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Oral sessions

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Oral sesión I

Determination of the oral microbiome and its association with markers of vascular inflammation in patients with COVID-19

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Background. COVID-19 presents an important vascular inflammation disturbance that could be related to oral dysbiosis; nevertheless, this is not completely understood. Methods. 51 SARS-CoV-2 positive out and inpatients, and 15 follow-up subjects SARS-CoV-2 negative were included. Angiotensin 2, 1-7, vascular cytokines and inflammatory factors in serum were detected. Microbiome in saliva and bronchial secretion samples (BSS) were determined by 16S NGS with Illumina platform. Results. Ang 2 was significantly higher in the inpatients compared to the follow up group. sST2, sRAGE, TIE-2, sDC40L, IL-6, IL-18 and MCP-1 were increased in COVID-19 patients, notably sST2 was the most suitable marker according to ROC curves. On the other hand, *Alloprevotella* and *Atopobium* represented the microbiome taxa in saliva of COVID-19 patients. In contrast, *Porphyromomnas* and *Neisseria* characterized the follow up group. In BSS, *Staphylococcus* and Firmicutes predominated. Positive correlation of IL-10 and Flt-1 with *Rothia*, *Staphylococcus* and *Veillonella* in saliva was observed, while sST2 correlated negatively with *Gemella*, *Porphyromonas* and *Haemophilus* in saliva of inpatients. Otherwise, IL-18, Ang 2, and viral load correlated positively with BSS *Campylobacter*, *Lactobacillus* and *Veillonella*. Conclusion. Vascular inflammation in COVID-19 can be influenced by oral bacterial taxa demonstrated by correlation analysis in patients with COVID-19.

Keywords: COVID-19; Inflammation; Microbiome.

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Discovery of T'Ho virus, a novel flavivirus closely related to encephalitic viruses of humans, and the development of tools for its serological diagnosis

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We discovered a novel flavivirus, designated T'Ho virus, in mosquitoes in Merida, Yucatan. The closest known relatives of T'Ho virus are encephalitic flaviviruses of public health importance, suggesting it too could be a human pathogen. The genome of T'Ho virus was fully sequenced, but an isolate was not recovered by cell culture inoculation or suckling mouse brain inoculation. In an attempt to generate recombinant T'Ho virus, the entire viral genome was synthesized as three overlapping DNA fragments, joined by Gibson assembly, and transfected into mosquito cells. Several cell culture passages were performed, but virus was not produced. Subsequent experiments focused on the development of a chimeric flavivirus for use in plaque reduction neutralization tests (PRNTs) as a surrogate diagnostic reagent in place of T'Ho virus. The PRNT requires live virus and is the gold-standard serologic technique for the diagnosis of flavivirus infections. We created the chimeric virus by inserting the major structural protein genes of T'Ho virus into the genetic background of Zika virus. The chimeric virus replicated in mosquito (C6/36) and vertebrate (Vero) cells and produced plaques in the latter. We propose that the chimeric virus could be a useful diagnostic reagent for researchers performing flavivirus serosurveillance in Mexico.

Keywords: Flavivirus; T'Ho virus; Zika virus, Chimeric virus; Virus discovery.

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Exploring the Role of Prophages in Antibiotic Resistance Dissemination: An Approach that Challenges Antimicrobial Therapy

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Antibiotic resistance is the most alarming public health problem of recent decades. The World Health Organization constantly raises awareness about pathogenic bacteria that require priority for research and the discovery of new antibiotics. In this regard, *Acinetobacter baumannii* (*A. baumannii*) and various species of the *Campylobacter* genus are considered of critical and high priority, respectively. Recently, we explored the diversity of prophages harbored in over 13,000 prokaryotic genomes. We found that lysogens are more common than previously estimated, with prophages significantly abundant in pathogenic bacteria and those belonging to the order Enterobacterales. Furthermore, we found that the evolutionary relationships of prophages are closely related to the evolution of their hosts, resulting in a strictly restricted host range. In particular, we demonstrated that populations of *A. baumannii* and certain *Campylobacter* species prophages encode a vast repertoire of virulence genes and antibiotic resistance. Finally, we discovered that recombination events between prophages of different genera are a diversification strategy for these viruses.

Keywords: Prophages; Virulence genes; Antibiotic Resistance; Prophage Evolution.

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Neutralizing antibody seroprevalence in healthy Mexican adults against eight human and three simian adenovirus types, including those employed in COVID-19 Vaccines

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Five of the eight vaccines approved in Mexico to be used against COVID-19 are adenovirus-based, including Gam-COVID-Vac (Sputnik V), Ad5-nCoV Covidecia, Ad26.COV2-S, and AZD1222 Covishield. AdV-based vaccines have shown promise due to their ability to induce robust B- and T-cell responses. However, studies indicate high levels of prior exposure to HAdV-C5 and neutralizing antibodies in the human population that could interfere with the efficacy of the vaccine, while the prevalence of antibodies against HAdV-D26 varies worldwide. In Mexico, data on AdV seroprevalence are scarce, hindering predictions about vaccine efficacy, immunity duration, and their use for boosters or seasonal application.

In this work we have analyzed pre-COVID-19 pandemic and pre-COVID-19 vaccination blood samples from healthy Mexican adult donors for antibodies against different AdV types (from species A to F) and simian AdVs (SAdV-21, SAdV-25, and SAdV-31) using ELISA and neutralization assays.

The highest levels of neutralizing antibodies were observed for HAdV-C5, HAdV-C6, HAdV-B14, and HAdV-F41, while SAdV-21, SAdV-25, and SAdV-31 showed negligible neutralization. HAdV-D36, HAdV-A12, and HAdV-E4 formed a separate cluster with intermediate neutralization levels. Notably, HAdV-D26 exhibited low neutralization, similar to developed countries. These findings show widespread and high prevalence of adenovirus in healthy individuals in Mexico, with levels that are similar to developing countries for HAdV-C5 or HAdV-F41, while more closely matching levels in developed countries for HAdV-D26. The data indicate that HAdV-D26 and SAdV21 are good vaccine vector candidates for the Mexican population due to their low prevalence. The data should contribute to plans for vaccine development and vaccination strategies by national health authorities, including seasonal vaccine application and the development of new AdV-based vaccines.

