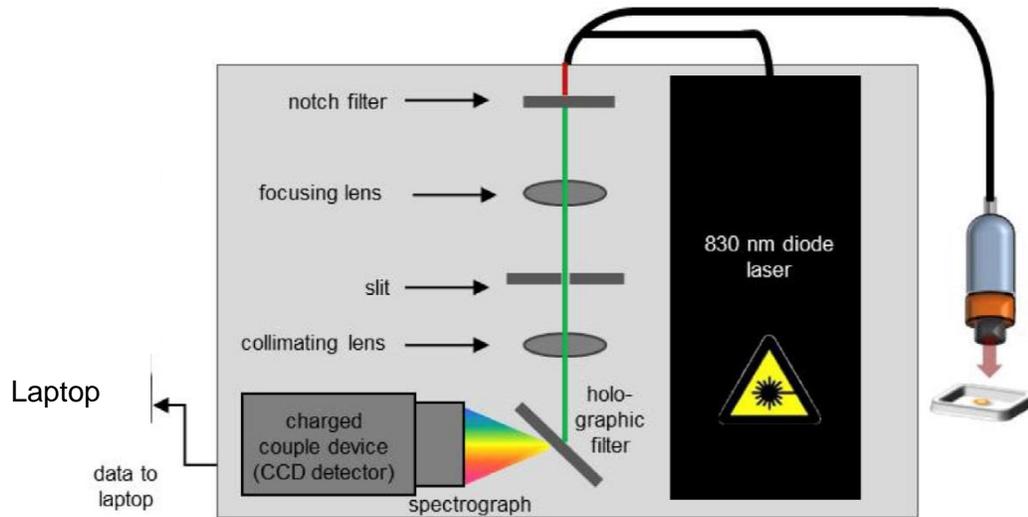


**Supplemental Figure 1:** Computed tomographic examination demonstrating a pancreatic pseudocyst at the end of the arrow adjacent to the stomach.

