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# Vimentin Cytoskeleton Rearrangements Induced by PRRSv Infections

## Reorganización del Citoesqueleto de Vimentina Inducido por Infecciones por PRRSv

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**SUMMARY:** Swine respiratory and reproductive syndrome (PRRS) is a highly infectious disease that has a significant negative economic impact on the Mexican pig farming industry. The virus responsible for PRRS relies on the functioning of cells' cytoskeleton, and vimentin is thought to play a role in the virus's replication and transport. Therefore, this study aimed to examine the vimentin cytoskeleton's morphology in MARC-145 cells infected with the PRRS virus, in order to create a cell morphology model that could help identify infection-induced changes in vitro. The results revealed altered patterns of vimentin expression in the perinuclear zone and the formation of syncytia, clusters of cells containing the same protein, starting from 24 hours post-infection (hpi). The study concluded with the destruction of the cell monolayer at 72 hpi, possibly due to the depolymerization of the cytoplasm, leading to a reorganization of vimentin. This reorganization could be linked to the virus's infectivity and underscores the importance of establishing a morphological model of vimentin for the study of PRRSv.

**KEY WORDS: Cytoskeleton; Intermediate filaments; PRRS; Cell culture.**

## INTRODUCTION

The swine industry is greatly affected by viral diseases, leading to annual economic losses of over 20 million dollars in Mexico. Swine Influenza (PI), Porcine Circoviruses (PCV), Porcine Epidemic Diarrhea (PED), and Porcine Reproductive and Respiratory Syndrome (PRRS) are the main causes of these losses (Castro, 2020). PRRS is a highly infectious swine disease found worldwide (Jackson & Cockcroft, 2007), caused by Betaarterivirus suid 1 and Betaarterivirus suid 2 (Zimmerman *et al*., 2019; International Committe on Taxonomy of Viruses, 2023). It is a disease caused by an enveloped RNA virus that grows in serum, homogenized lymphoid and lung tissues, and cell cultures, especially in the MARC-145 cell line (Zimmerman *et al*., 2019).

During an infection, the virus multiplies in monocyte-derived cells, which act as cellular receptors essential for virus binding, internalization, and replication. The virus then spreads to alveolar and intravascular pulmonary macrophages, monocyte-derived macrophages in lymphoid tissues, and to a lesser extent in dendritic cells through a receptor-mediated endocytosis mechanism. This mechanism triggers the release of proinflammatory cytokines such as TNF-alpha, IL-1, and IL-6 (Ding *et al*., 2012; Zimmerman *et al*., 2019). The virus has six receptors: heparan sulfate, CD163, vimentin, sialoadhesin, CD151, and CD169. Some cell lines like BHK-21, PK-15, and CHO-K1, which were previously not susceptible to the virus, can become infected when they express these receptor proteins (Shi *et al*., 2015; Qingzhan & Dongwan, 2015).

The cytoskeleton is a network of protein filaments composed of actin filaments, microtubules, and intermediate filaments. Vimentin filaments, which are part of the intermediate filaments, are expressed in mesenchymal cells and various cell lines, forming transcellular networks that provide support and strength to the cells (Alberts *et al*., 2022). There have been reports of viruses interacting with cytoskeletal filaments, leading to rearrangements of actin

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filaments caused by viruses such as SARS-CoV-2, influenza virus, picornavirus, and vesicular stomatitis virus (Acharya *et al*., 2022). This can result in disorganization of actin filaments and increased stress fibers in uninfected neighboring cells (Kanlaya *et al*., 2009), changes in cell morphology including rounding of cells, possibly due to alterations in actin filaments caused by viruses such as pseudorabies virus (Van Minnebruggen *et al*., 2002), abnormal organization of stress fibers caused by dengue virus (Suttitheptumrong *et al*., 2021), and degradation of stress fibers caused by Marek's virus (Schumacher *et al*., 2005). Other effects include stress fiber reduction (rubella virus) and actin filament disorganization (SV40 virus) (Nunbhakdi *et al*., 2003), as well as the formation of actin aggregates resulting in lamellipodia and filopodia formation in classical swine fever virus-infected cells (Cheng *et al*., 2021).

