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Tanapox Virus: Emerging Insights into Virology, Ecology, Pathogenesis, and Clinical Management

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ABSTRACT

Tanapox virus (TANV) is a zoonotic pathogen belonging to the genus Yatapoxvirus within the Poxviridae family. First identified in 1957 during outbreaks in the Tana River valley of Kenya, TANV is endemic to equatorial regions of East and Central Africa. Its ecological niche involves non-human primates, likely as natural reservoirs, with mechanical transmission to humans primarily via mosquito vectors. Clinically, TANV causes a self-limiting febrile illness characterized by one or a few localized, nodular skin lesions that resolve without scarring over several weeks. While benign in immunocompetent individuals, its clinical presentation can mimic more severe poxviral diseases, such as monkeypox, necessitating accurate laboratory diagnosis. TANV possesses a large, double-stranded DNA genome with a unique complement of genes, including those encoding immunomodulatory proteins that provide insights into viral immune evasion strategies. This review provides a detailed synthesis of the current understanding of TANV, encompassing its taxonomy, virion structure, genomic organization, replication cycle, and pathogenesis. We further elaborate on its geographical distribution, modes of transmission, clinical manifestations, and diagnostic methods. As no specific antiviral therapy or vaccine exists, management remains supportive, and prevention focuses on vector control and limiting exposure to primate reservoirs. The study of TANV not only addresses a neglected tropical zoonosis but also offers a valuable model for understanding poxvirus evolution, host interactions, and emerging potential.

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Keywords
Tanapox Virus, TANV, Yatapoxvirus, Poxviridae, Zoonosis, Emerging Infectious Disease, Viral Pathogenesis, Africa, Mosquito-borne Transmission.

Introduction

Tanapox virus (TANV) is a zoonotic pathogen belonging to the genus *Yatapoxvirus* within the family *Poxviridae*. It is closely related to Yaba monkey tumor virus (YMTV) and shares morphological and genetic features typical of poxviruses, such as large, double-stranded DNA genomes and complex brick-shaped virions (Moss, 2013). Tanapox virus was first identified during an outbreak in 1957 along the Tana River Valley in Kenya, from which its name is derived, and was later recognized in subsequent epidemics in both East and Central Africa (Downie et al., 1971; LeDuc et al., 1978). Its natural reservoir is not fully established, but it is believed to circulate among non-human primates, particularly baboons, with incidental transmission to humans (Bray & Buller, 2004).

Clinically, tanapox virus causes a self-limiting febrile illness characterized by the development of localized, nodular skin lesions that resemble those caused by other poxviruses such as Orf virus or molluscum contagiosum. The incubation period ranges from 4 to 5 days, after which patients typically present with fever, malaise, and a single or a few raised skin nodules that progress slowly before resolving without scarring over a period of 4–6 weeks (LeDuc et al., 1978; Damon, 2013). Unlike variola virus (smallpox), tanapox does not cause systemic, life-threatening disease in humans, making it a relatively benign infection. Nonetheless, its clinical similarity to other poxvirus infections highlights the importance of accurate laboratory diagnosis, particularly in regions where emerging and re-emerging poxviruses pose significant public health threats.

From an ecological and epidemiological perspective, tanapox virus infections are sporadic and often linked to riverside habitats, reflecting the ecology of its primate reservoirs and the role of insect vectors such as mosquitoes in transmission (LeDuc et al., 1978; Reynolds et al., 2010). This zoonotic pattern underscores its significance as a model for studying cross-species transmission of poxviruses and their potential to emerge as human pathogens.

In recent years, research interest in tanapox virus has expanded beyond its clinical and epidemiological aspects to include its role in immunology and virology. Like other poxviruses, TANV encodes immunomodulatory proteins that interfere with host immune responses, offering insights into viral immune evasion strategies and potential applications in biotechnology and vaccine development (Seet et al., 2003).

