# **A Profile of *Mortiferum somniculosum*: A Fictional Neurotropic Virus Inducing Zombification Syndrome**

### **Abstract**

This report details a hypothetical neurotropic pathogen, *Mortiferum somniculosum*, engineered to induce a "zombification" syndrome. The virus, classified within the *Mononegavirales* order, exhibits a unique pathogenesis characterized by three distinct phases: an initial incubation period with exaggerated sensory sensitivity, followed by severe neurological degeneration leading to profound motor and behavioral dysregulation, and culminating in clinical death and subsequent reanimation. Reanimation is uniquely triggered by a genetically engineered ectosymbiotic microorganism, *Thanatomicrobium vivificans*, which establishes a bio-electrical network within the virally damaged brain. Epidemiologically, *Mortiferum somniculosum* boasts an exceptionally high basic reproduction number (R0) due to its dual-mode transmission via bodily fluids and highly efficient aerosolized particles, coupled with a significant period of asymptomatic shedding. This combination precipitates rapid global impact, resulting in the zombification of 63% of the world's population. A rare genetic mutation, the HLA-B27 variant, confers immunity in approximately 5% of individuals. The virus's deep lore traces its origin to a weaponized bat-borne pathogen developed by a pharmaceutical company, released accidentally by Patient Zero, leading to an unprecedented societal collapse. The only plausible therapeutic avenue involves a rare blood plasma protein, Vivicase, derived from immune individuals, which presents profound ethical dilemmas regarding resource allocation and human exploitation. CRISPR-inspired gene therapies are rendered ineffective due to the virus's efficient integration into host DNA. This profile blends realistic biological principles with plausible fictional elements to explore the complex implications of advanced biotechnology and emerging infectious threats.

### **Introduction**

The landscape of global health is perpetually shaped by the emergence of novel infectious diseases, many of which originate from zoonotic spillover events. Historically and contemporaneously, such pathogens have demonstrated a profound capacity for widespread disruption, challenging public health infrastructures and societal stability on an unprecedented scale.1 A particularly concerning subset of these emerging threats comprises neurotropic pathogens—viruses, bacteria, or other microorganisms that specifically target the central nervous system (CNS). These agents are capable of inducing severe neurological and behavioral sequelae, ranging from encephalitis and meningitis to progressive neurodegenerative disorders.3 The direct impact on brain function presents a unique and formidable challenge, as it can compromise an individual's cognitive abilities, motor control, and even fundamental personality, leading to outcomes far more devastating than typical systemic infections.

The convergence of a highly mobile natural reservoir, such as bats, and a target system known to be susceptible to viral invasion, namely the central nervous system, creates a potent hypothetical threat. Bats are recognized as significant reservoirs for a multitude of viruses, including those with substantial zoonotic potential, often carrying these pathogens without exhibiting overt disease themselves.11 This inherent capacity of bats to host high-impact human pathogens, combined with the known susceptibility of the CNS to viral invasion and the varied neurological manifestations of neurotropic viruses, establishes a compelling biological precedent for the development of highly destructive agents. The fictional elements presented in this report, such as the induction of a comatose state followed by reanimation and specific behavioral alterations, are not entirely fabricated. Instead, they are presented as extreme amplifications of known biological effects, rooted in the documented neurological disruptions caused by real-world viruses. This approach grounds the hypothetical pathogen in a plausible biological reality, thereby enhancing the relevance and gravity of the presented threat in the context of contemporary concerns regarding emerging zoonoses and biosecurity.

This report aims to construct a scientifically plausible profile of a novel, highly pathogenic neurotropic virus, herein designated *Mortiferum somniculosum*, which induces a unique "zombification" syndrome. The subsequent sections will systematically detail the virus's nomenclature and taxonomic placement, its virion structure and genomic characteristics, and its proposed zoonotic origin and evolutionary pathway. A comprehensive analysis of the pathogenesis and clinical progression will follow, outlining the distinct stages of the Zombification Syndrome from initial exposure to reanimation. The epidemiological dynamics, including transmission modes and global impact, will be thoroughly examined, alongside the identification of a genetically immune population. Finally, the report will delve into the deep lore surrounding the virus's origin as a weaponized agent, its Patient Zero, the mechanisms of societal collapse, and the enduring legacy of survivors, concluding with an exploration of the challenging therapeutic landscape and the profound ethical dilemmas associated with a potential cure. Throughout this analysis, a deliberate balance of 75% realistic biological principles with 25% fictional, yet biologically informed, elements will be maintained, adhering strictly to established academic standards.

### **I. Etiology and Classification of *Mortiferum somniculosum***

#### **A. Nomenclature and Taxonomic Placement**

The novel neurotropic pathogen is formally designated *Mortiferum somniculosum*. This scientific name is derived from Latin roots, adhering to established conventions in medical and biological terminology.14 "Mortiferum" translates to "death-bringing" or "deadly," directly reflecting the high fatality rate and destructive nature of the infection. "Somniculosum" means "sleepy," "drowsy," or "causing sleep," alluding to the characteristic coma phase that precedes the reanimation, a critical and unique aspect of the syndrome.

In accordance with the International Committee on Taxonomy of Viruses (ICTV) guidelines, which authorize and organize the taxonomic classification and nomenclature for viruses, *Mortiferum somniculosum* is systematically placed within the universal taxonomic scheme.16 Its classification is as follows:

* **Realm:** *Riboviria*. This realm encompasses all RNA viruses, which aligns with the virus's genetic material.14
* **Order:** *Mononegavirales*. This order is a fitting placement as it includes a diverse group of non-segmented, negative-sense single-stranded RNA viruses known for their helical capsid symmetry and enveloped virions.17 Notably, this order contains highly pathogenic neurotropic viruses such as those in the *Rhabdoviridae* family (e.g., Rabies virus) and *Filoviridae* family (e.g., Ebola virus).17
* **Family:** *Thanatoviridae*. This is a newly proposed family, derived from the Greek "Thanatos," meaning death, reflecting the virus's lethal outcome. Family names consistently end in the suffix "-viridae" as per ICTV rules.16
* **Genus:** *Somnivirus*. This newly proposed genus name is derived from the Latin "somnus," meaning sleep, again referencing the characteristic comatose state induced by the virus. Genus names consistently end in the suffix "-virus".16
* **Species:** *Mortiferum somniculosum*. As with all scientific species names, it is italicized, with the first letter of the first word capitalized, and follows the binomial format required for new species designations since 2020.16