The movement of viruses between cells has been observed in different viral families. These viruses have evolved to avoid the immune response by altering the actin cytoskeleton. This can include the creation of syncytia, actin comets, nanotube tunnels, and filopodia (Labudova, 2020; Aliyu *et al*., 2021). PRRSv-infected cells show that virus transmission depends on cytoskeletal function. It has been found that microtubule inhibitors can suppress the secondary spread of the virus. In addition, there is a noticeable change in the actin filaments of PRRSv-infected cells, suggesting that actin plays a role in creating a protective barrier for cell-to-cell transmission (Cafruny *et al*., 2006). Furthermore, it has been reported that vimentin filaments, along with the ANXA2 protein, are necessary for viral infection, replication, and transcription (Chang *et al*., 2018).

The present study aims to determine the structural changes in vimentin filaments caused by PRRSv in MARC-145 cells.

## MATERIAL AND METHOD

**Cell culture.** The MARC-145 cell line is cultured in RPMI medium. For propagation phase/uninfected cultures, it is supplemented with 10 % fetal bovine serum, and for infected cultures, it is supplemented with 4 % fetal bovine serum along with antibiotics (5000 IU/ml penicillin and 5 µg/ml streptomycin). The cells are maintained sterile in a humidified incubator (Thermo Scientific) with a 95:5 air/ CO2 mixture at 37 °C.

**Infection with PRRSv**. PRRSv from a live attenuated commercial vaccine INGELVAC PRRS MLV from the Boehringer Ingelheim Laboratory with the ATCC-VR 2332 strain was used. PRRSv was propagated by removing the culture medium from the cell cultures, then 5 ml of the vaccine was added and left in the incubator for 3 h. They were removed from the incubator and the 5 ml of vaccine were recovered. Finally, 5 ml of 4 % RPMI medium was added to each infected culture and incubated to follow the course of infection at 24, 48 and 72 h.

**Vimentin and actin filament labeling.** Cells cultured on PRRSv-infected coverslips and uninfected controls were fixed with 10 % aqueous formalin in PBS for 20 min at room temperature (RT), permeabilized with 0.5 % triton X-100 in PBS for 5 min and then blocked with 1 % bovine serum albumin (BSA) in PBS for 20 min. Once the above steps were completed, the cells were incubated with the primary anti-vimentin antibody (mouse anti-vimentin IgG, Santa Cruz Biotechnology) at 4 °C overnight in a humid chamber, then incubated with the secondary antibody conjugated to fluorescein isothiocyanate (goat anti-mouse IgG-FITC, Santa Cruz Biotechnology) overnight at 4 °C, in a humid chamber. Once finished the cells were subjected to a final incubation with phalloidin conjugated to rhodamine isothiocyanate (Sigma-Aldrich) for 20 min at RT in the dark, to proceed to mounting with mounting medium added with 4'6-diamino-2-phenylidinol (DAPI) on slides (Ultracruz® Mounting Medium for Fluorescence, Santa Cruz, CA, USA). After each step of the process the samples were washed 3 times with PBS. The cell cultures were observed with a fluorescence microscope (Zeiss Axioskop 40).

**Image processing.**The free Image J software was used for image analysis together with the "Z project" function for joint visualization of actin, vimentin and DAPI (merge).

## RESULTS

In this work we present the rearrangements of cytoskeletal filaments (vimentin and actin) in PRRSv-infected MARC-145 cells. In these cells, the distribution of vimentin filaments showed a well-defined pattern with a radial arrangement throughout the cytoplasm in the form of transcellular networks. Actin filaments were observed with a well-defined distribution pattern and structure, showing in particular stress fibers. Nuclei were observed with good morphology and structure (Fig. 1).

At 24 hours post-infection, the presence of vimentin filaments was observed in a perinuclear pattern with intense labeling, with loss of the characteristic morphology of these filaments (Fig. 2A). The loss of stress fibers and the formation of numerous filopodia between cells that did not present a direct contact between them (Fig. 2B), the presence of larger cells with a poorly defined pattern of actin filaments and the formation of syncytia with a disorganized pattern of vimentin and around the syncytium there were small cells