Classification of Tanapox Virus

Tanapox virus (TANV) is a member of the family **Poxviridae**, a diverse group of large, double-stranded DNA viruses that infect both vertebrates and invertebrates. Within this family, TANV belongs to the **subfamily Chordopoxvirinae**, which encompasses poxviruses infecting vertebrate hosts. More specifically, TANV is classified under the **genus Yatapoxvirus**, which includes only two recognized members: *Tanapox virus* and *Yaba monkey tumor virus (YMTV)* (Moss, 2013; Damon, 2013).
The Yatapoxviruses are unique among the Poxviridae in that they are geographically restricted to equatorial Africa and are primarily associated with non-human primates as their natural reservoirs, with occasional zoonotic infections in humans

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(LeDuc et al., 1978; Reynolds et al., 2010). These viruses are distinguished from other poxvirus genera by their genomic organization, molecular signatures, and host range. Tanapox virus, in particular, is notable for its ability to cause self-limiting febrile illness with localized skin lesions in humans, setting it apart from more virulent members of the family such as *Variola virus* (Orthopoxvirus) or *Monkeypox virus*.

The official classification of Tanapox virus is as follows:

- **Order:** *Nidovirales* (disputed historical placement; poxviruses are sometimes treated as unassigned to order)
- **Family:** *Poxviridae*
- **Subfamily:** *Chordopoxvirinae*
- **Genus:** *Yatapoxvirus*
- **Species:** *Tanapox virus*

This classification reflects both genetic phylogeny and phenotypic characteristics such as virion morphology, replication strategy, and host specificity. Importantly, the genus *Yatapoxvirus* has attracted scientific attention due to its immunomodulatory proteins and evolutionary relationship with other poxviruses, which provide insights into host–pathogen interactions and viral adaptation (Seet et al., 2003).

Structure and Genome Nature of Tanapox Virus

The Tanapox virus virion is a large, complex particle measuring approximately 280–300 nanometers in length and 220–250 nanometers in width, with a characteristic brick-shaped or ovoid morphology (Moss, 2013; Damon, 2013). Its structure is composed of several distinct elements that work together to ensure stability and infectivity.

At the center of the virion is a biconcave core that contains the viral genome along with essential enzymes required for early transcription. Flanking the core are lateral bodies, protein-rich structures that are thought to carry viral factors critical for initiating infection once the virus enters host cells. Surrounding these internal components is a lipid membrane envelope, derived from host cellular membranes, in which viral proteins are embedded to facilitate attachment and entry into target cells (Moss, 2013).

TANV exists in two infectious forms: the intracellular mature virion (IMV) and the extracellular enveloped virion (EEV). IMVs are stable and adapted for transmission between hosts, while EEVs, although more fragile, are highly infectious and play a critical role in facilitating the spread of the virus within the host (Damon, 2013). This dual form of virion underscores the virus's adaptability and enhances its capacity for persistence and dissemination.

Genome Nature

The genome of Tanapox virus is composed of linear double-stranded DNA (dsDNA), with an estimated size of 145 kilobase pairs (kbp) (Esposito and Knight, 1985; Upton et al., 2003). This places TANV among the medium-sized poxvirus genomes, encoding a wide array of proteins required for replication, immune modulation, and virion assembly.
The genome is organized into three major regions: a central conserved core and two terminal variable regions. The central region encodes essential replication machinery and structural proteins that are highly conserved across the *Poxviridae*. By contrast, the terminal regions harbor genes responsible for host range determination and immune evasion, giving TANV a unique ability to modulate host defenses (Brunetti et al., 2003).

At its termini, the TANV genome contains inverted terminal repeats (TIRs) and covalently closed hairpin loops. These structures are critical for genome replication and resolution. In addition, TANV encodes its own set of replication enzymes, including a DNA-dependent RNA polymerase, transcription factors, and capping and polyadenylation enzymes, enabling it to complete its entire replication cycle in the cytoplasm without requiring nuclear machinery (Seet et al., 2003; Moss, 2013).

