

Supplemental File

Autophagy promotes non-lytic release of progeny rhinovirus in microvesicles from mucociliary differentiated airway epithelial cells

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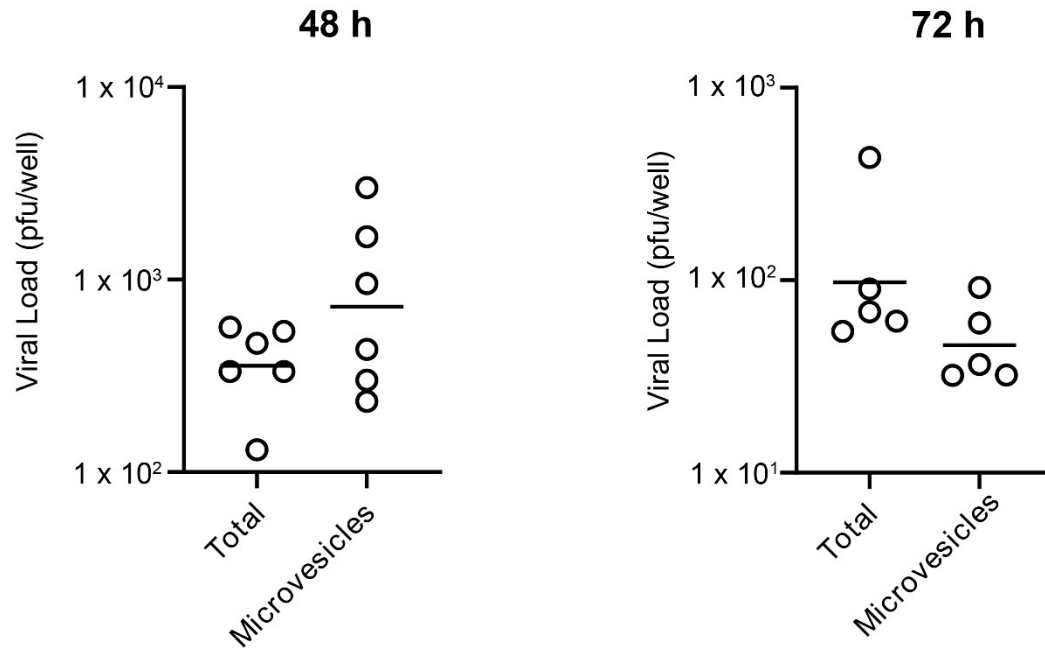
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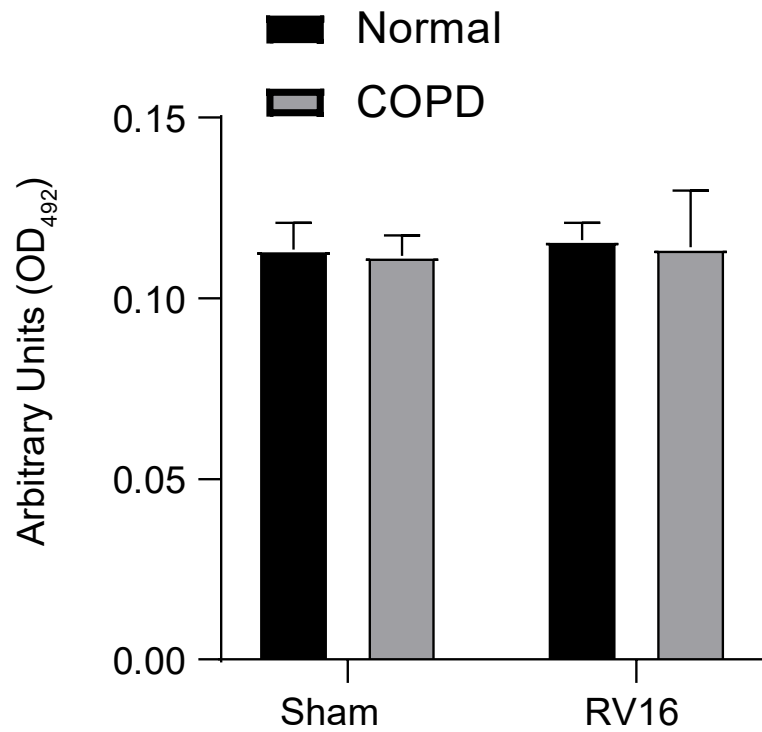
Supplemental Table 1. Characteristics of patients with COPD and healthy non-smokers

Donor	Age (Yrs)	Sex	Smoking History (pack-years)	FEV1 (% predicted)
COPD				
7	58	<i>Female</i>	70	25
10	49	<i>Female</i>	136	13
13	45	<i>Female</i>	31.25	42
1190	68	Male	84	42
1209	79	Male	50	34
1236	59	Male	30	18
Healthy non-smokers				
53	54	<i>Male</i>		
31	60	<i>Female</i>		
396	46	Male		
1201	70	Male		
1220	67	Female		
1259	77	Male		

Airway basal cells obtained from University Michigan are in italics. Rest of them are from Temple Lung Center, Department of Thoracic Medicine and Surgery, Temple University, Philadelphia



Supplemental Figure 1. RV virions in the apical wash at 48 and 72 h post-infection Normal mucociliary differentiated airway epithelial cell cultures were infected with RV16 and incubated for 2 h. Apical surface was washed and further incubated for 22, 48 or 72 h. Medium in the basolateral chamber was changed every 24 h and washed with PBS at 24, 48 and 78 h. Total and microvesicle encapsulated RV virions were quantified by plaque assay and expressed as PFU/well. The data represents median with range from 2 donor cells from 2-3 independent experiments.



Supplemental Figure 2. Rhinovirus does not induce cell death. Mucociliary differentiated COPD and normal subjects were infected with sham or RV16 apically and incubated for 2h. The unbound virus was removed and incubated for additional 22 h. Apical surface of the cultures was washed with 0.5 ml PBS twice. The washes were mixed and an aliquot was used to determine the LDH levels. The results represent mean $\pm$ SD calculated from 6 COPD and 6 normal cell cultures.