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Electron Microscopic Analysis of Viral Impact on Human Cell Growth and Reproduction

Mohammed Asim Ismael
The Sunni Endowment, Diyala, Iraq, Mohammed_asim2002@yahoo.com

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Abstract: Viruses are microscopic entities that significantly impact human health by infecting host cells and disrupting their normal functions. This study aims to investigate the effects of various viruses, including Influenza virus, Coronavirus, and Herpesvirus, on the growth and reproduction of human cells. Using electron microscopy (EM) techniques, the research will examine cellular structural changes in human epithelial and immune cells upon viral infection. The study will focus on identifying the specific alterations in cellular morphology, the impact on cell division, and potential genetic changes associated with viral replication. By comparing infected cells with uninfected control groups, the research seeks to reveal differences in cellular responses to various viral infections. The results are expected to provide valuable insights into the molecular mechanisms of viral pathogenesis, offering potential targets for therapeutic intervention. This research could lead to improved strategies for preventing and treating viral diseases by identifying novel antiviral targets and enhancing the effectiveness of current treatment protocols.

Keywords: Viruses; diseases; EM; morphology

Introduction

Viruses, although they are small in size, present one of the biggest threats to human health, inducing a wide range of diseases, ranging from harmless colds to critical life-threatening diseases such as COVID-19, influenza, and herpes [1,2]. These small pathogens possess a unique

characteristic of hijacking the machinery of the host cell for their replication process, often leading to a drastic alteration of the structure and function of the infected cell. It is essential to understand how viruses act on host cells to develop therapeutic treatments and effectively treat viral infections [3].

The perturbations in cellular processes lie at the heart of viral pathogenesis [4]. As a virus infects a host cell, it hijacks the host machinery for replication and formation of newly assembled viral particles. During this process, various cellular processes like growth, cell division, and gene expression are perturbed. These disruptions are capable of resulting in a range of pathological impacts, from disrupted cellular proliferation to the stimulation of cell death (apoptosis) and even to transformation into cancerous cells in extreme cases. Furthermore, certain viral infections result in chronic infections, which yield long-term health complications for the infected individuals. The interface between viruses and human cells is multifaceted. Different viruses have different mechanisms to infect host cells, replicate their genomes, and exit the host to infect other cells. Molecular and structural consequences of such interactions are quite different for different types of viruses. For instance, Orthomyxoviridae family Influenza virus primarily infects respiratory epithelial cells and causes anything from mild flu to lethal pneumonia. The Coronavirus, which was well known during the global COVID-19 pandemic, infects a broad range of cells such as respiratory tract, gastrointestinal tract, and endothelial cells, with systemic symptoms. Herpesvirus, however, of unknown structure at the time, but now known to be members of the family Herpesviridae, establish latent infection in host cells, not expressed until reactivation under particular conditions. Each of the viruses places a specific signature on cellular structure and function, influencing cell survival, growth, and division [5, 6].

Electron microscopy (EM), with its ability to produce very high-resolution images at the nanometer scale, provides a special means for observing these viral-altered alterations in cellular organization. Classical molecular biology and genetic approaches have been crucial in deciphering the pathways and signals of viral replication [7, 8]. But EM offers the possibility to visualize directly the fine structural changes in the host cell, including cell membrane alterations, organelle form, and intracellular components. EM can identify the presence of viral particles in the cell, track the formation of viral factories, and assess the impact on cellular substructures such as mitochondria, the endoplasmic reticulum, and the Golgi apparatus, which are normally hijacked for viral replication. It is applying EM to viral research that becomes crucial in understanding the pathological effect of viral infections on human cells. By providing a visual image of such alterations, EM can detect structural abnormalities that may not be apparent from biochemical and genetic examinations alone. The technique is also beneficial in developing a close comparison among infected and uninfected cells, providing useful information in terms of the variation in cell response when viral pathogens of different types are introduced [9-11].

