Factors That Influence Selection of Coding Resumption Sites in Translational Bypassing

MINIMAL CONVENTIONAL PEPTIDYL-tRNA:mRNA PAIRING CAN SUFFICE*§

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This study investigates bypassing initiated from codons immediately 5′ of a stop codon. The mRNA slips and is scanned by the peptidyl-tRNA for a suitable landing site, and standard decoding resumes at the next 3′ codon. This work shows that landing sites with potentially strong base pairing between the peptidyl-tRNA anticodon and mRNA are preferred, but sites with little or no potential for Watson-Crick or wobble base pairing can also be utilized. These results have implications for re-pairing in ribosomal frameshifting. Shine-Dalgarno sequences in the mRNA can alter the distribution of landing sites observed. The bacteriophage T4 gene 60 nascent peptide, known to influence take-off in its native context, imposes stringent P-site pairing requirements, thereby limiting the number of suitable landing sites.

Standard genetic decoding is dependent on the formation and maintenance of cognate codon:anticodon pairing. Once established in the ribosomal A-site, this interaction continues through the ribosomal P-site. However, at specific sites in mRNAs ranging from those in retroviruses to Escherichia coli, ribosomes can shift reading frame for gene expression purposes. Most of these specific changes in reading frame involve dissociation of codon:anticodon pairing in the ribosomal P-site and re-pairing at an overlapping codon. Although mechanisms for ensuring the fidelity of pairing in the A-site have been well studied (1), much less is known about the stringency of pairing in the ribosomal P-site. In single nucleotide frameshifting events, the stringency of re-pairing is often difficult to separate from the efficiency of dissociation because the codons share two nucleotides in common. Dissociation and re-pairing, however, are uncoupled in the related phenomenon of translational bypassing. Following initiation of mRNA slippage (or take-off), ribosomes move further downstream, bypassing a block of nucleotides, before coding resumes at the codon 3′ of the landing site. A careful analysis of landing site selection offers a means to investigate P-site pairing stringency.

Long range translational bypassing in response to A-site codons whose aminoacyl-tRNAs are limiting has been demonstrated (2–4). In these cases, the peptidyl-tRNA dissociates from the ribosomal P-site codon, scans the coding gap, and re-pairs to mRNA at complementary triplets. There are also examples of short distance hopping over stop codons (5), including β-globin in rabbits, where hopping produces some longer forms of the protein (6). In one of these studies, landing at sites with two out of three Watson-Crick or wobble base pairs was reduced by at least two-thirds compared with a matched codon (7).

Long distance bypassing is required for expression of phage T4 gene 60 (8, 9) where 50 nucleotides are bypassed by 50% of the ribosomes that initiate translation (10). Several different signals in the mRNA are required. Most important for the current work is a nascent peptide sequence encoded upstream of the 50-nucleotide coding gap that, while still within the ribosome, strongly stimulates take-off (9, 11, 12). Because the nascent peptide moves with the bypassing ribosome, the potential exists for this signal to also influence landing site selection. Forward ribosome slippage is normally constrained by ribosomal protein L9 (7, 13). In gene 60 bypassing the role of L9 is overridden through effects of a stem-loop structure at the 5′ end of the coding gap (14). Deletion of the L9 gene reduces the constraints on forward mRNA slippage and increases the proportion of bypass products relative to termination and stop codon readthrough or frameshift products (7, 13). How L9 exerts its effect on restraining mRNA slippage is unclear. Its N-terminal domain binds within domain V of 23 S rRNA close to the base of the L1 stalk (15, 16). The L1 stalk is actively involved in the translational movement of tRNA from the P-site to the E-site (17). L9 has a centrally located long α-helix (18) and a C-terminal region that undergoes substantial movement between two states in the ribosome cycle (19, 20). Strains with either WT L9 or lacking L9 were used in the current study, which explores factors that influence bypassing ribosomes to resume coding.

EXPERIMENTAL PROCEDURES

Bacterial Strains and Plasmids—The WT strain used in the experiments was CSH142 (21). The isogenic U34C-RNA−−expressing strain CSH142[su/SU34C] has been described (22) as well as another isogenic strain containing a precise deletion of the gene encoding L9 (AH156) (13). The parental expression vectors for the GST-MBP-6XHis constructs (GTM10) and TrxA-6XHis-MBP construct (TSM1) have been described (7). The insert for the nascent peptide fusion construct was constructed using standard PCR techniques. For all other constructs, complementary oligonucleotides were designed to generate compatible ends for the appropriate restriction sites.

