ADDITIONS AND CORRECTIONS

THE JOURNAL OF BIOLOGICAL CHEMISTRY VOL. 290, NO. 8, p. 4814, February 20, 2015

VOLUME 284 (2009) PAGES 25620–25629
DOI 10.1074/jbc.A109.000042

Long range communication between exosites 1 and 2 modulates thrombin function.

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The sequences reported for the HD22 and HD23 aptamers were not correct.

1) The aptamer that we called HD23, an inactive variant of HD22 that does not bind thrombin previously named ODN 4a (Dougan, H., Weitz, J. I., Stafford, A. R., Gillespie, K. D., Klement, P., Hobbs, J. B., and Lyster, D. M. (2003) Evaluation of DNA aptamers directed to thrombin as potential thrombus imaging agents. *Nucl. Med. Biol.* **30,** 61–72), was reported with a sequence that was missing three nucleotides. We are now calling this aptamer SV23, as shown below. However, HD23 was used in this study.

AGTCCGTAATAAAGCAGGTTAAAATGACT (HD23)

AGTCCG - - -TAAAGCAGGTTAAAATGACT (SV23)

2) Instead of using the aptamer HD22 that was previously named (60–18[29]) and was reported to bind thrombin (Tasset, D. M., Kubik, M. F., and Steiner, W. (1997) Oligonucleotide inhibitors of human thrombin that bind distinct epitopes. *J. Mol. Biol.* **272,** 688–698), we used an aptamer in which the TGG triplet at position 7–9 was duplicated. We are now calling this aptamer EV22, as shown below.

AGTCCGTGG - - -TAGGGCAGGTTGGGGTGACT (HD22)

AGTCCGTGG**TGG**TAGGGCAGGTTGGGGTGACT (EV22)

To determine how this error may have affected our results, we used surface plasmon resonance to compare the thrombin binding affinity of EV22 with that of HD22. Biotin-labeled aptamers were adsorbed onto separate streptavidin-modified flow cells and active thrombin was then injected. Analyses of on- and off-rates yielded K_d values of 39 nM and 0.3 nM for EV22 and HD22, respectively. Similar values were obtained when Phe-Pro-Arg-chloromethyl ketone-inhibited thrombin was injected in place of active thrombin. The K_d value of 0.3 nM for thrombin binding to HD22 is consistent with the K_d value of 0.5 nM reported previously for 60–18[29] (Tasset *et al.*). Thus, EV22 exhibited 130-fold weaker thrombin binding affinity than HD22. γ-Thrombin, which lacks exosite 1, bound EV22, whereas R93E thrombin, a variant with an impaired exosite 2, did not bind EV22, so EV22 appears to exhibit the same exosite 2 specificity as HD22. Because EV22 differs from HD22 in affinity but not specificity, we conclude that these errors do not affect the interpretation of the results or the conclusions of this work.