Plasticity of the RNA kink turn structural motif

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ABSTRACT

The kink turn (K-turn) is an RNA structural motif found in many biologically significant RNAs. While most examples of the K-turn have a similar fold, the crystal structure of the *Azoarcus* group I intron revealed a novel RNA conformation, a reverse kink turn bent in the direction opposite that of a consensus K-turn. The reverse K-turn is bent toward the major grooves rather than the minor grooves of the flanking helices, yet the sequence differs from the K-turn consensus by only a single nucleotide. Here we demonstrate that the reverse bend direction is not solely defined by internal sequence elements, but is instead affected by structural elements external to the K-turn. It bends toward the major groove under the direction of a tetraloop–tetraloop receptor. The ability of one sequence to form two distinct structures demonstrates the inherent plasticity of the K-turn sequence. Such plasticity suggests that the K-turn is not a primary element in RNA folding, but instead is shaped by other structural elements within the RNA or ribonucleoprotein assembly.

Keywords: RNA structure; X-ray crystallography; group I intron; reverse kink turn

INTRODUCTION

Analysis of structural elements that provide stability to global RNA complexes is important for understanding the rules of RNA folding. The kink turn (K-turn) is a well-studied RNA motif that forms both structured and flexible conformations within larger RNA molecules (Matsumura et al. 2003). The plasticity of the K-turn motif and the ability to analyze its properties at an atomic level make it a good model for studying RNA structure (Turner et al. 2005).

The K-turn was first described in the large ribosomal subunit of *Haloarcula marismortui* (Klein et al. 2001). Six examples of this motif are present within the *H. marismortui* 50S subunit, and two are present in the *Thermus thermophilus* 30S subunit (Ban et al. 2000; Schlutzen et al. 2000; Wimberly et al. 2000). Other K-turns are located in non-ribosomal structures such as riboswitches, small nucleolar RNAs, and ribozymes (Nottrott et al. 1999; Adams et al. 2004; Montange and Batey 2006; Blouin and Lafontaine 2007). The K-turn is composed of 15 nucleotides (nt) that form a helix–internal-loop–helix structure in duplex RNA

(Fig. 1). Ten of these 15 nt make up the K-turn's consensus secondary structure (Klein et al. 2001). A standard K-turn has a canonical helix (C-stem), formed from C–G pairs, followed by a 3-nt, asymmetric internal loop that ends with a noncanonical helix (NC-stem) of G–A mispairs. Tertiary interactions to the two closing G–A mispairs facilitate the K-turn bend and include a stabilizing A-minor motif contact to the A in the first G–A pair. The three nucleotides within the asymmetric loop create the kink in the phosphodiester backbone of the RNA. Nearly every K-turn adheres to the consensus secondary structure and forms a 120° bend in the helix toward the minor groove (Klein et al. 2001).

Unlike other K-turns, the crystal structure of the *Azoarcus* group I intron revealed a helix–loop–helix element that has many secondary structural features of a consensus K-turn, yet it is bent in the opposite direction (Adams et al. 2004). Similar to the consensus K-turn, the reverse K-turn has a 3-nt asymmetric bulge and is flanked on both sides by C- and NC-stems. A 90° bend forms at the asymmetric loop between the P9.0 and P9. The only difference in sequence between the K-turn consensus and that of the reverse K-turn is a G-to-A change (nucleotide 201, *Azoarcus* numbering) in the first base pair of the NC-stem, resulting in an A–A instead of a G–A mispair. In a standard K-turn, the 5'-most nucleotide of the loop stacks on the C-stem, allowing the second nucleotide in the bulge to stack on the NC-stem, and the third nucleotide to be displaced into solution. In the reverse K-turn, all three residues stack beneath either the C- or NC-stem with no residue extended into solution.

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Article published online ahead of print. Article and publication date are at <http://www.rnajournal.org/cgi/doi/10.1261/rna.1883810>.

