High on the list of the research tools necessary to develop medical interventions to treat severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infections are informative animal models with which to study viral pathogenesis. Gu et al. developed a mouse model in which a SARS-CoV-2 strain was infectious and could cause an inflammatory response and moderate pneumonia. Adaptation of this viral strain in the mouse appeared to be dependent on a critical amino acid change, Asn\textsuperscript{501} to Tyr (N501Y), within the receptor-binding domain of the viral spike protein. The new mouse model was used to study neutralizing antibodies and a vaccine candidate against the virus.

Mutations in the gene \textit{LMNA}, which encodes nuclear envelope proteins, can cause dilated cardiomyopathy associated with arrhythmia and sudden cardiac death. To understand the mechanisms contributing to this disease, Sayed and colleagues studied induced pluripotent stem cell-derived endothelial cells (iPSC-ECs) from a family harboring an \textit{LMNA} mutation. They found downregulation of a protein involved in mechanotransduction, which caused endothelial dysfunction. Lovastatin could induce this protein in iPSC-ECs, improving cardiomyocyte function in coculture and clinical endothelial cell function in two patients treated with the drug. This study demonstrates a workflow for identifying and validating potential drug treatments for patients with cardiolaminopathy.