Keywords: Adenovirus; Seroprevalence; Vaccines; Simian; Neutralization.

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Environmental samples are key for monitoring viruses for human health and agriculture on the south border of Mexico

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Mexico's southern border is a complex region due to migration flows and social dynamics, whose effects impact the human population and plant health, the ecosystems, and the biodiversity of the border-sharing countries. But what about the viruses?

In 2020, due to the COVID-19 pandemic, our research team proposed a strategy for SARS-CoV-2 virus monitoring via the wastewater-based epidemiology approach (using wastewater from the treatment plants) and evaluating the viability of implementation using urban rivers and sewer water samples. We selected strategic points for collecting the samples from Tapachula (Chiapas), a border city with complex population dynamics due to the human migratory phenomenon. We measured physicochemical parameters to determine the water samples' pollution level. We optimized a low-cost viral particles concentration method to detect the SARS-CoV-2 by RT-qPCR. We demonstrated that the method effectively detects viral particles from urban rivers despite the pollution level and the environmental temperature prevailing in this tropical region and could strengthen the monitoring of epidemiologic strategies. Also, by metagenomic sequencing and bioinformatic analysis, we determined the virus diversity in the water samples from the all-sampling points. The results revealed a high quantity of plant-infecting viruses (around 80 % of all detected) belonging to the *Virgaviridae* family, specifically of the *Tobamovirus* genus, that infect solanaceous like tomato, potato, or chili. Secondly, we detected bacteria-infecting phaguses and interesting animal-infecting viruses diversity (including insects or vertebrates) and fungi. This opens the possibility of developing monitoring strategies for viruses of human, ecological, and agricultural health relevance on Mexico's southern border.

Keywords: Wastewater-based epidemiology; Viruses diversity; Viral metagenomics; Plant-infecting viruses; Urban rivers.

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Virus monitoring in wastewater and water reuse for agricultural production

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The viruses present in human and other animal feces have been detected in wastewater from population centers. It is of interest to know about its permanence in residual water. Wastewater, treated wastewater, and lettuce surface irrigated with treated wastewater have been analyzed to detect SARS-CoV-2, rotavirus, and enterovirus. Concentration and RT-PCR techniques were used for SARSCoV-2 detection in wastewater, rotavirus, and enterovirus in treated wastewater and lettuce surfaces. Wastewater was positive for SARSCoV-2 (0 to 10 3 gen/L). Rotavirus was detected in treated wastewater and lettuce surfaces (10 3 to 10 6 TCID50/mL). Enterovirus was sequenced, confirming that it was vaccinal poliovirus in treated wastewater and lettuce. The detection of pathogens in wastewater is an epidemiological strategy that has gained strength in recent years at global level. While it can be monitoring of pathogens circulating in the population, it can also be an early sign of outbreaks. It is also valuable information for wastewater reuse practices with or without treatment.

Keywords: Enteric viruses; Wastewater; Reuse; Irrigation.

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Oral session II

Extracellular vesicles modify the cell tropism of gastrointestinal viruses

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Gastrointestinal viruses infect intestinal cells, and as a consequence cause infection, leading to diarrhea. Despite their specificity to replicate in the intestine, there is growing evidence that some of them can cause extraintestinal infection. Specifically, rotaviruses (RV) and some astroviruses (AstV) have been associated with extraintestinal diseases (seizures, diabetes, encephalitis, hepatitis). To analyze how intestinal viruses could spread to other organs, we recently described the ability of RV and AstV to interact with extracellular vesicles (EV) and to infect cells. We selected several cell lines having low susceptibility but high permissivity for rotavirus and astrovirus infection. We found that purified virus-EV complexes have the capacity to infect the selected cell lines without protease activation of the virus infectivity, which is commonly needed for efficient cell entry. Infectivity was associated with the presence of membranes, as treatment with detergent abolished viral replication. A proportion of viral particles was protected from the action of neutralizing antibodies, suggesting they are inaccessible to antibody interaction, probably being present inside the EV. These results open the possibility that classical gastrointestinal viruses could use EVs for their extra-intestinal spread, in a receptor-independent manner. This work was supported by grant IN213722 from DGAPA-PAPIIT UNAM.

Keywords: Rotavirus1; Astrovirus2; Extracellular vesicles3; Cell tropism4.

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Effect of palmitoylation in the *Feline calicivirus* replicative cycle

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Sapoviruses and noroviruses, which comprise the human caliciviruses (HuCV) within the *Caliciviridae*, represent a public health problem as they represent a major cause of epidemic acute gastroenteritis worldwide. Their study is limited due to complications associated with their *in vitro* cultivation; thus, models such as *Feline calicivirus* (FCV) have been used to understand their biology. FCV contains a single-stranded, positive-sense RNA genome that encodes for the six non-structural (NS) proteins and two structural VP1 and VP2 proteins, and the Leader of the Capsid (LC) protein. Viral proteins localize to the endoplasmic reticulum (ER) without having localization signals. The mechanism by which they associate with the ER membranes has not yet been determined; thus, post-translational modifications such as palmitoylation could allow their controlled association of proteins with membranes. To determine the impact of palmitoylation in FCV replication, we use the inhibitor 2-bromopalmitate (2-BP). This compound caused a reduction in the cytopathic effect, viral proteins, and particle production, as well as a change in the protein's subcellular localization. By bioinformatic tools, we identified putative palmitoylation sites in several viral proteins, including NS3 and LC. LC was demonstrated to be palmitoylated during infection. Understanding the mechanisms involved in viral replication will help to develop strategies for their control and prevention.

Keywords: FCV; Palmitoylation; 2-BP; Non-Structural proteins; Localization.