Overall, the TANV genome encodes more than 150 predicted open reading frames (ORFs), many of which share homology with other poxviruses, although yatapoxvirus-specific genes provide insight into its distinct biology and host interactions (Upton et al., 2003).

Biological and Functional Implications

The structural and genomic features of Tanapox virus collectively shape its pathobiology. Its complex virion structure ensures both environmental stability and efficient spread within host organisms. The cytoplasmic replication strategy allows the virus to evade nuclear antiviral mechanisms and maintain control over the replication process. Furthermore, the accessory genes encoded in the terminal genome regions contribute to immune evasion, including interference with apoptosis and cytokine signalling (Seet *et al*., 2003). These characteristics provide TANV with the ability to establish infections in primate hosts and maintain its ecological niche as a zoonotic pathogen.

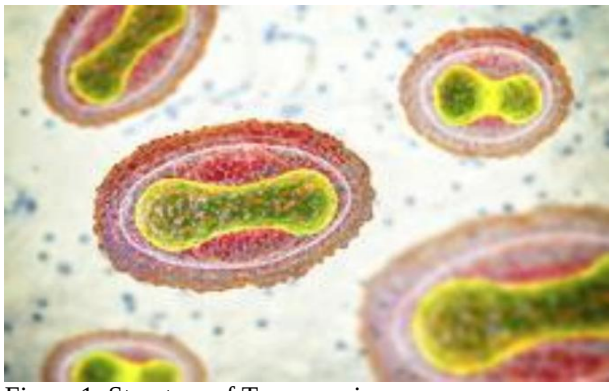

Figure 1: Structure of Tanapox virus
Source: Jezek *et al.* (2002)

Properties of the Virus

1. Biological Properties

1. The virus possesses a double-stranded DNA genome and appears pleomorphic under electron microscopy, with virion sizes ranging from 100–300 nm (Downie, 2004).

2. Structurally, it consists of two main components: an outer envelope with short surface projections and an inner nucleocapsid composed of DNA and glycoproteins.

3. Only a single strain of the virus is recognized, with no known antigenic variation. Reported alterations in virulence across regions are attributed to host

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immune responses and environmental conditions rather than genetic diversity.

4. The virus grows relatively slowly in human and monkey cell cultures, where it induces the formation of intranuclear inclusion bodies. This feature helps differentiate it from other closely related yatapoxviruses (Monroe et al., 2014).

2. Chemical Properties

1. Similar to other poxviruses, the Tanapox virus demonstrates resistance to 40% glycerol at low temperatures and remains stable in phosphate-buffered saline (Jezek et al., 2002).

2. The virus is highly stable when frozen, retaining infectivity for several months at –20°C and –70°C.

3. Virus suspensions exhibit an infective titre of approximately 10^7 focus-forming units (f.f.u.) per ml. However, concentrated extracts from infected tissue cultures do not agglutinate red blood cells from a wide range of species, including vervet monkeys, baboons, fowls, chicks, rabbits, guinea pigs, and humans (group O blood type) (Croitoru et al., 2002).

Distribution of Tanapox Virus

Tanapox virus was first described during epidemics in 1957 and 1962, which occurred in the lower Tana River region of Kenya and were temporally associated with seasonal flooding (Cornel et al., 2018). Serological surveys in the area have since demonstrated continuing transmission to humans, indicating persistent endemicity. Subsequent human infections have also been recorded in the forested regions of Zaire (now the Democratic Republic of Congo), confirming that Tanapox is geographically restricted to Eastern and Central Africa (Monroe et al., 2014). Although rare, a few cases have been documented in travelers returning to Europe and the United States after visiting endemic regions.

The primary maintenance hosts of Tanapox virus remain unidentified; however, several non-human primates, particularly vervet monkeys (*Cercopithecus aethiops*), are susceptible and are abundant in endemic areas (Downie, 2004). Epidemiological evidence suggests that transmission from monkeys to humans is mediated by mosquitoes or other arthropod vectors, which likely explains the seasonal variation in human infections corresponding with periods of increased vector activity (Downie and Espana, 2004).