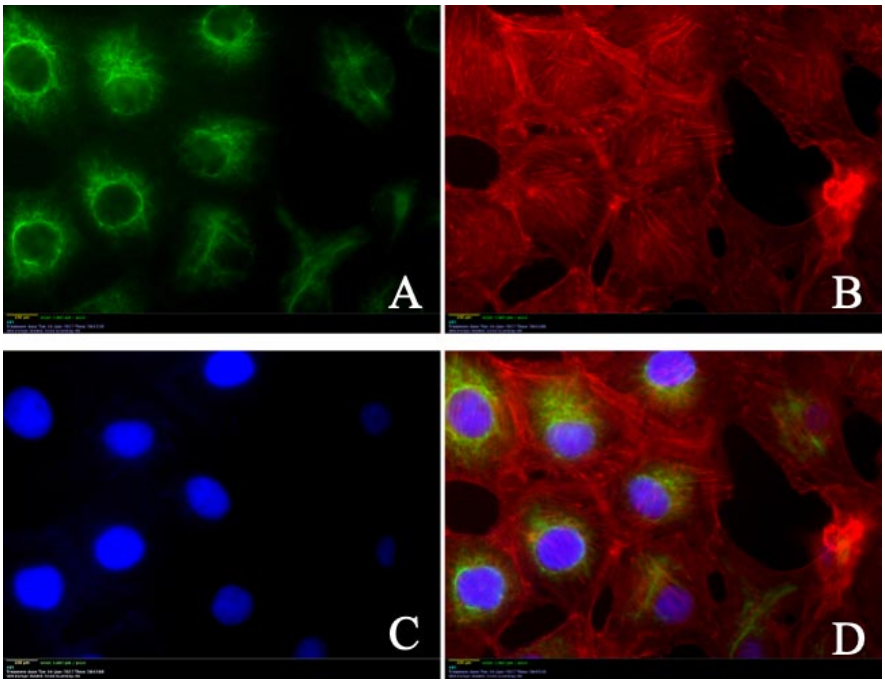


Fig. 1. Vimentin and actin filaments in MARC-145 cells. The arrangement of the (A) Vimentin filaments, (B) Actin filaments, and (C) Nuclei in the control cell cultures is shown, as well as signal splicing to visualize the colocalization of the cytoskeleton components with the cell nucleus. (D) Merge. Fluorescence microscopy triple-labeling, 40X.