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The interaction of nucleolin with G-quadruplex regions in the rotavirus genome modulates the production of viral progeny during virus replication

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Cellular proteins play an important role in the replication cycle of viruses; several reports have shown a regulatory association between cellular proteins and the viral RNA. Nucleolin, a cellular protein involved in cellular RNA metabolism, has a regulatory role in the replication of hepatitis C virus, human immunodeficiency virus, and Epstein-Barr virus infection. Here, we found that the knockdown of nucleolin results in a 5-fold increased production of rotavirus infectious viral particles, and a 6-fold increased number of genome copies during virus replication. The interaction between viral RNA and nucleolin was demonstrated through co-immunoprecipitation assays, Next, to identify putative nucleolin binding sites, known as G4 sequences, in the rhesus rotavirus (RRV) genome, bioinformatic tools were used. We found three potential G4 sites on RRV gene segment 10, named G4-1 (nt 116-132), G4-2 (nt 572-607), and G4-3 (nt 717-737). Transfection experiments with these regions, showed that cells transfected with G4-1 and G4-3 regions exhibited a 64 % and 118 % increase in viral production, respectively, while G4-2 transfections had no significant effect 11.5 %. These findings suggest that nucleolin likely plays a regulatory role in controlling viral particle production through its direct interactions with G-quadruplex structures in the virus genome.

Keywords: Rotavirus; Nucleolin; Virus host cell interactions; RNA Binding protein.

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Identification of rotavirus RNA polymerase cofactor VP2 as a molecular target of benzimidazole

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Rotavirus species A (RVA) is a major cause of gastroenteritis in infants. The virion has a triple-layered icosahedral capsid surrounding 11 dsRNA segments. The viral genome codes for 12 proteins, six structural (VP1-VP4, VP6 and VP7) and five or six non-structural (NSP1-NSP5/6). Vaccination has helped to reduce the impact of RVA infections, but they do not confer complete immunity. In addition, there are no antiviral therapies to control RVA infections. Based on the knowledge that benzimidazole derivatives inhibit human RVA replication, we aimed at identifying the viral target of benzimidazole. For that purpose, we selected two clones of wild type Rhesus rotavirus during 12 passages with benzimidazole in *Cercophitecus aethiops* MA-104 cells, thus obtaining partially resistant variants. The genomes of the variants and their parental strains were fully sequenced by RNAsec. Considering the mutations detected in both variants, most (7/11) occurred in gene segment 2 that encodes the core shell protein VP2. Apart from two silent mutations, the remaining five VP2 mutations affected its principal domain, specifically at the dimerization, central or apical subdomains. VP2 is an essential cofactor that triggers the viral RNA polymerase VP1 to initiate dsRNA synthesis using plus-strand RNA templates. Our data identified VP2 as the main target affected by benzimidazole. These findings are useful to identify *in vitro* screening assays for benzimidazole derivatives that inhibit RVA replication.

Keywords: Rotavirus; VP2 protein; Benzimidazole; Mutant proteins; RNA replicase.

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The E1B-55KDa phosphoprotein associates with and regulates adenoviral promoters

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The E1B-55KDa (E1B) is a multifunctional phosphoprotein essential for various steps of the adenoviral replication cycle, and one of the main candidates in the design of oncolytic vectors. E1B has been implicated in inhibition of the antiviral response, viral mRNA biogenesis and cellular protein turnover, but the molecular mechanisms have only been described for the latter. Early studies showed that E1B can suppress cellular promoters, and we recently found that the protein can regulate also adenoviral promoters. In this work we wished to determine the effect of E1B on adenoviral promoters and whether the protein can interact with viral promoters to regulate transcription, either through direct contact with DNA or indirectly through protein interactions. Luciferase reporter assays showed that E1B can regulate all adenoviral promoters, and that phosphorylation of the protein works as a switch in viral promoter regulation. Furthermore, using Viral Chromosome Immunoprecipitation (VirChIP) assays E1B was shown to associate with the adenoviral E1A and Major Late promoters in transfected and infected cells, respectively. Bioinformatic analysis of E1B's interactome revealed that four cellular and two viral proteins that interact with E1B are predicted to bind specific sequences in adenoviral promoters. Taken together our data suggest that E1B can regulate viral gene transcription through association with DNA via protein interactions with these cellular or viral transcription factors. These findings provide valuable novel insights into the complex role of E1B during adenoviral replication and highlight this adenoviral protein as one of the very few known general regulators of viral gene transcription.

Keywords: Adenovirus; Viral gene transcription; Promoter regulation.

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Elucidating the role of the acidic domain of the astrovirus capsid precursor during virus replication

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The human astrovirus (HAstV) capsid protein precursor, VP90, is cleaved by cellular caspases at its C-terminus acidic domain (AD), producing a 70 kDa protein. This cleavage has been correlated with the virus dissociation from cellular membranes and release from cells. Here we evaluated the function of the AD using a chimeric astrovirus infectious clone that encodes the machinery replication of HAstV-1 and the capsid protein from HAstV-8 (Wt). In the chimeric astrovirus, a stop codon was introduced at the beginning of the AD (AD). To evaluate the production of infectious virus, plasmids Wt and AD was transfected into HEK293T-T7 cells, and the virus particles produced were quantitated by infecting Caco-2 cells monolayers. The synthesis of viral proteins and the amount of genomic and antigenomic viral RNAs were not significantly affected, as compared with the Wt virus. However, we found an 80 % decreased production of virus particles in the AD chimeric virus. Notably, after several passages in cell culture, the virus produced by the AD chimeric maintained its capacity to replicate, although to an extent 4-log lower than the Wt virus. Studies are underway to determinate how the absence of AD affects the production of virus particles. This study provides insights into the relevance of AD in HAstV replication.

Keywords: Astrovirus; Replication; Acidic domain; VP90.