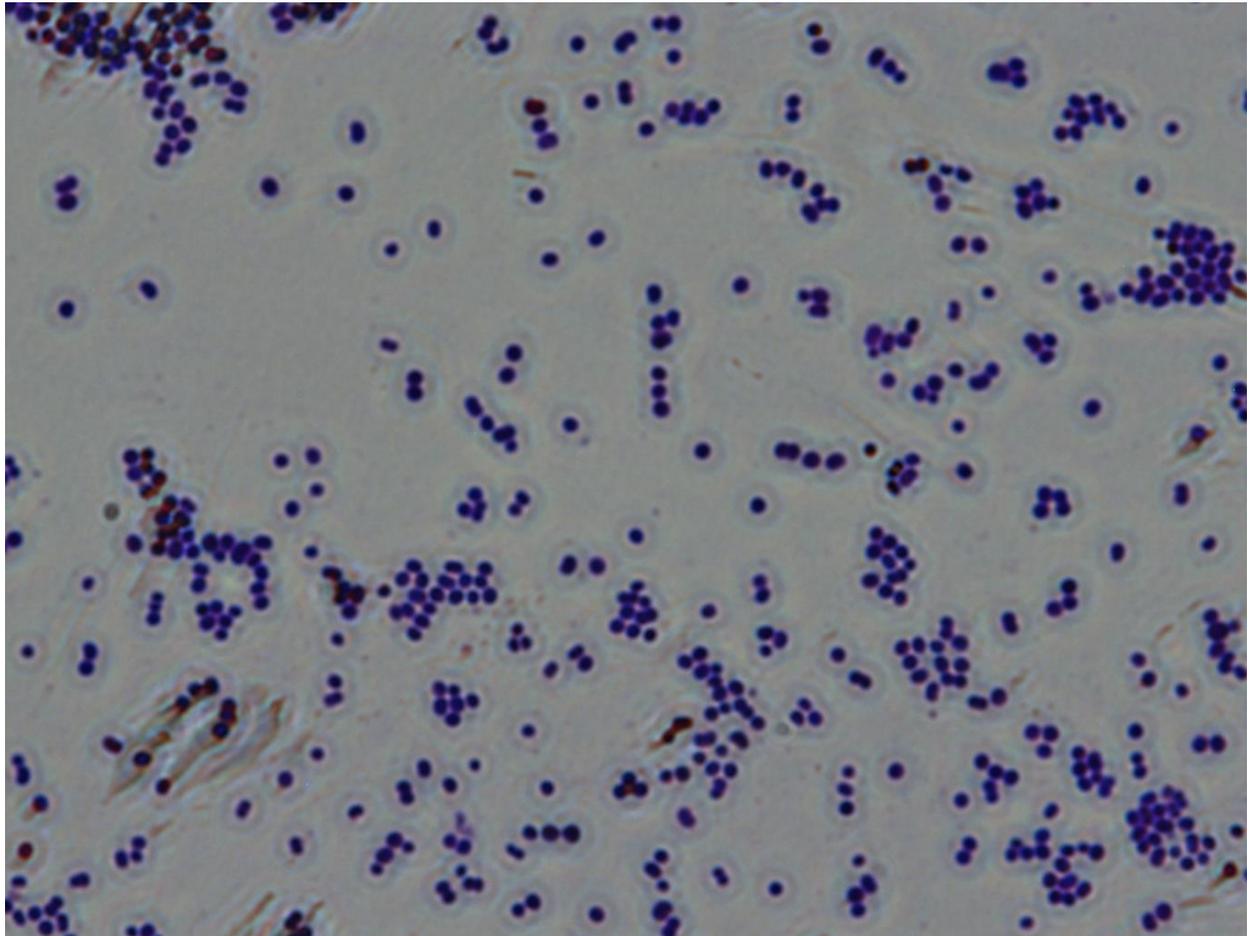


**Supplemental Figure 2: Schematic Diagram of near-infrared excitation Raman spectrometer with handheld fiberoptic probe.** Excitation wavelength light (830 nm) is focused on the sample using focusing lens with 1 mm illumination spot size. Collection fibers in the probe send scattered light back to the instrument. Excitation light is filtered off using a notch filter. Light is ultimately diffracted by a holographic filter to separate the wavelengths of Raman shifted light which are measured in separate pixel channels of a CCD camera.

