Virtual Poster Session 1:

Biological Sciences Collegiate Division
Developing a Visualization Tool for the Geographic Distribution of Linkage Disequilibrium

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Mentor(s): Prof. John Novembre, Human Genetics; Arjun Biddanda, Human Genetics

Linkage disequilibrium (LD) refers to the non-random association between alleles on chromosomes. Numerous factors affect LD, including recombination rates, population demographic changes, and natural selection. Differences in LD data between populations can be leveraged to determine the particular alleles responsible for a given phenotype, allowing us to improve our understanding of disease mechanisms and potentially improving our ability to treat these diseases. At present, a universal tool for accessing LD data across global populations through an API remains absent. With this project, we aimed to fill that gap by developing a tool to access LD data and compare it across populations with ease. In the process of developing this framework, the EmeraLD package and Flask API were used extensively. We also provide an exploration of strategies for storing LD data. The estimated storage requirements for chromosome 22 without the use of such strategies were roughly 5GB, necessitating such efforts. We utilized banded matrices, and adaptively banded matrices, in an attempt to improve efficiency through reducing the storage required by the data. Through performance-testing these strategies, we found that it was not viable to satisfy this aim without sacrificing an unacceptable level of LD data that we determined to be relevant. Through the development of a public facing API, we can enable broader access to LD data without requiring substantial data storage costs.
Automatic Segmentation and Analysis of COVID-19 Patient CT Scans Using Deep Learning
Beatrice Katsnelson, 2nd-Year, Biological Sciences, Computer Science, Molecular Engineering, Technology and Innovation
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Mentor(s): Prof. Maryellen Giger, Radiology; Jordan Fuhrman, Committee on Medical Physics, Giger Lab

COVID-19 is an infectious disease that can cause mild to severe respiratory symptoms, such as trouble breathing and pneumonia, with severe cases leading to death. An efficient and automatic method for analyzing COVID-19 presentation in the lungs on thoracic CT scans is desirable for informing treatment decisions and patient management. Accordingly, the purpose of this study is to create a deep learning model that can simultaneously produce segmentations of the lungs and tissue indicative of COVID-19 disease on CT scans in order to extract quantitative information, such as the percentage of COVID-19-infected tissue in the lungs, and to use this information to predict clinically relevant information.

Thoracic CT scan images from 41 patients each with multiple temporally separate CT acquisitions (total of 221 scans) were manually segmented, serving as “truth” for training, validation, and testing data using the MATLAB Image Labeler program. A multi-headed U-Net convolutional neural network model architecture was used for the automatic segmentation with two up-sampling paths, one for lung segmentation and one for COVID-19 lung involvement segmentation. The U-Net model is evaluated through the Dice coefficient, which assesses the overlap between the model segmentations of the lungs and COVID-19 involvement compared to manual segmentations. With the ability to produce lung and COVID involvement segmentations, a correlational analysis will be performed to determine the association between the COVID-19-infected tissue in the lungs and clinical information, such as the patient’s duration of hospitalization. Accurate prediction of a patient’s length of hospital stay from their thoracic CT scan upon admission to the hospital has broad reaching applications, including the hospital’s ability to better plan patient care and schedule hospital staff and rooms, as well as economic applications for the hospital, insurance, and the patient’s family.
Many types of cells need to adopt polarized morphologies to function. In metazoans, polarity often emerges from asymmetric distributions of proteins in the highly conserved partitioning-defective (PAR) network. The PAR network consists of two sets of proteins which locally compete for binding to the cell membrane and are typically enriched in complementary sub-cellular domains. The protein LGL-1 is a core member of the PAR network and plays a key role in maintaining polarity in multiple contexts, but how it does so remains unclear. In the *C. elegans* zygote, PAR proteins segregate along the anterior/posterior axis. LGL-1 localizes to the posterior, and is necessary for maintaining PAR asymmetries in sensitized genetic backgrounds. I will use this cell as a model to evaluate two hypothesized mechanisms for how LGL-1 maintains cell polarity: (1) LGL-1 sequesters the anteriorly localized heterodimer PAR-6/aPKC in the cytoplasm, and (2) LGL-1 binds to PAR-6/aPKC on the cell membrane and dissociates it into the cytoplasm. To resolve this question, I have represented these hypotheses as mathematical models, which I have constrained with empirical measurements. I will use these models to determine whether or not these hypotheses can plausibly explain a set of experimental observations where LGL-1 is required for stable polarity in the absence of other posterior PAR proteins. Ultimately, I hope to quantify the extent to which each hypothesis contributes to polarity maintenance in the *C. elegans* embryo.
Investigating the Role of B7-H3 in PTEN-Deficient Prostate Cancer