Protein Analysis—Overnight cultures of strains expressing the GST-
MBP-6XHis plasmids were diluted 1:100 in Terrific Broth containing 100 μg/ml ampicillin, grown for 2 h at 37 °C, and then induced with 1 mM isopropyl-β-D-thiogalactoside for an additional 4 h at 37 °C. Harvested cells were lysed using Novagen BugBuster reagent. Full-length GST-MBP-6XHis fusion protein was purified by sequential passages over glutathione-Sepharose (Amersham Biosciences) and nickel-nitrilotriacetic acid-agarose (Qiagen). Cultures containing the TrxA-6XHis gene 60-MBP plasmids were grown as described above except the inductions were carried out at 20 °C to maximize solubility of the gene 60 nascent peptide-containing fusion proteins. Full-length TrxA-6XHis gene 60-MBP fusion protein was purified by sequential passages over nickel-nitrilotriacetic acid-agarose (Qiagen) and amylose-agarose (New England Biolabs). Eluted proteins were concentrated and washed extensively with Nanopure H2O using a Centricon 30 (Millipore) filtration unit. Protein was digested with PreScission Protease according to the protocol provided by the supplier (Amersham Biosciences). The digestion buffer was 50 mM Tris-HCl, pH 7.0, 150 mM NaCl, 1 mM EDTA, and 1 mM dithiothreitol. The samples contained the proteins of interest, as well as the GST- or TrxA-containing N-terminal portions of the fusion proteins, PreScission protease, and nonspecific contaminants that copurified on the affinity columns. For Fig. 1, protein concentrations were determined by measuring absorbance at 280 nm. Final desalting and clean up for mass spectrometric analysis were performed using C4 P10 ZipTips (Millipore). These reverse-phase devices were prepared initially by washing with 80 μl of methanol + 1% (v/v) formic acid and then equilibrated with 80 μl of high pressure liquid chromatography-grade water. Protein samples in water were loaded onto the ZipTip and washed with high pressure liquid chromatography-grade water + 1% formic acid. Proteins were eluted with three 5-μl aliquots of 65% (v/v) methanol + 3% formic acid that were pooled and infused at 4 μl/min. Mass measurements were performed with a Quadrupole II mass spectrometer (Micromass, Inc.) using positive ion electrospray ionization. All spectra were acquired using Masslynx software (Micromass), and multiply charged ion species were processed into a neutral mass spectrum (mass range 42,550 to 44,750 Da). Molecular mass spectrum of a mixture of the same four proteins in a ratio of 10:5:2:1::1:2:3:4 shows the expected and observed masses of each product in Da.

**RESULTS**

**Significance of the Ratio of Products Inferred from Mass Spectrometry**—This study required the analysis of samples of multiple protein products differing by a very small number of internal amino acids. To assay if mass spectrometric analysis accurately corresponded to the relative abundance of each component of a mixture, four separate synthetic proteins were produced. These four proteins contained glutathione S-transferase (GST) at the N terminus, a PreScission protease cleavage site, a variable region, followed by maltose-binding protein (MBP) with a six-histidine (His6) tag. Fusion proteins were purified by affinity chromatography and digested with PreScission protease. Two samples were prepared for mass spectrometric analysis, one containing equimolar amounts of the four proteins (Fig. 1A) and one containing the four proteins, 1:2:3:4 in a 10:5:2:1 ratio (Fig. 1B). The ratio of peak heights in the mass spectra reflected the ratio of products in the mixed samples providing confidence in this sensitive method of product analysis.

**Bypassing Can Lead to Coding Resumption at Multiple Sites**—Previous work has shown that in-frame stop codons can promote peptidyl-tRNA bypassing. To explore the latitude in pairing requirements for coding resumption, a sequence containing few codons with potential for strong Watson-Crick interactions with peptidyl-tRNA was placed 3' of GGA UAA (Fig. 2A). GGA is the take-off codon for bacteriophage T4 gene 60 bypassing and is decoded by tRNA<sub>GGY</sub> (anticodon = 3'CCU*5' where the * is an unknown modification) (9, 22). None of the other stimulatory elements for gene 60 bypassing (the nascent peptide, the stem-loop in the coding gap, or matched landing codons) was included in this construct. Fusion protein resulting from readthrough and/or bypassing was purified, digested with PreScission protease, and then analyzed by electrospray mass spectrometry to characterize the translational event(s).