FIGURE 2. (A) Schematic representation of the *Azoarcus* group I intron used in kinetic assays. The reverse K-turn sequence (boxed region) differs from a consensus K-turn at position A201. A TL-TLR interaction forms between P9 and J5/5a. Exogenous GMP is added *in trans*, and cleavage of the 5' exon (represented by CAUAAA) is used to measure splicing activity. (B) Consensus K-turn sequence with G201. (C) Secondary structure of the base-paired construct.

the NC-stem; an A in the reverse K-turn is a G in the canonical sequence. We compared the activity of the WT ribozyme with one in which the sequence was mutated to that of a consensus K-turn (KT ribozyme with an A201G mutation) under the slightly destabilizing reaction conditions described above. The WT ribozyme reacted with a rate of $39 \times 10^{-3} \text{ min}^{-1}$, while the KT ribozyme was negligibly slower, reacting at a rate of $33 \times 10^{-3} \text{ min}^{-1}$ (Table 1). This suggests that the intron remains properly folded with P9.0 bent toward the major groove despite containing a consensus K-turn sequence that would be predicted to bend in the opposite direction.

Mutations within the internal asymmetric loop

The three nucleotides (residues 198–200) in the asymmetric loop of a standard K-turn do not display any sequence conservation and are not expected to be defining elements for the consensus K-turn. To determine if these nucleotides define an obligate reverse K-turn subclass, we mutated the nucleotides in the asymmetric loop. Of particular interest is nucleotide C199, which appears to form a hydrogen bond with the phosphate of G180 in the NC-stem. This interaction could facilitate the close stacking of bases under the C- or NC-helix, helping to stabilize the reverse K-turn bend. Constructs WT_{C199U} and KT_{C199U} disrupt this potential hydrogen bond in the WT and KT backgrounds, respectively. However, the C199U mutation had a small effect on the rate in either context (Table 1). These data suggest

that the internal hydrogen-bonding interaction plays at most only a minor role in stabilizing the conformation of the reverse K-turn and is not the defining element for the bend direction.

The asymmetric loop of the reverse K-turn is a relatively A-rich region with adenosines at the first and third nucleotides. Four additional constructs were developed to disrupt the two other positions in the asymmetric loop (constructs WT_{A198U}, (KT_{A198U}, WT_{A200U}, and KT_{A200U} mutate the adenosines at positions 198 and 200 in both the WT and KT backgrounds). For all constructs, the rates are within twofold of the wild-type values. These data further suggest that the three nucleotides in the asymmetric loop, the primary site of the bend, are not a significant determinant of K-turn bend direction. The sequence of the *Azoarcus* group I intron asymmetric loop does not define a reverse K-turn subclass.

Mutation of external elements

Given that sequence elements within the reverse K-turn do not appear to define bend direction, we next explored the role of external structural factors. We were particularly interested in the possibility that a TL-TLR interaction would have a strong influence on the reverse K-turn bend direction. The tetraloop at the end of P9 interacts with a tetraloop receptor within P5 of this intron (Fig. 2A). We mutated this GAAA tetraloop to UUGG in both the WT and KT backgrounds (WT_{UUGG} and KT_{UUGG}). This resulted in 740- and 925-fold losses in the rate of cleavage for the two constructs. We also examined a more conservative mutation of the tetraloop, A190C, which changes the GAAA tetraloop to GCAA. The result is a stable GNRA tetraloop, but the tetraloop forms a significantly less stable interaction with the receptor (Costa and Michel 1997; Murphy and Cech 1993). We found a moderate fivefold

FIGURE 3. Reaction rates for wild-type (WT), consensus (KT), and base-paired (BP) *Azoarcus* constructs. The fraction of unreacted substrate is plotted as a function of time with final rates determined by fitting the curves to a first-order rate equation.

