EBOLA

THE TROJAN HORSE

With outbreaks in 1976 and 1994 in central Africa, the recent 2014 outbreak has stirred major concerns about the spread of Ebola. Because symptoms present similarly to many other non-lethal viruses in the area, infected persons may remain misdiagnosed until it is too late. The filovirus spreads through contact with bodily fluids, breaks in the skin, or from mother to child. Awareness of personal protection for healthcare workers is paramount to keep the disease under control, as there is currently no vaccine, and no cure.

PHAGOCYTOSIS BY LYMPHOCYTES

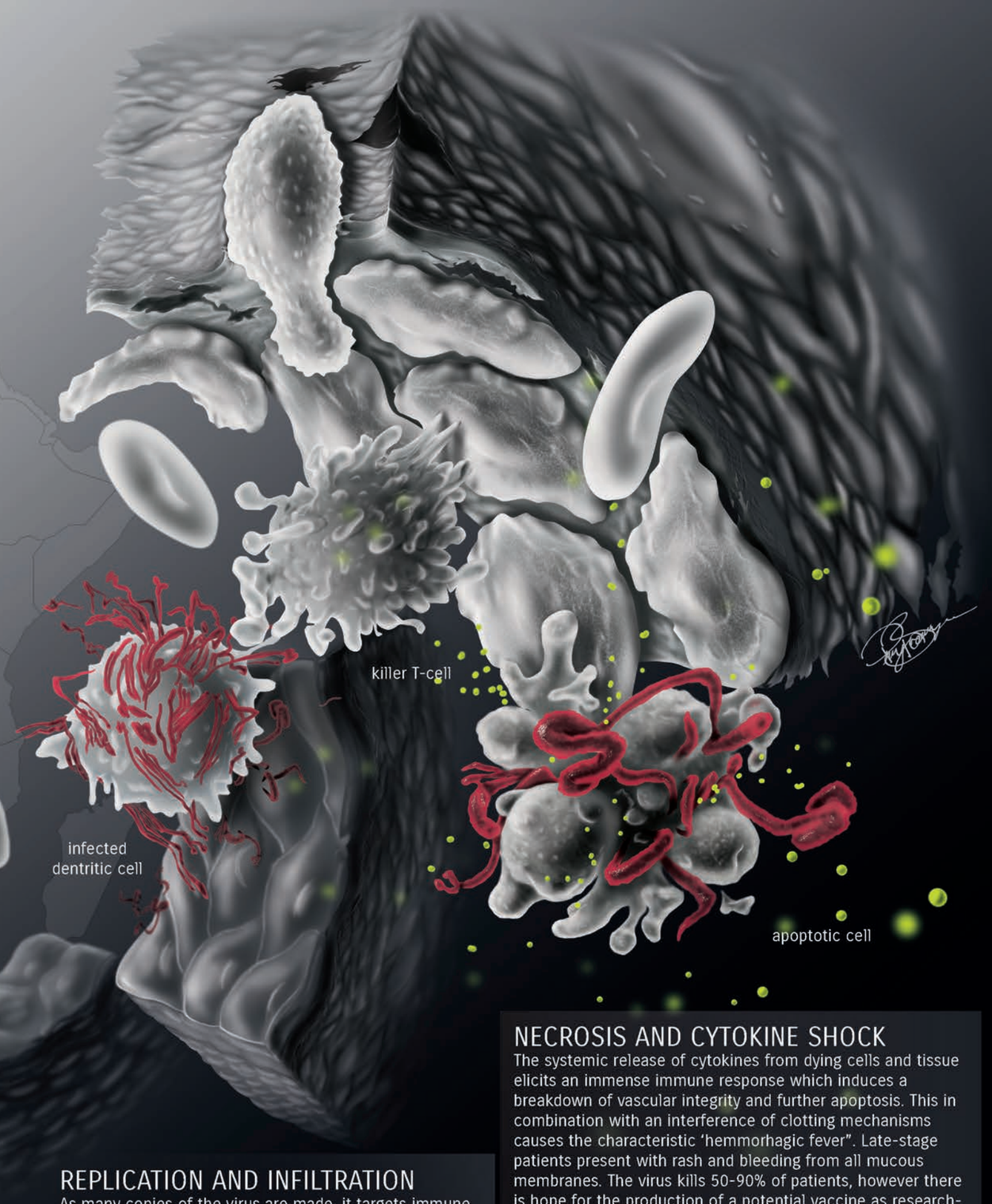
The Ebola virion is phagocytosed by a dendritic cell. It uses specific surface glycoproteins to evade destruction and antigen presentation, thus prevents activation of the adaptive immune system.

ADAPTIVE IMMUNE SYSTEM EVASION

Ebola-infected lymphocytes exhibit 10-50 fold fewer surface proteins than a normal activated cell. This conceptual illustration also shows fewer pseudopodia.

DEPLETION OF LYMPHOYCYTES

Lymphocytes are targeted for viral replication. This leads to rapid loss of lymphocytes, and causes 'bystander apoptosis', where nearby immune cells self destruct.



REPLICATION AND INFILTRATION

As many copies of the virus are made, it targets immune and endothelial cells. Because it has evaded host defenses, it spreads systemically via the vascular system.

NECROSIS AND CYTOKINE SHOCK

The systemic release of cytokines from dying cells and tissue elicits an immense immune response which induces a breakdown of vascular integrity and further apoptosis. This in combination with an interference of clotting mechanisms causes the characteristic 'hemmorhagic fever". Late-stage patients present with rash and bleeding from all mucous membranes. The virus kills 50-90% of patients, however there is hope for the production of a potential vaccine as researchers learn more about this debilitating virus .

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