Stem Cell Based Platform

for Studying **Virus Infection**

Cell Stem Cell

A Human Pluripotent Stem Cell-based Platform to Study SARS-CoV-2 Tropism and Model Virus Infection in Human Cells and Organoids

SARS-CoV-2 has caused the COVID-19 pandemic. There is an urgent need for physiological models to studySARS-CoV-2 infection using human disease-relevant cells. COVID-19 pathophysiology includes respiratoryfailure but involves other organ systems including gut, liver, heart, and pancreas. We present an experimentalplatform comprised of cell and organoid derivatives from human pluripotent stem cells (hPSCs). A Spike-enabled pseudo-entry virus infects pancreatic endocrine cells, liver organoids, cardiomyocytes, and dopa-minergic neurons. Recent clinical studies show a strong association with COVID-19 and diabetes. We findthat human pancreatic beta cells and liver organoids are highly permissive to SARS-CoV-2 infection, furthervalidated using adult primary human islets and adult hepatocyte and cholangiocyte organoids. SARS-CoV-2infection caused striking expression of chemokines, as also seen in primary human COVID-19 pulmonary au-topsy samples. hPSC-derived cells/organoids provide valuable models for understanding the cellular re-sponses of human tissues to SARS-CoV-2 infection and for disease modeling of COVID-19.

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링크 안의 Key Resources table을 확인해 보세요 ! (page. 8)

PLOS ONE

Characterization of zika virus infection of human fetal cardiac mesenchymal stromal cells

To develop a better understanding of potential causes for these pathologies at a cellular level, we characterized ZIKV infection of human fetal cardiac mesenchymal stromal cells (fcMSCs), a cell type that is known to contribute to both embryological development as well as adult cardiac physiology. Total RNA, supernatants, and/or cells were collected at various time points post-infection to evaluate ZIKV replication, cell death, and antiviral responses. We found that ZIKV productively infected fcMSCs with peak (~70%) viral mRNA detected at 48 h. Use of an antibody blocking the AXL receptor decreased ZIKV infection (by ~50%), indicating that the receptor is responsible to a large extent for viral entry into the cell. ZIKV also altered protein expression of several mesenchymal cell markers, which suggests that ZIKV could affect fcMSCs' differentiation process. Gene expression analysis of fcMSCs exposed to ZIKV at 6, 12, and 24 h post-infection revealed up-regulation of genes/pathways associated with interferon-stimulated antiviral responses. Stimulation of TLR3 (using poly I:C) or TLR7 (using Imiquimod) prior to ZIKV infection suppressed viral replication in a dose-dependent manner. Overall, fcMSCs can be a target for ZIKV infection, potentially resulting in CHD during embryological development and/or cardiovascular issues in ZIKV infected adults.

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링크 안의 Material and Method를 확인해 보세요 ! (page. 3)

이 모든 게 가능한 것은 R&D systems의 믿을 수 있는
Growth factor와 Cytokine이 함께 하기 때문입니다.

R&D systems Recombinant Protein

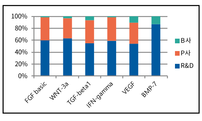
당신의 실험 Quality를 바꿉니다.

믿을수 있는 QC
성능이 보장된 Protein 제공

낮은 Variation
모든 Lot 에서 동일한 결과 제공

높은 Activity
시간의 절약 비용의 절감

다양한 Reference



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