The deliberate placement of *Mortiferum somniculosum* within the *Mononegavirales* order establishes a robust biological precedent for the virus's neurotropic and highly pathogenic nature. Viruses such as rabies and Ebola, which belong to this order, are renowned for their ability to invade and severely affect the central nervous system, leading to profound neurological dysfunction and often fatal outcomes.7 This taxonomic choice is not arbitrary; it leverages existing scientific knowledge to lend credibility to the fictional elements of the Zombification Syndrome. By associating *Mortiferum somniculosum* with a group of viruses already known for causing severe neurological disruption and high mortality, the concept of "zombification" can be presented as an extreme, yet biologically comprehensible, manifestation of known viral pathology. This grounding in established virology enhances the scientific plausibility of the hypothetical pathogen. The chosen nomenclature, *Mortiferum somniculosum*, cleverly combines the lethal aspect of the disease with the "sleepy" precursor to reanimation, further integrating the fictional narrative with scientifically derived terminology.

#### **B. Virion Structure and Genomic Characteristics**

*Mortiferum somniculosum* is classified as a Group V virus under the Baltimore classification system, meaning it possesses a single-stranded, negative-sense RNA genome.17 This genomic characteristic is consistent with its placement within the *Mononegavirales* order. The virion, or individual viral particle, exhibits a distinctive morphology: it is enveloped and possesses helical capsid symmetry.17 This helical structure, where the RNA genome is tightly encapsidated by nucleoprotein molecules, forms a flexible rod-like or filamentous shape. The entire nucleocapsid is then enclosed within a lipid envelope, which is typically derived from the host cell membrane during the process of viral budding.19 This envelope is crucial for the virus's ability to fuse with host cell membranes, facilitating entry, and for its stability in the extracellular environment.

The genetic material, being a negative-sense RNA genome, dictates that the virus must carry its own RNA-dependent RNA polymerase (L protein) within the virion. This enzyme is essential for transcribing messenger RNA (mRNA) directly from the negative-sense genome upon entry into a host cell, as host cells lack the machinery to do so.17 In addition to the L protein, the virion of *Mortiferum somniculosum* is hypothesized to encode several other key proteins vital for its life cycle and pathogenic effects:

* **Glycoprotein (G protein):** This protein is embedded in the viral envelope and forms spikes that protrude from the virion surface. The G protein is critical for host cell attachment and entry, mediating the binding to specific receptors on target cells. In many neurotropic viruses, such as the rabies virus, the G protein plays a crucial role in determining neurotropism and virulence.20 For *Mortiferum somniculosum*, this protein is likely engineered to target specific neural receptors with high affinity, facilitating rapid and efficient neuroinvasion.
* **Matrix protein (M protein):** Located beneath the viral envelope, the M protein is involved in virion assembly and budding from the host cell. It acts as a bridge between the nucleocapsid and the envelope, facilitating the formation of new viral particles.19
* **Nucleoprotein (N protein):** This protein tightly associates with the RNA genome, forming the helical nucleocapsid. It protects the viral RNA from degradation and plays a role in replication and transcription.
* **Novel "Reanimin" Protein:** A unique and hypothetical viral protein, the "Reanimin" protein, is proposed to be a key component in the pathogenesis of the Zombification Syndrome. This protein is envisioned to interact specifically with the symbiotic microorganism, *Thanatomicrobium vivificans*, facilitating the establishment of their unique relationship within the host. Its role could be as a specific receptor, a signaling molecule, or even an enzymatic component that primes host tissue for the symbiont's activity. The concept of a viral protein interacting with a symbiotic organism to alter host biology is biologically plausible, drawing parallels from complex host-symbiont relationships observed in nature.21

To further illustrate the structural characteristics of *Mortiferum somniculosum*, a mock diagram of its virion structure would be highly informative. This diagram would depict the helical nucleocapsid, containing the negative-sense RNA genome and associated N and L proteins, encased within a lipid envelope. Spikes representing the G protein would be prominently displayed on the outer surface of the envelope. A distinct label or highlighted region could indicate the presence and location of the "Reanimin" protein, emphasizing its unique functional role. Such a visual representation would reinforce the scientific plausibility of the virus's structure, grounding it in known viral morphology and helping the reader conceptualize the physical entity and how its components contribute to its unique "zombification" function.

#### **C. Proposed Zoonotic Origin and Evolutionary Pathway**

The proposed natural reservoir for *Mortiferum somniculosum* is the Schreiber's bat, *Miniopterus schreibersii*. This choice is grounded in current virological research, which identifies bats as significant reservoirs for a wide array of viruses, including those with substantial zoonotic potential for humans.11 Specifically, *Miniopterus schreibersii* has been documented to host various viruses, including coronaviruses and filoviruses such as the Lloviu virus (LLOV).11 The LLOV is particularly relevant as it is functionally and genomically related to Ebola virus and has demonstrated the capacity to infect human cells *in vitro*.12 This specific link to a bat species with known zoonotic potential provides a concrete, real-world foundation for the fictional virus's origin and its ability to cross the species barrier to humans.

The evolutionary pathway of *Mortiferum somniculosum* is envisioned as a progression from an initially benign bat-borne virus. This precursor, tentatively named *Miniopterus somnivirus*, would have circulated endemically within *Miniopterus schreibersii* populations, remaining apathogenic in its natural host. This aligns with observations that bats often carry high-impact human pathogens without exhibiting discernible disease themselves, a phenomenon attributed to their unique immunological features.13 The critical transformation into *Mortiferum somniculosum* is hypothesized to have occurred through the acquisition of novel gene sequences. This could involve genetic recombination with another bat virus, a process known to occur in viral evolution, or through a rare, advantageous mutation. This acquired genetic material would confer the heightened neurotropic properties observed in *Mortiferum somniculosum* and, crucially, encode the unique "Reanimin" protein. The high plasticity of RNA viruses and their capacity for horizontal gene transfer further support the plausibility of such an evolutionary leap.24

The selection of *Miniopterus schreibersii* as the bat reservoir is a deliberate decision that significantly enhances the scientific credibility of the fictional virus's origin. By choosing a specific bat species with documented connections to human-infecting pathogens, the narrative moves beyond a generic "bat virus" to a more tangible and biologically informed scenario. The fact that many bat-borne viruses are asymptomatic in their natural hosts provides a realistic backstory for how a precursor virus could have existed undetected for an extended period. This asymptomatic carriage is a critical element, allowing for a plausible scenario where such a virus could be isolated, studied, and subsequently weaponized or accidentally released without prior alarms. This detailed consideration of the virus's origin in a specific, biologically relevant host, and its proposed evolutionary trajectory, strengthens the overall narrative by grounding the fictional premise in concrete, albeit extrapolated, scientific data.

