TABLE 1 Half-lives of mutant RNA I transcripts

RNA I allele	RNA structure†	Mutant RNA I half-life (min)‡	Wild-type RNA I half-life (min)‡
RNA I (wild-type)			2.7 ± 0.4
RNA I.10		13 ± 2	2.8 ± 0.3
RNA I.20		13 ± 2	3.1 ± 0.3
RNA I.30		7.0 ± 1.0	3.1 ± 0.4
RNA I.40		13 ± 2	2.9 ± 0.2
RNA I.4		2.6 ± 0.2	3.2 ± 0.5
RNA I.15		3.0 ± 0.5	2.6 ± 0.2
RNA I.25		2.1 ± 0.2	2.3 ± 0.2
RNA I.15ΔE		13 ± 1	2.6 ± 0.5

† The expected secondary structure of each RNA I mutant is represented diagramatically (for a detailed secondary structure of wild-type RNA I, see Fig. 1). I, II, III, RNA I stem–loops. *, Synthetic hairpin hp*. X, synthetic hairpin hpX. 1, *ompA* hairpin hp1 (GAUCACCAGGGGUGCUCGGCAUAAGCCGAAGAUAUCGGUAGAGUUAAUAUUGAGCAGAUCCCCCGGUGAUC)[19]. Arrow, RNase E cleavage site[24,25]. RNA I.10 is RNA I with hp* added at the 5′ terminus. RNA I.20 is RNA I with hpX added at the 5′ terminus. RNA I.30 is RNA I with *ompA* hp1 added at the 5′ terminus. RNA I.40 is RNA I.10 with four unpaired nucleotides (GAUC) added immediately downstream of hp*. RNA I.4 is RNA I with four unpaired nucleotides (GAUC) added at the 5′ terminus. RNA I.15 is RNA I.10 with five unpaired nucleotides (GAUCC) added upstream of hp*. RNA I.25 is RNA I.20 with five unpaired nucleotides (GAUCA) added upstream of hpX. RNA I.15ΔE is RNA I.15 with a deletion of seven internal nucleotides (AGUAUUU) surrounding the RNase E cleavage site.

‡ The half-life of each mutant RNA I and of wild-type RNA I was measured simultaneously at 35 °C in *E. coli* strain N3433 (*rne*$^{+}$, *lacZ43, relA, spoT1, thi1*)[7] containing a plasmid that encodes both RNAs. See Fig. 2 for experimental details. The half-life of RNA I.15 increases to 22 ± 2 min in the isogenic *rne*^{ts} *E. coli* strain N3431 (*rne*-3071^{ts}, *lacZ43, relA, spoT1, thi1*)[7] within 30 min after a temperature upshift from 35 to 43 °C (half-life of wild-type RNA I = 14 ± 1 min); its half-life at 43 °C in N3433 is 3.3 ± 0.2 min (half-life of wild-type RNA I = 3.7 ± 0.2 min). The half-life of RNA I.25 increases to 18 ± 1 min in N3431 after a temperature upshift to 43 °C (half-life of wild-type RNA I = 11 ± 1 min); its half-life at 43 °C in N3433 is 5.6 ± 0.4 min (half-life of wild-type RNA I = 5.6 ± 0.9 min).

ling the rate of RNA degradation by RNase E *in vivo*. Although RNase E cleavage can be inhibited by a 5′-terminal stem–loop, it is not abolished completely, as RNA I.10 and RNA I.20 are slowly cleaved *in vivo*. This finding suggests either that RNase E can bypass the 5′ end with low efficiency or that RNase E can interact, albeit less effectively, with a base-paired 5′ terminus. The latter interpretation might explain why not all 5′ stem–loops are equally effective barriers to degradation by this enzyme.

The ability of 5′-terminal unpaired nucleotides to aid RNase E cleavage at a downstream cleavage site in an untranslated RNA suggests that this enzyme has an intrinsic orientation of digestion that might explain the many previous reports of 5′-to-3′ mRNA decay in prokaryotes[26–31]. Such an explanation obviates any need to invoke ribosome movement or the existence of an undiscovered bacterial 5′ exoribonuclease to account for this phenomenon. □

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ERRATA

Abrogation by c-*myc* of G1 phase arrest induced by *RB* protein but not by p53

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Nature **360,** 177–179 (1992)

IN this letter in the 12 November issue, the two-part Fig. 1 and single-part Fig. 2 were transposed. The figure legends are correct as printed.

Mutations in T-cell antigen receptor genes α and β block thymocyte development at different stages

Peter Mombaerts, Alan R. Clarke, Michael A. Rudnicki, John Iacomini, Shigeyoshi Itohara, Juan J. Lafaille, Lili Wang, Yoshiaki Ichikawa, Rudolf Jaenisch, Martin L. Hooper & Susumu Tonegawa

Nature **360,** 225–231 (1992)

IN Fig. 3 of this article the layout of parts *a* and *b* on page 227 was misleading. The three panels in part *b* correspond to the first three panels in part *a*, that is, *WT*, $\alpha-/-$ and $\beta-/-$ respectively. In addition, the last sentence in paragraph 4 of the Discussion on page 231 should read: 'However, rearrangements may occur sequentially at the TCR-α loci, but *without* a causal relationship.'