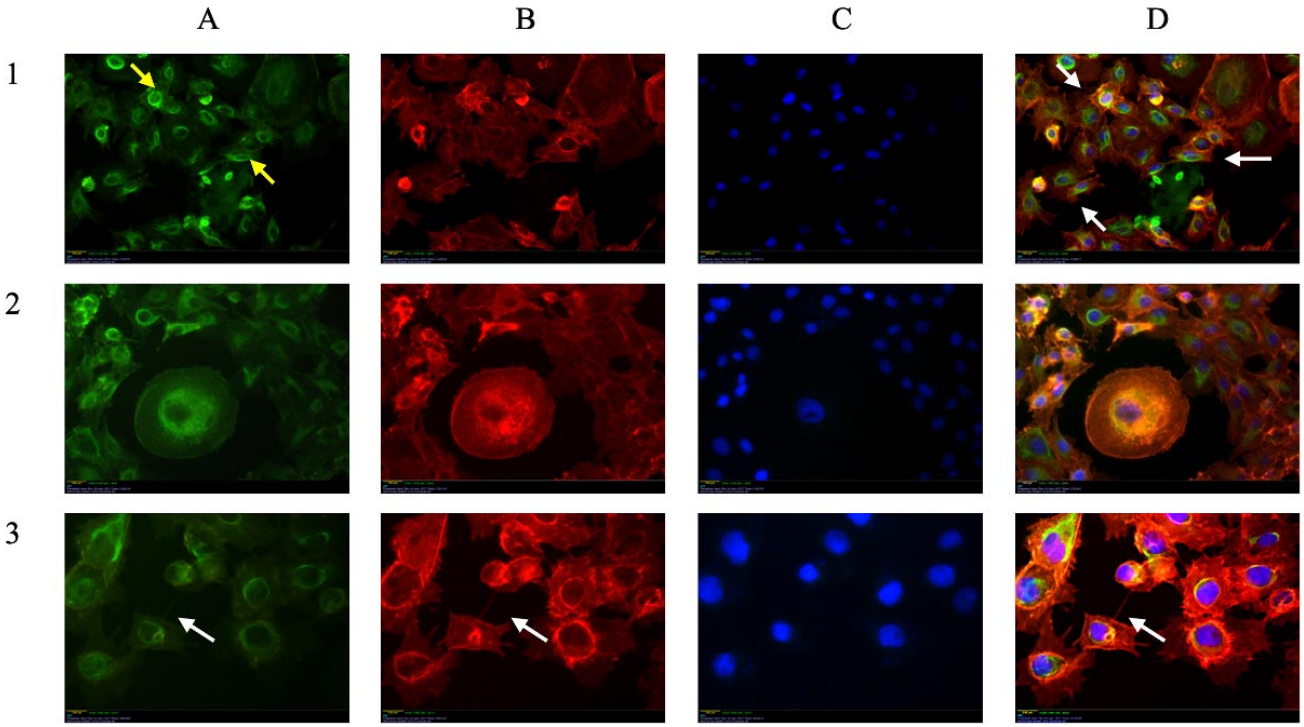


Fig. 2. MARC-145 cell line at 24 hpi with PRRS virus. At 24 hpi, positive labeling to vimentin was observed in the cultures with a predominant localization pattern in the perinuclear cortex (1A, yellow arrows), these aggregates do not colocalize with the perinuclear actin cortex in the merge (1:D, white arrows) and remain surrounding the nuclei, which at this post-infection time do not show changes in their morphology or centric position in the cells. The formation of giant cells is also observed, which do not present the normal morphology of MARC-145 cells, these cells remain isolated from the others, and at the cytoplasmic level, the actin filaments are observed depolymerized without the characteristic radial patterns and stress fibers (2:B), while the vimentin pattern is limited to the perinuclear zone without radiating towards the cell cortex (2A and 2D). Finally, the white arrows (3A, 3B & 3C) show the presence of filopodia, a higher-order structure of actin specialized in sensing the environment surrounding the cell. (A) vimentin; (B) actin; (C) DAPI; (D): Merge. Fluorescence microscopy, 20X (1& 2) and 40X (3).

with abundant aggregation of vimentin, which appears colocalized with aggregations of actin (Fig. 2D).

At 48 hpi a notorious alteration in the integrity of the monolayer was evidenced, characterized by changes in cell size, syncytia formation and decrease in cell population. Disorganization of vimentin and actin filaments was observed, in addition to nuclear damage with loss of morphology both in position and structure characterized by fragmentation, disordered with irregular sizes, suggestive of an advanced process of cell death (Fig. 3). At 72 hpi, visualization of cells was difficult since the integrity of the monolayer was almost completely affected, allowing only isolated cells with altered morphological patterns to be located. Vimentin filaments were observed forming a perinuclear ring suggesting their depolymerization and aggregation around the nucleus while actin filaments show a diffuse arrest with loss of stress fibers (Fig. 4).