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Mentor(s): Prof. Akash Patnaik, Medicine; Dr. Jordan Shafran, Medicine, Patnaik Lab

Prostate cancer (PCa) is the second leading cause of cancer-related mortality in the United States. The standard of care for advanced prostate cancer patients is androgen deprivation therapy, but disease progression to metastatic castration-resistance prostate cancer (mCRPC) remains inevitable. In addition to genomic alterations in the androgen receptor (AR) pathway, PTEN loss-of function (LOF) occurs in 50-75% of mCRPC patients and correlates with disease progression and poor clinical outcomes. PTEN LOF leads to hyperactivation of the PI3K pathway to promote growth, proliferation, and angiogenesis. It has also been shown to induce an immunosuppressive tumor microenvironment (TME) with low T-cell infiltration. Prior studies have shown that the androgen/AR signaling axis can suppress the expression of a B7 superfamily member, B7-H3 (CD276), and that B7H3 expression correlates with high Gleason score and progression towards metastatic/hormone refractory PCa. Furthermore, activation of PI3K/AKT/mTOR signaling upregulates B7-H3 in non-small cell lung cancer. We hypothesized that PTEN loss-of-function coupled with AR suppression enhances the expression of B7-H3, leading to an adaptive resistance mechanism that results in an immunosuppressive, pro-tumorigenic tumor microenvironment. In vitro results have confirmed that loss of PTEN via CRISPR/CAS9 deletion or transient knockdown in murine and human PCa models increases the expression of B7-H3. Mechanistically, preliminary findings suggest that B7-H3 is regulated by PTEN in a PI3K/AKT/mTOR-independent manner. Experiments are in progress to evaluate the immunosuppressive function of B7-H3 in vivo using MYC-driven murine PCa models, in order to better utilize novel immuno-oncology strategies to treat PTEN-deficient PCa.
More Cases of Cry Wolf? The Effect of False Alarms and Misses on Air Traffic Controllers  
Grace A. Richards, 3rd-Year, Statistics  
Mentor(s): Prof. Keith J. Ruskin, Anesthesia and Critical Care

Air Traffic Control (ATC) facilities are high-stress environments that require rapid decision making in dynamic situations; errors can be life-threatening and are unacceptable. This project is a critical component of NextGen, an FAA project to modernize the nation’s airspace, focusing specifically on improving the safety and effectiveness of alarms, alerts, and warnings (collectively called signals) for air traffic controllers. My aim was to identify both the scope and nature of any signal problems by modeling data collected from NASA’s Aviation Safety Reporting System (ASRS) database. Through simple odds ratio calculations, I found that the automated alarms were much more prone to signal response error than the air traffic controllers themselves, and using logistic regression, I found that signal errors may be associated with human error. The data suggest, then, that signals may contribute to human error. Moreover, I intend to use techniques such as multinomial logistic regression to identify which alarms are most prone to signal error. Altogether, this information will help my research team to both identify the problem and suggest enhancements to alarms, alerts, and warnings, as part of a handbook that the FAA will use to design signals for new equipment.
Actin cytoskeleton networks are necessary for a diverse number of cellular processes, such as migration, polarization, trafficking, vesicle transport, and division. Assembly and organization of these networks are mediated by a variety of actin-binding proteins. TIRF microscopy is a valuable strategy to directly visualize how individual actin filaments in these networks are formed and maintained; however, current analysis of this microscopy data is time-consuming and requires manual tracking. To improve analysis efficiency, we worked on developing an algorithm that 1) tracks user-specified filaments with high fidelity, 2) allows manual adjustments to correct any potential mistakes, 3) handles events such as filament annealing, breaking, and crossing over, and 4) analyzes the data to output filament elongation rates. With further work, we plan to increase filament detection sensitivity and robustness against noise, optimize computing speed, and develop modules that will output different measurements and analyses. This algorithm will greatly reduce the amount of time it takes to analyze actin filaments in microscopy movies and will be useful in my own analysis as I move forward with biochemical characterization of the actin-binding protein \textit{Diaphanous}, which is a member of the formin class of actin-binding proteins. These proteins typically work in tandem with another actin-binding protein, profilin, to increase the rate at which individual actin filaments elongate. However, preliminary data has suggested that \textit{Diaphanous} is capable of mediating increased rates of actin filament elongation in the absence of profilin. Through in vitro pyrene assays and TIRF microscopy, I will characterize the biochemical properties of \textit{Diaphanous} and determine whether it does enhance filament elongation rates in the absence of profilin. This property in the formin would suggest a novel mechanism by which the protein mediates actin filament elongation and deepens our understanding of how \textit{Diaphanous} and actin interact to form cellular networks.
Effect of Public Policies to Reduce Smoking: A Narrative Review
Jacob Gillis, 3rd-Year, Public Policy, Health and Society
Mentor: Prof. Marcia Tan, Public Health Sciences

