Role for Protein Deamidation in Viral Immune Evasion

USC Case #2017-042

Market Opportunity:
The United States spends over $500 million on total lifetime care of herpes simplex viral infections (HSV-1 and HSV-2) and complications arising from them. Most current herpes drugs are nucleoside analogs that are only marginally effective in improving lesion-healing time. In addition, they only work if treatment is started within a narrow time window after symptoms begin. Novel treatment approaches like an HSV vaccine can reduce costs and improve patient care, but the $40 billion global vaccine market still awaits a breakthrough in HSV research.

USC Solution:
USC researchers have discovered that herpes viruses evade immune clearance by modifying the cytosolic receptor RIG-I with a viral deamidase. The deamidation-resistant RIG-I maintains the ability to induce immune responses in presence of the herpes virus and stops viral replication. The team has also created a non-replicating mutant virus as a potential vaccine strain. This mutant virus cannot deamidate RIG-I and therefore induces strong immune responses in the host.

Value Proposition
• Reveals a novel molecular pathway involved in immune evasion by herpes viruses
• Provides a target for anti-viral drug therapy
• Novel herpes strain created for vaccine development

Keywords:
Infectious disease, biomarker, innate immune response, RIG-I, protein deamidation, herpes virus immune evasion

Applications
• Viral deamidase provides a target for anti-viral drug discovery
• Novel strain for vaccine development

Stage of Development
• Tested in vitro in human embryonic kidney cell lines
• Available for exclusive and non-exclusive license

Intellectual Property
Status:
Patent pending

Key Publication:
“A Viral Deamidase Targets the Helicase Domain of RIG-I to Block RNA-Induced Activation.” Cell Host Microbe, 2016.

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