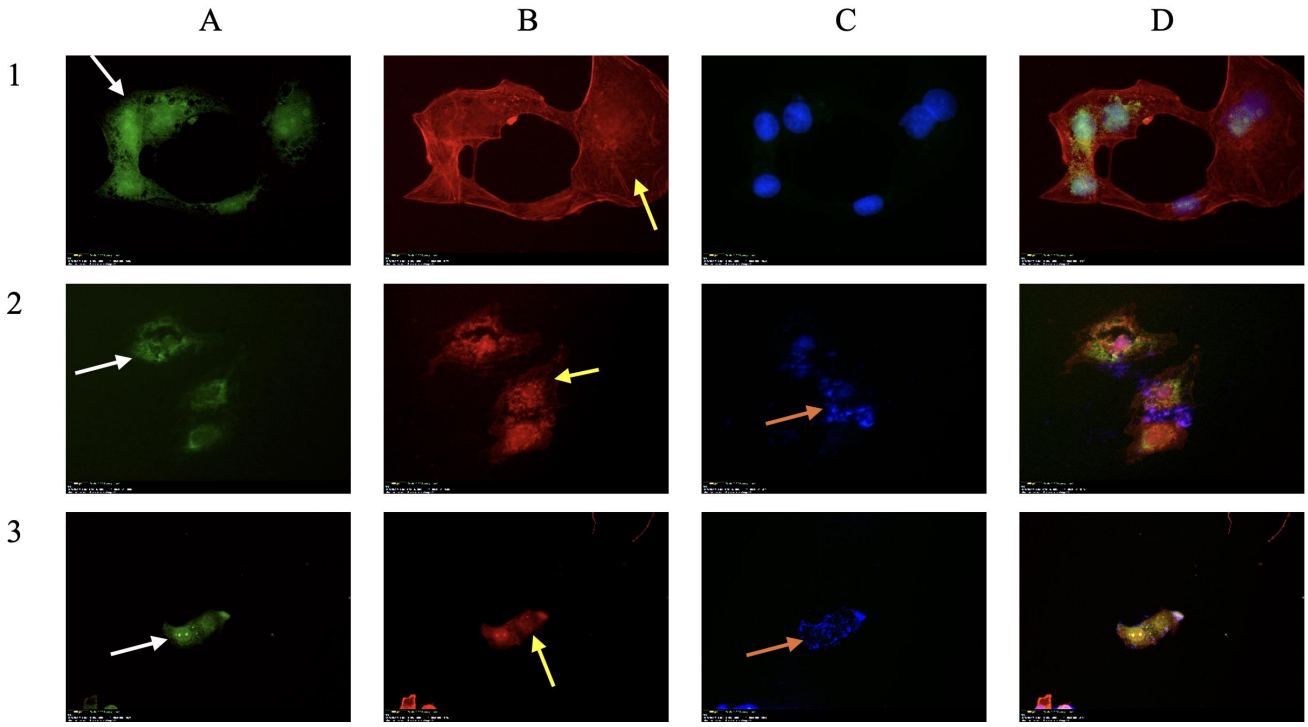


Fig. 3. MARC-145 cell line at 48 hpi with PRRS virus. At 48 hpi the cultures showed a marked depolymerization of the vimentin and actin cytoskeleton. In the cytoplasmic region, the distribution pattern of vimentin radiating from the perinuclear zone towards the cell membrane disappeared, showing labeling without a defined distribution (white arrows); actin depolymerization presented a lumpy pattern without visualization of stress fibers (yellow arrow). At the nuclear level, a diffuse punctate pattern of material was generated throughout the cytoplasm (orange arrow). Figs. 1, 2, & 3 show cells from 3 random fields where damage is evident (A) vimentin; (B) actin; (C) DAPI; (D): Merge. 40X fluorescence microscopy.

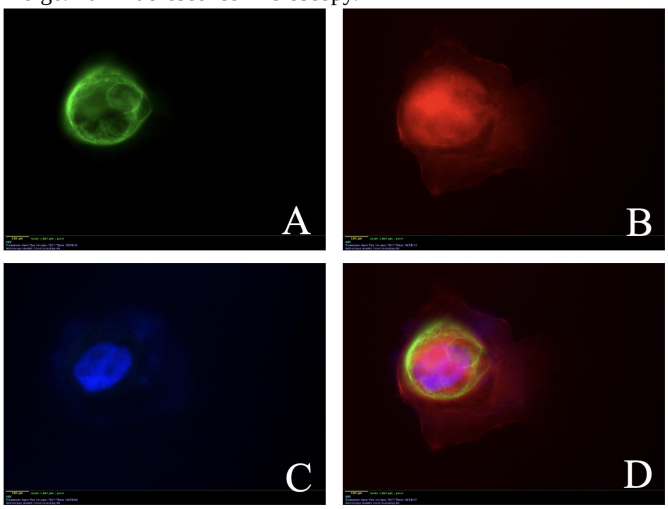


Fig. 4. MARC-145 cell line at 72 hpi with PRRS virus. Cells isolated by massive monolayer destruction and the morphological patterns of actin and vimentin at the cytoplasmic and perinuclear levels were severely affected (Figs. A & C). A: vimentin; B: actin; C: DAPI; D: Merge. Fluorescence microscopy, 40X.

## DISCUSSION

In this work we studied the rearrangements of actin and vimentin filaments in MARC-145 cells in PRRSv-infected cells. Infected cultures showed changes in their morphology and in the structure of actin and vimentin filaments. Cafruny *et al*. (2006), reported that viral transmission is dependent on an intact cytoskeleton, in particular vimentin filaments (Chang, 2018). (Jardon, 2020; Jardon *et al.* 2023), corroborated that the morphology of actin filaments is altered during the process of viral infection, which seems to indicate that there is an important modification of the cytoskeleton induced by PRRSv, so that its infectivity capacity could be dependent on its manipulation.