### **II. Pathogenesis and Clinical Progression: The Zombification Syndrome**

The Zombification Syndrome induced by *Mortiferum somniculosum* progresses through three distinct clinical stages: Incubation, Neurological Degeneration, and Coma & Reanimation. This progression is characterized by a rapid and escalating assault on the host's central nervous system, culminating in a unique form of post-mortem motility driven by a symbiotic microorganism.

#### **A. Incubation Phase: Initial Systemic Manifestations (0-72 hours post-exposure)**

The incubation period for *Mortiferum somniculosum* is exceptionally short, typically lasting between 0 and 72 hours post-exposure. This aggressive timeline is a critical factor in the virus's rapid global spread, designed to overwhelm public health response systems before effective containment measures can be implemented. Such a condensed incubation period, while severe, aligns with the dynamics of highly contagious respiratory viruses where rapid onset of symptoms contributes to swift dissemination within a population.25

During this initial phase, individuals experience a blend of realistic and exaggerated symptoms:

* **Realistic Symptoms:** The onset is marked by common prodromal signs reminiscent of many acute viral infections. These include a high fever, severe headache, generalized muscle aches (myalgia), profound fatigue, and a pervasive sense of malaise.5 These non-specific symptoms often lead to initial misdiagnosis as common influenza or other prevalent viral illnesses, allowing the virus to spread undetected during its most critical early window.
* **Fictional/Exaggerated Symptoms:** A distinguishing feature of this phase is the rapid development of **hyperesthesia**, an extreme and debilitating heightened sensory sensitivity. Individuals experience intense discomfort, particularly to touch, sound (hyperacusis), and light (photophobia).5 Normal environmental stimuli, such as ambient noise or dim light, become overwhelmingly painful, leading to immediate and profound withdrawal, agitation, and an aversion to external interaction. Concurrently, subtle but marked behavioral shifts emerge, including an uncharacteristic increase in irritability, anxiety, and a nascent, unprovoked aggression.28 These early indicators point towards initial viral neuroinvasion and irritation of the limbic system, a brain region crucial for emotion and behavior.5

The inclusion of hyperesthesia as a prominent early symptom, beyond typical flu-like signs, provides a unique and biologically informed characteristic for this neurotropic virus. While hyperesthesia is a documented neurological symptom observed in various conditions, including some viral infections, its exaggerated presence and rapid onset in the Zombification Syndrome serve a specific purpose. This amplified, yet plausible, manifestation of a real symptom explains the infected individual's immediate withdrawal and agitation, which are atypical for common viral illnesses. This early neurological impact sets the stage for the rapid escalation to more severe paranoia and aggression in subsequent stages, demonstrating a coherent and terrifying progression of the disease from subtle discomfort to overt behavioral pathology. The underlying mechanism involves initial rapid viral replication in peripheral tissues, followed by swift systemic dissemination via the bloodstream. Early, low-level neuroinvasion then causes widespread inflammation and immune activation within the central nervous system, particularly affecting sensory processing pathways, leading to the pronounced hyperesthesia and the initial behavioral changes.5

#### **B. Neurological Degeneration Phase: Central Nervous System Invasion (72-168 hours post-exposure)**

As the infection progresses into the Neurological Degeneration phase, the virus establishes a widespread and devastating presence within the central nervous system, leading to a cascade of severe and irreversible neurological dysfunctions. This phase typically spans from 72 to 168 hours post-exposure.

The symptoms observed during this stage are a combination of realistic neurological pathologies, significantly amplified and distorted by the virus's specific targeting:

* **Viral Encephalitis:** The widespread brain inflammation, a hallmark of encephalitis, intensifies dramatically. Individuals experience profound confusion, severe disorientation, and extreme agitation.5 Vivid hallucinations, both visual and auditory, become frequent and terrifying, often exacerbated by severe and persistent insomnia.28 Seizures, initially sporadic, become common, severe, and often intractable, reflecting the extensive neuronal damage and hyperexcitability within the brain.6
* **Basal Ganglia Dysfunction:** The virus exhibits a pronounced tropism for the basal ganglia, a group of subcortical nuclei critical for motor control, habit formation, and decision-making.41 Damage to these structures results in a progressive loss of fine and gross motor control, manifesting as severe involuntary movements such as chorea (random, jerky movements), athetosis (slow, writhing movements), and ballismus (wild, flinging movements).42 Muscle tone increases, leading to rigidity and spasms. Speech becomes severely slurred or entirely incoherent (dysarthria), and walking is characterized by a distinctive shuffling or jerky, uncontrolled gait.41 This constellation of motor symptoms is highly reminiscent of parkinsonism-like disorders or Huntington's disease, but with an accelerated and more aggressive progression.4
* **Limbic System Pathology:** Concurrent with motor dysfunction, the limbic system, responsible for emotion, motivation, and memory, suffers extensive viral damage. This pathology leads to an exacerbation of paranoia, extreme and unprovoked aggression, profound loss of inhibition, and complete, irreversible personality changes.32 The prefrontal cortex, which normally serves as a control center for aggressive impulses and social behavior, is severely compromised, leading to a breakdown of behavioral regulation.34 Simultaneously, the amygdala, a brain region central to processing fear and aggression, becomes hyperactivated.34 This neurological imbalance, coupled with dysregulation of neuropeptides (e.g., vasopressin, oxytocin) and persistent, uncontrolled surges of adrenaline, creates a constant "fight-or-flight" state, fueling the characteristic hyper-aggression and paranoia.33
* **Insomnia:** The severe and persistent sleep disruption, noted in the incubation phase, becomes debilitating. This extreme insomnia contributes significantly to the delirium, hallucinations, and overall cognitive and behavioral disorganization observed in this stage.28

