logical adaptations to overwintering or lack of efficient sinks for carbon (in non-reproducing plants) may set constraints on the potential response to an increase in carbon dioxide. There is insufficient evidence to evaluate the potential combined role of genome size, ploidy level, life cycle and phylogeny in other herbaceous species of plants and in trees.

Although multiplication of noncoding DNA comprises most of the increase in genome size, coding regions also become more numerous[9]. The amount of Rubisco or the amount of enzyme responsible for regenerating the supply of ribulose bisphosphate, the substrate for Rubisco, may be directly correlated with the number of copies of relevant genes in the nuclear genome. Consequently, a larger genome may be able to support production of larger amounts of Rubisco and counterbalance the CO_2-related reduction in Rubisco activity, thus removing one possible limitation to photosynthesis[8].

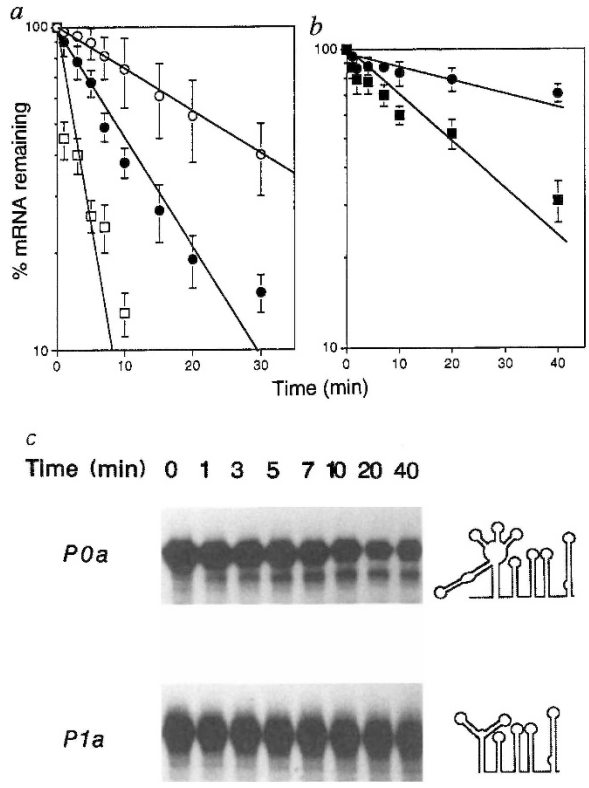
M. Jasieński, F. A. Bazzaz
Department of Organismic and Evolutionary Biology, Harvard University, 16 Divinity Avenue, Cambridge, Massachusetts 02138, USA

1. Bennett, M. D. & Smith, J. B. *Phil. Trans. R. Soc. Lond.* B**334,** 309–346 (1991).
2. Bennett, M. D. *New Phytol.* (Suppl.) **106,** 177–200 (1987).
3. Szarski, H. *J. theor. Biol.* **105,** 201–209 (1983).
4. Grime, J. P., Shacklock, J. M. L. & Band, S. R. *New Phytol.* **100,** 435–445 (1985).
5. Macgillivray, C. W. & Grime, J. P. *Funct. Ecol.* **9,** 320–325 (1995).
6. Bennett, S. T. & Bennett, M. D. *Genome* **35,** 1050–1053 (1992).
7. Poorter, H. *Vegetatio* **104/105,** 77–97 (1993).
8. Bowes, G. *A. Rev. Pl. Physiol. Pl. molec. Biol.* **44,** 309–332 (1993).
9. Lapitan, N. L. V. *Genome* **35,** 171–181 (1992).
10. Hunt, R., Hand, D. W., Hannah, M. A. & Neal, A. M. *Funct. Ecol.* **7,** 661–668 (1993).

Gene expression and mRNA

SIR — The balance between messenger RNA synthesis and decay has a significant effect on the level of gene expression and the fast turnover of mRNA is crucial for rapid changes in the pattern of gene expression[1]. Many bacterial genes have multiple promoters which respond to different signals to facilitate quick adaptation to changes in growth conditions. The selective degradation of mRNAs originating from different promoters in a multiple-promoter gene would enable cells to change the level of gene expression very efficiently and precisely while maintaining a basal level of expression.

