



CORRIGENDUM

Corrigendum to “An Integrated Systems Biology Approach Identifies the Proteasome as A Critical Host Machinery for ZIKV and DENV Replication” [Genomics Proteomics Bioinformatics 19 (1) (2021) 108–122]



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The authors regret that there were errors in Figure 6E published in Issue 1, 2021. The correct **Figure 6** is shown below.

The authors would like to apologize for any inconvenience caused.

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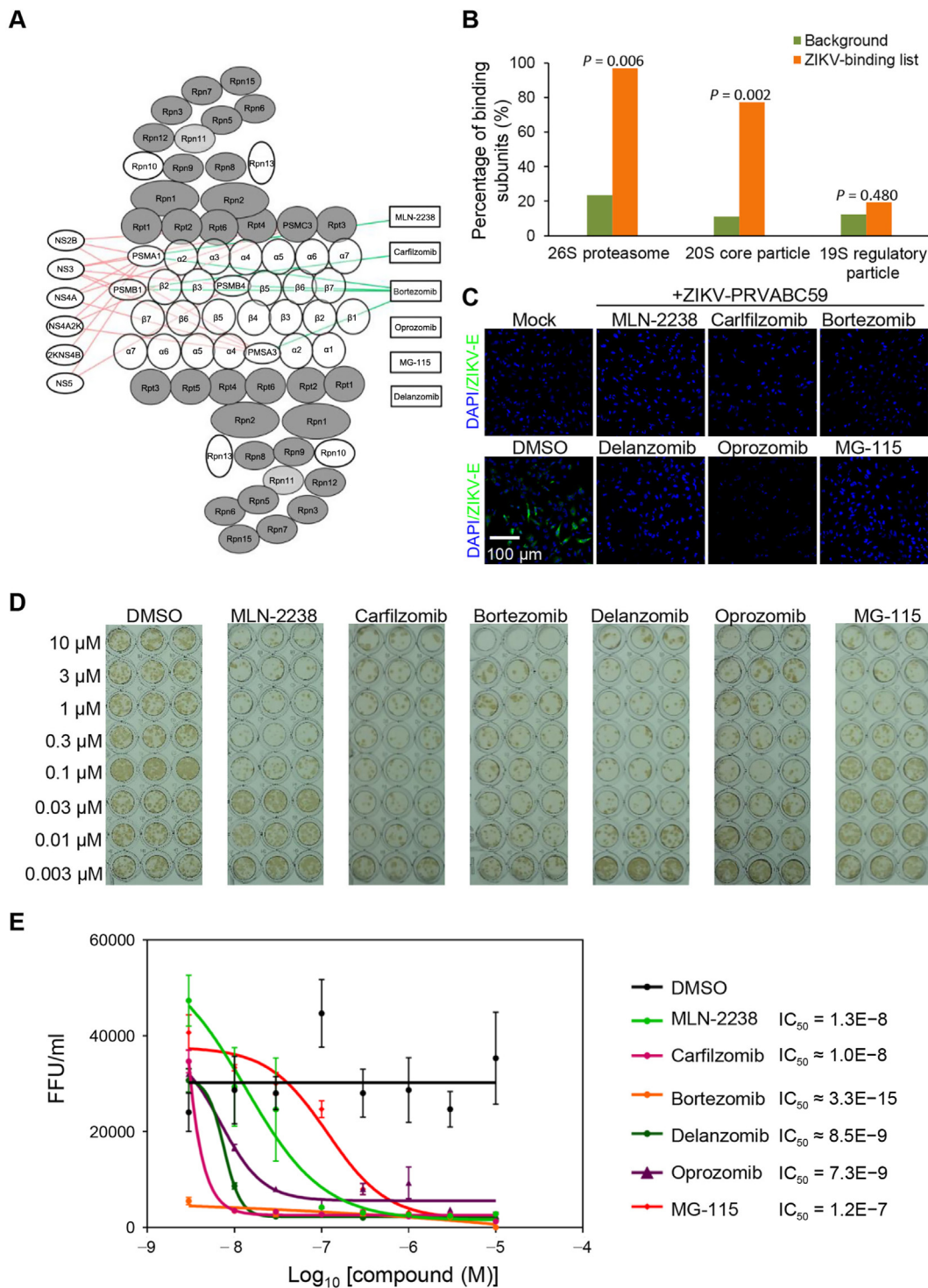


Figure 6 Experimental validation of the proteasome inhibitors

A. PPI network analysis of virus proteins and human proteasome subunits reveals that most of the interacting proteasome subunits are part of the 20S core particle. **B.** Percentage of the ZIKV-binding subunits in 26S proteasome and its two sub-complexes, the 20S core particle and the 19S regulatory particle. **C.** Inhibition of ZIKV expression in human glioblastoma cell line SNB-19 by a panel of proteasome inhibitors. The SNB-19 cells were infected by ZIKV PRVABC59 (MOI = 1) in the presence of 1 μ M of each inhibitor and then incubated for 48 h before the cultures were analyzed for ZIKV-E protein expression by immunostaining. Mock indicates cells without ZIKV infection. Scale bar: 100 μ m. **D.** and **E.** Sample images (**D.**) and quantification (**E.**) of titer assay to assess the potency of the proteasome inhibitors against infectious ZIKV production in SNB-19 cells. All data were normalized to that of 0 μ M for each compound. Dose-dependent antiviral activity is presented as fluorescent focus-forming units per ml (FFU/ml) and data are represented as mean \pm SD ($n = 6$). Curves represent best fits for calculating IC₅₀ values (listed to the right). MOI, multiplicity of infection; ZIKV-E, ZIKV envelope.