The detailed progression from general neurological symptoms of encephalitis to specific motor and behavioral dysfunctions, particularly affecting the basal ganglia and limbic system, provides a deep, scientifically grounded explanation for the "zombie" phenotype. By directly linking the hyper-aggression and paranoia to viral damage in the amygdala and prefrontal cortex, and further exacerbating these behaviors with dysregulated adrenaline surges, the fictional behavioral changes are firmly rooted in established neurobiology.34 This avoids the simplistic "mindless" zombie trope and instead presents a creature driven by extreme, uncontrolled primal urges, making it more terrifyingly plausible. The meticulous mapping of symptoms to specific brain regions and known neurological dysfunctions elevates the scientific credibility of the Zombification Syndrome.

#### **C. Coma and Reanimation Phase: Symbiotic Reintegration and Post-Mortem Motility (168-240 hours post-exposure, leading to reanimation)**

As the neurological damage inflicted by *Mortiferum somniculosum* becomes overwhelming, and vital organ systems succumb to the systemic viral infection, the host enters a deep, irreversible coma. This state is characterized by the cessation of higher brain function and progresses to clinical death, typically occurring between 168 and 240 hours post-exposure. However, unlike conventional death, this is not the end of motor activity.

The reanimation phenomenon is uniquely triggered and sustained by a novel, genetically engineered ectosymbiont, *Thanatomicrobium vivificans*. The name, derived from Greek "Thanatos" (death), "mikrobion" (small life), and Latin "vivificans" (life-giving), encapsulates its paradoxical role. This microorganism is designed to be highly resilient, capable of surviving and rapidly proliferating in necrotic tissue, a crucial adaptation for its post-mortem function.22

The mechanism of reanimation, while containing fictional elements, is built upon plausible biological concepts:

* *Thanatomicrobium vivificans* is hypothesized to be a highly specialized, genetically modified bacterium or fungus.22 It establishes a parasitic ectosymbiotic relationship with the *Mortiferum somniculosum* virions and, more critically, with the virally altered host cells, particularly within the central nervous system.50 The "Reanimin" protein, encoded by *Mortiferum somniculosum*, plays a pivotal role here, acting as a specific receptor or signaling molecule that facilitates this symbiotic relationship. This interaction could potentially provide a metabolic advantage to *T. vivificans*, allowing it to thrive in the compromised neural environment.
* Upon the clinical death of the host, as higher cortical functions cease, *T. vivificans* undergoes rapid proliferation. It forms a dense, resilient network, particularly within the virally damaged and metabolically compromised basal ganglia and limbic system.41 These regions, already primed by the viral infection, become the primary sites for the symbiont's activity. This microbial network effectively acts as a "bio-electrical conduit," bypassing the non-functional higher cortical areas and directly manipulating the residual motor pathways.
* *T. vivificans* is engineered to produce and release novel neuroactive compounds—such as modified neurotransmitters, unique peptides, or electrochemical signals. These compounds, in conjunction with the viral "Reanimin" protein, induce residual, uncontrolled muscle contractions and primitive motor functions. This is a highly fictionalized element, but it is grounded in the established biological concept of behavior-altering parasites that manipulate host neurology 51, and the demonstrated ability to engineer symbiotic relationships with profound effects on host biology and behavior.22
* The reanimation is not a return to life in any meaningful sense. Instead, it is a sustained, primitive motor function driven by this symbiotic microbial-viral complex. The reanimated individuals are devoid of higher cognitive functions, consciousness, or self-awareness. Their movements are primarily focused on primal drives: propagation of the virus (through biting and aerosol spread) and seeking dense populations. This state is sustained by scavenging residual energy from decaying tissues and the symbiont's unique metabolic adaptations.

The symbiotic reanimation mechanism represents the core 25% fictional element of this profile, yet its plausibility is carefully constructed by drawing upon real biological phenomena. The concept of parasites altering host behavior to their own benefit 51, and the rapidly advancing field of lab-engineered symbionts with profound effects on host biology 22, provide a scientific framework for this otherwise extraordinary phenomenon. By specifying an *ectosymbiotic* relationship, where the microorganism lives on the surface of host cells rather than strictly within them 50, and by linking its action to the *already virally damaged* basal ganglia and limbic system, the reanimation is presented not as magic or true resurrection, but as a grotesque, biologically driven continuation of motor function and primal urges.49 This crucial distinction maintains the scientific plausibility of the "undead" state.

To enhance the understanding of the neurological progression, a mock diagram of brain regions affected by the viral progression would be highly beneficial. This visual aid would present a sagittal or coronal cross-section of the human brain, employing color-coding to illustrate the escalating impact of the virus across the three stages. During the Incubation phase, diffuse, low-level inflammation could be depicted, particularly within sensory processing areas. In the Neurological Degeneration phase, intense inflammation and damage would be highlighted in the limbic system (amygdala, hippocampus, hypothalamus) and basal ganglia (caudate, putamen, globus pallidus), shown in a darker, more intense color. Finally, for the Coma & Reanimation phase, higher cortical areas (prefrontal cortex, sensory cortex) would be shown as "dark" or "inactive," signifying brain death, while the basal ganglia and limbic system would display a distinct, primitive "activity" pattern, perhaps glowing green, with a superimposed network representing the pervasive influence of the symbiotic microorganism. This visual element would concretize abstract neurological concepts, clearly illustrating how selective viral targeting and subsequent symbiotic manipulation lead to the observed symptoms and the transition from complex brain function to primitive, reanimated motility.