Tanapox infections occur in both males and females and are especially prevalent among individuals who have occupational or domestic exposure to primates, such as zookeepers or those keeping rare monkeys as pets (Downie and Espana, 2004). Rare instances of transmission through direct contact with an infected primate have been described; however, there is currently no evidence of human-to-human transmission (Dhar et al., 2007).

Mode of Transmission of Tanapox Virus

Tanapox virus is classified as a zoonotic pathogen, meaning it is transmitted from animals to humans (Stich et al., 2002). Epidemiological evidence suggests that the virus circulates primarily among wild non-human primates, which are implicated as potential reservoirs. Transmission to incidental hosts, including humans, is thought to occur mechanically via mosquito bites, particularly through contaminated mouthparts (Axford and Downie, 2009). Sero-prevalence studies have shown that non-human primates are the only animals testing positive for Tanapox antibodies, further supporting their role as the primary reservoir.

Culicine mosquitoes are considered the likely vectors responsible for transferring the virus from infected primates to humans. This hypothesis is supported by the observation that reported Tanapox cases often follow periods of high rainfall, when mosquito populations increase substantially (Axford and Downie, 2009).

Experimental studies have demonstrated that transmission from primates to humans can occur in controlled laboratory settings through direct inoculation, such as scratches from infected animals. However, there have been no documented cases of human-to-human transmission, indicating that natural person-to-person spread is extremely unlikely (Carlson et al., 2022).

Replication of Tanapox Virus

Tanapox virus, like other poxviruses, is a large, double-stranded DNA virus that replicates entirely in the cytoplasm of host cells, which is unusual for DNA viruses (Downie, 2004). The replication process involves several distinct steps, including entry, early gene expression, genome replication, late gene expression, assembly, and release.

1. Viral Entry

Tanapox virus gains entry into host cells primarily via receptor-mediated endocytosis or macropinocytosis. The viral envelope fuses with the host cell membrane, allowing the nucleocapsid to enter the cytoplasm. The viral core contains enzymes necessary for early transcription, enabling replication to begin immediately upon entry (Monroe et al., 2014; Iheukwumere *et al*., 2025a).

2. Early Gene Expression

Once in the cytoplasm, the viral core releases its DNA and initiates early gene transcription using virally encoded RNA polymerase. Early genes encode proteins that modulate host defenses, facilitate viral DNA replication, and prepare the cytoplasm for viral assembly (Downie and Espana, 2004; Iheukwumere *et al*., 2025b).

3. Genome Replication

Tanapox virus replication occurs entirely within cytoplasmic viral factories, specialized sites where viral DNA is synthesized. The virus encodes all enzymes necessary for DNA replication, including DNA polymerase, helicase, and ligase. The genome is replicated through a concatemeric intermediate, which is subsequently processed into unit-length genomes for packaging (Downie, 2004; Iheukwumere *et al*., 2025c).

4. Late Gene Expression

After genome replication, late genes are transcribed and translated. These genes encode structural proteins for the nucleocapsid, envelope proteins, and enzymes required for virion maturation. This stage is critical for assembling

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infectious viral particles (Monroe et al., 2014; Iheukwumere *et al*., 2025d).

5. Assembly and Morphogenesis
New viral particles are assembled in the cytoplasm, where nucleocapsids are wrapped with membranes derived from host organelles, forming the intracellular mature virion (IMV). Some IMVs acquire an additional membrane to form extracellular enveloped virions (EEVs), which are important for cell-to-cell spread and dissemination within the host (Downie and Espana, 2004; Iheukwumere *et al*., 2025e).

6. Release and Transmission
Mature virions are released either by cell lysis or through budding as EEVs, enabling the virus to infect neighboring cells and, ultimately, to transmit to new hosts. The stability of Tanapox virus in the environment allows it to remain infectious long enough to be transmitted via mosquito vectors or direct contact with infected primates (Axford and Downie, 2009; Iheukwumere *et al*., 2025f).

