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Analysis of Current Clinical Antiviral Treatment Approaches and Medications and Related Suggestions for Future Research

Daniel Sunderland
Colby College

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Introduction

- The term “virus” describes a group of nonliving obligatorily-pathogenic particles that are capable of infecting host cells, taking over their metabolic machinery, and reproducing at the cost of the host’s own energetic and molecular resources.
- The viral replication cycle has six basic steps:
  - adsorption
  - penetration/uncoating
  - viral component replication
  - assembly, and
  - release
- Treatment methods fall into discrete categories, all based on known viral infection and replication methods, to slow the replication and spread of the virus.

Immunomodulation

- Immunomodulatory treatments are promising because they may enhance innate immunity.
- Only interferon is prescribed for viral infection
  - Natural interferons are released by a cell infected by a viral pathogen to warn nearby cells, triggering a series of non-specific antiviral genes. GCSF is generally used to help cancer patients recover from chemotherapy, though it could also be used to increase immunological response, especially in advanced AIDS patients.
  - GCFSF has been shown to cause Sweet’s Syndrome, an autoimmune condition. Further study would be needed to confirm efficacy in a virology setting.
  - Imiquimod is prescribed as a topical cream to treat warts and other skin irritations. It activates (TLR7), acting upstream of interferon in the immune signaling pathway and triggering the release of several cytokines. Its efficacy against viral infections is largely unknown.

Sulfated polysaccharides as immunomodulatory elements

- These compounds have been shown to increase the immune response. Sulfated polysaccharides from Enteromorpha prolifera and the red seaweed Nemalion helminthoides were shown to increase the proliferation of macrophages and stimulated nitric oxide and cytokine production.

Viral Vectors

- The persistence of viral infections, such as dengue fever in the more arid parts of the world, can be directly attributed to non-human vectors in those areas.
- A recent study explored the immune response of the Aedes aegypti mosquito when it has been infected by dengue virus. Two genes silenced in A. aegypti elevated resistance to the virus in the mosquito’s midgut, suggesting that the virus affected the immune response of the host by somehow modulating the expression of those genes.

Resistance

- Interferon modulation
  - Example: A Borna virus nucleoprotein inhibits interferon production by counteracting the TBK1–IRF3 pathway.
- Amantadine and rimantadine
  - Inhibits the viral M2 ion channel needed to uncoat influenza A viruses after host cell penetration.
  - Amantadine is no longer recommended because due to widespread resistance, particularly in all relevant strains of H1N1. Amantadine was highly encouraged as both a prophylactic and a treatment as early as the 1970s.
- Raltegravir was hailed as major progress in the search for new integrase inhibitors in 2005 and has now been approved for use in very young children. Merck & Co. are currently researching MK-2048, a compound they refer to as a “second generation integrase inhibitor,” capable of lasting up to four hours longer than raltegravir.
- Oseltamivir can be seen in the very early stages of this resistance process.
  - A fact sheet produced by Roche in 2006 claimed that resistance was infrequent at that time. As of 2009, the WHO believes that “there is no evidence to indicate the development of widespread antiviral resistance among pandemic H1N1 viruses.”
  - Some viruses have also developed resistance to acyclovir and penciclovir, which act at a very direct level on viral replication mechanisms, after decades of treatment.

Works Cited

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