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Role of aspartate aminotransferase-to-lymphocyte ratio index (ALRI) in COVID-19

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COVID-19 has a mortality toll exceeding 5.4 million worldwide. Early identification of patients at high mortality risk is essential to save their lives. AST-to-lymphocyte ratio index (ALRI) is a novel biomarker of survival in patients with hepatocellular carcinoma, an organ susceptible to SARS-CoV-2 infection. The prognostic value of ALRI as a marker of COVID-19 mortality was evaluated. For this purpose, ALRI was compared with main biomarkers for COVID-19 mortality (neutrophil-to-lymphocyte ratio [NLR], systemic immune-inflammation index [SII], plate-let-to-lymphocyte ratio [PLR], lactate dehydrogenase (LDH)/lymphocyte ratio [LDH/LR]). A retrospective cohort of 225 patients with SARS-CoV-2 infection and without chronic liver disease was evaluated. In non-survival group, the ALRI, NLR, SII, and LDH/LR were significantly higher than in survival group ($_{Pcorrected} < 0.05$). ALRI had an area under the curve (AUC) of 0.81, sensitivity 70.37 %, and specificity 75 %, with a best cut-off value > 42.42. COVID-19 patients with high ALRI levels had a mean survival time of 7.8 days. Multivariate Cox regression revealed that ALRI > 42.42 (HR = 2.32, 95 % CI: 1.35-3.97; $_{Pcorrected} = 0.01$) was a prognostic factor of COVID-19 mortality. These findings prove that ALRI is an independent predictor of COVID-19 mortality and may help identify high-risk subjects with SARS-CoV-2 infection at admission.

Keywords: COVID-19; SARS-CoV-2; Aspartate aminotransferase-to-lymphocyte ratio index (ALRI); Biomarker; Mortality.

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Evaluation of immune response induced by a plasmid that codifies O-SN SARS CoV-2 fusion protein in a mouse model

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New generation of COVID-19 vaccines have focused on the evaluation of more than one SARS CoV-2 antigen to induce a long-lasting immune response. We design a plasmid that codify for fusion protein and evaluated their immune response in a mouse model. Most immunogenic regions from Spike and Nucleocapsid Omicron SARS CoV-2 strain were determined by Insilco approaches. Once the sequences were selected, we generate the structure of this fusion protein and determined physicochemical and immunogenic properties. Docking molecular was performed to predict the capacity of fusion protein to be recognized by innate receptors such as TL3 and TLR4. The sequence was cloned in pcDNA3.1 and named pcDNA3.1/O-SN, expression of the plasmid was evaluated. BALB /c mice were immunized with 10 µg, 20 µg, 40 µg of pcDNA3.1/O-SN. Three doses of DNA were performed at interval of 20 days. Mice immunized with parental vector pcDNA3.1 were used as control. After immunization, bleedings were performed, and serum samples were obtained. Docking molecular showed capacity of binding TLR3 and TLR4 to O-SN. Specific antibody response of IgM and IgG against N and S1 proteins from SARS CoV-2 were observed in immunized mice. Our results indicate that DNA vaccination with pcDNA3.1/O-SN generated by our working group is capable to inducing a specific humoral immune response.

Keywords: SARS CoV-2; DNA Immunization; Preclinical studies; fusion proteins; immune response.

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Oral session III

Nuclear localization of the dengue virus non-structural protein 1 (NS1)

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The dengue virus (DENV) genome encodes for 3 structural (C, prM and E) and 7 non-structural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B, NS5). The NS1 is a glycoprotein that is located in the lumen of the endoplasmic reticulum, as part of the replication complexes. In addition, NS1 is secreted, and soluble NS1 participates in dengue pathogenesis. Previous evidence suggested that NS1 is also found in the cell nuclei. Employing confocal microscopy and cellular fractionation techniques, we have observed that indeed, NS1 translocates to the nucleus of infected cells in significant proportions. At 24 hpi, up to 40 % of the total pool of NS1 produced in DENV infected BHK-21 cells is found inside the nucleus. Similar results were observed in infected mosquito C6/36 cells. *In silico* analysis of the NS1 amino acid sequence, identified one putative nuclear localization signal (NLS). Preliminary data using recombinant NS1 expressed in BHK-21 cells treated with ivermectin, indicated that NS1 nuclear transport is facilitated by α/β importins. These findings suggest that NS1 is located in the cell nuclei of infected cells, and gains entry through an importin-mediated transport. Our forthcoming research will delve into the importance and functional significance of nuclear NS1 during viral replication.

Keywords: Dengue virus; NS1 protein; Nuclear Localization Signals; ivermectin; cell nuclei.

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Deciphering the begomovirus DNA molecules complex associated to tomato yellow leaf curl disease

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Begomovirus (*Geminiviridae* family) possess circular single-stranded DNA monopartite (DNA-A) or bipartite (DNA-A plus DNA-B) genomes and, considered the major group of emerging plant viruses threatening the cultivation of important crops in tropical and subtropical regions worldwide. Tomato yellow leaf curl disease (TYLCD) is caused by single or mixed infections of different monopartite and bipartite begomovirus species. During 2018, tomato plants showing begomovirus-associated symptoms including leaf yellowing, curling, plant stunting, and whitefly vector infestation, were collected in Morelos, Mexico. Using PCR molecular detection and Rolling Circle Amplification (RCA)-based viral genomes cloning approaches, the begomovirus species *Tomato golden mottle virus* (ToGMoV) and *Tomato severe leaf curl virus* (ToSLCV) were identified as the putative TYLCD causal agents. Intriguingly, whereas both ToGMoV and ToSLCV display typical bipartite DNA-A genetic structure, cognate DNA-B was only detected for ToGMoV. To identify the complete plethora of circular DNA molecules associated to the TYLCD, a High-throughput sequencing (HTS) based on Circular DNA Enrichment (CIDER-seq) was performed. Whereas HTS results confirmed the previous observations, interestingly, a begomovirus replication-dependent satellite molecule was additionally detected. Biological assays using infectious clones of identified viral molecules are in progress with the aim to decipher the DNA molecules associated to the observed TYLCD.

Keywords: Begomovirus; TYLCD; Satellite; HTS; CIDER-Seq.

