigenes assayed, the highest accumulated fraction was reached in all cases within a 30-min induction period (Fig. 2B). Therefore, all subsequent determinations of peptidyl-tRNA concentration were made at 30 min (Fig. 1B, columns 4 and 5). The relative concentrations of peptidyl-tRNA in C600[pTh[Ts]] varied from 40 to 70% for toxic, 10 to 20% for partly toxic, and 5 and 10% for non-toxic minigenes (Fig. 1B, fourth column in
each cell). In C600rap, the relative concentrations of peptidyl-tRNA from toxic and partly toxic minigenes were proportionally higher (Fig. 1B, fifth column in each cell), whereas the concentrations of peptidyl-tRNA for non-toxic minigenes were lower, although similar to those observed for \( \text{pth} \) (Ts). Wild-type transformants did not accumulate peptidyl-tRNA (data not shown). Except for minigene AAT, encoding Asn, which intriguingly promotes accumulation of peptidyl-tRNALys (Fig. 2A), heterologous peptidyl-tRNAs did not accumulate upon minigene expression: minigene AAA did not accumulate peptidyl-tRNAAsn; minigene GAT did not accumulate peptidyl-tRNAGlu; minigene GAA did not mediate accumulation of peptidyl-tRNAAsp (Fig. 2A); and minigene AGA did not accumulate peptidyl-tRNALys.

Peptidyl-tRNA Accumulation Depends upon the Specific Codon-tRNA Interaction

In six of the genetic code boxes (dashed boxes in Fig. 1B), minigenes showed mixed degrees of toxicity. To understand the basis of these differences, we analyzed peptidyl-tRNA accumulation upon minigene induction in \( \text{pth} \)-defective mutants. For example, in the set of minigenes

![Fig. 1. Constructs and degree of toxicity of two-codon minigenes. A, schematic diagram of a linear version of the used constructs: each construct contained a single minigene cloned between the EcoRI and HindIII sites (in italics) in pKQV4; the relative positions of other genetic markers are also indicated. Minigenes were under the control of the \( \text{P}_{\text{lac}} \)/\( \text{O}_{\text{lac}} \) transcriptional promoter and were followed by the transcriptional terminator Trrnt. Translation was controlled by a SD sequence (underlined) and initiation (ATG) and stop (TAA) triplets. B, genetic code indicating the variable codon of the 64 possible two-codon minigene constructs and, for the analyzed cases, the corresponding tRNAs and amino acid residues (columns 2, 3, and 1, respectively, in each box). The viability of C600pth(Ts) and C600rap cells transformed with each minigene was measured after IPTG induction of minigene expression (see “Experimental Procedures”). Different degrees of toxicity are given in different colors: red, minigenes toxic to both \( \text{pth} \)-deficient strains; blue, partly toxic minigenes that inhibit the growth of \( \text{pth} \)(rap) cells only; green, non-toxic minigenes. The figures in each box correspond to estimated peptidyl-tRNA fractions, expressed as a percentage of the corresponding tRNA present in either C600pth(Ts) (columns numbered 4) or C600rap (columns numbered 5) after 30 min of expression. Underlined codons are decoded by tRNAs having concentrations comparable with that of tRNAArg-4, typically considered as a scarce tRNA species (22). Codons in half-brackets are cognate to a unique tRNA.

