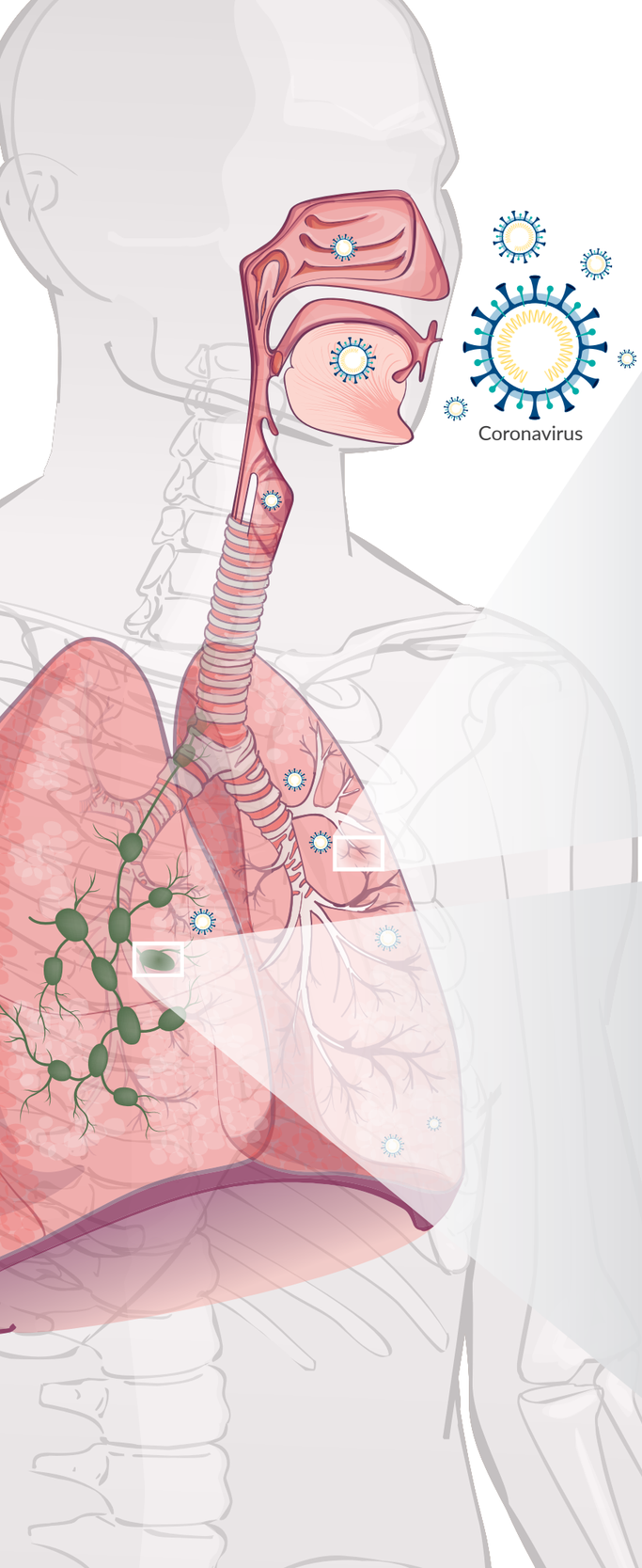
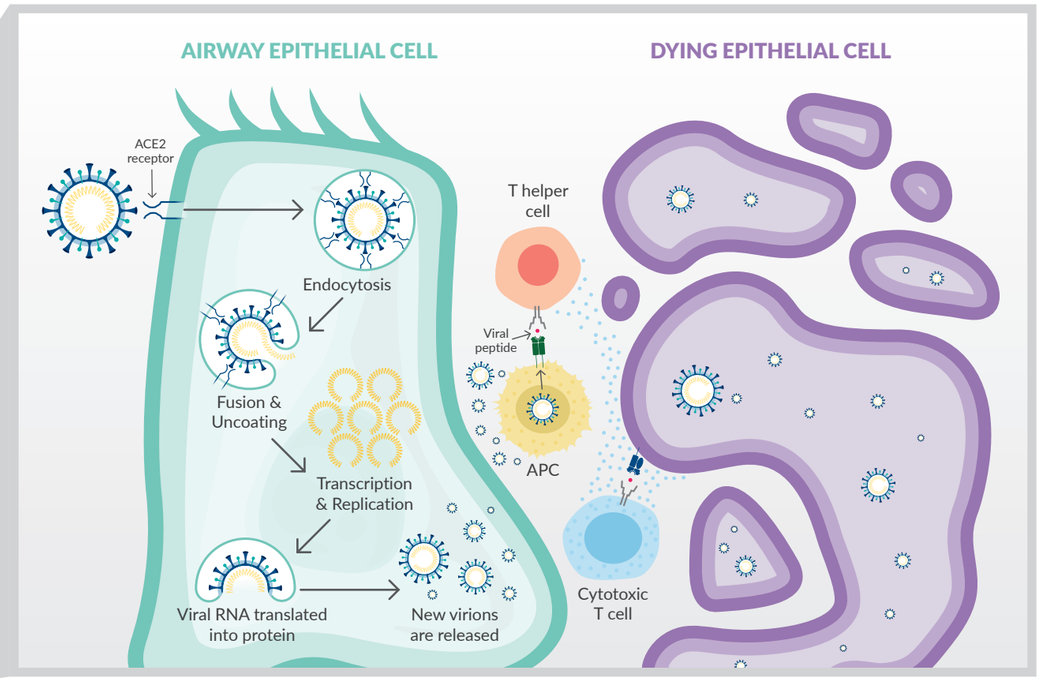
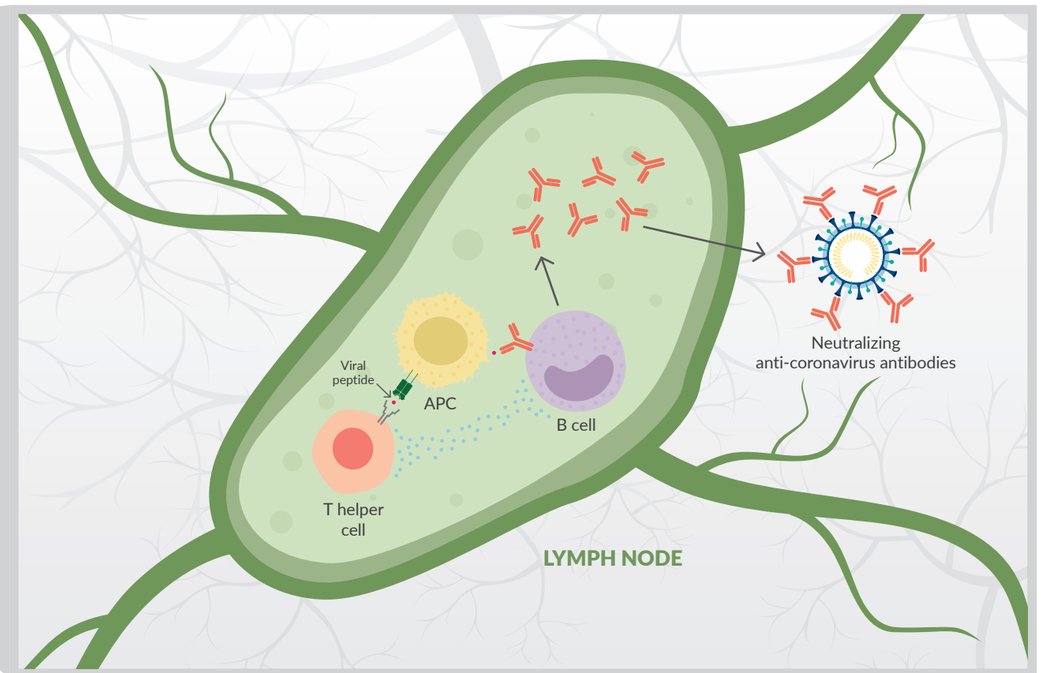
VIRAL INFECTION LIFE CYCLE