Tobacco smoking poses health risks, and, consequently, governments have taken a role in efforts to reduce tobacco usage. Governments have focused on increasing taxes on tobacco products, requiring smoke-free laws in public establishments, and sponsoring mass media campaigns to reach this goal. The objective of this project was to review the effectiveness of these policies and provide recommendations for future policies. In this qualitative narrative review, articles examining the success of initiatives to reduce smoking were analyzed. These articles were found in public health and economics journals located using the search engine Google Scholar. Methodologies referenced in these articles included time-series analyses and population-based studies. Metrics, including the smoking rate and cigarette usage, were used to evaluate the success of these policies. The findings of the current study indicate that cigarette taxation is regressive and leads smokers with lower incomes to ingest nicotine more intensely. Advertisements evoking strong emotions are most successful in convincing viewers to quit. Finally, smoking bans within public establishments play a critical role in reducing the likelihood that individuals begin smoking and incentivizing current smokers to quit, particularly among persons with low income. Recommendations based on the current study’s findings include (a) taxes on tobacco products should be increased; however, the revenue should be directed to low-income populations adversely impacted by smoking, (b) governments should invest increased resources in anti-tobacco media campaigns, especially advertisements featuring strong emotions and graphic images, and (c) governments should expand the scope of public facilities where smoking bans apply. Tobacco control research is valuable as it helps inform policymakers at the local, state, and federal levels on the most effective strategies to minimize the consumption and societal effects of tobacco products. An extension of the current research will focus on the role of school curricula in curbing smoking among young adults.
Optimization of DNA Extraction and Characterization of DNA Damage Patterns in Genome-Wide Data from Formalin-Fixed Paraffin-Embedded (FFPE) Human Tissues

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Mentor(s): Prof. Maanasa Raghavan, Human Genetics

Genomic analysis of formalin-fixed paraffin-embedded (FFPE) tissue samples offers a window into disease evolution and the genetic basis of observed epidemiological patterns over time. Key limitations in the analysis of FFPE DNA include inadequate DNA extraction methods and formalin-induced DNA damage. The aim of this project was twofold: (1) to optimize DNA extraction from archival FFPE tissue samples spanning the last ~100 years and (2) to characterize FFPE-specific DNA damage patterns using published genomic datasets. For the first aim, systematically testing and modifying 4 commercial FFPE-DNA extraction protocols using freshly prepared FFPE blocks revealed that the Covaris truXTRAC FFPE DNA kit with a modified incubation step yields the largest amount of DNA (mean = 4881 ng). Subsequent DNA extractions from archival FFPE tissue samples dating back to 1927 revealed that the novel extraction method recovers enough high-quality DNA for next-generation sequencing from samples that are over 30 years old. For the second aim, comparatively analyzing data from 26 pairs of FFPE and fresh-frozen (FF) tissue showed that the FFPE-derived DNA is significantly more fragmented than FF-derived DNA (FFPE mean = 141 bp, FF mean = 148 bp; p = 2.10E-08). Additionally, an average of 30.5% of the sequencing reads in FFPE-derived DNA were enriched in adenine immediately 5’ upstream of the read start, but only 23.3% of the sequencing reads in FF-derived DNA (p = 1.22E-20), suggesting that fragmentation in FFPE DNA predominantly occurs at an adenine base. In contrast, FF DNA showed a significant increase in the frequency of cytosine at the same position (FF mean = 28.3, FFPE mean = 24.8%; p = 7.61E-16). In summary, my research proposes an optimized method for DNA extraction and establishes preliminary DNA damage patterns resulting from formalin fixation. These insights are useful to researchers generating sequencing data from historical FFPE tissue.
Genetic Underpinnings of Coinfection Effects in a Natural Plant-Pathogen System

