PRESERVATION POTENTIAL OF LIPID-CONTAINING VIRUSES UNDER SILICIFYING CONDITIONS. J. E. Kyle[1,2,3], L. L. Jahnke[2], and K. M. Stedman[1]. [1]Portland State University, Portland, OR, [2]NASA Ames Research Center, Moffett Field, CA, [3]NASA Astrobiology Institute Postdoctoral Fellow (jennifer.kyle@pdx.edu)

Introduction: Viruses are the most abundant biological entities on Earth. Viruses are present in all environments on Earth where life is found, even the most extreme, at one to two orders of magnitude greater numbers than that of prokaryotes [1]. It is probable that all life on Earth has at least one associated virus [2]. Viruses can be removed from solutions by precipitatating minerals [3,4,5]. Their small size allows them to be strongly bound to mineral surfaces, concentrating them within sediments, and potentially enalbing them to exist for long periods of time in the absence of a suitable host. In addition, viral lipids can be distinguished from that of their host, which creates the possibility of unique viral signatures. Recent analyses have identified unique lipids within ocean algal viruses as potential biomarkers for virus infections [6]. In order to assess potential virus lipid biosignatures in the rock record, the preservation potential of lipid-containing viruses under silica saturated conditions was investigated by analyzing virus infectivity and lipid composition for up to 6 months.

Methods: Two spherical lipid-containing viruses, PRD1 and PBCV1, were used in this study. PRD1 is 65 nm in diameter, contains a 4 nm thick internal lipid membrane [7], and infects enterobacteria, including *Salmonella typhimurim* LT2. PBCV1 is 190 nm in diameter, contains a 40nm thick internal lipid membrane [8,9], and infects the algae *Chlorella* NC64A. Both viruses are lytic, killing their host upon release of the viruses from the cells. Large amounts of virus, from ~40L and 15L, respectively, was produced and purified in order to determine if and how the virus lipids degrade over time. 400 ppm silica solution (final concentration) was added to the respective viruse stocks and was sampled over the course of 6 months. Control microcosms of virus without silica, host only, and host plus virus in the presence and absence of silica were also sampled. Samples were collected immediately after silica addition, after 1 month, 3 months, and 6 months (PRD1 only). Virus infectivity, dissolved silica concentration, and pH were determined for each sample. Additional samples were collected for lipids analysis where they underwent a total lipid extraction, thin layer chromatography, and analysis on a gas chromatography-mass spectrometer (GC-MS).

Results: The role of silica in virus lipid preservation is still under investigation. GC-MS analysis of PRD1 and PBCV1 samples after 1 month of incubation does not reveal differences in the presence or absence of silica. Interestingly, month old PBCV1 only samples contained a fatty acid within the glycerophospholipids that was dominant within the virus but absent within the host cells. In addition, microcosms with PBCV1 particles only and PBCV1 plus host contained large amounts of an unknown lipid that was only present in small amounts in the host culture. PRD1 phospholipids, as described elsewhere [7], were the same as the host but present in different relative concentrations.

PRD1 infectivity was greatly affected by silica addition decreasing by 3 orders of magnitude within one month, whereas PBCV1 infectivity only decreased by approximately a third. Over the course of 6 months, PRD1 infectivity increased slightly. In The PRD1 plus host microcosms, the presence of silica only influenced infectivity within the first month. After an additional 5 months, the infectivty in both systems (plus and minus silica) decreased by the same order of magnitude.

The concentration of dissolved silica immediately dropped by half upon addition of silica into the respective micocosms, and then remained stable for the remainder of the experiment. The only exception was PRD1 plus silica, where silica precipitation was immediate and complete. The pH for all systems remained stable except where host and viruses were mixed resulting in a decrease in pH by at least two orders of magnitude.

Conclusions: Investigations on the influence of silica on the preservation potential of viruses are ongoing. The presence and precipitation of silica has a greater influcence on PRD1 than PBCV1, which could be due to the smaller size of PRD1 particles. PRD1 particles, however, seem to regain infectivity over time despite dissolved silica concentrations remining close to zero. Lastly, but most importantly, the production of unique lipids, and fatty acids within the glycerophospholipids within the PBCV1 particles creates a potential environmental virus biosignature.

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