TABLE 1. First-order rate kinetics for *Azoarcus* group I intron mutants

Mutant	Location of mutation	k_{obs} [(10 ⁻³) min ⁻¹]	Relative rate decrease
WT	—	37 ± 3.0	1.0
KT	A201G	33 ± 2.7	1.2
BP	Base-pairing nucleotides 198–200	0.3 ± 0.09	123
WT _{C199U}	C199U	41 ± 3.2	1.0
KT _{C199U}	C199U, A201G	22 ± 3.0	1.7
WT _{A200U}	A200U	35 ± 1.0	1.1
KT _{A200U}	A200U, A201G	26 ± 0.8	1.4
WT _{A198U}	A198U	15 ± 1.0	2.4
KT _{A198U}	A198U, A201G	16 ± 0.5	2.3
WT _{UUGG}	G189U, A190U, A191G, A192G	0.05 ± 0.02	740
KT _{UUGG}	G189U, A190U, A191G, A192G, A201G	0.04 ± 0.03	925
WT _{A190C}	A190C	7.3 ± 0.6	5
KT _{A190C}	A190C, A201G	1.4 ± 0.7	26

Mutations were made to internal and external positions within the P9/P9.0 helix.

rate effect in the wild-type background (WT_{A190C}), but an almost 30-fold effect for the RNA with mutations in both external and internal regions (KT_{A190C}). The combination of internal and external mutations appears to destabilize the intron more than either point mutation alone.

Structure determination of the consensus sequence

To confirm that we made the proper structural interpretation of this kinetic result, we crystallized the A201G mutant RNA (KT) and obtained the structure by molecular replacement. The construct was assembled in a manner similar to that used to obtain the original *Azoarcus* group I intron crystal structure (Adams et al. 2004). A 22-residue oligonucleotide, dcircm, which represented the 3'-exon and the 3'-end of the intron and included the A201G point mutation, was crystallized with the 5'-exon CAT and the remainder of the intron produced as an RNA transcript. We obtained a 4.2 Å resolution structure of the mutant group I intron in the same space group (P4₁22) as previously reported for other *Azoarcus* intron structures (Table 2; Adams et al. 2004; Stahley and Strobel 2005; Lipchick and Strobel 2008). Both the mutant and the original structures have the same overall architecture (Fig. 4). Although the resolution was limited, there was no observable change in conformation of the phosphate backbone of the P9 helix resulting from the point mutation. Thus, despite a consensus sequence, the P9.0 helix remained bent toward the major grooves. This provides structural support to the kinetic data and suggests that a consensus K-turn sequence can fold in the opposite direction within the context of the *Azoarcus* ribozyme.

DISCUSSION

This study demonstrates that the K-turn motif is an example of a plastic secondary structural element that can

adopt multiple conformations in the presence of different external factors. We explored the sequence and structural determinants of the reverse K-turn within the context of the *Azoarcus* ribozyme and found that docking of a TL-TLR can mold a consensus K-turn into an unusual bend direction. The ability of the K-turn to form two distinct, yet stable structures from the same primary sequence supports the proposal that K-turns are not principal organizing elements of RNA structure (Goody et al. 2004; Turner et al. 2005). While other internal loop motifs, such as the bulged-G motif, are defined as rigid regions (Correll et al. 2003), K-turns are dynamic with kinked and unkinked conformations influenced by metal ion concentration, tertiary

TABLE 2. Crystallographic data collection and refinement statistics

Parameter	Result
Data Collection	
Cell dimensions	
$a = b$ (Å)	109.8
c (Å)	250.2
$\alpha = \beta = \gamma$ (°)	90
Wavelength (Å)	0.9783
Resolution (Å) ^a	50.0–4.2 (4.4–4.2)
R_{merge} (%) ^a	10.1 (78.9)
$\langle I /\sigma(I) \rangle$ ^a	15.5 (2.2)
Completeness (%) ^a	99.4 (100)
Redundancy ^a	6.3 (6.4)
Refinement	
Resolution (Å)	50.0–4.2
Number of Reflections ^b	11,715 (562)
$R_{\text{work}}/R_{\text{free}}$ (%)	25.7/30.3
RMSD bond (Å)	0.009
RMSD angle (°)	1.806
Protein database accession code	3IIN

^aValues in parentheses are for the highest-resolution shell.

