Supporting Information for

## Effects of storage conditions on the performance

## of an electrochemical aptamer-based sensor

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**Figure S1.** Shown are the properties of the vancomycin-detecting EAB sensor prior to storage. (A) The estimated packing density is 9.5 ( $\pm$  0.8) × 10<sup>9</sup> aptamers/mm<sup>2</sup> across n = 32 sensors, (B) the signal gain is 95 ( $\pm$ 4)%, and the binding midpoint is 17 ( $\pm$ 1)  $\mu$ M. Error bars in panel B reflect 95% confidence intervals derived from four electrode replicates.



**Figure S2.** The drying process itself does not detectably damage EAB sensors. Specifically, we observe no significant change in packing density when we dry EAB sensors and then immediately (within 15 min) return them to buffer for characterization.



**Figure S3.** The use of potential stabilizers, such as 5% (w/v) trehalose or 5% (w/v) bovine serum albumin, does not increase aptamer retention under either wet or dry storage conditions. To see this, here we have normalized the average signal from each set of sensors to its signal prior to storage, yielding "% aptamer retained;" the "best" conditions are thus those that yield aptamer retention of near 100%. The red arrow signifies the "simplest" conditions that achieve effectively perfect aptamer retention. In some of these experiments we included 0.1% (w/v) sodium azide to prevent microbial growth.<sup>1,2</sup>



**Figure S4.** Stored sensors retain their ability to perform well in undiluted whole blood held at 37°° C. Shown are binding curves collected under these conditions from four freshly made sensors (grey) and four sensors that had been stored at -20° for 14 days (red). The curve is a fit to a Hill-Langmuir binding isotherm with  $n_{\rm H} = 0.80 \pm 0.15$ ,  $K_{\rm D} = 125 \pm 70 \,\mu$ M, and Gain = 163 ± 30%.

## References

- (1) Snyder, M. L.; Lichstein, H. C. Sodium Azide as an Inhibiting Substance for Gram-Negative Bacteria. J. Infect. Dis. **1940**, 67 (2), 113–115.
- (2) Kleinhofs, A.; Owais, W. M.; Nilan, R. A. Azide. *Mutat. Res. Genet. Toxicol.* **1978**, *55* (3–4), 165–195. https://doi.org/10.1016/0165-1110(78)90003-9.