

Rabies glycoprotein, an external component of the rabies viral cell wall, inhibits cellular apoptosis produced by infected virion.

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ABSTRACT

The rabies glycoprotein exterior component of the rabies viral cell wall reduces the cellular apoptosis caused by infected virion. The virulence of a virus is inversely correlated with cellular apoptosis. The beta bungarotoxin in krait (*Bungarus Caeruleus*) venom shares a molecular structure with the rabies virus's glycoprotein. The antibodies or immunoglobulin against the krait venom (beta-bungarotoxin) para-specifically inhibit the viral glycoprotein and enhance the death of infected neuron, accompanied by a decrease in cell viability. The virulence of the rabies virus may halt the disease's progression in the brain. A sign of recovery hope. A single-strand RNA virus is the rabies virus. Virions are circular particles with a ribonucleoprotein and nucleocapsid core that are encased in a lipid bilayer. The virus encodes five structural proteins; ribonucleoprotein, matrix protein, and glycoprotein are linked to genomic RNA in nucleoprotein, phosphoprotein, and transcriptase, respectively.

The principal target for antibodies that neutralise substances is the glycoprotein, which protrudes from the outer layer. The viral particle buildup is represented by the Negri bodies. In muscles and nerve endings near the canine bite site, the rabies virus reproduces. The

virus enters the neuromuscular and neurotendinal spindles within 24 hours of the bite; this penetration represents a deep site into the nervous system. In the neuromuscular junction, it binds to the nicotinic acetylcholine receptor [2]. Virus A sign of recovery hope. A single-strand RNA virus is the rabies virus. Virions are circular particles with a ribonucleoprotein and nucleocapsid core that are encased in a lipid bilayer. The virus encodes five structural proteins; ribonucleoprotein, matrix protein, and glycoprotein are linked to genomic RNA in nucleoprotein, phosphoprotein, and transcriptase, respectively.

INTRODUCTION

The most lethal disease is rabies. regardless of advanced intensive care treatment, regardless of a specialised therapy regimen that includes ketamine, interferon alpha, ribavirin, immunoglobulin, monoclonal antibodies, the rabies vaccine, and other medications [1]. Rabies is still one of the most serious and dreaded hazards to public health in the twenty-first century. Worldwide, rabies probably causes 75000 human fatalities each year. Our knowledge and comprehension of the causes of rabies in people are lacking. However, a recent study on the viral structure, including its glycoprotein and the corresponding affected human receptors, revealed that pharmacological agonists and antagonists can be used to overcome the negative effect of the virus on various nervous system receptors, offering hope to alleviate the suffering, extend survival, and give us a better quality of life. A sign of recovery hope. A single-strand RNA virus is the rabies virus. Virions are circular particles with a ribonucleoprotein and nucleocapsid core that are encased in a lipid bilayer. The virus encodes five structural proteins; ribonucleoprotein, matrix protein, and glycoprotein are linked to genomic RNA in nucleoprotein, phosphoprotein, and transcriptase, respectively.

The principal target for antibodies that neutralise substances is the glycoprotein, which protrudes from the outer layer. The viral particle buildup is represented by the Negri bodies. In muscles and nerve endings near the canine bite site, the rabies virus reproduces. The virus enters the neuromuscular and neurotendinal spindles within 24 hours of the bite; this penetration represents a deep

site into the nervous system. In the neuromuscular junction, it binds to the nicotinic acetylcholine receptor [2]. Virus The afferent sensory nerves travel down from the infected brain to the salivary glands, lacrimal glands, and highly innervated tissues, including the heart. Apoptosis occurs after neuronal malfunction as a result of the virus. The brain stem, thalamus, basal ganglia, and spinal cord are where the virus concentrates. Because the pharyngeal and laryngeal (bulbar) spasms have been misinterpreted as a fear of drinking water, hydrophobia has been listed as a symptom. Aerophobia has also been mistakenly labelled as a comparable reaction to air blowing over the face. Important sequelae of autonomic instability include diabetes insipidus, spontaneous ejaculation, excessive sweating, excessive salivation, piloerection, priapism, sinus tachycardia, respiratory failure, tachycardia, heart failure, arrhythmias, coronary sinus rhythm, and hypertension or hypotension. when the virus is introduced the immune system, and as a result, antibodies cannot be used to cleanse the central nervous system. There is strong evidence that the glycoprotein serves as the unique attachment component of the enveloped virus, and that several negative stranded viruses become non-infectious when the virus spike glycoprotein is removed enzymatically or genetically [3]. It has been determined which glycoprotein helps the virus cling to the cell surface. The rabies virus can be rendered non-pathogenic by substituting the amino acid arginine at position 333 of the glycoprotein molecule [4]. The virus enters the spinal cord from the neuromuscular and neurotendinal spindles by retrograde transport within the axon because rupture of the axon or inhibition of axoplasmic flow prevents the virus from spreading centripetally. The virus is spread in the central nervous system by direct transmission from Synaptic connections between neurons. Specialized surface regions with an abundance of AChR are infected by the rabies virus.

