

# Molecular basis of viral neurotropism: Experimental reovirus infection\*

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Clinical sequelae of human infection by neurotropic viruses have been recognized for nearly 4,000 years. A funeral stela of a NIXth dynasty Egyptian priest showed the effects of prior poliomyelitis.<sup>1</sup> The epitome of classic clinical and pathologic studies of neurotropic viruses can be found in Pasteur's work on rabies.<sup>2</sup> Until recently, it was not possible to explain the fundamental clinical properties of viruses at the level of the viral gene. Nowhere is this difficulty better exemplified than in the problems of neurotropism and the spread of virus within the nervous system.

It has been known for over a century that the striking clinical variability in infection by polio or rabies implies selective infection of discrete neuronal populations. Using experimental reovirus infection, it has been possible to establish the molecular and genetic basis of viral neurotropism.<sup>3,4</sup>

Reoviruses are nonencapsulated viruses 600 to 900 Å in diameter, with icosahedral symmetry.<sup>5</sup> "Reo" is an acronym for respiratory and enteric orphan virus and reflects the original sites of isolation of these viruses as well as the initial lack of associated disease (hence "orphan"). The viral core contains 10 unique segments of double-stranded RNA (dsRNA) that can be subdivided into three classes based on molecular weight. Each dsRNA segment encodes a single strand of messenger RNA that is translated on host ribosomes into a unique single polypeptide. Two of the small dsRNA segments (S1 and S4) and one medium segment (M2) code for structural polypeptides ( $\sigma_1$ ,  $\sigma_2$ , and  $\mu_2$ ), which together form the outer capsid shell.<sup>6,7</sup> The three distinct serotypes (types 1, 2, and 3) of reovirus identified by hemagglutination inhibition and antibody neutralization tests also differ in electrophoretic mobility of the RNA segments and proteins. Stable recombinants containing specific desired gene combinations can be isolated and perpetuated unchanged

in tissue culture.<sup>8,9</sup>

If two serotypes differ in a biologic property, such as the ability to infect a discrete population of nerve cells, it is possible to identify the molecular and genetic basis of the property by studying recombinant viruses of known genetic and structural composition. Neurovirulence has been attributed to the S1 gene<sup>3,4,10</sup>—ie, the type 1 S1 gene segment is responsible for reovirus tropism for ependymal cells, and the type 3 S1 gene segment is responsible for tropism for neurons leading to the necrotizing encephalitis induced by reovirus type 3.<sup>3,4,10</sup>

We wished to extend our prior studies of neurotropism to discrete populations of neurons in the CNS. The spread of reovirus from brain to retina provides an ideal situation to study neurotropism more directly and to examine a related property—ie, spread of virus within the nervous system. The discrete and orderly arrangement of structurally distinct groups of cells in the retina provides an excellent background for investigating both hemagglutination and neural spread of viruses, and allows us to quickly and reproducibly evaluate specific patterns of viral tropism.

In this report, we have found that reovirus type 3 infects a discrete population of neural cells, the retinal ganglion cells, and that this property is a function of a single gene (S1) that encodes the  $\sigma_1$  polypeptide on the viral surface. The differing patterns of viral growth and tropism shown by reovirus type 1 and type 3 suggest that these reovirus serotypes spread by different routes. Type 3 may reach the eye by neural pathways. By contrast, the rapidity of spread and the lack of viral infection of neural cells by type 1 suggest that this type spreads through vascular routes.

**Materials and methods.** *Viruses.* Reovirus type 3 (Dearing) and reovirus type 1 (Lang) were from laboratory stocks.<sup>11</sup> The two types of recombinant vi-

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# Molecular Basis Of Neurology

**VM Jensen**

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## **Molecular Basis Of Neurology:**

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In a fast-paced world fueled by information and interconnectivity, the spellbinding force of linguistics has acquired newfound prominence. Its capacity to evoke emotions, stimulate contemplation, and stimulate metamorphosis is truly astonishing. Within the pages of "**Molecular Basis Of Neurology**," an enthralling opus penned by a very acclaimed wordsmith, readers embark on an immersive expedition to unravel the intricate significance of language and its indelible imprint on our lives. Throughout this assessment, we shall delve into the book's central motifs, appraise its distinctive narrative style, and gauge its overarching influence on the minds of its readers.

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### **Table of Contents Molecular Basis Of Neurology**

1. Understanding the eBook Molecular Basis Of Neurology
  - The Rise of Digital Reading Molecular Basis Of Neurology
  - Advantages of eBooks Over Traditional Books
2. Identifying Molecular Basis Of Neurology
  - Exploring Different Genres
  - Considering Fiction vs. Non-Fiction
  - Determining Your Reading Goals
3. Choosing the Right eBook Platform
  - Popular eBook Platforms
  - Features to Look for in an Molecular Basis Of Neurology
  - User-Friendly Interface
4. Exploring eBook Recommendations from Molecular Basis Of Neurology
  - Personalized Recommendations
  - Molecular Basis Of Neurology User Reviews and Ratings

- Molecular Basis Of Neurology and Bestseller Lists
- 5. Accessing Molecular Basis Of Neurology Free and Paid eBooks
  - Molecular Basis Of Neurology Public Domain eBooks
  - Molecular Basis Of Neurology eBook Subscription Services
  - Molecular Basis Of Neurology Budget-Friendly Options
- 6. Navigating Molecular Basis Of Neurology eBook Formats
  - ePub, PDF, MOBI, and More
  - Molecular Basis Of Neurology Compatibility with Devices
  - Molecular Basis Of Neurology Enhanced eBook Features
- 7. Enhancing Your Reading Experience
  - Adjustable Fonts and Text Sizes of Molecular Basis Of Neurology
  - Highlighting and Note-Taking Molecular Basis Of Neurology
  - Interactive Elements Molecular Basis Of Neurology
- 8. Staying Engaged with Molecular Basis Of Neurology
  - Joining Online Reading Communities
  - Participating in Virtual Book Clubs
  - Following Authors and Publishers Molecular Basis Of Neurology
- 9. Balancing eBooks and Physical Books Molecular Basis Of Neurology
  - Benefits of a Digital Library
  - Creating a Diverse Reading Collection Molecular Basis Of Neurology
- 10. Overcoming Reading Challenges
  - Dealing with Digital Eye Strain
  - Minimizing Distractions
  - Managing Screen Time
- 11. Cultivating a Reading Routine Molecular Basis Of Neurology
  - Setting Reading Goals Molecular Basis Of Neurology
  - Carving Out Dedicated Reading Time
- 12. Sourcing Reliable Information of Molecular Basis Of Neurology
  - Fact-Checking eBook Content of Molecular Basis Of Neurology
  - Distinguishing Credible Sources

13. Promoting Lifelong Learning
  - Utilizing eBooks for Skill Development
  - Exploring Educational eBooks
14. Embracing eBook Trends
  - Integration of Multimedia Elements
  - Interactive and Gamified eBooks

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