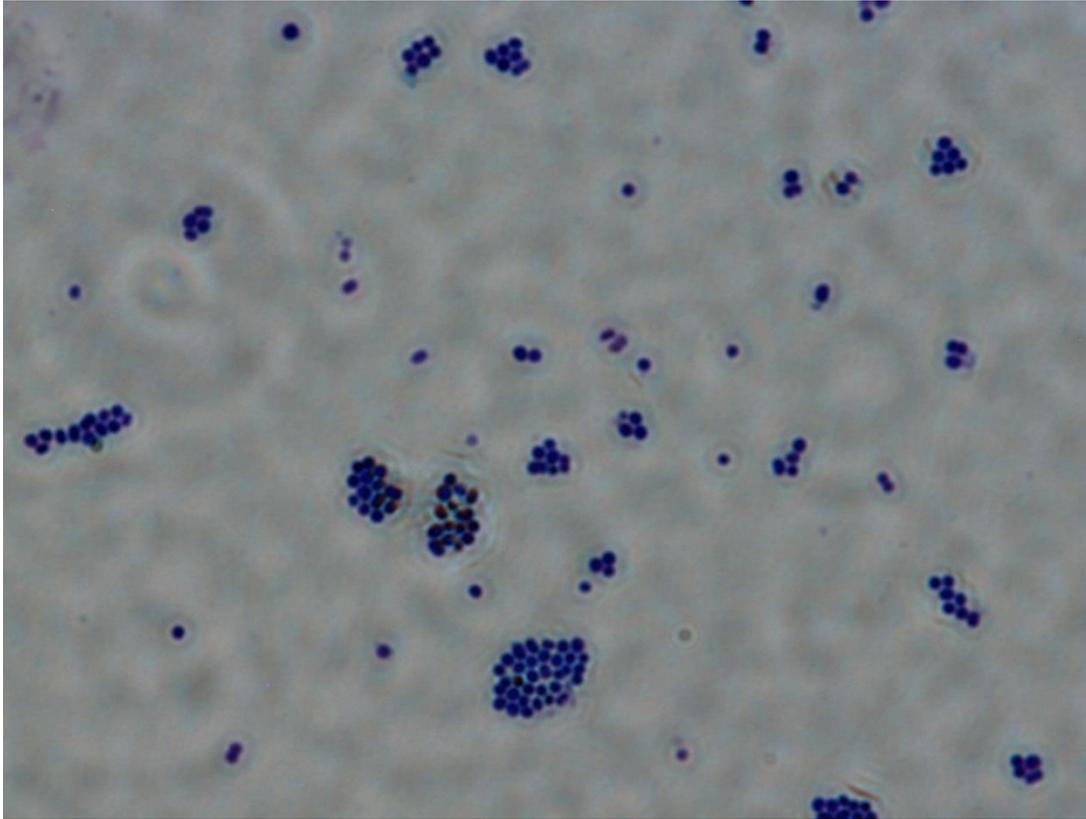
**Supplemental Figure 2:**



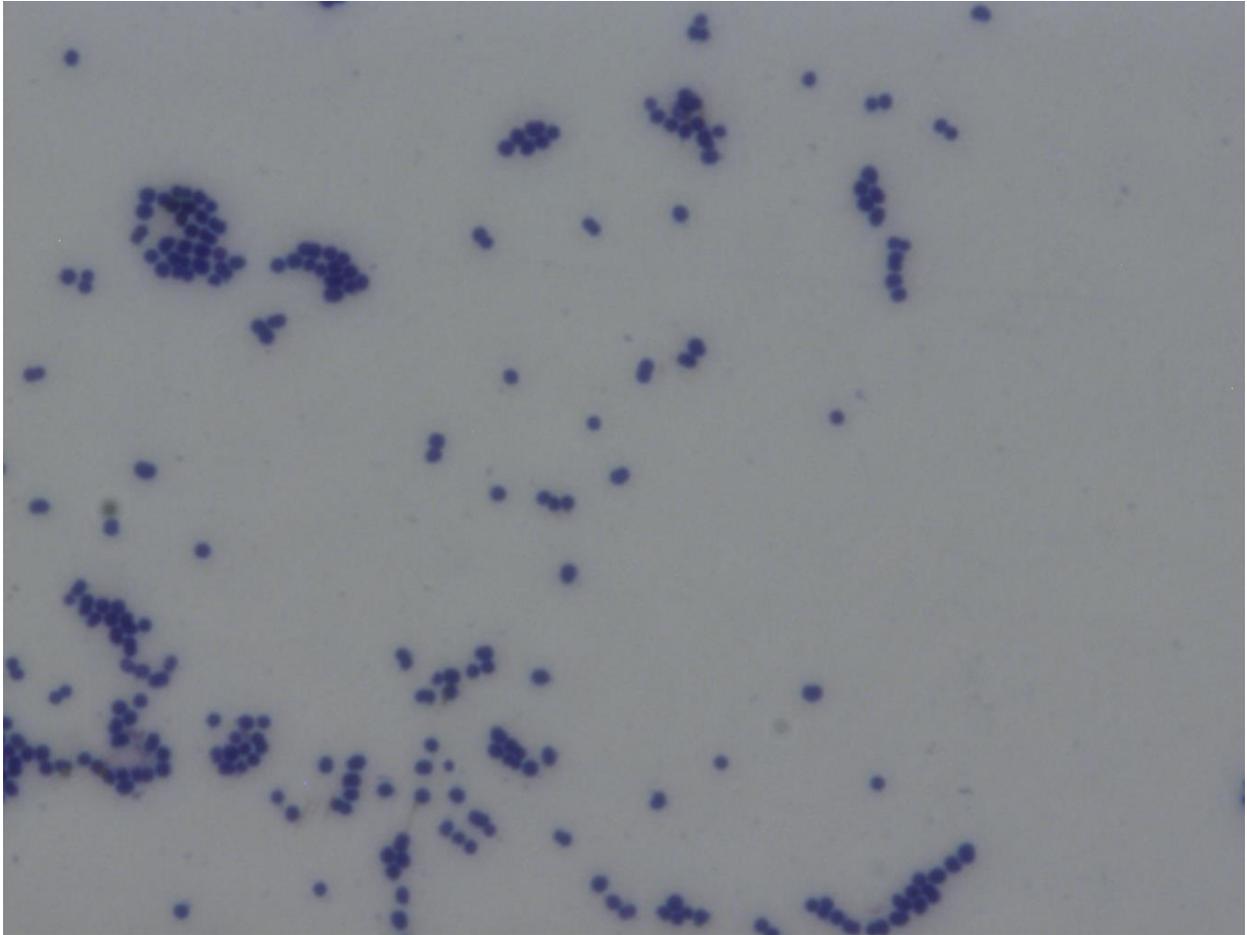
**Supplemental Figure 3: Full image showing capsule staining of isolate TP1 (entire field of view).**