![Fig. 2. Northern blot analysis and time course of peptidyl-tRNA accumulation following expression of toxic and partly toxic minigenes. A, Northern blot of peptidyl-tRNA (peptRNA) accumulation at different times after IPTG induction of toxic, AAT and AAA, and partly toxic, GAT and GAA, minigenes in \( \text{E. coli} \) C600rap. The peptidyl-tRNAs and tRNAs (arrows), treated previously with CuSO₄, were revealed by complementary, \(^{32}\text{P}\)-labeled, oligos (see “Experimental Procedures”). B, time course graph of percentage of peptidyl-tRNAs accumulated in Northern blots shown in A.](http://www.jbc.org/content/26067/1/13404/F1.large.jpg)
carrying Arg codons CGU, CGC, and CGA, for which tRNA\(^{\text{Arg}-2}\) is the sole cognate species, the generated peptidyl-tRNAs must be chemically identical. If we assume an equal decoding rate for the mini-ORFs in each of the three minigenes, toxicity would depend on the drop-off rate, because the rate of peptidyl-tRNA hydrolysis by Pth is identical. The relative amounts of peptidyl-tRNA\(^{\text{Arg}-2}\) accumulated after expression of each minigene in Pth-defective strains ranked in the order CGA>GCT>GCG, as expected from their degree of toxicity (Fig. 3). Accordingly, the peptidyl-tRNA concentrations should reflect the rate of peptidyl-tRNA\(^{\text{Arg}-2}\) drop-off due to differences in tRNA\(^{\text{Arg}-2}\) interaction with each of the three codons (see “Discussion”).

\(\text{tRNA}\^{\text{Sec-1}}\) is cognate to the codons in partly toxic minigenes TCT and TCA and clearly accumulates as peptidyl-tRNA\(^{\text{Sec-1}}\) in strain \(\text{prfA1}\) (40°, Fig. 1B); by contrast, accumulation of peptidyl-tRNA\(^{\text{Sec-1}}\), produced by both the partly toxic TCT and the non-toxic TCC minigenes, was not promoted in either strain (Fig. 1B). This suggests strongly that the toxicity of minigenes TCT and TCA is associated with peptidyl-tRNA\(^{\text{Sec-1}}\) accumulation. The data for the minigenes CTA and CTG could be explained in a similar way.

**Non-toxic Minigene mRNAs Are Translated**—For the expression of different minigene variants, the cellular levels and stability of minigene mRNA correlate with the strength of toxicity. It has been speculated that the increased messenger stability results from a longer interaction period with the ribosome during translation (25). Results with Northern blot assays confirmed that toxic minigenes (e.g., AAA, GTT, and CGA) accumulated high levels of mRNA, whereas non-toxic minigenes (e.g., GCC, GGC, and CGC) did not (data not shown). We then asked whether non-toxic minigene mRNAs were translated at all and, if they were, why their expression did not drive peptidyl-tRNA accumulation. Minigene AGA, lethal to \(\text{C600pth(Ts)}\), and the non-lethal minigene GGC, were expressed in the presence of antibiotics (Fig. 4). The antibiotics used were pactamycin, which causes ribosome stalling soon after initiation of protein synthesis (27), and erythromycin, which enhances the dissociation of peptidyl-tRNAs containing at least six to eight amino acids (6, 28). As expected, the expression of the lethal minigene AGA resulted in mRNA accumulation even in the absence of the antibiotics. The non-toxic GGC minigene, on the other hand, accumulated mRNA only in the presence of pactamycin. Similar results were obtained with the lethal CGA and the non-toxic CGC minigenes (data not shown). These results indicate that mRNAs of these non-toxic minigenes are translatable; furthermore, in the absence of antibiotics translation termination should have occurred readily, suggesting that the mRNA-ribosome complex may be short lived. Erythromycin did not mediate accumulation of peptidyl-tRNA nor did it stabilize the ribosome-mRNA complex as deduced from the observation that it did not favor mRNA accumulation (Fig. 4). Given that the erythromycin binding site is at the entrance of the 50 S ribosomal subunit tunnel (29), it is not expected to affect dipeptidyl-tRNAs which are too short to reach the site.