In a strain wild type for ribosomal protein L9, readthrough of the UAA stop codon was detected, product 5, and multiple bypass products from peptidyl-tRNA landing at +1 frame. Assays were performed as described (21) except that cell were grown in MOPS medium (23) supplemented with 20 amino acids.

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it is in the second position, the most important interaction of the three positions. In the absence of L9, the efficiency of readthrough and/or bypassing increased from 0.8 to 1.6% (Fig. 2D). As expected, when L9 is not present to constrain forward mRNA slippage, bypassing increases relative to readthrough (in the L9/H11002 strain, bypass products constitute 94% of the total, whereas in the L9/H11001 strain, bypass products account for 82% of the total; comparing product 5 with 6–11). Comparison of the distributions of bypass products in the isogenic strains, except for L9, shows that there may be an effect of L9 on landing site preference. However, the most striking result is the detection of landing at an AAU codon, product 6, with no Watson-Crick or wobble pairs possible.

Effect of Gene 60 Nascent Peptide—An effect of the gene 60 nascent peptide on dissociation of peptidyl-tRNA:mRNA pairing to initiate bypassing is known. Whether it also plays a role in landing site selection merited investigation. In the constructs designed, competition was set up between +1 frameshifting and bypassing, both of which require forward mRNA movement (Fig. 3A). These constructs contained thioredoxin A sequences in place of GST in order to increase the solubility of the highly insoluble gene 60 nascent peptide-containing products. In the control lacking the nascent peptide signal in a wild type L9 strain, +1 frameshifting from GGA to GAU, product 12, predominates, although there is bypassing to GGU, product 14, +31 GAA, product 15, and +37 GGU, product 17 (Fig. 3, B and C). When the nascent peptide is present in a wild type L9 strain, +1 frameshifting is eliminated, whereas bypassing to +49 GGU, product 19, is the most frequent event. These results could be because of the increased stringency requirement for re-pairing in the P-site.
of the nascent peptide; bypassing to +49 GGU allows for two Watson-Crick pairs, whereas +1 frameshifting to GAU allows for only a single, first position pair. Alternatively, these results could implicate the nascent peptide signal in promoting long distance mRNA slippage or an effect of landing site context. In the strain lacking L9 and the nascent peptide, where overall net shifting to the +1 frame is increased, bypassing is favored over +1 frameshifting, as expected. The diversity of landing sites was also increased as revealed by landing at +13 AAU, product 13, +34 GAA, product 16, and +49 GGU, product 19 (Fig. 3, B and C). However, in the strain lacking L9 but containing the nascent peptide, the predominant landing site, +49 GGU, did not change, although the distribution of the lesser products was altered.

In summary, the results from these experiments raise an issue concerning the possible effect of the nascent peptide on bypassing distance which is addressed below.

**Nascent Peptide Effect on Forward mRNA Movement**—To test directly for the ability of the peptide signal to affect distant forward message movement, two constructs were designed that contained similar downstream landing sites. The take-off site was changed to GGG UAG to allow the +1 frameshifting site (underlined) to duplicate the downstream landing sites, GGU. Many of the suboptimal landing sites observed in Fig. 3 were changed to less favorable sites to more easily monitor landing at GGU. In this experiment, the immediate context of the downstream GGU landing sites was the same, CAA-5' and AAA-3', to

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**Fig. 3. Effect of the gene 60 nascent peptide on landing site selection.** A, diagram of constructs containing or lacking the gene 60 nascent peptide signal. Maltose-binding protein (MBP) sequences are in the +1 frame relative to TrxA. Landing sites are underlined, and codon:anticodon pairings are shown below. B, molecular mass spectra of PreScission protease-treated fusion protein lacking the nascent peptide (mass range 41,630–44,130 Da) or containing the nascent peptide (mass range 46,300–48,800 Da). Increasing mass is on the x axis and relative abundance of product on the y axis. L9 was WT or deficient as indicated. C, table of predicted and observed masses in Da. The sizes of the products from the construct lacking the nascent peptide are 4670 ± 1 Da smaller than the products from the construct containing the nascent peptide, the difference corresponding to the mass of the nascent peptide.
preclude adjacent sequence effects on landing. A strain containing tRNA2^Gly with a mutation of its anticodon base 34, U^*, to C (22, 24, 25) was utilized because its anticodon is complementary to the modified take-off site, GGG. The strain was also WT for L9. In the construct lacking the nascent peptide signal, there is +1 frameshifting, product 20, as well as bypassing to the closer +22 GGU, product 21, and a trace amount to the more distant +49 GGU, product 22 (Fig. 4, B and C). When the nascent peptide signal is present, only the +1 frameshifting product, 20, is detected. The presence of the nascent peptide increased transition to the +1 frame by 30-fold when assayed in a lacZ reporter system (from 0.3 to 9 β-galactosidase units). It follows that the nascent peptide does not promote distant forward mRNA movement, but rather imposes a greater stringency requirement for P-site codon:anticodon pairing.