^bValues in parentheses are for the cross-validation test set.

FIGURE 4. Comparison of a crystal structure of an *Azoarcus* mutant with a consensus K-turn sequence (green/orange) to the original *Azoarcus* intron structure (gray). The P9-J5/5a tetraloop-tetraloop receptor interaction is highlighted (blue) as well as a scissile phosphate in the intron core (red).

interactions, and protein binding (Nottrott et al. 1999; Goody et al. 2004).

A tetraloop-tetraloop receptor interaction stabilizes the reverse K-turn bend direction

Solution studies of isolated K-turn motifs show flexible RNAs that are able to form tightly kinked or extended conformations in the presence or absence of metal ions (Goody et al. 2004). Other K-turns, like many in the ribosome, rely on external ligands and tertiary interactions to mold their conformations (Matsumura et al. 2003). Because K-turns can be shaped by external elements we investigated the role of the TL-TLR interaction between P5 and P9 in the bend direction of the reverse K-turn sequence. We observed that the disrupted tetraloop mutants had the most significant impact on reaction rate, which is consistent with the conclusion that mutations to the GAAA tetraloop of L9 disrupted proper intron folding (Rangan et al. 2003). In fact, it was only when this interaction was lost that we were able to observe any effect from mutation of the reverse K-turn to the consensus sequence.

This view of the K-turn is similar to the bent sequence within the P4-P6 domain of the *Tetrahymena* group I intron, which also requires external tertiary interactions in order to adopt a bend (Cate et al. 1996a,b). The P4-P6 domain consists of 160 nucleotides and forms an independent tertiary structure including a 180° hinge at J5/5a (Szewczak and Cech 1997; Cate et al. 1996a,b). To fold correctly, the motif requires a TL-TLR and A-minor motif

interactions. The nucleotide sequence that makes up the J5/5a hinge has only a minor role in specifying folding (Szewczak and Cech 1997). Similar to the J5/5a hinge, we found that the reverse K-turn loop nucleotides do not direct folding of the K-turn in the *Azoarcus* group I intron; instead, a TL-TLR interaction is the main determinant of the reverse K-turn bending, and the reverse K-turn is simply a flexible region that can be molded into a major groove bend.

In the hierarchy of RNA folding, a TL-TLR has a dominant role in structure stabilization relative to the KT-bend direction. The plasticity of the reverse K-turn enables it to be shaped in different conformations using distal tertiary contacts and protein binding. While it is certainly possible to design RNA to have a K-turn bend, the actual direction of that bend may be more difficult to predict suggesting that the knowledge of structural plasticity for an RNA secondary structure could enhance RNA tertiary structure prediction.

MATERIALS AND METHODS

Assembly of kinetic assay constructs

The WT reverse sequence template consisted of an *Azoarcus* group I intron construct (L-6 C204) that terminated at nucleotide position C204. Twelve additional constructs used in kinetic experiments were generated using site-directed mutagenesis (QuikChange, Stratagene) with the WT construct serving as template DNA (Table 1).

In vitro transcription

DNA was prepared for in vitro transcription by performing an EarI restriction enzyme digestion. RNA was transcribed using T7 RNA polymerase and purified by electrophoresis in a 6% polyacrylamide gel containing 7 M urea. Following an overnight elution step in water, the RNAs were concentrated to 0.1–1 mg/mL using Amicon Centrifugal Filter devices.

Short oligonucleotide radiolabeling

A small oligonucleotide was 5'-end-radiolabeled in a 10-μL reaction with [γ - 32 P]ATP using T4 PNK. Reagents were incubated for 1 h at 37°C and quenched by the addition of formamide loading buffer (95% formamide, 2.5 mM EDTA, 0.1% bromophenol blue, and 0.1% xylene cyanol). The labeled oligonucleotide was purified by 20% non-denaturing PAGE, and the labeled oligonucleotide was eluted overnight in water.