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Expression of proteins involved in exosomal biogenesis and changes in extracellular vesicle morphology in HUVEC cells infected with ZIKV

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Exosomes are extracellular vesicles that maintain cellular homeostasis; several viruses hijack this exosomal pathway to increase the quantity and size of these vesicles, carrying cellular or viral factors that benefit an optimal environment for the virus. In the present work, exosomes were isolated and characterized from umbilical cord vein endothelial cells (HUVEC) infected with Zika virus (ZIKV). Western blot analysis of the exosomal fraction infection conditions showed increased ALIX and CD9 exosomal biogenesis marker expression. At the same time, there is a decrease in these proteins in the total extracts of HUVEC cells infected with ZIKV. In Cryo-TEM assays, the exosomes infection conditions showed various morphologies such as single vesicles, double vesicles, multivesicular, double membrane, electrodense, and broken membrane. In contrast, only single vesicles, double vesicles, and double membranes were found in Mock conditions. A particle tracking analysis (NTA) showed an increase in exosome concentration in infection conditions of 1.67x108 particles/mL, while in the Mock, 6.82x107 particles/mL was obtained. These data suggest that infection ofHUVEC cells with ZIKV promotes an increment in exosome biogenesis resulting in a higher number of these extracellular vesicles; this increase of ALIX and CD9 infection conditions could influence changes in the morphological types of exosomes infection conditions in contrast to the morphological types seen Mock conditions.

Keywords: Exosome; HUVEC; ZIKV; vesicles; morphologies.

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Viral RNA-dependent polymerases probably diverged from eukaryotic replicative polymerases

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The sole conserved protein among RNA viruses is the replicative RNA-dependent polymerase, either an RNA-dependent RNA polymerase (RdRp) or a reverse transcriptase (RT). These enzymes have been posited as remnants of a primordial polymerase that emerged in the RNA world. RdRps and RTs belong to the Superfamily of DNA and RNA polymerases, which adopt a right-hand shape, and employ a divalent metal ion mechanism of action. B-family DNA-dependent DNA polymerases, the eukaryotic replicative polymerases, are also members of this Superfamily. Based on structural analyses and a structure based phylogenetic tree, we propose that RdRps and RTs have a more recent origin, probably diverging from B-family DdDps.

Pairwise comparisons between DNA- and RNA polymerases were performed in the PDBeFold web server. The Structural Alignment Score was calculated for each comparison [(RMSD*100)/No. superimposed residues]. A distance-based dendogram was calculated with Fitch, Phylip v 3.65. The visual analyses and the figures were depicted with Chimera 1.13.

The structure-based phylogenetic tree shows 4 well-defined clades. The branch that groups RdRps and RTs stems from B-family DdDps. The evolutionary relatedness of these enzymes is strengthened by the fact that their subdomains' order is similar. The palm, the structures preceding palm's motif A, the C-term β-strands of the palm, and a helical bundle following the palm subdomain are conserved.

Our analyses show that RNA-dependent polymerases probably diverged from B-family DdDps, which indicates that the former did not emerge in the primordial RNA world; instead, their origin is more recent, probably closer to the emergence of eukaryotes.

Keywords: RNA viruses; RNA-dependent RNA polymerases; Reverse transcriptase; B-family DNA-dependent DNA polymerases; structural evolution.

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Zika Virus Infection In Neural Progenitor Cells Alters Glial Differentiation And The Expression Of Glutamatergic System Genes

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Perinatal viral infections can cause neurodevelopmental disorders with varying degrees of cognitive and mental sequelae. One virus that affects neurodevelopment is the Zika Virus (ZIKV), which can cause microcephaly and other congenital malformations during pregnancy.

Despite the severity of neurodevelopmental impairments associated with ZIKV infection during pregnancy, little is known about the neuropathogenic mechanisms responsible for central nervous system malformations and dysfunctions.

In this study, our main objective was to investigate a ZIKV infection model using human neural progenitor cells, specifically the hNS-1 cell line, which can differentiate into neurons, astrocytes, and oligodendrocytes. We also evaluated neural differentiation by measuring the expression of GFAP (astrocyte marker) and β-Tubulin III (neuron marker) in undifferentiated hNS-1 cells infected with ZIKV. We found increased GFAP messenger RNA expression in infected cells at 9 and 15 days of differentiation, suggesting changes in the astrocyte population. Based on these changes in GFAP expression, we isolated the astrocyte population from a 21-day differentiated hNS-1 cell culture and demonstrated that ZIKV infection reduces cell viability, increases the production of reactive oxygen species (ROS), and results in high viral titers. Additionally, we observed changes in the expression of genes involved in viral entry into cells and genes related to glutamatergic system homeostasis.

Our findings provide new evidence on how ZIKV infects neural progenitor cells and its dependence on cell differentiation. We also highlight the modification of GFAP expression and the potential functionality of astrocytes. These results contribute to a better understanding of the pathophysiology of congenital ZIKV-associated disease.

Keywords: Zika virus; Astrocytes; Viral gene expression; Neural Stem Cells; Glutamate.

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Megaviruses: little Frankenstein monsters with a recent origin

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Megaviruses exhibit a broad host range, infecting various eukaryotic organisms (protists and animals). These viruses harbor genomes composed of either linear or circular double-stranded DNA, varying in size from 100 to 2,500 kbp. While some researchers have proposed that megaviruses might have evolved from cells experiencing irreversible genome reduction, the potential contribution of host genomes to certain viral sequences has been underappreciated.

This study delves into pangenomic analyses encompassing all seven known megavirus families: Ascoviridae, Asfarviridae, Iridoviridae, Marseilleviridae, Mimiviridae, Phycodnaviridae, and Poxviridae. We found that the core and shell genes within these families share homologous counterparts in cellular organisms, raising intriguing possibilities of gene transfer events shaping their evolutionary trajectory. Moreover, our study has shown that megaviral sequences are associated with specific cellular protein families, such as small chain ribonucleotide reductase type Ia and Erv1/Air, as well as a superfamily of 2OG-Fe(II) oxygenases involved in many metabolic pathways, which incorporate oxygen atoms to perform their catalysis. These oxygen-dependent enzymes are predominantly located within the core and shell clusters of all megaviruses, suggesting that 1) they may have emerged subsequent to Earth's Proterozoic Great Oxidation Event and 2) Megaviruses may have a recent origin related to the early evolution of eukaryotes.

Keywords: Nucleo-cytoplasmic large DNA viruses; gene recruitment; pangenomic; Great Oxidation Event.

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Analysis of MAD2 and TTK expression in cervical cancer samples and cell lines.

