were not predictive for a prolonged duration of NV GE in SOT patients. Interestingly, a reduction of the immunosuppression (IS) in SOT patients (dose reduction or drug termination) was associated with a prolonged duration of symptoms (median 47 vs. 14 days; Figure 3, \( P = 0.0007 \)).

**Conclusion.** In this series of patients, SOT was associated with a prolonged duration of NV GE. A reduction of the immunosuppression was associated with a prolonged disease duration in SOT patients. It remains unclear whether this observation is due to a selection bias, or aggravation of symptoms were caused by immune reconstitution with reduced immunosuppressive therapy.

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1899. The Cellular Kinase Inhibitor OSU-03012 Inhibits Enterovirus 71 In Vitro
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**Background.** Enterovirus 71 (EV-71) is a nonenveloped, single-stranded positive-sense RNA virus belonging to genus Enterovirus, family Picornaviridae. EV-71 has caused recurrent outbreaks of hand, foot, and mouth disease especially among children in Asia. Some patients develop severe complications, such as meningitis, encephalitis, poliomyelitis-like paralysis, myocarditis, and pulmonary edema. There are currently limited treatment options for EV-71 infection. OSU-03012 is a celastrol derivative cellular kinase inhibitor with no inhibiting activity on cyclooxygenase that has antiviral activities against a broad-spectrum of viruses, including flaviviruses, filoviruses, and arenaviruses.

**Methods.** Two clinical isolates of EV-71 obtained from patients with laboratory-confirmed EV-71 infections were included in the study. We evaluated the in vitro anti-EV-71 activity of OSU-03012, using virus yield reduction assays (by quantitative reverse transcription-polymerase chain reaction), cell protection assay; and plaque reduction assay in multiple cell lines.

**Results.** OSU-03012 inhibited both EV-71 strains in U251 (neuronal) and RD (rhabdomyosarcoma) cells. The half maximal inhibitory concentration (IC50) of OSU-03012 against EV-71 was consistently ~2 µM in these cell lines in the virus yield reduction assay. At 2 µM of OSU-03012, there was a nearly 2-log reduction in viral load in both U251 and RD cells. There was a dose-dependent increase in the percentage of viable cells after the addition of 0 to 2 µM of OSU-03012 in EV-71-infected U251 and RD cells in the cell protection assay. In the plaque reduction assay, there was >70% reduction in plaque numbers with the addition of 2 µM of OSU-03012.

**Conclusion.** OSU-03012 exhibits anti-EV-71 activity in vitro. The treatment effects of OSU-03012 should be further evaluated in representative animal models of severe EV-71 infection to provide further data for potential clinical evaluation in the future.

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