Pathogenesis of Tanapox
The pathogenesis involves viral entry, local replication, immune response modulation, lesion formation, and eventual resolution, and is typically self-limiting in immunocompetent individuals.

1. Viral Entry and Initial Infection
The virus enters the host via mechanical inoculation, usually through the bite of an infected mosquito or, in rare cases, via scratches or direct contact with infected primates. The virus targets epithelial cells of the skin, initiating localized infection (Downie, 2004; Iheukwumere *et al*., 2024a).

2. Local Viral Replication
Upon entry, Tanapox virus replicates in the cytoplasm of keratinocytes and other epithelial cells. Viral replication leads to cytopathic effects, including cell swelling and necrosis, and triggers the formation of intracellular inclusion bodies, which are characteristic of poxvirus infections (Monroe et al., 2014; Iheukwumere *et al*., 2024b).

3. Lesion Formation
Local viral replication induces a localized inflammatory response, characterized by erythema, swelling, and infiltration of immune cells such as neutrophils and macrophages. Clinically, this manifests as papular or nodular lesions, which evolve into umbilicated vesicles or pustules, often followed by crusting and scab formation. Lesions are usually solitary or few in number and are most commonly located on exposed areas of the body (e.g., forearms, face, or legs) (Iheukwumere *et al*., 2024c).

4. Immune Response
The host immune system plays a key role in controlling the infection. Innate immunity, including interferon responses and natural killer (NK) cell activity, limits viral spread, while adaptive immunity, involving virus-specific antibodies and T-cell responses, contributes to lesion resolution and long-term immunity (Downie and Espana, 2004; Iheukwumere *et al*., 2024d). Unlike smallpox or monkeypox, Tanapox infection rarely causes systemic symptoms, and viremia is minimal.

5. Resolution and Recovery
In immunocompetent hosts, lesions typically heal spontaneously within 3–6 weeks, leaving mild hypopigmentation but no permanent scarring in most cases. Immunocompromised individuals may experience more extensive lesions or delayed healing. No human-to-human transmission has been documented, limiting the spread of the virus (Carlson et al., 2022; Iheukwumere *et al*., 2024e).

Clinical Manifestations of Tanapox Virus
Tanapox virus infection in humans typically presents as a mild, self-limiting disease, characterized by localized skin lesions and systemic symptoms. The incubation period is not precisely established; however, in cases of voluntary inoculation, erythema and central thickening at the inoculation site have been observed by the fourth day post-exposure (Stich et al., 2002).

1. Signs
- Skin Lesions: Infection usually begins with a small, painless nodule that develops into a papular lesion. The lesion gradually enlarges, reaching a diameter of approximately 15 mm by the end of the second week (Carlson et al., 2022).
- Nodular Ulceration: During the third week, the nodule may ulcerate before gradually healing over five to six weeks, typically leaving a scar (Downie, 2004).
- Lymphadenopathy: Draining lymph nodes become enlarged and tender around the fifth day following lesion onset, indicating local immune response activation (Dhar et al., 2007).

2. Symptoms
- Pre-eruptive Fever: Most patients experience a mild fever lasting 3–4 days before lesion formation.
- Systemic Discomfort: Patients often report severe headaches, backaches, and localized itching at the lesion site (Stich et al., 2002).

3. Syndromes and Lesion Distribution
- Kenya: Lesions are typically solitary, found predominantly on the upper arms, face, neck, and trunk.
- Zaire (Democratic Republic of Congo): Approximately 22% of patients present with multiple lesions, usually two or three, with the maximum observed being ten lesions. In Zaire, lesions occur mostly on the lower limbs, with occasional involvement of the upper limbs, trunk, and head (Stich et al., 2002; Dhar et al., 2007).

4. Course of Disease
The infection generally follows a self-limiting course, with lesions progressing from papule to nodule, occasionally ulcerating, and ultimately healing over 5–6 weeks, leaving minimal scarring in most cases (Downie, 2004). Severe or disseminated disease is rare and usually limited to immunocompromised individuals.