Kim *et al*. (2006), evaluated the effect of PRRSv vimentin expression on receptors on the surface of MARC-145 cells and other cell lines not permissive to PRRSv infection, and reported that infection resulted in opsonization and endocytosis of the virus, whereas BHL-21 and CRFK cultures (not sensitive to PRRSv infection) were administered simian vimentin and became susceptible to PRRSv infection, suggesting an important involvement of the vimentin cytoskeleton in viral infection. The reorganization of vimentin produced by PRRSv promotes viral replication (Zheng *et al*., 2021). Our results show changes in the structure of vimentin filaments supporting these results. In a study by Zhuang *et al*. (2012), they identified proteins expressed in MARC-145 and PAM cells infected with PRRSv by proteomic methods (Western Blot and RT-PCR), they found increased levels of cofilin-1 and vimentin, the authors mention that despite not being specific proteins of the disease, the changes detected were caused by PRRSv infection causing a reorganization of vimentin which probably leads to an unstable cytoskeletal structure. However, PRRS is not the only agent reported with changes in the cytoskeleton of infected cells, an example is the Hepatitis C virus where vimentin interacts with the central protein of the virus which regulates its expression and affects virus replication (Nitahara, *et al*., 2009, Ramos *et al*. 2020). As mentioned by Yang *et al*. (2016), in Dengue virus (DENV) infections, vimentin interacts with the virus envelope protein in endothelial cells and favors virus uptake and subsequent infection. Another case is Influenza virus type A (IAV) where vimentin is required for the release of viral ribonucleoproteins (vRNPs) into the cytoplasm by altering lysosomal trafficking during entry, this alteration could be related to reduced acidification of early endosomes in cells lacking vimentin, which are required for membrane fusion during the early stages of infection (Wu & Pante, 2016). Additionally, vimentin abundance has also been found to increase in both cytoplasmic and membrane fractions in infected cell cultures (DeBoer, *et al*., 2018).

Our results showed that during infection with PRRSv, evaluated at 24 hpi, there was an altered pattern in vimentin expression characterized by increased labeling in the perinuclear zone and in colocalization zones with alterations of actin stress fibers, the formation of these vimentin clusters was more evident in the initial sites of syncytia formation. As for the actin filaments, disorganization was observed that as the infection time progresses (24 to 72 hpi) ends with the loss of stress fibers and a diffuse distribution of the actin label. Cells infected with dengue virus showed disorganization of actin filaments and an increase in stress fibers was observed in neighboring uninfected cells (Kanlaya *et al*., 2009), which is consistent with our results. The changes in cell morphology as well as the loss of stress fibers is in agreement with previous reports that indicate that these changes may be due to alterations in actin filaments caused by viruses such as pseudorabies (Van Minnebruggen *et al*., 2002), as well as abnormal organization of stress fibers caused by dengue virus (Suttitheptumrong *et al*., 2021) and degradation of stress fibers caused by Marek's virus (Schumacher *et al*., 2005).

In well-established syncytia, vimentin expression was predominantly from the perinuclear zone at the cytoplasmic level, radiating to the cell membrane. During replication of some enveloped viruses, viral fusion proteins are inserted into the membrane of the infected cell that induce fusion with other membranes of adjacent cells. The result is that the cytoplasm of the cells involved are combined, but not the nuclei, if this occurs by bringing together several cells forming syncytia. For the virus this means being able to infect cells without leaving the extracellular space, where it could be neutralized by antibodies, this could be a short-term solution because the syncytia eventually die, as observed at 72 hpi.

## CONCLUSIONS

During the viral infection process in cell cultures, infection with PRRSv affects the pattern of vimentin filaments at different times of infection. These findings suggest that vimentin plays an important role in the process of viral infection. Therefore, this cytoskeleton protein has significant potential to be a therapeutic target for new strategies against this disease.

## ACKNOWLEDGEMENTS

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**RESUMEN:** El síndrome respiratorio y reproductivo porcino (PRRS) es una enfermedad altamente infecciosa que tiene un impacto económico negativo significativo en la industria porcina mexicana. El virus responsable del PRRS depende del funcionamiento del citoesqueleto de las células, y se estima que la vimentina juega un papel en la replicación y el transporte del virus. Por lo tanto, este estudio tuvo como objetivo examinar la morfología del citoesqueleto de vimentina en células MARC-145 infectadas con el virus PRRS, con el fin de crear un modelo de morfología celular que pudiera ayudar a identificar cambios inducidos por la infección in vitro. Los resultados revelaron patrones alterados de expresión de vimentina en la zona perinuclear y la formación de sincitios, cúmulos de células que contienen la misma proteína, a partir de las 24 horas post-infección (hpi). El estudio concluyó con la destrucción de la monocapa celular a las 72 hpi, posiblemente debido a la despolimerización del citoplasma, lo que llevó a una reorganización de la vimentina. Esta reorganización podría estar relacionada con la infectividad del virus y subraya la importancia de establecer un modelo morfológico de la vimentina para el estudio del PRRSv.

**PALABRAS CLAVE: Citoesqueleto; Filamentos intermedios; PRRS; Cultivo celular.**

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