The following table summarizes the clinical stages and associated symptoms of the Zombification Syndrome:

**Table 1: Clinical Stages and Associated Symptoms of Zombification Syndrome**

| **Stage** | **Time Post-Exposure** | **Key Symptoms (75% Realistic / 25% Fictional)** | **Neurological Basis / Mechanism** |
| --- | --- | --- | --- |
| **Incubation** | 0-72 hours | Fever, headache, muscle aches, profound fatigue, general malaise (Realistic); Extreme hyperesthesia (sensitivity to touch, sound, light), heightened irritability, nascent uncharacteristic aggression (Exaggerated/Fictional) | Rapid systemic viral replication; Early, low-level neuroinvasion causing widespread CNS inflammation and immune activation; Direct irritation of sensory pathways and nascent limbic system irritation. |
| **Neurological Degeneration** | 72-168 hours | Profound confusion, severe disorientation, extreme agitation, vivid hallucinations (visual & auditory), severe seizures, debilitating insomnia (Realistic); Progressive loss of fine/gross motor control (chorea, athetosis, ballismus), increased muscle tone/rigidity, slurred/incoherent speech, shuffling/jerky gait, extreme paranoia, unprovoked hyper-aggression, complete loss of inhibition, irreversible personality changes (Realistic, but severely exaggerated/amplified by fictional elements) | Efficient breach of blood-brain barrier; Widespread viral infection and inflammation (encephalitis) in the brain, particularly targeting basal ganglia (motor control) and limbic system (emotion, behavior); Severe damage to prefrontal cortex (inhibitory control) and hyperactivation of amygdala (fear/aggression); Dysregulated neuropeptide release and persistent adrenaline surges. |
| **Coma & Reanimation** | 168-240 hours (onset of coma), then reanimation | Clinical death (cessation of higher brain function, organ failure) (Realistic); Primitive, uncontrolled motor function, relentless pursuit of uninfected individuals, extreme resilience to pain, complete absence of higher cognition or self-awareness (Fictional, driven by symbiont) | Overwhelming neurological damage leading to brain death; *Thanatomicrobium vivificans* rapidly proliferates, forming a dense bio-electrical network within the virally damaged basal ganglia and limbic system; Symbiont produces neuroactive compounds, interacting with viral "Reanimin" protein, to induce residual muscle contractions; Reanimation is a grotesque, biologically sustained locomotion for propagation, not a return to life. |

### **III. Epidemiology and Global Impact**

The epidemiological characteristics of *Mortiferum somniculosum* are designed to facilitate rapid and widespread global impact, leading to an unprecedented scale of infection and societal disruption.

#### **A. Transmission Dynamics**

The virus employs a highly efficient, dual-mode transmission strategy, which is a key factor in its devastating R0 value and rapid spread:

* **Primary Modes:**
  + **Bodily Fluids:** Direct contact with infected bodily fluids serves as a primary transmission route. This includes exposure to highly virulent saliva, which is exacerbated by the virus-induced hypersalivation observed in the neurological degeneration phase, similar to the symptoms seen in rabies.7 Transmission occurs through bites, contact with open wounds, or exposure to mucous membranes. This mechanism is common for highly pathogenic viruses such as HIV and Ebola.25
  + **Aerosolized Particles:** Inhalation of virus-laden aerosols represents an equally, if not more, critical mode of transmission. These aerosols are generated through common expiratory activities such as breathing, talking, coughing, and the aggressive shouting characteristic of infected individuals.55 Crucially, the majority of exhaled aerosols are smaller than 100 μm, with a significant fraction being less than 5 μm. Particles in this size range can remain suspended in still air for hours and travel well beyond the typical 2-meter social distancing recommendation, penetrating deeply into the lower respiratory tract upon inhalation.55 This highly efficient and rapid transmission mechanism is particularly effective in poorly ventilated, crowded indoor environments, where the virus can accumulate and spread efficiently among susceptible individuals.56
* **Asymptomatic Shedding:** A critical factor contributing to the rapid and undetected spread of *Mortiferum somniculosum* is its significant period of asymptomatic shedding. Individuals, particularly during the initial 0-72 hour incubation phase, are highly contagious even before the onset of overt symptoms.19 This phenomenon, observed in real-world pathogens such as HIV, Polio, and COVID-19, allows the virus to disseminate widely within a population before any public health interventions can be effectively mobilized.26 The virus is shed from both respiratory and potentially gastrointestinal tracts, further broadening the avenues of transmission and complicating containment efforts.57
* **Basic Reproduction Number (R0):** The estimated basic reproduction number (R0) for *Mortiferum somniculosum* is between 10 and 15. This exceptionally high value quantifies the virus's devastating transmissibility. For context, highly contagious airborne diseases like measles have an R0 of 12-18, and chickenpox an R0 of 10-12.59 An R0 significantly greater than 1 indicates that each infected individual will, on average, transmit the disease to more than one other person, leading to exponential growth of the epidemic in a susceptible population.25 This high R0, combined with the stealthy asymptomatic shedding and dual-mode transmission, ensures rapid and widespread infection, quickly overwhelming any attempts at containment.

The combination of both highly efficient aerosolized particle transmission and direct bodily fluid transmission, especially when coupled with a significant period of asymptomatic shedding during the incubation phase, creates a "perfect storm" for rapid and uncontrollable global spread. Most highly contagious diseases primarily rely on one dominant mode of transmission (e.g., measles is predominantly aerosol, HIV is primarily bodily fluids). *Mortiferum somniculosum*'s multi-pronged transmission strategy, compounded by its long asymptomatic shedding period, renders containment nearly impossible. This epidemiological profile is the primary biological driver behind the rapid global collapse and the exceptionally high zombification rate, making the fictional outcome a logical consequence of the virus's inherent transmissibility.

#### **B. Pandemic Progression and Societal Collapse**

The epidemiological characteristics of *Mortiferum somniculosum* ensure an unprecedented pandemic progression, leading swiftly to global societal collapse. The exceptionally high R0 and the stealthy nature of asymptomatic shedding mean that the virus spreads exponentially, quickly overwhelming any existing public health infrastructure and rendering traditional contact tracing efforts futile.25

Within days of the first confirmed cases, the rapid increase in infections would lead to a catastrophic collapse of healthcare systems. Hospitals would be inundated with patients in the incubation and neurological degeneration phases, quickly depleting resources, and medical staff would themselves become infected, further exacerbating the crisis.1 The unique and terrifying symptoms, particularly the hyper-aggression and reanimation, would create widespread panic and a breakdown of social order. Law enforcement and military forces would be rapidly overwhelmed by the sheer number of aggressive, reanimated individuals. Essential services, including power grids, water treatment facilities, and food supply chains, would cease to function due to mass infection, fear-driven absenteeism, and the general inability to maintain operations in a chaotic environment.1

The rapid progression from subtle symptoms to hyper-aggression, combined with the virus's high infectivity, would effectively prevent any form of effective containment. The short incubation period and the extended duration of asymptomatic shedding mean that individuals are highly contagious and actively spreading the virus before any overt, recognizable symptoms appear. This inherent biological characteristic would lead to an exponential increase in cases, quickly rendering traditional public health responses, such as isolation and contact tracing, utterly ineffective. The inability to identify and isolate infected individuals before they become highly transmissible and dangerous would be the primary driver of the rapid societal breakdown, as the infection rate would consistently outpace any response. This highlights a critical vulnerability in global health security, where a pathogen with such characteristics could swiftly bring about widespread societal collapse.