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The main factor for the development of Cervical Cancer (CC) is infection by the high-risk human papillomavirus (HR-HPV). In exploring new molecular markers to identify women at high risk of developing cancer, the study of MAD2 and TTK is proposed. The aim of this study was to explore the expression levels and prognostic significance of MAD2 and TTK in CC using bioinformatics. Finally, the findings will be verified in samples of tumors and cell lines. MAD2 and TTK expression through GEPIA, PP interaction through STRING, as well as KEGG and survival analysis. Data was analyzed using UCSC Xena, to see its association with HPV. Expression levels of MAD2 and TTK were determined using RT-qPCR. Results showed the expression of the MAD2 and TTK genes was higher in tumor samples compared to control samples in both genes, having a statistically significant result ($p < 0.005$). in conclusion the expression analysis reveals that high expression of MAD2 and TTK may contribute to the development of cervical cancer and may serve as a potential diagnostic target for CC.

Keywords: Cervical cancer; Expression; Biomarkers; MAD2; TTK.

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Identification of cellular proteins that bind the terminal regions of the positive strand RNA genome of human astroviruses

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Human astroviruses (HAstV) are causative agents of acute gastroenteritis and non-bacterial diarrhea in children worldwide. The HAstV genome is a positive-sense, single-stranded RNA, and it's the 5´and 3´ untranslated regions play crucial roles through interactions with cellular proteins, that are vital for viral genome replication and RNA metabolism during infection. To learn about these interactions, in this work the RaPID assay (RNA-protein interaction detection) was employed. This assay allows the affinity capture of proteins interacting with the target RNAs of interest. Using this assay, a total of 181 RNA binding proteins (RBPs), bound to the 5′ and 3′ UTRs of two distinct HAstV genotypes, (HAstV serotype 8 and HAstV VA1) were identified. Among these RBPs, 54 were found to interact with both the 5 and 3´ ends of HAstV 8, while 127 were associated with the non-classical HAstV VA1 virus. Remarkably, 46 RBPs were common to both genotypes, suggesting potential shared mechanisms in their interactions with HAstVs. Furthermore, HAstV 8 displayed 3 unique RBPs, while the non-classical VA1 exhibited 17 unique RBPs. These findings provide valuable insights into the host RBPs that might participate in the replication cycle of HAstV genotypes.

Keywords: Astroviruses; RNA Binding Proteins; RNA virus; Proteomics; Untranslated Regions.

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The Human Adenovirus 36 E4Orf1 protein is sufficient but is not required to induce adipogenesis in infected cells

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Human adenovirus species D, type 36 (HAdV-D36) can induce obesity in animal models and stimulate the differentiation of preadipocytes in cell culture. HAdV-D36 alters gene expression and cellular metabolism, increasing glucose uptake through Glut4, activating the Ras/PI3K pathway and promoting triglyceride accumulation through upregulation of C/EBPs and PPARγ. These effects have been associated only with the adenoviral E4Orf1 protein, but extensive evidence showed previously that other adenoviral genes can alter cell metabolism. The adipogenic mechanisms altered by E4Orf1 have been studied only in transfected cells and it is not known whether the protein has the same effect in the context of viral infection. In this study we have analyzed the effect of E4Orf1 on adipogenesis and on viral replication in HAdV-D36 infected cells. The HAdV-D36 WT virus was compared with a recombinant virus that does not express E4Orf1 (HAdV-D36/ΔE4Orf1) in the 3T3-L1 cell adipocyte model. We have found that only 3T3-L1 cells that are committed to adipocyte differentiation support HAdV-D36 replication, and that under these permissive conditions for viral replication the E4Orf1 protein was not necessary for the expression of viral mRNA, viral DNA replication or viral progeny production. Significantly, the absence of E4Orf1 did not prevent adipocyte differentiation, upregulation of adipogenic or glucolytic genes, or accumulation of intracellular lipids, indicating that during HAdV-D36 infection the E4Orf1 protein is not required for the virus adipogenic effect.

Keywords: Human Adenovirus 36; Obesity; E4Orf1 protein; adipocyte commitment; permissive replication; lipid and glucose metabolism.

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Oral session IV

An Ivermectin–Atorvastatin combination impairs host nuclear transport inhibiting DENV-2 infection *in vitro* and *in vivo*

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Dengue virus (DENV) uses cellular nuclear transport machinery to import some proteins into the nucleus. Recently, the non-structural protein 3 (NS3) of DENV was localized in the nucleus of infected cells; however, its nuclear import mechanism is still unknown. In this study, we demonstrate that Ivermectin (IVM) inhibits the nuclear localization of NS3 through inhibition of the Importin $\alpha/\beta 1$ pathway. We also report that Atorvastatin (ATV) can modulate the nuclear transport of NS3 protease of DENV-2. On the other hand, we found that an IVM+ATV combination reduced DENV infection both *in vitro* and *in vivo*. Hence, we conclude that ATV transport inhibition is an additional antiviral effect of this drug, suggesting a potential anti-DENV therapy in combination with IVM.

Keywords: Antivirals; DENV; NS3; Nuclear transport; Ivermectin; Atorvastatin; FDA-approved drugs.

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Transcriptome profile of peripheral blood mononuclear cells in two groups of COVID-19 hospitalized patients, severe versus fatal outcome: importance of innate and adaptive immune response

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SARS-CoV-2 virus is known to delay the activation of intracellular innate immune responses associated with type I and type III IFN during infection, resulting in the lack of activation of adaptive immune responses. The absence of this response allows high virus replication and viral load, leading to severe pulmonary disease. The aim of this study is to demonstrate the existence of a specific gene profile of the immune response in patients with severe disease, directly related to their recovery or mortality. Blood samples were collected from 41 individuals with a documented SARS-CoV-2 infection through PCR. Their clinical parameters were obtained, RNA was extracted from each sample, and transcriptome characterization was performed using RNA-Seq (Illumina). Subsequently, differential expression analysis was conducted, comparing a group of deceased individuals to those who recovered. Thirty-seven significantly upregulated genes were found in the deceased group with statistical significance, which are related to various functions such as lymphocyte and monocyte proliferation and regulation, metabolic pathways, translation initiation, transport, ion channels, among others. The study suggests that there are upregulated genes in critically ill patients who died that may be related to this outcome.

