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AUTHOR'S CORRECTION

Role of Iron in Human Serum Resistance of the Clinical and Environmental *Vibrio vulnificus* Genotypes

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Volume 73, no. 23, p. 7501–7505, 2007. Due to an inaccurate description of PCR primers for the amplification of *viuB* in *Vibrio vulnificus* (G. Panicker, M. Vickery, and A. K. Bej, Can. J. Microbiol. **50:**911–922, 2004 [Erratum, **53:**671, 2007]), some strains in our paper were inaccurately designated as *viuB* negative. Upon our discovery of this mistake, published *viuB* and *venB* sequences from *Vibrio vulnificus* strain MO6-24 (GenBank accession no. U32676) were BLAST aligned with chromosomes one and two of the published *V. vulnificus* genomes (YJ016 [GenBank accession no. NC_005139 and NC_005140, respectively] and CMCP6 [accession no. NC_004459 and NC_004460, respectively]). Results showed that *viuB* aligned with 97% nucleotide identity with VVA1303 from YJ016 and VV2_0837 from CMCP6. *venB* aligned with 99% nucleotide identity with VVA1304 from YJ016 and VV2_0838 from CMCP6. The following primers for *viuB* and *venB* were subsequently developed from regions conserved among all three strains: *viuB* primers viuBF2 (5′-CTCGGTCAGACAATATCGTGC-3′) and viuBR2 (5′-CGGCAGTGGACTAATAC GCAGC-3′) and *venB* primers venBF (5′-CTAACCAACATCCGACAGAC-3′) and venBR (5′-CAAGATCAACCTCGTCTG G-3′). Upon PCR screening using these primers, all strains used in this study were found to be positive for both *viuB* and *venB*.

As a result of the misdesignation of isolates as *viuB* negative, the observed differences in serum survival cannot be attributed to differences in the presence of *viuB*. Thus, the third paragraph of Results and Discussion (p. 7503) should be disregarded. Also, as opposed to what was suggested in the concluding sentence of the paper, the apparently ubiquitous nature of *viuB* makes this gene an unlikely marker for screening of clinically important strains. However, the fact that significant differences in serum survival were observed between the C and E types keeps the virulence predictive value of C/E subtyping intact. Thus, we feel that the overall findings and value of our paper remain unchanged. Ultimately, the discovery that all C- and E-type isolates possess *viuB* (and *venB*) and the lack of obvious differences in gene content between the two genotypes highlight the critical need to further investigate genetic regulators that may account for the differing serum survival levels of C and E strains.

We sincerely regret the error that was made.