**Shine-Dalgarno Effect on Peptidyl-tRNA Landing Site Selection**—In Figs. 2 and 3, particular landing sites may be utilized because of an mRNA signal that could direct landing. One such
element known to affect framing during translation is a Shine-Dalgarno-like sequence (reviewed in Ref. 26). In these constructs, the take-off site, GGA, itself a weak Shine-Dalgarno sequence, could be affecting landing, in particular, at AAU with no potential for Watson-Crick or wobble pairing. To test directly the effect of a Shine-Dalgarno sequence on landing, a take-off site lacking As or Gs was utilized, and a Shine-Dalgarno sequence was introduced downstream. The take-off site, chosen from a set of all possible matched codons for the gene 60 take-off and landing sites, was UCC. The constructs shown in Fig. 5 contain a suboptimal UAC landing site 17 nt 3′ of the take-off site and an optimal UCC landing site 29 nt 3′ of the take-off site (Fig. 5A). In one construct, a Shine-Dalgarno-like sequence (GGAGG) is positioned 6 nt 5′ of the suboptimal UAC. In the other construct, this Shine-Dalgarno-like sequence is mutated to CUACU. Because take-off is less efficient with UCC, detailed product analysis was undertaken only in an L9-deficient strain that favors forward mRNA slippage. In the presence of the Shine-Dalgarno-like sequence, landing occurred at UAC by two out of three pairings (Fig. 5, product 23), at AAC by one out of three pairings (product 24), and at the matched UCC (product 25). In the absence of the Shine-Dalgarno-like sequence, landing was detected only at the matched UCC codon (product 25). Based on previous proof of the effect of Shine-Dalgarno-like mRNA sequences and 16S rRNA interactions on frameshifting (27, 28) and the present results, it was concluded that Shine-Dalgarno:16S rRNA interactions can exert a significant influence on landing site selection.

**DISCUSSION**

The take-off site utilized for the translational bypassing required for synthesis of the topoisomerase component encoded by T4 gene 60 has been utilized to set up a system to explore bypassing initiated by a 3′ stop codon. This study has yielded three conclusions about decoding. (a) Landing sites with strong potential Watson-Crick or wobble base pairing between peptidyl-tRNA and mRNA are preferred but weaker sites may be

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**Fig. 5.** The effect of a Shine-Dalgarno sequence on landing at suboptimal codons. A, construct in the GST-MBP context (see Fig. 2) containing a Shine-Dalgarno-like sequence (SD), overlined, and a suboptimal landing site, UAC, separated by 6 nucleotides. In the construct lacking a Shine-Dalgarno-like sequence GGAGG was changed to CUACU. The UCC take-off site is boxed, and the landing sites are underlined. B, molecular mass spectra (mass range 42,000–44,000 Da) of PreScission protease-digested fusion protein purified from an L9-deficient strain. Increasing mass is shown on the x axis, and relative abundance of product is indicated on the y axis. C, table of expected and observed masses of the products in Da.
utilized. The likely influences of tRNA structure, pairing position(s) within the anticodon, the immediate codon context, and distance from the take-off site are difficult to apportion for any particular landing site. (b) Shine-Dalgarno sequences in mRNAs as they are translating is associated at take-off and increased base pairing requirements at downstream Shine-Dalgarno sequence, as discussed above. How- ever, P-site pairing may not be necessary in other situations upstream gene (29). The 30 S subunit, which contains the antico- don:codon pairing as has been shown previously to occur in the P-site while still within the ribo- somes, presumably in the exit tunnel. Previous work has shown effects of nascent peptide sequences on inhibition of peptidyl-tRNA transfer or cleavage or translation elongation in docking of various genes including tryptophanase, the upstream open reading frame preceding the coding region of mammalian S-adenosylmethionine decarboxylase, a human cytomegalovirus gene and a fungal argen gene (40–42). The present finding of a nascent peptide effect on the fidelity of codon-anticodon interaction in the ribosomal P-site strengthens appreciation of the versatile effects that nascent peptides can have on decoding.

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