Diagnosis of Tanapox Virus
The diagnosis of Tanapox virus infection relies on a combination of clinical evaluation, epidemiological history,

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and laboratory confirmation, as the clinical features can resemble other poxvirus infections. Early and accurate diagnosis is important for differentiating Tanapox from similar diseases, such as monkeypox, smallpox, or cowpox.

1. Clinical Diagnosis
   - History and Exposure: A patient's recent travel to endemic areas in Eastern or Central Africa and potential contact with non-human primates or mosquito bites can suggest Tanapox infection.
   - Lesion Characteristics: Tanapox lesions are usually solitary or few in number, progressing from papules to nodules, possibly ulcerating, and eventually healing within 5–6 weeks (Downie, 2004; Stich et al., 2002).
   - Systemic Symptoms: Mild pre-eruptive fever, headache, backache, and lymphadenopathy can support a presumptive clinical diagnosis.

2. Laboratory Diagnosis

Laboratory confirmation is essential because clinical signs can overlap with other poxvirus infections. Methods include:

a. Electron Microscopy
   - Examination of lesion material reveals pleomorphic virions characteristic of Yatapoxviruses, with nucleocapsid and envelope structure visible (Downie, 2004; Iheukwumere *et al*., 2024f).

b. Serology
   - ELISA or neutralization assays can detect specific antibodies against Tanapox virus in human or non-human primate sera (Axford and Downie, 2009; Iheukwumere *et al*., 2024g).
   - Serological studies help confirm exposure and identify reservoirs but are limited in acute cases due to delayed antibody production.

c. Molecular Methods
   - Polymerase chain reaction (PCR) is used to detect viral DNA in lesion swabs, scabs, or biopsy samples. PCR allows rapid and specific identification, distinguishing Tanapox from other poxviruses (Monroe et al., 2014; Iheukwumere *et al*., 2025h).

d. Virus Isolation
   - Rarely, the virus can be cultured in monkey or human cell lines, producing characteristic cytopathic effects and intranuclear inclusion bodies. This method is largely restricted to specialized research laboratories (Downie and Espana, 2004; Iheukwumere *et al*., 2025i and Iheukwumere *et al*., 2025j).

3. Differential Diagnosis

Because Tanapox lesions can resemble those caused by monkeypox, cowpox, or herpesvirus infections, laboratory confirmation is critical to avoid misdiagnosis, especially in regions where multiple poxviruses are present.

Treatment of Tanapox Virus

Currently, no specific antiviral treatment or vaccine exists for Tanapox virus infection. The disease is generally self-limiting, and lesions typically heal spontaneously within 5–6 weeks without medical intervention (Croitoru et al., 2002). Management is primarily supportive, focusing on alleviating symptoms such as itching or discomfort at the lesion site. In immunocompromised individuals, close monitoring is recommended to prevent secondary bacterial infections.

Prevention of Tanapox Virus

Although Tanapox lesions can be unsightly and uncomfortable, they are not life-threatening. Preventive measures focus on reducing exposure to the virus and its potential vectors:

1. Vaccination: No vaccine currently exists for Tanapox. Vaccination against smallpox or monkeypox does not confer protection against Tanapox virus (Axford and Downie, 2009).
2. Mosquito Avoidance: Since the virus may be transmitted mechanically by mosquito vectors, the use of insect repellents according to manufacturer instructions is recommended. Additional measures include wearing light-colored, loose-fitting clothing and minimizing outdoor exposure during peak mosquito activity (Jezek et al., 2002; Dhar et al., 2007).
3. Lesion Management: Individuals diagnosed with Tanapox should cover lesions loosely with a clean cotton bandage to reduce potential risk of accidental mechanical transmission, although human-to-human spread has not been documented (Croitoru et al., 2002).
4. Environmental Awareness: Limiting direct contact with wild or captive non-human primates in endemic areas also reduces the risk of infection.

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