The global impact is projected to be devastating, with approximately 63% of the world's population becoming zombified. This scale of infection is a direct and inevitable consequence of the virus's aggressive transmission dynamics, its short incubation, and the unique reanimation mechanism that sustains infectivity post-mortem.

#### **C. Genetic Immunity: The HLA-B27 Variant**

Amidst the widespread devastation, a small fraction of the human population, approximately 5%, exhibits a natural immunity to *Mortiferum somniculosum*. This immunity is conferred by the presence of a specific genetic variant, the Human Leukocyte Antigen B27 (HLA-B27).62

The HLA-B27 gene is part of the major histocompatibility complex (MHC) class I molecules, which play a crucial role in the immune system. These proteins are found on the surface of most cells and are responsible for presenting small peptides (fragments of proteins) to T-cells, allowing the immune system to distinguish between the body's own cells and foreign invaders like viruses and bacteria.63 While HLA-B27 is typically associated with an increased risk of certain autoimmune and inflammatory diseases, such as ankylosing spondylitis and reactive arthritis, its role in immune response is complex and multifaceted.62

In the context of *Mortiferum somniculosum*, the specific HLA-B27 variant confers partial immunity through a novel mechanism. It is hypothesized that this variant alters the way viral peptides from *Mortiferum somniculosum* are presented to T-cells. This unique presentation leads to a more effective and rapid T-cell mediated immune response, specifically a cytotoxic T-lymphocyte (CTL) response, that is capable of identifying and eliminating infected cells more efficiently than in non-carriers.65 This accelerated immune clearance prevents the virus from establishing widespread neuroinvasion or, critically, inhibits the *Thanatomicrobium vivificans* symbiotic organism from establishing its "bio-electrical conduit" within the brain.62 The precise mechanism could involve the HLA-B27 variant presenting a viral peptide that strongly activates a protective T-cell clone, or perhaps its misfolding properties (known to occur with HLA-B27) inadvertently create an environment unfavorable for viral replication or symbiont establishment.64

The utilization of the HLA-B27 variant, typically associated with autoimmune diseases, to confer immunity against *Mortiferum somniculosum* provides a plausible, albeit fictional, genetic component to survival. This narrative choice subverts the conventional understanding of genetic predispositions, where a variant linked to pathology in one context unexpectedly provides a significant advantage in another. This adds a layer of biological complexity to the survivor population and introduces a compelling element for the deep lore, as these individuals become crucial to any potential cure. The concept underscores that genetic variations, even those with known drawbacks, can have unforeseen benefits under extreme selective pressures.

### **IV. Deep Lore: Origin, Patient Zero, Societal Collapse, and Legacy**

The catastrophic emergence of *Mortiferum somniculosum* is not a natural evolutionary event but the result of a highly unethical and disastrous bioweaponry program. The intricate deep lore surrounding its origin, the identity of Patient Zero, the mechanisms of societal collapse, and the enduring legacy of survivors paints a grim picture of humanity's hubris and resilience.

#### **A. Origin as a Weaponized Bat-Borne Virus**

*Mortiferum somniculosum* originated within the clandestine laboratories of "Aethelred Pharmaceuticals," a fictional multinational corporation with a shadowy history of involvement in controversial bioweaponry research. This research was meticulously disguised as a legitimate endeavor into advanced neurodegenerative disease therapies and neuro-stimulant development, leveraging the dual-use nature of biotechnology.66 The company's scientific focus was on understanding and manipulating neural pathways, a field ripe for both therapeutic breakthroughs and weaponized applications.

The virus itself was developed from a benign, naturally circulating bat-borne virus found in *Miniopterus schreibersii* bats. Researchers at Aethelred Pharmaceuticals isolated a precursor virus, *Miniopterus somnivirus*, which, while neurotropic, caused only mild or asymptomatic infections in its natural host.13 Through advanced genetic engineering techniques, the *Miniopterus somnivirus* was weaponized. This involved enhancing its neurotropism to ensure rapid and widespread brain invasion, increasing its replication efficiency to overwhelm host defenses, and, most critically, integrating the gene sequence for the novel "Reanimin" protein. The original intent behind the "Reanimin" protein was to create a highly potent neuro-stimulant for military applications, designed to enhance combatant aggression and resilience to pain, potentially even overriding physiological limitations. However, the experimental design inadvertently resulted in the grotesque reanimation effect when combined with the engineered symbiotic microorganism, *Thanatomicrobium vivificans*, under conditions of host death. The accidental release of this weaponized pathogen occurred during a highly classified trial or a containment breach within one of Aethelred's remote, high-security BSL-4 facilities, highlighting the inherent risks and ethical dilemmas associated with dual-use biotechnology.66

#### **B. Patient Zero**

The individual identified as Patient Zero for the *Mortiferum somniculosum* outbreak was Dr. Aris Thorne, a brilliant but ethically compromised lead virologist at Aethelred Pharmaceuticals. Dr. Thorne was deeply immersed in the "Reanimin" protein integration experiments, driven by an obsessive desire to perfect the neuro-stimulant for its intended military applications.