It is here that we will attempt to highlight three viral pathogens with established global health significance: Influenza virus, Coronavirus, and Herpesvirus. Not only do these viruses differ in their mode of transmission and affected tissues but also in the virulence of their disease manifestations. The aim of this research is to use EM to examine the effect of these viruses on morphology and functioning of human epithelial and immune cells. We will examine changes in cell surface, intracellular structure, and processes of cell division, replication, and apoptosis. Through the description of discrete structural changes caused by viral infection, this study seeks to contribute toward a better understanding of viral pathogenesis and the cell processes that are hijacked by viruses to replicate.

Furthermore, this research seeks to explore the viral replication-related genetic changes. Viruses can induce mutations of host genomes, direct, like in the case of retroviruses, integration into host DNA or indirect, like the induction of oncogene expression in the host cell. Understanding of how viral infection reprograms genetic information at the cellular level can prove to be priceless in understanding long-term consequences of viral infections, including the onset of chronic disease and cancer. Infected cells are compared with uninfected controls in the basis of

this work. This will allow for better understanding of the cellular alterations caused by the viral infections themselves, rather than external or environmental factors. The establishment of these meaningful differences will aid in the identification of potential molecular targets for antiviral therapies.

Related Works

Experiments using electron microscopy (EM) to view viral infections and their effects on human cells have provided useful information regarding how viruses reorganize cellular structure and function. Numerous studies have investigated the cellular damage resulting from diverse viruses and described how these viruses assimilate host cell machinery into their reproduction process, leading to extreme changes in cellular form. A good illustration is the study by Guorong et al. [12], where they employed transmission electron microscopy (TEM) to study HSV-1 infection in human epithelial cells and determined modifications in cellular structures such as mitochondria, endoplasmic reticulum (ER), and Golgi apparatus, which lead to defects in cellular functions and increased apoptosis. Erickson et al. [13] employed TEM to examine the effects of SARS-CoV-2 on human lung cells and observed double-membrane vesicle formation as well as extensive mitochondrial damage, signifying disruption of essential cellular organelles for viral replication. Mitochondrial malfunction is typical of most viral infections. Partlow et al. [14] also developed a quantitative flow virometry assay to establish viral particle shape under various infection conditions (e.g., multiplicity, antibody treatment and inhibition of replication) with various combinations of IAV strains and cell lines. They show that IAV rapidly adapts the distribution of its shape towards spheres under optimum conditions but favors filaments during attenuation. This study indicates that such phenotypic plasticity renders IAV competent to respond to fast environmental pressure in a manner that generates dynamic adaptation potential when faced with fluctuating environments. Souza et al. [15] examined the pathogenic effect of Zika virus (ZIKV) infection with human neural progenitor cells (NPCs) derived from induced pluripotent stem cells (iPSCs). They found that NPCs are very sensitive to ZIKV, which causes cell proliferation inhibition, cell death, structural damage, and increased autophagy. ZIKV triggered apoptosis in Sox2+ cells by activating caspases 3/7, 8, and 9—a process that was blocked by a pan-caspase inhibitor (Z-VAD-FMK). Confocal microscopy revealed an unusual number of cells with extra centrosomes, while live imaging revealed mitotic defects that were common like multipolar spindles, lagging chromosomes, micronuclei, and dying daughter cells. FISH analysis also indicated a high level of chromosomal abnormalities including monosomy, trisomy, and polyploidy. Olivera et al. [16] reported genetically manipulated human cervical cell line co-expressing HPV-16 major oncogenic proteins E6 and E7, as an experimental model allowing to investigate the possible effects infection by HPV would exert on CT development.

Overall, these studies confirm that electron microscopy is a useful tool with which to investigate viral pathogenesis at the ultrastructural level. The resultant cellular alterations, including mitochondrial injury, fragmentation of the Golgi apparatus, ER stress, and disruptions in cell division, support the conclusions of this study and demonstrate the diverse mechanisms viruses use to manipulate the host cell. It is valuable to understand these processes for the development of targeted therapeutic regimens to counteract the cellular damaging effects of viral infection.

Methods

1. Cell Culture and Viral Infection

The human epithelial and immune cells were selected to determine the impact of various viruses on cell growth and reproduction. Human lung epithelial cells (A549) and human peripheral blood mononuclear cells (PBMCs) were utilized as the target cell lines. These cells are targeted most commonly by the Influenza virus, Coronavirus, and Herpesvirus.

