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CORRECTION
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Corrigendum: Antibiotic Resistance Mediated by the MacB ABC Transporter Family: A Structural and Functional Perspective

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A Corrigendum on

Antibiotic Resistance Mediated by the MacB ABC Transporter Family: A Structural and Functional Perspective
by Greene, N. P., Kaplan, E., Crow, A., and Koronakis, V. (2018). Front. Microbiol. 9:950. doi: 10.3389/fmicb.2018.00950

In the original article, there was a mistake in **Figure 6** as published. The labels "ATP hydrolysis" and "ATP binding" were inverted; the corrected **Figure 6** appears below. The error does not change the scientific conclusions of the article in any way.

The original article has been updated.

REFERENCES

Crow, A., Greene, N. P., Kaplan, E., and Koronakis, V. (2017). Structure and mechanotransmission mechanism of the MacB ABC transporter superfamily. *Proc. Natl. Acad. Sci. U.S.A.* 114, 12572–12577. doi: 10.1073/pnas.1712153114

Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

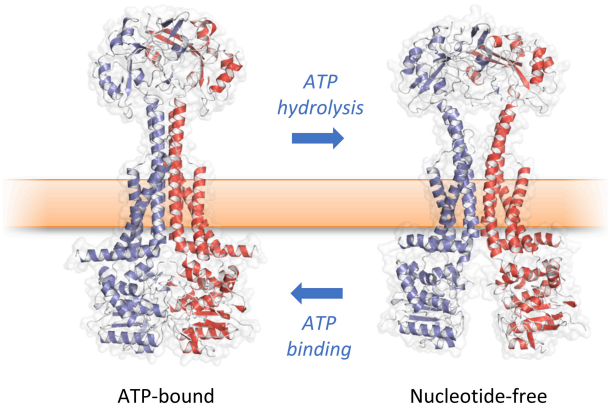

FIGURE 6 | Mechanotransmission mechanism of MacB. ATP binding and hydrolysis cause large, transmembrane conformational changes in MacB structure. Rather than transporting substrates across the inner membrane, MacB-like proteins coordinate reversible dimerization of their NBDs with periplasmic conformational changes. TEP-forming MacB homologs use periplasmic conformational change to drive substrates across the bacterial outer membrane via TolC-like exit ducts. MacB homologs that do not form TEPs are proposed to use similar motions during lipoprotein trafficking and transmembrane signaling. Adapted from Crow et al. (2017).