both species. Next, to study putative targets of *Fx-mir* miRNAs, we searched for miRNAs predicted to target *Fmr1* using the gene of interest as input in specialized target finding softwares. We also made the reverse search, finding every possible target for each miRNA of *Fx-mir*. We found that miR-880 is a possible regulator of *Fmr1*.

Finally, we extended the search to all of the X chromosome genome sequence, finding 8 more candidates predicted to target *Fmr1* that might be of interest.

17. (155) ALTERATIONS IN THE INTERFERON PATHWAY WITHIN COVID-19 INFECTION

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Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a novel virus that emerged in late 2019 in Wuhan, China. Although much attention has been placed in virus host cell receptors, little has been described about the anti-viral proteins. It is well accepted that type I interferon (IFN) is essential in fighting viral infection by induction of IFN-stimulated genes (ISGs), which work in synergy to inhibit viral replication via multiple mechanisms. In this work, we aimed at evaluating the expression profiles of several genes associated with the IFN pathway in response to the infection with SARS-CoV-2 in COVID-19 positive patients vs. COVID-19 negative patients. We performed bioinformatics analyses in a case-control study from SARS-CoV-2 positive (n=403) and negative (n=54) patients. Samples were obtained from nasopharyngeal swabs. We analyzed the differential expression of the IFN-associated genes alongside with their correlation with other clinical parameters such as age, sex and viral load. Results show a significant downregulation of IFGNR1, STAT6, JUN, MAP3K1, CEBPB, and RAPGEF1 in COVID-19 positive patients. We also found a significant correlation between most of the genes and the viral load. No significant differences were observed between gene expression and age or sex. In summary, our study findings support the role of IFN and IFN-associated genes in SARS-CoV-2 infection, pointing out to new potential drugable targets in order to achieve a better anti-viral response.

18. (177) COMBINED ACTIVITY OF IVERMECTIN PLUS ATOR-VASTATIN ON NUCLEAR LOCALIZATION OF IMPOR-TIN ALPHA AND THERAPEUTIC TARGET EXPRESSION PROFILING IN HOST CELLS FROM NASOPHARYNGEAL SWABS OF SARS-COV-2-POSITVE PATIENTS

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Nuclear transport and vesicle trafficking are key cellular functions in-

volved in the pathogenesis of RNA viruses. Among other pleiotropic effects on virus-infected host cells, ivermectin (IVM) inhibits nuclear transport mechanisms mediated by importins and atorvastatin (ATV) affects actin cvtoskeleton-dependent trafficking controlled by Rho GTPases signaling. In this work we first analyzed the response to infection in nasopharyngeal swabs from SARS-CoV-2-positive and -negative patients by assessing gene expression of the respective host cell drug targets importins and Rho GTPases. COVID-19 patients showed alterations in KPNA3, KPNA5, KPNA7, KPNB1, RHOA and CDC42 expression compared with non-COVID-19 patients. An in vitro model of infection with Poly(I:C), a synthetic analog of viral double-stranded RNA, triggered NF- κB activation, an effect that was halted by IVM and ATV treatment. Importin and Rho GT-Pases gene expression was also impaired by these drugs. Further, by confocal microscopy we analyzed the effects of IVM and ATV on nuclear to cytoplasmic importin a distribution, alone or in combination. Results showed a significant inhibition of importin α nuclear accumulation under IVM and ATV treatments. For gene expression analysis we performed two-tailed Welch's t tests or Wilcoxon rank sum test. For correlations Spearman's rank coefficient was calculated. In cellular studies, Mann-Whitney or t-tests were used. In case of more than 2 experimental groups, ANOVA or Kruskal-Wallis analysis were used. Differences were considered statistically significant at a level of p<0.05. Data processing and statistical analysis was performed using Prism 6.1 Software and R. These findings confirm transcriptional alterations in importins and Rho GTPases upon SARS-CoV-2 infection and point out to IVM and ATV as valid drugs to impair nuclear localization of importin a when used at clinically-relevant concentrations

19. (186) HUMAN GUT MICROBIOTA ASSOCIATED WITH SYSTEMIC LUPUS ERYTHEMATOSUS: A PILOT STUDY IN AN ARGENTINE POPULATION

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BACKGROUND: The ability of a microorganism, including commensals, to trigger disease is highly dependent on the host's activation state, the location of the particular microorganism, as well as the genetic predisposition of the individual. In this sense, studying possible changes in the intestinal microbiota related to the initiation, progression and response to treatment of patients with autoimmune diseases, such as Systemic Lupus Erythematosus (SLE), is an interesting field which promotes a comprehensive view of chronic inflammatory processes of increasing incidence worldwide. In the present study we aim to describe the unknown gut microbiota of SLE-patients of the Argentine population in comparison with healthy individuals in order to find novel SLE-biomarkers in our region.

METHODS: We evaluated 24 non-SLE-controls and 13 SLE-patients, from the metropolitan area of

Buenos Aires, Argentina. Fecal DNA was extracted and hypervariable regions V3-V4 of the bacterial 16SR-gene were sequenced using a MiSeq-Platform. Sequences were analyzed with the QIIME2 environment and differential functional pathways were evaluated using PICRUSt. RESULTS: In SLE-patients we found no significant differences in alpha diversity compared to non-SLE-control. However, Beta diversity was significantly different between groups (UniFrac distances, PERMANOVA, p-value <0.05). Additionally, functional metabolic pathways were analyzed and it was found that SLE patients have different metabolic capabilities compared to the control group. Six metabolic pathways were found from the Metacyc database mainly associated with the degradation of aromatic compounds and fatty acid biosynthesis. CONCLUSIONS: Overall, our study provides new knowledge on the gut microbiota composition of our population, allowing the association of local changes in gut