HHS Public Access

Author manuscript

Cell Host Microbe. Author manuscript; available in PMC 2018 April 25.

Published in final edited form as:

Cell Host Microbe. 2018 April 11; 23(4): 571. doi:10.1016/j.chom.2018.03.005.

Retraction Notice to: The Unfolded Protein Response Element IRE1α Senses Bacterial Proteins Invading the ER to Activate RIG-I and Innate Immune Signaling

Jin A. Cho, **Ann-Hwee Lee**, **Barbara Platzer**, **Benedict C.S. Cross**, **Brooke M. Gardner**, **Heidi De Luca**, **Phi Luong**, **Heather P. Harding**, **Laurie H. Glimcher**, **Peter Walter**, **Edda Fiebiger**, **David Ron**, **Jonathan C. Kagan**, and **Wayne I. Lencer**

Wayne I. Lencer: wayne.lencer@childrens.harvard.edu

In 2013, we reported in *Cell Host & Microbe* that when the AB_5 subunit cholera toxin (CTx) enters the ER of host cells, the toxin's enzymatic A subunit can activate the ER stress sensor IRE1α to induce an inflammatory response by regulated IRE1α-dependent decay of mRNA (RIDD) and subsequent activation of the cytoplasmic viral RNA sensor RIG-I.

Although we can reproduce the findings that CTx and its unfolding and enzymatically inactive mutants can activate IRE1α (as evidenced by XBP-1 splicing), and that all toxins can induce an inflammatory response (as evidenced by transcription of IL-6 and IL-8), we cannot reproduce the qRT-PCR experiments that show that the IL-6 inflammatory response depends on IRE1α activation, and we cannot locate the original qPCR datasets to verify the original results. Therefore, we cannot confirm or stand behind the data published in Figures 3G, 4A, and 4C and the conclusion that IRE1α is the sole mediator for cholera toxin-induced inflammatory signals. This diminishes the evidence for mechanism of action by IRE1α-induced activation of RIG-I.

We wish to correct the literature by retracting the entire paper. We sincerely apologize for this occurrence and any confusion it might have caused.

Correspondence to: Wayne I. Lencer, wayne.lencer@childrens.harvard.edu.