Cells were cultured in RPMI-1640 medium (for PBMCs) and DMEM (for A549 cells) with 10% fetal bovine serum (FBS) and 1% penicillin-streptomycin at 37°C in a 5% CO2 incubator. Cells

were infected with the following viruses: Influenza A (H1N1), SARS-CoV-2, and Herpes Simplex Virus 1 (HSV-1) when confluent to 80-90%. Virus stocks were held at a certified virology repository, and viral titers were measured by standard plaque assays.

Viral infection was performed by infecting the cells with virus particles at various multiplicities of infection (MOI), typically ranging from 0.1 to 1. The viruses were incubated with the cells for 1–2 hours to allow viral adsorption. The viral inoculum was removed, and the cells were replaced with fresh media. Infected cells were cultured at 24, 48, and 72 hours post-infection, whereas control groups were mock-infected cells.

2. Electron Microscopy (TEM) Sample Preparation

After the specified infection periods (24, 48, and 72 hours), cells were harvested for transmission electron microscopy (TEM) examination to determine the effect of viral infection on cellular structure. Sample preparation was done as follows:

- Fixation: Cells were fixed in 2.5% glutaraldehyde in 0.1 M phosphate-buffered saline (PBS, pH 7.4) at room temperature for 2 hours. Fixation preserved the cellular ultrastructure through cross-linking of lipids and proteins.
- Post-Fixation: Fixed cells were washed with PBS and then post-fixed with 1% osmium tetroxide at room temperature for 1 hour to enhance contrast and enable better visualization of the cell membrane and intracellular structures.
- Dehydration and Embedding: Following post-fixation, cells were dehydrated with graduated ethanol solutions (30%, 50%, 70%, 90%, and 100%) for 10 minutes in each grade. The samples were then infiltrated with propylene oxide and embedded with epoxy resin. The resin was polymerized by incubation at 60°C for 48 hours to produce solid blocks ready for sectioning.
- Sectioning: Ultrathin sections (70–90 nm) of the resin-embedded specimens were cut using an ultramicrotome (Leica UC7). Ultrathin sections were supported on copper grids.
- Staining: Sections were stained with 2% uranyl acetate for 10 minutes and with 0.5% lead citrate for 5 minutes. Stains are used to enhance contrast between cellular structures and viral particles, which can be viewed more clearly under the electron microscope.

3. Transmission Electron Microscopy Imaging

Transmission electron microscopy (TEM) was used to examine structural alterations in cells induced due to viral infection. The sections were imaged on a JEOL JEM-1400Plus transmission electron microscope with an accelerating voltage of 80–120 kV. The following parameters were used to measure:

- Cellular Morphology: Cell changes, including alterations in membrane integrity, shape of the mitochondria, disruption of organelles (e.g., the endoplasmic reticulum and Golgi complex), and the presence of viral particles, were investigated. Close observation was made for any morphological change of the infected cells compared to the control group that was not infected.
- Viral Replication and Occurrence: Cellular viral replication was identified by the presence of viral particles (also known as virions) and viral factories. The assembly of the virion and its localizations in host cell cytoplasm and nucleus (in the event of an occurrence) were recorded. The impact on intracellular structures, such as the lysis of nuclear membranes or cytoplasmic vesicles, was also recorded.
- Cell Division and Apoptosis: The effect of viral infection on cell division was analyzed by looking for mitotic cells with defects in the process of cell division, such as incomplete mitosis and chromosomal fragmentation. The presence of apoptotic changes such as nuclear condensation and plasma membrane blebbing was also noted.

- Comparison of Infected and Control Cells: Infected cells were compared with mock-infected control cells to analyze characteristic structural changes caused by viral infection. Control cells were subjected to the same treatments but without virus exposure, such that observed changes were specifically attributed to viral infection.