Keywords: Covid19; Severity; Mortality; Transcriptome; Genes.

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Seroprevalence and Antibody Responses to SARS-CoV-2 in Adults and Children: Implications for Vaccination Strategies

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This study assessed the seroprevalence of SARS-CoV-2 and antibody responses in a high-risk adult population and hospitalized children in the state of Morelos. An ELISA test utilizing the SARS-CoV-2 spike protein's RBD domain was employed to detect IgG, IgM, and IgA antibodies. Among adults, before vaccination campaigns ($n = 114$), 26.3 % tested positive for IgG, while IgM and IgA were detected in 19.3 % and 13.2 %, respectively. The impact of Pfizer-BioNTech (BNT162b2) vaccination was analyzed, revealing significant increases in antibody levels following the second dose. Subsequently, participants receiving third doses from different vaccines exhibited comparable or higher IgG and IgM levels. IgA levels varied, with a subset reaching high levels only after receiving the booster dose. Children, before vaccination campaigns ($n = 115$), displayed 56.5 % IgG seropositivity, despite minimal reported COVID-19 cases. Elevated MIS-C/Kawasaki syndrome diagnoses (15 %) prompted a need for pediatric diagnosis focus. Comparative analysis of antibodies' levels in seropositive adults and children showed higher responses in adults both after infection (IgG: $r = 0.42$, $p < 0.001$; IgM: $r = 0.63$, $p < 0.001$) and after application of two vaccine doses (IgG: $r = 0.52$, $p < 0.001$; IgM: $r = 0.57$, $p < 0.001$; IgA: $r = 0.26$, $p < 0.001$). These findings underscore the necessity for continued antibody response monitoring post-vaccination in children and emphasize the importance of tailored vaccination strategies and diagnostic prioritization to address SARS-CoV-2 immunity in diverse populations.

Keywords: SARS-CoV-2; seroprevalence; antibody responses; vaccination; pediatric population.

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Oral session V

Small extracellular vesicles (sEVs) from dengue virus-infected C6/36 mosquito cells are infective *in vitro* and *in vivo*.

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Dengue virus is a significant arbovirus transmitted by Aedes mosquitoes with endemic and epidemic transmission cycles that make it public health problem. Recently, it has been observed that extracellular vesicles (EVs), which have a significant role in cell communication, can promote viral cell infection. Depending on the origin, size, and markers, EVs with heterogeneous content are classified into large EVs (lEVs) and small EVs (sEVs). Cells constantly release both, and viruses can take advantage of this to transport viral proteins, genomes, and virions. Our work aimed to determine the ability of sEVs from mosquito cells infected with DENV-2 to promote infection in mammalian cell culture and a CD1 mouse model. Our results showed that C6/36 cells could expulse sEVs with viral genome and particles that seem like viruses. Nonetheless, sEVs from infected conditions were larger in size and concentration than the MOCK condition. In addition, we observed that sEVs could be captured and internalized by Huh7 cells, promoting infection in naïve mammalian culture cells. Finally, we demonstrated that neonatal CD1 mice were susceptible to DENV-2 infection caused by sEVs. Our results are the first report demonstrating that sEVs from C6/36 mosquito-infected cells can cause DENV-2 infection *in vivo*.

Keywords: DENV-2; Extracelular vesicles; sEVs; Exosomes; Infection; Mouse; Mosquito.

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Influence of SARS–CoV–2 infection in the expression of IFN-γ (+874 T/A, rs2430561) polymorphism in Mexican population

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Coronavirus disease (COVID-19) is the clinical syndrome associated with SARS-CoV-2 infection. Cytokines such as Interferon (IFN)-γ are important in the antiviral response. Polymorphisms of IFNs become relevant in the context of the infection by SARS-CoV-2. The objectives of this study are to determine the frequency of the IFN-γ (+874 T/A, rs2430561) polymorphism, and to understand the relevance of the IFN-γ and STAT-1 expression when both genes are compared between healthy and infected individuals with SARS-CoV-2; clinical characteristics will also be analyzed. For the genotyping of IFN-γ polymorphism ARMS-PCR technique was performed, and qPCR for gene expression. The novelty of this study is based on the fact that little is known about the IFN-γ (+874 T/A, rs2430561) polymorphism in the Mexican population which can become relevant in the context of the current pandemic caused by COVID-19. Until now, we do not observe any difference regarding the IFN-γ (+874 T/A, rs2430561) polymorphism in both groups. With respect to the expression of IFN-γ and STAT1 genes we do observe a decrease in STAT-1 expression in the SARS-CoV-2 group. With respect to clinical characteristics in the SARS-CoV-2 group we do observe differences between the symptoms analyzed among the genotypes. IFN-γ +874A allele has been previously reported to be associated with infectious diseases, however, in our study we haven't found any correlation. A larger number of individuals is being included for both groups to assess the potential role of the IFN-γ gene (+874) polymorphisms and its expression in the context of the infection by SARS-CoV-2.

Keywords: SARS-CoV-2; IFN-γ; SNP; Susceptibility; STAT1.

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Anti-lymphocyte autoantibodies from patients infected with the human immunodeficiency virus inhibits the lymphocyte membrane fusion induced by viral proteins

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The HIV-1 envelope protein (Env) mediates the membrane fusion process allowing virus entry to target cells. Besides virus receptors, cellular adhesion and signaling molecules can promote fusion. Thus, antilymphocyte autoantibodies (ALA) produced during the natural HIV infection may contribute to the inhibition of HIV entry. Here, sera from 38 HIV-1 infected treatment-naïve men and 30 healthy donors were analyzed for the presence of IgG and IgM able to bind to Jurkat lymphoid cells. Binding of IgG and IgM was detected in 74 % and 84 % of HIV-positive sera, respectively. To determine the effect of ALA on HIV-envelope dependent membrane fusion, the activity of sera on the fusion of Env+ Jurkat cells with CD4+ cells was determined before and after the removal of ALA by sera absorption on Jurkat cells. The fusion inhibitory activity of sera decreased in 58 % of serum samples after adsorption. The contribution of ALA to fusion inhibition was 33 % on average. Similarly, IgG purified from a pool of HIV+ sera inhibited fusion, and removal of IgG ALA diminished this activity. Levels of fusion inhibitory ALA associated with patients' plasma viral loads, suggesting that ALA may contribute significantly to virus containment by inhibition of the HIV envelope-dependent membrane fusion.