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Mentor(s): Prof. Joy Bergelson, Ecology and Evolution

Infectious disease has impacted the world in a myriad of ways. In agricultural settings, infection alone accounts for $220 billion in damage and majorly affects 20 – 40 percent of global agricultural efforts. While the molecular basis of single infection in many diseases is well-established in many agricultural and model species, efforts to provide lasting resistance to disease have failed. One important reason for this is that plants interact with complex microbial communities, whose interactions can greatly influence both pathogen evolution (including resistance) and disease outcomes. In particular, coinfection of multiple pathogen strains is common in nature, and has been shown to drastically alter pathogen performance. Although the outcome of a given host-pathogen interaction often differs drastically between single and co-infections, the genetic basis of these differences remains poorly understood. The model plant Arabidopsis thaliana, and one of its dominant bacterial pathogens in nature, Pseudomonas viridiflava, provide a well-characterized, practical study system to characterize how coinfection shapes the genetic architecture of pathogen virulence. Using a diverse set of 224 viridiflava strains with sequenced genomes, isolated from Arabidopsis, and transformed with luciferase, we have developed a high-throughput luminescence-based assay which quantifies in-planta bacterial growth, a common measure for pathogenicity. This system has provided insight into the differential outcomes associated with coinfection. From this data, we conduct GWAS using both phylogeny-based and kinship-based approaches to discover loci associated with coinfection effects. P. viridiflava strains have revealed genetic variants strongly associated with coinfecting strains but absent in singular infection, suggesting evolution of traits particular to coinfection settings. We infer aspects of the genetic underpinnings of coinfection in a dominant Arabidopsis pathogen, and provide further evidence of the importance of microbial interactions in pathogen evolution and plant disease.
Impact of Infections on Endogenous Alloreactive T cell Populations
Peter Wang, 3\textsuperscript{rd}-Year, Biological Sciences, History
Mentor(s): Prof. Maria-Luisa Alegre, Medicine, Rheumatology

Donor-specific transplantation tolerance can be achieved in an experimental model of heterotopic heart transplantation where, at the time of transplantation, recipients receive an injection of donor splenocytes (DST) + an antibody that blocks CD40/CD40L costimulation (anti-CD154). Using this model, we have previously shown that endogenous CD4\textsuperscript{+} conventional T cell (Tconv) populations specific for highly expressed constitutive graft alloantigen fail to expand their high avidity clones, in contrast to the same T cell populations in rejecting mice. This low avidity population-level state is fixed long term and resistant to alloantigen rechallenge and occurs even in mice treated with DST+anti-CD154 in the absence of a graft. We have also reported that infections with \textit{Listeria monocytogenes} (Lm) after tolerance induction can precipitate T cell-dependent transplant rejection. Our goal is to investigate if Lm infection at the maintenance phase of tolerance can expand high avidity Tconv clones, perhaps explaining Lm-dependent loss of tolerance. We found that infection with Lm engineered to express the same constitutive alloantigen shared with the DST could expand high avidity alloreactive Tconv clones in mice previously treated with DST+anti-CD154 in the absence, but not in the presence of a DST-matched allograft. In transplanted recipients, Lm-mediated loss of tolerance did not alter the allospecific Treg:Tconv ratio, nor the expression of the anergy markers CD73 and FR4. This suggests that antigen persistence in the graft results in stronger cell-intrinsic and cell-extrinsic control of alloreactive Tconvs than transient alloantigen expression in the form of DST without a graft. Our results also raise the hypothesis that rejection after Lm infection may be driven by T cells specific for alloantigens upregulated during inflammation, rather than those constitutively expressed at high levels.
Developing Computational Tools to Predict COVID-19 Symptom Severity  
Rahul Gupta, 3rd-Year, Biological Sciences, Classical Studies  
Mentor(s): Prof. Tao Pan, Biochemistry and Molecular Biology; Dr. Chris Katanski, Biochemistry and Molecular Biology, Pan Lab

