CORRIGENDUM

In the communication by **V. Thuillier, M. Famulok, and co-workers** (*Angew. Chem. Int. Ed.* **2000**, *39*, 4369 – 4373), a correction is necessary in Figure 3: The authors noticed that clone D16-05 contains an additional C residue in position 42 between G41 and G43 (5′-end of the sequence region shown in orange). This mutation slightly alters the computer-generated secondary structure of the point mutant D16-05 to the folding shown in Figure 3b. The RNA decoys incubated with D16-05 (Figure 3a, lanes 7, 8, 10, and 11) did not contain C42 because they were transcribed from a template DNA that was obtained by means of PCR with a 5′ primer that lacks C42. The authors therefore repeated the analysis represented in Figure 3 with the correct primers. The analysis revealed that constructs 10 and 11 in Figure 3a can effectively restore the cleavage activity. As a consequence, the previous statement that helix I (red) plays an important structural role in doxycycline binding is still valid but it now appears that the effect of helix I can be exerted in a sequence-independent manner. All other conclusions of this communication remain unaffected..

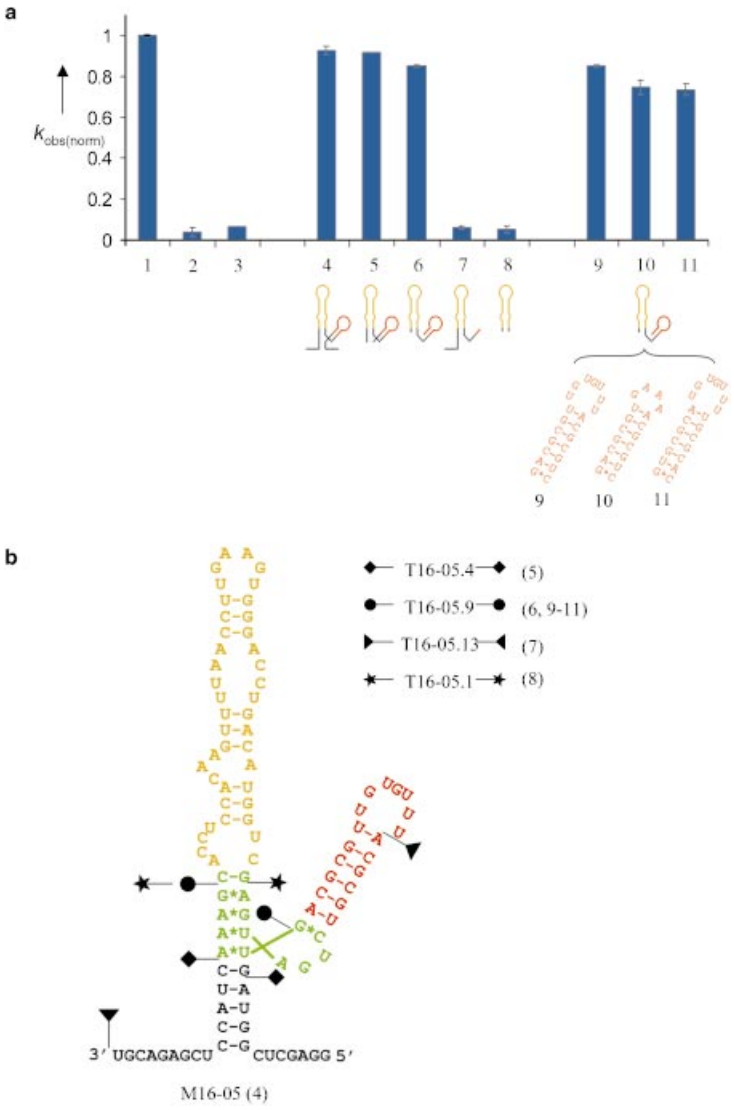

Figure 3. Determination of the minimal sequence of clone D16-05. a) Relative activity $k_{obs(norm)}$ of the cleavage reaction under various conditions (always in the presence of 1 μM tRNA as an unspecific competitor). 1: Cleavage activity of D16-05 (1 nM). 2: Same in the presence of 200 nM **1**. 3: Same as in 2 in the presence of 2.0 μM unselected pool as an unspecific competitor. 4–8: Same as in 2 in the presence of 2.0 μM cleavage inactive point mutant M16-05 (4), construct T16-05.4 (5), construct T16-05.9 (6), construct T16-05.13 (7), construct T16-05.1 (8). 9–11: Same as in 2 in the presence of 2.0 μM T16-05.9 with the depicted variations in helix I. b) Computer-generated secondary structure of the point mutant D16-05. The various marker symbols flank the sequences of the truncated versions tested. To enable T7-transcription two guanosine residues, which are not shown here, were inserted into the 5′-position of the truncated constructs. Orange: The original randomized region; red: constant region from the HHR (helix I); green: catalytic core. The guanosine residue shaded in black shows the point-mutation C–G. The numbers in parentheses refer to the numbering in (a).

In the communication by **D. Zhao and co-workers** in Issue 11, 2001, pp. 2166 – 2168, two relevant references were not cited, for which the authors apologize: a) A. K. Cheetham, G. Férey, T. Loiseau, *Angew. Chem.* **1999**, *111*, 3466 – 3492; *Angew. Chem. Int. Ed.* **1999**, *38*, 3268 – 3292; b) N. Guillou, Q. Gao, M. Nogues, R. E. Morris, M. Hervieu, G. Férey, A. K. Cheetham, *C.R. Acad. Sci. Ser. IIc* **1999**, *2*, 387 – 392.

Table 1 in the communication by **J. T. Groves, S. Shaik, and co-workers** in Issue 15, 2001, pp. 2874 – 2878 contains an incorrect sign. The first energy datum in entry 2 should be $+0.07$ kcal mol^{-1} and not -0.07.

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