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Editorial

DRUG MECHANISM OF TECOVIRIMAT

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EDITORIAL

Orthopoxvirus VP37 Envelope Wrapping Protein Inhibitor Tecovirimat. Tecovirimat works by inducing cytochrome P450 3A, inhibiting cytochrome P450 2C8, cytochrome P450 2C19, and inhibiting the breast cancer resistance protein. In 1980, the World Health Organization pronounced smallpox, a viral disease that can be contagious and occasionally lethal, to be extinct. But there have long been worries that smallpox could be turned into a bioweapon. Tecovirimat, also known as ST-246, was the first medication for smallpox to be licensed. For the treatment of smallpox, monkeypox, orthopoxvirus, and orthopoxviral disease, tecovirimat has been investigated. On July 13, 2018, the U.S. Food and Drug Administration approved the use of tecovirimat (marketed under the brand name TPOXX) to treat smallpox.

Health Canada approved the use of tecovirimat in December 2021. The FDA approved Tecovirimat in July 2018 as the first medication ever authorized to treat smallpox. Later, in

December 2021, Health Canada approved Tecovirimat, and in January 2022, the European Commission approved it as well. In addition to treating smallpox, tecovirimat is also prescribed to treat adult and pediatric cases of monkey pox, cowpox, and problems caused by the replication of the vaccinia virus after smallpox immunization. There are oral and intravenous versions of tecovirimat.

Tecovirimat is prescribed for the treatment of human smallpox sickness in adults and children weighing at least 3 kg. It is an inhibitor of the orthopoxvirus VP37 envelope wrapping protein. Patients with immunodeficiencies may experience a decrease in tecovirimat's effectiveness. After receiving a smallpox vaccination, it is also recommended in Europe to treat issues brought on by the vaccinia virus replicating. Successful viral replication causes a variety of infectious virion types to develop. Although they are contagious, mature viruses stay inside cells until cell lysis. On the other hand, mature viruses wrap with late endosomal membranes to form enveloped virion shape.

The orthopoxvirus P37 protein mediates the development of a wrapping complex for enveloped virions. These egress-competent encapsulated virions are necessary for the long-distance transmission of the virus within the host and are expelled from the cell in a nonlytic manner. All members of the orthopox virus genus share a highly conserved gene that produces the P37 protein. P37 interacts with TIP47 and the Rab9 GTPase, which are both found in transport vesicles produced from late endosomes. The virus-specific wrapping complex for enveloped virions is created as a result of interaction between TIP47, P37, and Rab9 GTPase. The P37 inhibitor tecovirimat prevents P37 from interacting with Rab9 and TIP47, hence inhibiting the assembly of the wrapping complex [1-3].

Tecovirimat is easily absorbed after being administered orally. When taken with food, tecovirimat has a higher oral bioavailability. When tecovirimat was taken orally together with food, the drug exposure (AUC) rose by 39%, which is equivalent to a modest fat and calorie meal. Following oral administration of 600 mg of tecovirimat and intravenous administration of 200 mg of tecovirimat, respectively, the volume of distribution was 383 L and 1030 L,

respectively. Blood to plasma ratios can be anything between 0.62 and 0.90. elimination

Renal elimination and metabolic elimination are the main methods of elimination. After being taken orally, around 73% of the dosage was eliminated in the urine, mainly as glucuronidated metabolites.

About 23% of the dose was found in the patient's stool, mostly as the parent medication in its original form. The most prevalent components in urine were primary tecovirimat glucuronide conjugate and M4 glucuronide conjugate, which accounted for means of 24.4 percent and 30.3 percent of dose, respectively. Following intravenous injection of 200 mg of tecovirimat and oral administration of 600 mg of tecovirimat, the elimination half-life (CV%) was 21 (45%) hours and 19 (29%) hours, respectively. Before being transported to the cell surface and released, the virus is triple-wrapped thanks to the protein's interaction with trans-Golgi component wrappers. Poxviruses spread by creating numerous double-membraned infectious viral "factories" in the cytoplasm.

Before being released into the extracellular area, a portion of the infectious virus is transformed into a triple wrapped form that bonds with the cellular membrane. Both *in vitro* and *in vivo*, these extracellular viruses hasten the infection's propagation. Given that the F13L gene is substantially conserved across all species, it suggests that this method of viral dissemination is universal to all orthopox viruses. The antiviral activity seen in the tissue culture screening came from preventing viral spread from cell to cell rather than from preventing viral multiplication [4,5].

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