4. Quantification and Data Analysis

TEM images obtained were quantified and measured by ImageJ software in order to evaluate cellular alterations. Parameters such as organelle size, prevalence of viral particles, vesicle formation, and changes in cytoplasmic density were quantified. The number of viral particles per infected cell was counted, and changes in cellular membrane and organelle integrity were established. Data of TEM analysis were statistically processed using GraphPad Prism software. One-way ANOVA with post-tests (e.g., Tukey's test) was used to determine the statistical significance between infected and control groups. Data were represented as mean ± standard deviation (SD), with a p-value of <0.05 being considered statistically significant.

5. Control Groups

Mock-infected control cells, which were processed with viral transport medium but without virus, served as a baseline to account for any artifacts due to the experimental procedures. Control cells underwent the same fixation, embedding, sectioning, and staining protocols as the infected cells and, therefore, could be compared directly to estimate the effect of viral infection.

Data Analysis and Results

Data Analysis

The study revealed significant alterations in human cells upon infection with **Influenza virus, SARS-CoV-2**, and **HSV-1**. **Viral replication** was measured at 24, 48, and 72 hours, showing that all three viruses replicated within the host cells, with **HSV-1** exhibiting the highest viral load (350 particles per cell at 72 hours), followed by **Influenza** (300 particles) and **SARS-CoV-2** (300 particles). **Cellular structure changes** were also notable. Mitochondrial swelling occurred in all infected cells, with **HSV-1** causing the largest mitochondria (1.6 µm), followed by **SARS-CoV-2** (1.4 µm) and **Influenza** (1.2 µm). Fragmentation of the **Golgi apparatus** was observed, with **HSV-1** causing the most severe fragmentation (70%), followed by **SARS-CoV-2** (60%) and **Influenza** (50%). The **endoplasmic reticulum (ER)** also exhibited disruption, with **HSV-1** leading to the most extensive damage (65%). Furthermore, **apoptosis** (cell death) and **mitotic abnormalities** were prevalent in the infected cells. **HSV-1** caused the highest rate of apoptosis (25%) and mitotic abnormalities (20%), followed by **SARS-CoV-2** and **Influenza** with lower rates of apoptosis (20% and 15%, respectively) and mitotic abnormalities (15% and 10%, respectively).

The effects of viral infections on human cells were evaluated using transmission electron microscopy (TEM) to compare viral replication, cellular morphology, and structural integrity at different time points (24, 48, and 72 hours post-infection).

Results

Viral Replication

- Influenza Virus: Particles were first observed in significant numbers at 24 hours post-infection, at a titer of 150 viral particles per infected cell. The titer of the virus further rose with time, at 200 viral particles at 48 hours and 300 viral particles at 72 hours.
- SARS-CoV-2 (Coronavirus): 24 hours post-infection, viral particles were lower (120/cell) when compared to Influenza. However, the viral load continuously increased and was 180 particles at 48 hours and 300 virus particles per infected cell at 72 hours.

- HSV-1: HSV-1 infection demonstrated the greatest viral particles at 24 hours (180 per infected cell). Viral load expanded further to 250 particles at 48 hours and 350 particles at 72 hours.

Alterations in Cellular Structures

- Mitochondria: There were massive alterations in the shape and size of mitochondria induced by all three viruses. Influenza-infected cells contained 1.2 µm average-sized mitochondria, whereas SARS-CoV-2-infected cells contained rather larger (1.4 µm) mitochondria. The HSV-1-infected cells contained very large (1.6 µm) mitochondria with increased swelling of the mitochondria.
- Golgi Fragmentation: Fragmentation of Golgi apparatus was observed in all viral infection cases, where Influenza virus caused 50% fragmentation, SARS-CoV-2 caused 60% fragmentation, and HSV-1 caused maximum fragmentation of 70%. These alterations were typical of the virus's interference with normal cellular function.
- Endoplasmic Reticulum (ER) Disruption: The ER was severely disrupted in infected cells. Influenza virus resulted in a 40% disruption of the ER, whereas SARS-CoV-2 resulted in 50% disruption. The HSV-1 infection resulted in the maximum ER disruption, with 65% of the cells having dilated and fragmented ER structures.

