TABLE 1 Effect of *virR*::Tn*10* on *proU–lacZ* and *bgl* expression in *E. coli*

Strain*	β-Galactosidase activity	
	No NaCl	0.3 M NaCl
GM37	8	1,336
BRE2076	166	Dead†
CJD389	423	2,316
CJD390	7	1,270

Strain*	β-Glucosidase activity
GM37	10
BRE2076	301
CJD389	233
CJD390	8

* Genotypes of strains are given in the legends to Figs 2 and 3.
† Osmotically sensitive[2].

β-Galactosidase assays for *proU–lacZ* expression were performed on SDS–chloroform permeabilized cells outgrown in 2 ml trypticase soy broth cultures in test tubes to mid-log (with or without 0.3 M NaCl) as described[2,18]. β-Glucosidase assays for *bgl* expression were on cells grown in MMA (minimal medium A) + 0.4 M succinate in the presence of 5 mM β-methyl-D-glucoside, a gratuitous inducer of *bgl*. Cells were assayed[2] for their ability to transport, phosphorylate and metabolize the β-glucoside derivative, *o*-nitrophenyl-β-D-pyranoglucoside. MMA + 0.4 M succinate was a poor growth medium for CJD389. This is also an unexplained feature of several *osmZ* alleles in *E. coli* (unpublished data). Values are averages of three independent experiments. Standard errors were less than 10%.

of *vir* gene expression in *S. flexneri*. Furthermore, we have shown that there is a close genetic relationship between *virR* and *osmZ*, a gene known to be important in the environmental control of DNA supercoiling. As we predicted[2,16], one of the products of the complex *osmZ*(*virR*) locus is a small, histone-like protein (known as H1 or HNS) (C. J. Hulton, A. Seirafi and C.F.H., manuscript in preparation). We have argued elsewhere[7,16] that environmentally determined levels of DNA supercoiling are important in regulating gene expression and have suggested that such a mechanism could be exploited by bacterial pathogens to adapt to new environments encountered at each stage of the infection process. Environmentally induced changes in DNA supercoiling must act with specific regulators of gene expression (such as the well characterized family of two-component regulators[17]). We have demonstrated that changes in DNA supercoiling in response to environmental factors do indeed contribute to the control of bacterial virulence. □

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CORRECTION

A new mechanism for induced vitamin D deficiency in calcium deprivation

M. R. Clements, L. Johnson & D. R. Fraser

Nature **325,** 62–65 (1987).

FURTHER work by the authors has revealed that the above paper contained a systematic miscalculation in the determination of plasma elimination half times for 25-hydroxyvitamin D in rats. The values quoted in the paper (mainly in Fig. 1 and Table 1) are over-estimates by a factor of log 10*e*. To obtain the correct values the published figures need to be divided by a factor of 2.3. This correction does not alter the validity or the interpretation of the published observations. Indeed, because the half time for 25-hydroxyvitamin D in the circulation is significantly shorter than previously indicated, the detrimental effect of calcium deprivation on vitamin D status is event greater than the authors had suggested. □

ERRATUM

DNA cleavage catalysed by the ribozyme from *Tetrahymena*

Daniel Herschlag & Thomas R. Cech

Nature **344,** 405–409 (1990).

IN Figs 1 and 2 of the above article, the shaded regions referred to in the figure legend did not show up because of an oversight in the reproduction process. The 5′ sequence (GGGAGG-5′) of the ribozyme (shown seven times) should have been shaded in these figures. Also, the complex formed between the ribozyme (E) and its oligonucleotide product (P) was changed to E ~ P in the editing process. Because this bond symbol might wrongly implicate participation of a high-energy phosphate bond in complex formation, this complex should have been represented by E · P. Other complexes involving the ribozyme, its oligonucleotide substrate (S), and G should then have been written as E · G · S, or E · G. □