VIRAL-HOST FUSION

Infection starts when the virus spike protein binds to the cell surface ACE2 receptors of the host and gains entry into cells through endocytosis or host-membrane fusion.

THERAPEUTIC TARGETS	
Arbidol	Thwarts the hemagglutinin envelope glycoprotein fusion machinery.
Imatinib	Inhibits kinase activation of viral effector proteins that facilitate fusion.
Camostat/E-64d	Prevents spike protein priming by host proteases TMPRSS2 and cathepsin B/L.
Chloroquine	Increases endosomal pH since viruses prefer an acidic environment.

VIRAL REPLICATION

Viral RNA is released, replicated by the viral RNA polymerase, and translated into proteins that are assembled into new virions.

THERAPEUTIC TARGETS	
Lopinavir/Ritonavir	Halts M^{pro} and PL^{pro} activity to prevent the production of replication proteins.
Remdesivir/Favipiravir	Inhibits RNA-dependent RNA polymerase replication of viral RNA.
Itraconazole	Disrupts the role of oxysterol-binding protein in producing replication organelles at the ER and Golgi.
Ivermectin	Inhibits nuclear transport of viral proteins such as integrases, nucleocapsid proteins, and non-structural proteins.
Auranofin	Controls ER stress and the unfolded protein response.

IMMUNE ACTIVATION

Virus-infected cells release immunostimulatory proteins, recruiting innate immune cells to help clear virus and cytotoxic T cells to target infected cells. Local antigen-presenting cells (APCs) ingest the virions and display viral peptides to T helper cells, which coordinate an antibody response from the lymph node.

THERAPEUTIC TARGETS	
STING Agonists	Activate pattern recognition receptors.
Immunosuppressive Agents	Control the circulation of inflammatory cytokines.
Immunopeptidome Profiling	Identifies viral peptides to develop antibodies against.

IMMUNE SURVEILLANCE

Memory B and T cells that recognize the virus will continue to patrol the body. A strong initial response bodes well for longer term immunity to the virus.