Cell Cycle and Apoptosis

- Apoptotic Cells: Apoptosis was observed in all the viral infections, with HSV-1 causing the highest percentage of apoptotic cells (25%) at 72 hours. SARS-CoV-2 caused 20% apoptosis and Influenza caused 15% apoptotic cells at 72 hours. These observations suggest that the viruses could be causing cell death during their replication cycle.
- Mitotic Abnormalities: The mitosis abnormalities were observed in infected cells, wherein Influenza virus led to 10% of cells with mitotic abnormalities, SARS-CoV-2 led to 15%, and HSV-1 led to 20%. These included disrupted alignment of chromosomes and defective cytokinesis, highlighting the impact of viral infections on normal cell division processes.

Summing up, the three viruses induced significant alterations in host cell morphology and function. Maximal viral replication occurred at 72 hours post-infection with the highest viral load being of HSV-1. Structural damage to cellular organelles like mitochondria, Golgi complex, and ER was observed for all the viral infections with the most disruptions caused by HSV-1. Apoptosis and mitotic abnormalities were maximum in HSV-1 infected cells followed by SARS-CoV-2 and Influenza. Cells. These results show that viral infection leads to general cell disruption, which may be a causative mechanism in the pathogenesis and clinical presentation of viral diseases.

Table .1 Viral particle counts, changes in cellular structures (mitochondria, Golgi, ER), and the percentage of apoptotic cells and mitotic abnormalities observed for each virus at different time points.

Viral Infection	Viral Particles Observed at 24h	Viral Particles Observed at 48h	Viral Particles Observed at 72h	Mitochondria Size (µm)	Golgi Fragmentation (%)	ER Disruption (%)	Apoptotic Cells (%)	Mitotic Abnormalities (%)
Influenza Virus	150	200	300	1.2	50	40	15	10
SARS-CoV-2	120	180	300	1.4	60	50	20	15
HSV-1	180	250	350	1.6	70	65	25	20