The *pts* operon, which encodes several factors in the phosphoenolpyruvate : carbohydrate phosphotransferase system (PTS)[2], is regulated in a complex fashion by at least five promoters, *P1a*, *P1b*, *P0a*, *P0b* and *Px* (ref. 3). The overall expression of PTS is modulated only two- to threefold, depending on changes in the environment[3,4]. The mRNAs from *P1a* and *P0a* are two of the major transcripts expressed *in vivo*. The mRNA from *P1a* is expressed almost constitutively regardless of the growth conditions whereas *P0a* expression is increased when cells grow on glucose[3,4]. The mRNAs from these *pts* promoters have long 5′-untranslated regions (translation starts at +164 from the *P1a* start site) with potential secondary structures. The predicted secondary structure of each of the mRNAs has a key difference in the 5′ end[3]. Only the mRNA from *P1a* has a stem–loop structure at its 5′ end which has been known to protect the mRNA from RNase E, an endonuclease which plays an important role in mRNA degradation in *Escherichia coli*[1,5,6].



The differential degradation of mRNAs from *pts* promoters. *a*, Semi-logarithmic graph of *in vivo* mRNA degradation as a function of time. Each point represents the mean of five independent experiments. ○, *P1a* mRNA in *rne*⁻ condition, ●, *P1a* mRNA in *rne*⁺ condition, □, *P0a* mRNA in *rne*⁻ condition. *b*, Semi-logarithmic graph of *in vitro* mRNA degradation as a function of time. Each point represents the mean of three independent experiments. ■, *P0a* mRNA, ●, *P1a* mRNA. *c*, The *in vitro* degradation of mRNAs which have the 5′-end regions originating from *P0a* and *P1a* promoters. The predicted secondary structure of each mRNA is shown on the right. The mRNA with the 5′-end region of *P1a*, which has a paired nucleotide at the 5′ end, is more stable than that from *P0a*.

The degradation of mRNAs from *P0a* and *P1a* was monitored by a primer extension assay after treating the cells with rifampicin to prevent new RNA synthesis. There were large differences in the stability of mRNAs originated from each promoter: the *P1a* message was much more stable than the *P0a* message (*a* in the figure). The half-life of mRNA from *P0a* and *P1a* was below 30 s and 10 min, respectively, in wild-type cells. The stability of mRNA was increased significantly overall in the *rne*^{ts} strain (the mutant strain with heat-sensitive RNase E)[7] after the heat-sensitive RNase E had been inactivated: the half-life of *P0a* was increased to 3 min and that of *P1a* to 23 min. The *Px* synthesized by σ^{32} RNA polymerase was also stabilized. The effect of RNase E was shown more clearly by an *in vitro* experiment using partially purified RNase E (ref. 8). mRNA from *P1a* was more stable than *P0a* (*b*, *c* in the figure), in agreement with the *in vivo* results. A new band appearing above the *P1a* band after 7 min (data not shown) and the band below the *P0a* mRNA (*c* in the figure, top panel) have the same 5′ end, indicating that it is a processing product of the *P0a* transcript.

These results demonstrate the differences in the stability of *P0a* and *P1a* mRNA. The differential degradation of mRNAs produced from multiple *pts* promoters should have a major role in the fine control of *pts* expression. There are clear advantages for cells if they maintain the basal level of *pts* expression through the expression of a stable mRNA from *P1a*, while fine modulation of *pts* expression is achieved through the expression of the highly unstable mRNAs from other promoters regulated by various external signals.

Sangryeol Ryu
Laboratory of Molecular Biology, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20892, USA

1. Belasco, J. G. in *Control of Messenger RNA Stability* (eds Belasco, J. G. & Brawerman, G.) 3–12 (Academic, San Diego, 1993).
2. Postma, P. W., Lengeler, J. W. & Jacobson, G. R. *Microbiol. Rev.* **57,** 543–594 (1993).
3. Ryu, S. & Garges, S. *J. biol. Chem.* **269,** 4767–4772 (1994).
4. De Reuse, H., Kolb, A. & Danchin, A. *J. molec. Biol.* **226,** 623–635 (1992).
5. Apirion, D. *Genetics* **90,** 659–671 (1978).
6. Bouvet, P. & Belasco, J. G. *Nature* **360,** 488–491 (1992).
7. Mudd, E. A., Krisch, H. M. & Higgins, C. F. *Molec. Microbiol.* **4,** 2127–2135 (1990).
8. Mackie, G. A. *J. Bact.* **173,** 2488–2497 (1991).