**Supplemental Figure 4: Full image showing capsule staining of isolate TP2 (entire field of view).**



**Supplemental Figure 5: Full image showing capsule staining of isolate TP3 (entire field of view).**



**Supplemental data.**

**Inactivation of phage by patient plasma.**

We performed an experiment with plasma collected 90 days following cessation of phage therapy. In this experiment, patient serum was diluted to a concentration of 1:100 in phosphate buffered saline (PBS). Phage were tittered to a concentration of  $10^7$  pfu/mL in PBS. 100  $\mu$ l of the phage preparation was mixed with 900  $\mu$ l of the serum preparation at 37<sup>0</sup> C. At specified intervals 100  $\mu$ l aliquots of the incubation mixture was removed and diluted 100-fold to temper further neutralizing activity at the tested ratio. 100  $\mu$ l of this diluted mixture was then plated at serial dilutions on an agar lawn of the bacterium and the reduction in phage titer in the presence of patient plasma was compared to a parallel experiment in which the initial incubation of the phage preparation was with PBS. The velocity constant (V) for phage inactivation was calculated by determining the rate of phage inactivation at each time interval from time zero using the following equations:

$$-dp/dt = Kp/D$$

and

$$K = 2.3 D/t \times \log p_0/p$$

Serum 8/19/2016 Time Course Phage sample	0	60	120	180	1440
Ab $\phi$ 1 – PBS (control)	270	138	179	132	178
Ab $\phi$ 1 - Serum	273	63	5	0	0
Ab $\phi$ 1 -K value	#DIV/0!	19.93586123	12.07697416	#DIV/0!	#DIV/0!
Ab $\phi$ 4 – PBS (control)	287	205	235	153	198
Ab $\phi$ 4 - Serum	263	133	80	20	0
Ab $\phi$ 4- K value	#DIV/0!	18.69190204	9.769077525	7.282017228	#DIV/0!
Ab $\phi$ 71 – PBS (control)	128	90	127	126	104
Ab $\phi$ 71 - Serum	172	86	60	21	0
Ab $\phi$ 71 - K value	#DIV/0!	19.41775594	10.00854344	7.254942012	#DIV/0!
Ab $\phi$ 97 – PBS (control)	21	12	4	3	5
Ab $\phi$ 97 - Serum	21	4	0	0	0
Ab $\phi$ 97 - K value	#DIV/0!	24.5254367	#DIV/0!	#DIV/0!	#DIV/0!
AbTP3 $\phi$ 1 – PBS (control)	54	38	24	22	14
AbTP3 $\phi$ 1 - Serum	43	49	17	12	3
AbTP3 $\phi$ 1 - K value	#DIV/0!	20.35424836	11.05830623	7.56549063	1.041848688

As demonstrated in the table, there was a more rapid decay of phage activity of each of the Navy-derived phages used in his therapy over time in the presence of patient serum than in the presence of PBS. It is possible this is, at least in part, antibody mediated

but we cannot be certain since a parallel experiment with antibody-depleted plasma was not conducted. If this increased reduction in phage activity in the presence of plasma represents antibody-mediated neutralization these (or other) antibodies could also be playing an opsonizing role *in vivo* and contributing to the decay we observed in peripheral blood phage activity by additional mechanisms.