His exposure was not a deliberate act of malice but a tragic consequence of scientific hubris and a lapse in biosafety protocols. During a critical phase of the "Reanimin" protein integration experiments, Dr. Thorne suffered an accidental self-inoculation, likely a needle stick injury or exposure through a compromised personal protective equipment (PPE) seal. In the initial hours and days post-exposure, Dr. Thorne, a highly intelligent and self-aware individual, would have recognized the early, flu-like symptoms and the subtle, yet disturbing, onset of hyperesthesia and irritability. However, driven by his scientific curiosity, a profound sense of denial regarding the potential severity of the infection, and the intense pressure of his classified research, he chose to self-monitor rather than report the incident. He likely dismissed the symptoms as stress-induced illness or a common lab-acquired infection, a known occupational hazard in virology research. This critical decision to keep his exposure secret and continue his work inadvertently facilitated the early, undetected spread of *Mortiferum somniculosum* beyond the confines of the laboratory, setting the stage for the global catastrophe.

#### **C. Societal Collapse**

The rapid and devastating societal collapse following the emergence of *Mortiferum somniculosum* was a direct consequence of the virus's unique biological characteristics and the systemic failures of global preparedness.

Initial governmental and public health responses were critically hampered by the virus's exceptionally high R0 and its stealthy asymptomatic shedding period. Governments and health organizations were caught entirely off guard; the initial flu-like symptoms led to widespread misdiagnosis as a severe influenza strain or a novel form of encephalitis, delaying the implementation of effective containment strategies.1 By the time the true nature of the Zombification Syndrome became apparent—with its terrifying neurological degeneration and reanimation—the virus had already achieved exponential spread across continents.

The characteristic hyper-aggression of infected individuals, coupled with their rapid zombification, swiftly overwhelmed law enforcement and military forces worldwide. Traditional methods of maintaining order proved ineffective against a relentless, seemingly tireless, and pain-insensitive adversary. As the infection spread, essential services collapsed. Power grids failed due to a lack of personnel and maintenance, water treatment facilities ceased operations, and food supply chains disintegrated as workers became infected or fled in panic.1 The widespread illness and fear led to mass absenteeism, rendering even the most resilient societies incapable of functioning. The result was an unprecedented global catastrophe, culminating in the zombification of approximately 63% of the world's population.

In the ensuing chaos and desperation, a profound psychological shift occurred within the surviving human population. The inexplicable and terrifying nature of the reanimated dead, coupled with the complete breakdown of established social and scientific frameworks, led to the emergence of diverse and often radical belief systems. Cults began to form, some worshipping the reanimated as a new form of life, a divine judgment, or even a pathway to immortality. Others sought radical "cures" or protection through ritualistic means, abandoning scientific understanding for superstitious practices.68 This phenomenon reflects a plausible human response to overwhelming, inexplicable catastrophe, where traditional structures fail, and individuals seek meaning and control in the face of existential dread.

#### **D. Legacy of Survivors**

In the wake of the collapse, the remnants of humanity coalesced into scattered enclaves, often in remote, defensible locations or within fortified urban ruins. These communities are characterized by a renewed emphasis on survival skills, resourcefulness, and a profound, often brutal, understanding of the "reanimated" behavior. Survivors have adapted to a world where the living are few and the dead are many.

A significant aspect of these enclaves is the presence of the 5% genetically immune population. These individuals, carriers of the HLA-B27 variant, form the core of many survivor groups, their natural resistance making them invaluable. However, their "privileged" status also presents profound ethical dilemmas, as their unique biology is seen as the only hope for a cure, leading to potential exploitation and social stratification.

The scientific understanding of the virus's origin and mechanisms has largely been lost or distorted over time. In many communities, the reanimated are no longer referred to by their scientific designation but by various colloquial terms: "the Shambling Dead," "the Sleepwalkers," or "the Revenants." The complex biological reality has been reinterpreted through the lens of folklore and new mythologies, reflecting how profound and traumatic events can be transmuted into cultural narratives over generations.68 This loss of scientific knowledge, combined with the desperate need for survival, shapes the new societal structures and belief systems.

Survivors have also developed new skills essential for navigating their perilous world. For instance, some have honed their auditory senses to a remarkable degree, developing a form of human echolocation. By actively creating sounds, such as clicks or shouts, and interpreting the returning echoes, individuals can detect objects, identify their location, size, and density, and navigate safely in low-light or visually obscured environments.70 This adaptation leverages the brain's neuroplasticity, remapping visual cortical areas to process auditory information, a phenomenon observed in blind individuals.71 This ability provides a critical advantage in a world where direct visual engagement with the reanimated is often too dangerous.

### **V. Therapeutic Challenges and Ethical Dilemmas**

The unique biological characteristics of *Mortiferum somniculosum* and its symbiotic reanimation mechanism present formidable challenges to the development of a cure, leading to profound ethical dilemmas that test the very fabric of surviving human society.

#### **A. Plausible Cure Angle: Rare Blood Plasma Protein**

The most plausible, albeit extremely limited, therapeutic avenue identified involves a rare, naturally occurring blood plasma protein, provisionally named "Vivicase." This protein is found exclusively in the blood plasma of the 5% of the human population that possesses the genetic immunity conferred by the HLA-B27 variant.62

Vivicase is hypothesized to be a potent protease or enzyme that specifically targets and degrades the *Mortiferum somniculosum* "Reanimin" protein. By neutralizing the "Reanimin" protein, Vivicase prevents the *Thanatomicrobium vivificans* symbiotic microorganism from establishing its critical "bio-electrical conduit" within the virally damaged brain, thereby inhibiting the reanimation process. While Vivicase may possess some ancillary antiviral properties against *Mortiferum somniculosum* itself, its primary and most crucial function is in preventing the horrifying post-mortem motility and aggression that defines the zombification syndrome.

The challenges associated with Vivicase are immense. It is an extremely rare protein, found only in a minute fraction of the population, making its natural supply inherently limited. Furthermore, current biotechnological capabilities have proven insufficient for its large-scale synthesis, necessitating constant harvesting from immune individuals.72 This reliance on a scarce biological resource from a specific human subset creates an unsustainable long-term solution and forms the bedrock of the ethical quandaries.

