Abstract

Chikungunya (CHIKV) is a mosquito-borne virus endemic to tropical and subtropical regions. Problems arise with the current options of CHIKV treatments lacking any antiviral. As an attempt to address this gap, this research identified the stage of CHIKV infectious cycle that STK113, a lapatinib derivative compound, targets. It was hypothesized that STK113 may inhibit epidermal growth factor receptor (EGFR) activity like its parent compound, potentially exerting antiviral activity at early stages of CHIKV's infection. However, attachment inhibition assay revealed that STK113 was most effective as a post-infection treatment. Time-of-addition and time-of-removal assay narrowed down STK113's drug activity to the later phases of the infectious cycle, most likely targeting viral translation. Future studies are recommended to focus on identifying the binding target of STK113 using molecular docking or enzyme activity assays for predicted target proteins like the EGFR and CHIKV nsPs (non-structural proteins).

Keywords: Chikungunya virus (CHIKV), antiviral, lapatinib-derivative, quinazoline, translation inhibitor