Existing nasal swab tests for SARS-CoV2 can diagnose patients infected with COVID-19, however, tests that can predict severe symptom development are still needed. The wide range of symptom intensities is a major challenge in the ongoing COVID-19 pandemic, even looking into the far future. In response to viral infection, many features of cellular physiology change as cells mount a defense and viruses hijack the cells’ translation machinery for their own replication. One set of molecules involved in these changes are transfer RNAs (tRNAs). These abundant non-coding RNA molecules decode mRNA and deliver amino acids for protein translation. During viral infection, tRNAs can be cleaved and undergo chemical modifications and changes in expression. Recent work in the Pan lab has studied how cellular tRNAs respond to SARS-CoV2 infection using patient nasal swabs and identified several possible biomarkers that predict symptom severity at the time of diagnosis. However, these data were generated with Next-generation sequencing. Such measurements are slow and expensive, and are not suitable for rapid scalable testing. Here, I develop computational tools to identify potential qPCR-based targets from tRNA-sequencing data. In contrast to mRNA, qPCR targets for tRNA are not trivial, as one must consider strong secondary structure, abundant modifications, and incomplete sequencing coverage. These tools will facilitate translation of tRNA-sequencing results to scalable qPCR-based tests for predicting COVID-19 symptom severity, and potentially other diseases beyond COVID-19 as well, such as various cancers in which tRNAs are implicated.
Investigating the Connection Between Arginine Synthesis and Other Metabolic Pathways in Pancreatic Adenocarcinoma
Riona (Rio) Chen, 3rd-Year, Psychology
Mentor(s): Prof. Alexander Muir, Cancer Biology

Pancreatic adenocarcinoma (PDAC) is extremely deadly, with only a 9.3% five-year survival rate, highlighting the need for novel therapeutic approaches. We are working to target metabolic alterations that PDAC requires due to abnormal tumor nutrient availability. Previously we have found that PDAC uses argininosuccinate synthase 1 (ASS1) to de novo synthesize arginine in an arginine-deprived tumor microenvironment. Perturbing de novo arginine synthesis either by using CRISPRi technology to knockdown ASS1 expression or removing citrulline, a necessary precursor of arginine, significantly slows PDAC growth. Although slowed, PDAC cells are still able to grow. Thus we want to explore how we can further decrease PDAC growth by inhibiting other metabolic pathways in combination with inhibiting de novo arginine synthesis. Madiraju and colleagues recently proposed a novel link in the liver between ASS1-mediated arginine synthesis and the AMPK nutrient sensing pathway. They found in hepatocytes that locally generated AMP, produced by ASS1 when generating arginine, activates AMPK. AMPK is also known to help tumors adapt and survive under nutrient stress. I hypothesized that AMPK activity helps nutrient-deprived PDAC cells survive in an ASS1-linked manner. However, after using CRISPRi technology to knockdown AMPK expression, we found no change in PDAC growth even under ASS1-inhibited conditions. As a result we are now pivoting to investigate other adaptations that PDAC cells use to cope with ASS1 inhibition. From previously published work identifying mechanisms of cellular adaptation to amino acid starvation, we identified several candidate pathways that may mediate PDAC’s ability to grow without ASS1 activity. We are currently determining if targeting these pathways in combination with perturbed de novo arginine synthesis might be effective at further slowing PDAC growth. We hope these results can provide insight into developing more effective PDAC treatments by targeting a combination of metabolic adaptations that PDAC is reliant on.
Predicting Tumor Grade and Mutation Status Using Computed Tomography Radiomic Features in Pancreatic Neuroendocrine Liver Metastases

Ryan Hoang, 2nd-Year, Biological Sciences
Mentor(s): Dr. Etay Ziv, Memorial Sloan Kettering Cancer Center Department of Interventional Radiology