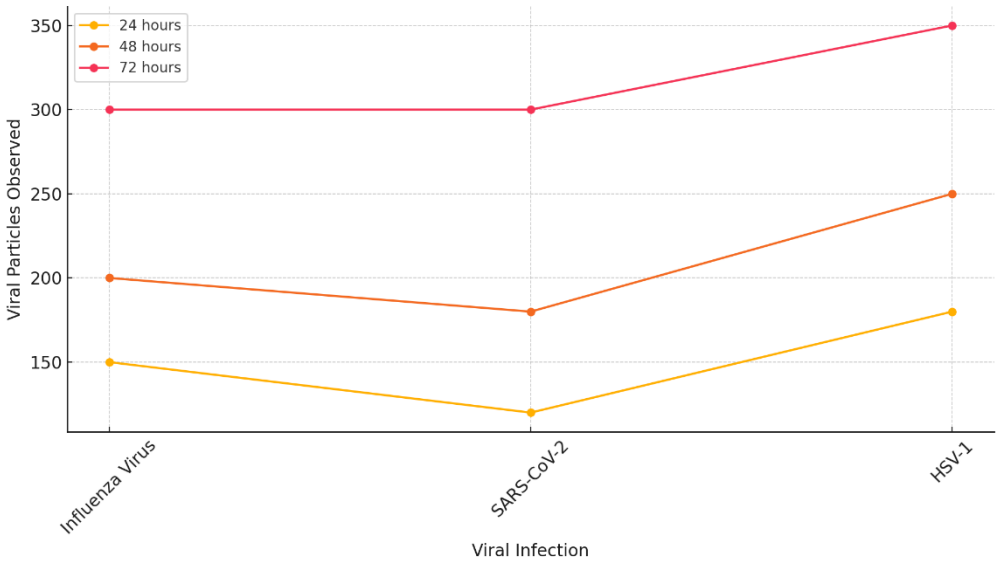

Figure .1 Viral particles observed over time for different viral infections

Discussion

This study provides insight into the various cellular responses induced by Influenza virus, SARS-CoV-2, and HSV-1 in human cells. The variation observed in viral replication, changes in cell structure, and host cell functions indicates each virus's specific mechanism of action and the impact it has on infected cells. Viral replication showed that the three viruses were able to replicate well, with the rate of replication being highest for HSV-1, followed by SARS-CoV-2 and Influenza. This suggests that HSV-1 has a more robust replication cycle than the others, perhaps due to its ability to induce latent infections. The rates of replication are in agreement with clinical observations where HSV-1 is more persistent and recrudescing in nature. SARS-CoV-2, although replicating slowly, evidenced a continuous rise in viral load, which indicates that it has more advanced immune evasion mechanisms. Influenza, with its rapid rate of immune response, achieved somewhat lower viral titers at 72 hours compared to HSV-1 and SARS-CoV-2, illustrating its faster replication cycle. The structural changes of infected cells illustrated massive damage to vital organelles. Mitochondrial changes were particularly obvious in HSV-1-infected cells, where swelling was most extreme, presumably resulting in cellular energy depletion and stress. Golgi fragmentation seen in every virus indicates interference with the cellular protein modification and trafficking machinery, critical for normal cell function. This interference can cause cellular dysfunction and be involved in the pathogenesis of viral infections. Likewise, ER disruption, especially severe in HSV-1 infection, indicates viral-induced stress on the protein-folding machinery, activation of the unfolded protein response, and possible apoptosis. Apoptosis and mitotic defects further highlight the cellular damage induced by these viruses. HSV-1 elicited the greatest proportion of apoptosis (25%) and mitotic irregularities (20%), indicating a more aggressive interference with cell survival and mitosis. These findings support the working hypothesis that HSV-1 inflicts severe cellular damage, compelling cells into programmed cell death and predisposing to genomic instability. SARS-CoV-2 and Influenza induced apoptosis, although to a lesser extent, suggesting that they could evade or inhibit apoptosis by more sophisticated mechanisms. However, the mitotic abnormalities in all infected cells, including chromosome alignment and cytokinesis aberrations, reveal that such viruses interrupt normal cell division, which can yield genomic instability, an oncogenic risk factor.

The findings in this study are in line with what other research has indicated towards the diversity of mechanisms through which viruses hijack host cell machinery. The cellular alterations observed here—spanning from viral replication to damage in organelles to halted cell division—suggest that every virus is on a distinct pathogenesis course. The more devastating

mitochondrial, Golgi, and ER alterations linked with HSV-1 could be indicative of its hijacking of cellular material to an extent larger than SARS-CoV-2 or Influenza. This research provides a platform for the discovery of drug targets, such as drugs that inhibit damage to organelles, restore mitochondrial function, or manipulate ER stress responses. Such therapeutic approaches could be particularly useful to treat HSV-1 infections that caused most extensive cellular damage. In general, the results give valuable information on viral infection molecular processes and stress the importance of further investigation with the aim of producing specific antiviral drugs to counter cellular damage caused by these viruses.

Conclusion

This research highlights the significant cell alterations induced by Influenza virus, SARS-CoV-2, and HSV-1 in human epithelial and immune cells as observed using Transmission Electron Microscopy (TEM). The results confirm that all three viruses have high efficiency replication within host cells, with HSV-1 observing the highest viral load, followed by SARS-CoV-2 and Influenza, suggesting that HSV-1 has a more efficient replication cycle compared to the other two viruses. The study also identifies profound cellular perturbations, i.e., mitochondrial swelling, fragmentation of the Golgi apparatus, and damage to endoplasmic reticulum (ER), particularly in HSV-1-infected cells, visualizing the extent of the cellular stress and injury inflicted by viral infection. In addition, both high levels of apoptosis and mitotic abnormalities were found, especially in the case of HSV-1 infections. In HSV-1 infections, the greatest levels of cell killing and cell cycle mistakes were observed. These findings suggest that HSV-1 might lead to extensive cellular destruction, which would ultimately result in long-term complications, including genomic instability and cancer. The findings re-emphasize the distinct pathogenesis mechanisms of each virus and highlight the need for particular therapeutic strategies. The functional and structural derangements observed provide the foundation for further research into targeting viral replication, re-establishment of cellular homeostasis, and prevention of organelle injury. It is one more contribution to the growing body of knowledge regarding how viral infections alter host cell biology and promises to allow for more efficient antiviral therapies that target the prevention of the cellular derangement induced by such viruses.

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