**Efficient Translation Termination Prevents Peptidyl-tRNA Accumulation**—To test whether toxic and non-toxic minigenes differ in the efficiency of translation termination, the levels of peptidyl-tRNA, the intermediate previous to termination hydrolysis, were assayed under a condition of defective termination (Fig. 5). An \(\text{E. coli prfA1-pth(rap)}\) double mutant, defective for both RF1 (thermosensible) and Pth activities, was transformed with constructs carrying minigenes causing different degrees of lethality. In mutant \(\text{prfA1}\) the activity of RF1 is greatly reduced at 43°C (16). We used the partly toxic CGT minigene and the non-toxic variant CGC, for which tRNA\(^{\text{Arg}-2}\) is the sole cognate isoacceptor. When RF1 was defective, expression at 32°C of the CGC minigene did not promote detectable peptidyl-tRNA\(^{\text{Arg}-2}\) accumulation, whereas it did at 43°C (lane 4 versus lane 8). The partly toxic CGT minigene promoted peptidyl-tRNA\(^{\text{Arg}-2}\) accumulation at both temperatures (lanes 2 and 6), and accumulation was enhanced at 43°C. These results suggest that efficient translation termination of non-toxic minigene mRNAs shortens peptidyl-tRNA residence time on the ribosome so that drop-off events remain undetectable. In strain \(\text{prfA1}\), which harbors the wild-type \(\text{pth}\) allele (lanes 10 and 12), no peptidyl-tRNA was identified during toxic minigene expression at 43°C; this suggests that all the generated peptidyl-tRNA eventually dissociates from the ribosomes and is cleaved by Pth in solution.

**DISCUSSION**

We used a library of two-codon minigenes to analyze the effect of the variable second codon on Pth-defective cells. The minigenes were ranked as fully toxic, partly toxic, and non-toxic. All analyzed minigenes showed that these categories correlated directly with relative levels of the respective peptidyl-tRNA concentrations. With few exceptions, the four minigenes bearing codons from the same genetic code box showed similar degrees of toxicity (Fig. 1B). This rule held for unmixed boxes, i.e. those encoding only one type of amino acid, as well as for mixed boxes, encoding two amino acids. A nucleotide sequence relationship has been reported for the tRNAs cognate to...
A third factor affecting ribosomal pausing could be the interaction between tRNA in the ribosomal P-site and the release factor in the A-site. For example, in the presence of peptidyl-tRNA\textsubscript{Met}^-\textsubscript{3} in the P-site at codons GGA/G, termination efficiency at UAG is higher than in the presence of peptidyl-tRNA\textsubscript{Gly}^-\textsubscript{2} at the same codons, suggesting an unusual interaction between tRNA\textsubscript{Gly}^-\textsubscript{2} and RF1 (36). This hypothesis could explain why toxicity and peptidyl-tRNA accumulation for TCT and CTG in the corresponding minigenes is associated to only one of the two cognate tRNAs (Fig. 1B).

What is the distribution of “toxic” and “non-toxic” codons in bacterial genes? Interestingly, AAA/G and AAU/AAC codons in minigenes, which promote high rates of peptidyl-tRNA accumulation (Fig. 2), are frequently located at the beginning of the E. coli ORFs. These codons enhance efficiency of translation when substituted at positions two and three of a reporter gene (37, 38). By contrast, codons that promoted low rates of peptidyl-tRNA accumulation are rarely located among the first three positions (e.g. CUN, GCN, and GGN gene code boxes; Refs. 37 and 38). In the \textit{pht(ts)} mutant, families of tRNAs cognate to these codons are among those with the lowest rates of accumulation of the corresponding peptidyl-tRNAs at non-permissive temperatures (11). The presence of codons prone to drop-off in minigenes may represent an advantage for protein synthesis when located at the initial positions in the mRNA ORFs. These codons might act as sensors of the general availability of charged tRNAs. If the availability is appropriate, elongation proceeds, otherwise abortive drop-off occurs. This strategy would prevent wasteful protein synthesis elongation under limiting tRNA availability.

Interestingly, a high frequency of AAA/G codons, associated to high drop-off rates in minigenes, has been found at the last sense position of the \textit{E. coli} ORFs where they also promote drop-off (13). This non-random codon distribution at the ends of ORFs has been considered as an important factor in the modulation of translation termination (39). This could be explained, because such codons provide an alternative translation termination mechanism including drop-off and Pht-mediated hydrolysis of the final peptidyl-tRNA.

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The Rate of Peptidyl-tRNA Dissociation from the Ribosome during Minigene Expression Depends on the Nature of the Last Decoding Interaction
L. Rogelio Cruz-Vera, Elena Hernández-Ramón, Bernardo Pérez-Zamorano and Gabriel Guarneros

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