Tumor grade and mutation status are putative biomarkers of response to trans-arterial embolization of pancreatic neuroendocrine liver metastases (PNLM). We sought to evaluate the effectiveness of using computed tomography (CT) radiomic features to classify grade and mutation status of PNLM. 40 patients with NLM who underwent CT scans and biopsies at a single center from April 2009 to February 2018 were included in the database. Biopsied tumors with recorded tumor grade (Grade 1-3) and mutation status (presence or absence of MEN1/DAXX/ATRX mutation) were segmented in both the hepatic arterial (HAP) and portal venous (PV) phases using 3D Slicer. Quantitative features (n=115) from each scan were extracted from segmented tumors using PyRadiomics. Standard feature normalization was performed. Principal component analysis (PCA) was used for dimensionality reduction. Linear and radial basis function support vector machine (SVM) were used for training. K-fold cross-validation was used to estimate testing accuracy. Permutation testing was used to assess statistical significance. There were 25/40 (62.5%) patients with MEN1, DAXX, and/or ATRX mutation. There were 9/40 (22.5%) G1 tumors, 16/40 (40%) G2 tumors, and 37.5% G3 tumors. Over 95% of the variance in the data was described by the top 6 principal components, which were used for training and testing. Testing accuracy was 75% +/- 11% for predicting mutation status (p=0.008). Testing accuracy was 52.5% +/- 11% for predicting tumor grade (p=0.004). CT radiomic features can be used to predict mutation status and tumor grade in patients with PNLM.
Impact of Household Air Pollution on Olfactory, Pulmonary, and Cognitive Function in Rural Srinivaspura in Karnataka, India
Simatul Rashid, 4th-Year, South Asian Language and Civilizations, Biological Sciences
Mentor(s): Prof. Jayant Pinto, Surgery

Household air pollution (HAP) is the cause of increased morbidity and mortality in India. The health risks from household air pollution include ischemic heart disease, stroke, chronic obstructive pulmonary disease (COPD), and lung cancer. Exposure to HAP results from the burning of biomass fuel such as coal, wood, dung, or agricultural residues for cooking in India. As rural inhabitants are more reliant on these cooking methods than urban residents, they are at increased risk from HAP. Dr. Jayant Pinto from the University of Chicago Department of Surgery and his colleagues from the Indian Institute of Science are evaluating the negative impact of HAP on olfactory, pulmonary, and cognitive functions in rural households of Srinivaspura in Karnataka, India. Since we are in the initial stages of the project, we are currently trying to quantify air pollution levels in rural households in Srinivaspura. Due to the COVID-19 pandemic, this project was suspended until recently. In March 2021, the team in India set up the first Edimax air pollution monitor in Srinivaspura. Another Edimax air pollution monitor was installed in a nearby location on a separate network on April 1st, 2021. The team in India is currently in the process of installing a third device made by Purple Air that stores data for intermittent transmissions. We anticipate seeing higher than healthy levels of air pollution within the households in Srinivaspura from the data. The overall effort of this project to quantify and characterize the impact of HAP on rural Indians’ health will help inform public health measures in India to mitigate adverse health related outcomes from HAP and add substantially to the current literature on this subject.
Physiologic changes during pregnancy alter ways in which the body responds to infectious and inflammatory insults. In this study, we aimed to describe the symptoms and clinical presentation of pregnant patients diagnosed with COVID-19, a novel virus characterized by respiratory, inflammatory, and vascular complications. Understanding how COVID-19 manifests in high risk black pregnant patients can help inform the medical management of this vulnerable, understudied population. A retrospective, descriptive analysis of COVID-19 positive pregnant patients admitted at an urban tertiary care center in the south side of Chicago was conducted to collect maternal medical information/outcomes. Overall, 56 subjects were included. Common obstetric diagnoses included labor/contractions (n=25, 44.6%), ruptured membranes (9, 16.1%) and bleeding (3, 5.4%). COVID testing was prompted by symptoms in 18 (32.1%) patients and universal screening in 38 (67.9%) patients. Of those patients included 15 (27.3%) had a cough, 8 (15.4%) had a fever, 5 (9.3%) had diarrhea, 10 (18.5%) had shortness of breath, 6 (11.1%) had chest pain, 7 (12.9%) had a headache, 7 (12.9%) had myalgia, 6 (10.9%) had malaise, and 9 (16.7%) lost their smell or ability to taste. There were no maternal deaths, but 2 (3.9%) patients were admitted to the ICU antepartum and 1 (2.0%) postpartum. Overall, 5 patients were intubated. The median (IQR) GA of delivery was 39.07 (37.29, 39.71) weeks, with 11 (22.0%) experiencing preterm (<37 weeks) labor. Overall, 19 (37.2%) patients underwent a cesarean delivery. A large majority of patients were asymptomatic and diagnosed with COVID-19 by universal screening. The rates of preterm labor and cesarean delivery were similar or higher than the national average. This data will enable the analysis of the risk factors and inform the treatment of this virus throughout this understudied population.