At the tip of the junctional folds of the neuromuscular junction, AChR is found in the highest density. By concentrating the virus at the postsynaptic locations close to the presynaptic axon terminals, binding to AChR at central synapses may also be responsible for the transfer and spread of virus from neuron to neuron.

Discussion

With the appearance of rabies symptoms, quinucidinyl benzylate's binding, an antagonist of the muscarinic AChR, is significantly reduced in the rat brain. When cultivated myotubes were exposed to the rabies virus, the antigen distributed on the

cell surface in patches in a manner resembling that seen after staining with alpha-bungarotoxin that was rhodamine-labeled. An isolated polypeptide from elapid snake venom called alpha bungarotoxin binds exclusively and almost irreversibly to the nicotinic AChR [5]. The amount of myotubes that became infected was significantly decreased by pretreating them with alpha-bungarotoxin and another AChR ligand, d-tubocurarine. Both of these ligands attach to the AChR's acetyl choline binding site, which is located in the 40000 dalton alpha subunit.

Recently, there has been suggestion that the NMDA receptor be a receptor for the rabies virus [6]. Virus receptor identification has Practical importance in that it establishes a foundation for preventing infection by preventing the attachment phase. This may be helpful in the case of the rabies virus because the human defence mechanism cannot stop the disease.

Delaying the infection may give the body more time to respond normally to immunisations, whether active or passive. Potentiated immune response has the potential to cause harm in rabies.

With rabies, brain death may occur together with preserved cerebral blood flow. The extract from *Datura* seeds (atropine), according to an Indian Vaidya (a physician who is knowledgeable about Ayurveda), increases the life of rabies-stricken animals [8, 9]. Atropine may thereby reduce bronchial and salivary production. It has been proposed that the N-methyl-D-aspartate subtype R1 and GABA neurotransmitter receptors in the central nervous system are potential rabies receptors.

Magnesium sulphate inhibits ligand-gated calcium channels, which reduces presynaptic terminal acetyl choline release and CNS overstimulation brought on by NMDA receptor activation. Moreover, this stops the spread of the infection [1,9]. The NMDA receptor, a neuroexcitator and virus carrier, is inhibited by it.

The CNS inhibition is accelerated by zolpidem, a GABA agonist.

REFERENCES

1. Warrell MJ, Warrell DA. Rabies and other lyssavirus disease. *Lancet*. 2004;363(9413):959-69.
2. Ientz TL, Burrage TG, Smith AL, Crick J, Tignor GH. Is the acetyl choline receptor a rabies virus receptor? *Science*. 1982;215(4529):182-4.
3. Riffkin DB, Compans RW. Identification of the spike proteins of a Rous sarcoma virus. *Virology*. 1971;46(2):485-9.

4. Dietzschold B, Wunner WH, Wiktor TJ, Lopes AD, Lafon M, Smith CL, et al. Characterization of an antigenic determinant of the glycoprotein that correlates with pathogenicity of rabies Virus . Proc Natl Acad Sci U S A. 1983;80(1):70-4.
5. Lee CY. Chemistry and pharmacology of polypeptide toxins in snake venoms. Annu Rev Pharmacol. 1972;12:265-86.
6. Gosztanyi G, Ludwig H. Interaction of virus proteins with neurotransmitter receptors may protect or destroy neurons. Curr Top Microbiol Immunol. 2001;253:121-44.
7. Jackson AC. Rabies in the critical care unit: diagnostic and therapeutic approaches. Can J Neurol Sci. 2011;38(5):689-95.
8. Shastri AD, Mehata PM. Sushrutsahita. Kashi Sanskrit Granth Mala. 156:46-63.
9. Hemachudha T, Lasothamatas J, Rupprecht CE. Human rabies: a disease of complex neuropathogenetic mechanisms and diagnostic challenges. Lancet Neurol. 2002;1(2):101-9.
10. Dietzschold B, Morimoto K, Hooper DC. Mechanism of virus induced neuronal damage and the clearance of viruses from CNS. Curr Top Microbiol Immunol. 2001;253:145-55.