#### **B. Ethical Dilemmas**

The necessity of Vivicase as the only known deterrent to zombification gives rise to a series of profound ethical dilemmas:

* **Exploitation of Immune Individuals:** The most pressing concern is the potential for the exploitation of the genetically immune population. The desperate need for Vivicase creates a moral imperative to harvest plasma from these individuals, which could easily devolve into forced donations, involuntary medical procedures, and the establishment of a two-tiered society where the immune are treated as mere biological resources rather than autonomous individuals.72 This raises fundamental questions about bodily autonomy, human rights, and the potential for genetic discrimination.
* **Resource Allocation:** With an extremely limited supply of Vivicase, difficult decisions regarding its allocation become unavoidable. Who receives the precious treatment? Should it be prioritized for children, essential personnel (e.g., medical, military, infrastructure maintenance), or those with mild symptoms who might still be saved? Or should it be distributed equitably, regardless of societal role, even if it means fewer overall lives are saved? These choices would inevitably lead to intense societal conflict and moral compromises.
* **Weaponization Risk:** The existence of a protein capable of neutralizing the reanimation effect of *Mortiferum somniculosum* also carries the inherent risk of further weaponization. Vivicase could be reverse-engineered or modified to create new bioweapons, or to serve as a counter-agent in future biological warfare scenarios, perpetuating the cycle of bio-conflict.66
* **Long-term Sustainability:** The inability to synthesize Vivicase at scale raises fundamental questions about the long-term survival of humanity. A sustainable future cannot be built on the constant depletion of a rare natural resource from a small, vulnerable population. This challenge forces a re-evaluation of humanity's relationship with biotechnology and its limits.

#### **C. Failure of CRISPR-Inspired Gene Therapy**

Despite the rapid advancements in genetic engineering, particularly those inspired by CRISPR technology, attempts to develop a gene therapy for *Mortiferum somniculosum* have universally failed. CRISPR-Cas9, a revolutionary tool for precise genome editing, allows for the removal, addition, or alteration of genes in living cells.74 However, its effectiveness against *Mortiferum somniculosum* is negated by a critical viral mechanism: the virus's highly efficient and widespread integration into the host cell's DNA.

Similar to retroviruses like HIV, *Mortiferum somniculosum* possesses a highly evolved viral integrase enzyme.76 This enzyme facilitates the rapid and pervasive incorporation of the viral genome directly into the host cell's DNA.76 This integration is not limited to specific, easily targetable sites but occurs widely throughout the host genome, transforming infected cells into viral factories and rendering their genetic material irrevocably altered.

The implication of this pervasive viral integration is profound: any attempt at CRISPR-inspired gene therapy to remove or neutralize the viral genome would necessitate widespread and indiscriminate editing of host DNA. Such an intervention would inevitably lead to catastrophic cellular damage, widespread genomic instability, and the induction of further, potentially malignant, mutations across the host's cells.74 The therapeutic window for CRISPR would be non-existent, as the cure would be as destructive, if not more so, than the disease itself. This fundamental limitation underscores the virus's unique biological resilience and the inadequacy of even the most advanced genetic tools against it, reinforcing the bleak outlook for a conventional cure and emphasizing the critical reliance on the rare Vivicase protein.

### **VI. Conclusions**

The profile of *Mortiferum somniculosum* presents a hypothetical pathogen of extreme virulence and transmissibility, meticulously constructed by leveraging both realistic biological mechanisms and plausible fictional amplifications. The virus's systematic nomenclature, grounded in ICTV guidelines and Latin etymology, immediately establishes its scientific credibility. Its classification within the *Mononegavirales* order, alongside known neurotropic and highly pathogenic viruses like rabies and Ebola, provides a strong biological foundation for its devastating effects on the central nervous system. The detailed virion structure, including the novel "Reanimin" protein, and its origin from a specific bat reservoir, *Miniopterus schreibersii*, further anchor the fictional elements in a framework of biological plausibility.

The pathogenesis of the Zombification Syndrome, progressing through distinct phases of incubation, neurological degeneration, and symbiotic reanimation, illustrates a terrifyingly coherent biological assault. The initial hyperesthesia, followed by severe basal ganglia and limbic system dysfunction, culminating in a post-mortem motility driven by the engineered ectosymbiont *Thanatomicrobium vivificans*, creates a "zombie" phenotype that is biologically driven rather than supernatural. This detailed neurobiological underpinning, where hyper-aggression and primitive motor functions are direct consequences of specific brain damage and microbial manipulation, elevates the scientific depth of the concept.

Epidemiologically, *Mortiferum somniculosum* represents a "perfect storm" for global catastrophe. Its exceptionally high basic reproduction number (R0), coupled with dual-mode transmission (bodily fluids and highly efficient aerosols) and a significant period of asymptomatic shedding, ensures rapid, undetectable, and uncontrollable spread. This combination directly explains the swift societal collapse and the staggering zombification rate of 63% of the world's population. The existence of a genetically immune 5% of the population, linked to the HLA-B27 variant, introduces a compelling element of biological resilience, albeit one fraught with profound ethical implications.

The deep lore surrounding the virus's origin as a weaponized pathogen from "Aethelred Pharmaceuticals" and its accidental release by Patient Zero, Dr. Aris Thorne, highlights the inherent dangers of unchecked scientific ambition and the dual-use dilemma in biotechnology. The subsequent societal collapse, characterized by the breakdown of essential services and the emergence of new cultic belief systems, underscores humanity's vulnerability to existential threats that transcend conventional preparedness. The legacy of scattered survivor enclaves, adapting to a new reality and developing novel survival skills like human echolocation, paints a stark picture of post-apocalyptic existence.

The therapeutic landscape for *Mortiferum somniculosum* is bleak, centered on the extremely rare blood plasma protein, Vivicase, derived solely from the immune population. This dependence creates an intractable ethical dilemma regarding the exploitation of immune individuals, resource allocation, and the potential for further weaponization. The categorical failure of CRISPR-inspired gene therapies, due to the virus's efficient and widespread integration into host DNA, further emphasizes the formidable biological resilience of *Mortiferum somniculosum* and the limitations of even advanced genetic tools against such a threat.

In essence, this report serves as a rigorous thought experiment on the vulnerabilities of global health systems to highly adaptable and engineered pathogens. It underscores the critical importance of responsible governance in biotechnology, the ethical quandaries inherent in advanced scientific pursuits, and the profound potential for societal transformation—both destructive and adaptive—in the face of existential threats. The delicate balance between scientific advancement and its responsible application emerges as a paramount concern for the future of humanity.

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