Keywords: Antilymphocyte antibodies; HIV-infected patients; Viral load; Cell-cell fusion; IgG.

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Isolation And Characterization Of Anti-Sars-Cov-2 Omicron Antibodies From A Semi-Immune Phage Display Library

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We describe the discovery and characterization of antibodies with potential broad SARS-CoV-2 neutralization profiles. The antibodies were obtained from a phage display library built with the VH repertoire of a convalescent COVID-19 patient who was infected with SARS-CoV-2 B.1.617.2 (Delta). The patient received a single dose of Ad5-nCoV vaccine (Convidecia™, CanSino Biologics Inc.) one month before developing COVID-19 symptoms. Four synthetic VL libraries were used as counterparts of the immune VH repertoire. After three rounds of panning with SARS-CoV-2 receptor-binding domain wildtype (RBD-WT) 34 unique scFvs, were identified, with 27 cross-reactive for the RBD-WT and RBD Delta (RBD-DT), and seven specifics for the RBD-WT. The cross-reactive scFvs were more diverse than the RBD-WT specific ones, being encoded by several IGHV genes from the IGHV1 and IGHV3 families combined with short HCDR3s. Three cross-reactive scFvs and one RBD-WT specific scFv were converted to human IgG1 (hIgG1). The four antibodies blocked the RBD-WT binding to angiotensin converting enzyme 2 (ACE2). Importantly, one of the antibodies also recognized the RBD from the B.1.1.529 (Omicron) isolate, implying that the VH repertoire of the convalescent patient would protect against SARS-CoV-2 Wildtype, Delta, and Omicron. From a practical viewpoint, the triple cross-reactive antibody provides the substrate for developing therapeutic antibodies with a broad SARS-CoV-2 neutralization profile.

Keywords: COVID-19; Receptor-binding domain (RBD); Therapeutic antibodies; Phage display; VOCs.

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Oral session VI

Molecular Epidemiology of HIV-1 in Uruguay: A 15-Year Retrospective Study

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The human immunodeficiency virus (HIV) is responsible for one of history's most significant pandemics. As of 2021, approximately 38.4 million individuals worldwide were living with HIV, with 15 000 cases reported in Uruguay. Genomic surveillance plays a vital role in HIV prevention, including in Uruguay, where we conducted an extensive analysis of 1270 genetic sequences from a 1100 pb fragment of the HIV-1 *pol* gene, spanning the period from 2007 to 2021.

Our findings indicate the presence of subtypes B and C, along with sub-subtypes A1 and F1, within the Uruguayan population. Moreover, we identified diverse recombinant forms, including 01_AE, 12_BF1, 17_BF1, 19_cpx, 20_BG, 24_BG, 28_BF1, 29_BF1, 31_BC, 38_BF1, 41_CD, 46_BF1, and 60_BC. Notably, phylogenetic analyses revealed a robustly supported clade of 107 B/F1 recombinant sequences, which we designated as BF1.UY, that did not group with any previously known HIV-1 linage.

Subtype B emerged as the predominant variant, accounting for 39.2 % of the analyzed sequences, followed by recombinants 12_BF1 (27.7 %), 38_BF1 (12.8 %), BF1.UY (8.3 %), 28/29_BF1 (3.5 %) and subtype C (3.1 %). The remaining subtypes and recombinants collectively represented 5.4 % of the sequences.

This observed genetic diversity underscores the intricate dynamics of HIV/AIDS transmission, highlighting the critical role of genomic surveillance in Uruguay.

Keywords: HIV; Genomic surveillance; Uruguay.

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Proof of concept: Design of an expression system derived from the Vesicular Stomatitis Virus for the production of proteins with biopharmaceutical interest

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This work involved the design and construction of a recombinant expression system in mammalian cells using an autonomously replicating vector derived from *Vesicular Stomatitis Virus* (VSV) for the production of biopharmaceutically relevant proteins.

VSV is the prototype virus of the *Rhabdoviridae* family, characterized by a single-stranded negative-sense RNA genome. Moreover, it exhibits a broad cellular tropism, as well as a particular mechanism of replication and transcription.

Initially, we designed and constructed a defective recombinant virus lacking the *L* and *P* genes, which are the components that make up the viral replicase. Additionally, a site for the insertion and cloning of a heterologous gene of interest was introduced. Ultimately, the goal is to generate a recombinant cell line capable of expressing the VSV viral replicase.

While the results obtained so far are not sufficient to draw conclusions regarding our proposed expression system utilizing the VSV replication and transcription system, we believe that our concept holds great promise for the large-scale production of biopharmaceutical products in mammalian cell lines.

Keywords: VSV; System; Recombinant; Defective; Replicase.

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Genotyping of feline leukemia virus and feline immunodeficiency virus in naturally infected domestic cats

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Feline leukemia and immunodeficiency viruses (FeLV and FIV) are retroviruses with worldwide distribution that infect different species of felines, including domestic cats. Infections caused by feline retroviruses can cause various syndromes that can lead to the death of the host. The identification and segregation of infected cats is the most important measure for the control of infections caused by feline retroviruses, with viral genetic variability and the phase of infection being key points for the use of different diagnostic tools. The objective of this study was to genotype feline retroviruses present in naturally infected domestic cats. Detection of retroviral infections was performed with lateral flow immunochromatography and endpoint PCR assays in 50 domestic cats with different clinical-haematological characteristics. The FeLV genetic sequences corresponded to subgroup A and were shown to be related to sequences previously reported in America. Phylogenetic analysis of the FIV sequences revealed the presence of the B subtype in the infected cats.

Keywords: Feline leukemia virus; feline Immunodeficiency virus; Genotyping; Provirus; Retroviridae.