

## Supplementary Materials for

### **SARS-CoV-2 infection of human iPSC-derived cardiac cells reflects cytopathic features in hearts of patients with COVID-19**

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#### **This PDF file includes:**

##### Materials and Methods

Fig. S1. Expression of SARS-CoV-2 entry factors in iPSC-derived cardiac cells, productive infection assay and drug pretreatment effects.

Fig. S2. Transcriptional disruption and cellular pathway dysregulation due to SARS-CoV-2 exposure in cardiac cells.

Fig. S3. Heat maps for gene pathways of interest, comparing transcriptional differences in mock and SARS-CoV-2-infected cardiomyocytes.

Fig. S4. Cardiomyocyte infection by SARS-CoV-2 leads to dramatic transcriptomic disruption in genes related with the nuclear envelope and sarcomere structures.

Fig. S5. iPSC-CM cultures infected with SARS-CoV-2 display myofibrillar fragmentation.

Fig. S6. Myofibrillar fragmentation phenotype is partially recapitulated by proteasomal inhibition, but not by cardiotoxic drug doxorubicin or infection by coronavirus NL-63 or OC-43.

Fig. S7. Additional representative images from heart autopsy samples from patients with and without COVID-19.

Table S1. List of reagents.

Table S2. List of dyes, primary and secondary antibodies used for immunocytochemistry and paraffin sections.

Table S3. List of RT-qPCR primer and probe sequences.

References (75–84)

**Other Supplementary Material for this manuscript includes the following:**  
(available at [stm.sciencemag.org/cgi/content/full/scitranslmed.abf7872/DC1](http://stm.sciencemag.org/cgi/content/full/scitranslmed.abf7872/DC1))

Data file S1. Individual-level data.