Kinetic measurements

Kinetic reactions were performed to measure the rate of splicing activity for each construct. Reactions contained 3 μM RNA, 1× kinetic buffer [3 mM MgCl₂, 25 mM MES at pH 6.0, and 1 mM Mn(OAc)₂], 5 mM exogenous guanosine monophosphate (GMP), 1 M urea, and trace amounts of 32 P-radiolabeled CAUAAA. Reaction mixtures were preincubated for 2 min at 50°C before the addition of labeled oligonucleotide and exogenous GMP. Time points were taken by removing 2 μL of the reaction mixture and quenching with 8 μL of formamide loading buffer. A 20%

denaturing polyacrylamide gel was used to separate the unligated substrates from the ligated products. RNA fragments were visualized using a Storm PhosphorImager (GE Healthcare), and data were quantized using ImageQuant (GE Healthcare). For each construct, a reaction rate was found by plotting the fraction of unligated product versus time and fitting the data with Kaleida-Graph to an exponential decay endpoint using Equation 1:

$$f_{\text{unreact}} = (1 - F_{\text{unreact}}) * \exp(-k_{\text{obs}} * t) + f_{\text{unreact}}, \quad (1)$$

where f_{unreact} = fraction unreacted; F_{unreact} = fraction unreactive.

Crystallization construct assembly and structure determination

A single crystallization construct was assembled from an RNA transcript (UP62) and two synthetic oligonucleotides (CAT and dcircm; Dharmacon). The oligonucleotide, dcircm, represents the 3'-end of the intron and the 3' exon. It contains a G at position 201, instead of A, to create the KT sequence, and a deoxyG at position 206. Oligonucleotides were deprotected according to the manufacturer's protocol. The UP62 RNA (120 μ M) and dcircm (150 μ M) were combined and heated for 2 min to 50°C in 10 mM sodium cacodylate (pH 6.5) and 15 mM Mg(OAc)₂. After slow cooling for 15 min, CAT (150 μ M) and the RNA binding domain of the U1A protein (140 μ M) were added. Hanging drop crystallization trials were performed as described previously (Adams et al. 2004). After 3 h, streak seeding was performed using crystals containing an unmutated oligonucleotide. The average crystal size was 120 μ m $\times$ 100 μ m $\times$ 100 μ m, and crystals typically appeared 12 h after seeding. Crystals were stabilized in 30% MPD, 50 mM sodium cacodylate (pH 6.8), 10 mM Mg(OAc)₂, 10 mM KOAc, and 0.2 mM Co(NH₃)₆³⁺; and were flash frozen in liquid nitrogen.

Diffraction data were indexed and scaled using HKL2000, and the *Azoarcus* deoxy pre-2S group I intron structure (PDB ID 1U6B) was used to perform molecular replacement to generate a model of the mutant RNA (Otwinoski and Minor 1997; Adams et al. 2004). As previously reported, this construct yielded crystals of space group P4₁22. Modeling and rigid body refinement of the structure was performed with COOT and REFMAC5 (Winn et al. 2001; Emsley and Cowtan 2004).

Protein Data Bank entry

Atomic coordinates and structure factors have been deposited in the Protein Data Bank, <http://www.rcsb.org>, under PDB ID 3IIN.

ACKNOWLEDGMENTS

We thank the Beamline staff at the National Synchrotron Light Source (NSLS) for assistance with X-ray data collection. This work was supported by NIH PO1 grant GM022778 to S.A.S.

Received August 17, 2009; accepted December 9, 2009.

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RNA 2010 16: 762-768 originally published online February 9, 2010

Access the most recent version at doi:[10.1261/rna.1883810](https://doi.org/10.1261/rna.1883810)

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