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Background. Human coronaviruses (HCoV) OC43, 229E, NL63 and HKU1 commonly cause upper respiratory tract infections, but can also cause severe lower respiratory tract disease. Increased use of diagnostic assays for respiratory viruses has facilitated detection and, since 2014, voluntary reporting of HCoV to the National Respiratory and Enteric Virus Surveillance System (NREVSS).

Methods. We reviewed weekly aggregate test results for HCoV OC43, 229E, NL63 and HKU1 voluntarily reported to NREVSS by U.S. hospital and clinical laboratories from July 1, 2014-April 30, 2017. Laboratories reporting any HCoV result using PCR were included, and the weekly percentage of positive HCoV tests by type was calculated. For a subset of HCoV detections reported to NREVSS via the Public Health laboratory Interoperability Project (PHLIP), which collects individual-level demographic data, we described age distribution and sex. Age distribution by HCoV type was compared using the Kruskal–Wallis test.

Results. 154 laboratories, across all 9 U.S. census divisions, reported 834,742 tests for HCoV. 18,514 (2.2%) were positive for HCoV-OC43, 8,363 (1.0%) for HCoV-NL63, 6,828 (0.8%) for HCoV-229E, and 5,170 (0.6%) for HCoV-HKU1. The percentage of tests positive for HCoV generally peaked between December and March (Figure 1). HCoV-OC43 showed distinct annual peaks with variation in magnitude by year. HCoV-HKU1 and NL63 had similar patterns, each with notable peaks during winter 2016 compared with 2015 or 2017. HCoV-229E showed a discernable peak in 2017 compared with the previous 2 years. Of 26,533 individuals with HCoV test results by year. HCoV-HKU1 and NL63 had similar patterns, each with notable peaks during winter 2016 compared with 2015 or 2017. HCoV-229E showed a discernable peak in 2017 compared with the previous 2 years. Of 26,533 individuals with HCoV test results reported via PHLIP, 1,589 (7.7%) tested positive for any HCoV; 50% of HCoV-positive tests for HCoV; 18,514 (2.2%) were positive for HCoV-OC43, 8,363 (1.0%) for HCoV-NL63, 6,828 (0.8%) for HCoV-229E, and 5,170 (0.6%) for HCoV-HKU1. The percentage of tests positive for HCoV generally peaked between December and March (Figure 1). HCoV-OC43 showed distinct annual peaks with variation in magnitude by year. HCoV-HKU1 and NL63 had similar patterns, each with notable peaks during winter 2016 compared with 2015 or 2017. HCoV-229E showed a discernable peak in 2017 compared with the previous 2 years. Of 26,533 individuals with HCoV test results reported via PHLIP, 1,589 (7.7%) tested positive for any HCoV; 50% of HCoV-positive individuals were male, and the median age was 22 (range 0–96) years. Age distribution differed between HCoV types (P < 0.01, Figure 2).

Conclusion. Over approximately 3 seasons, peak positivity for HCoV occurred during winter months, and annual differences in circulation by HCoV type were observed. Continued testing and surveillance for HCoV will allow for further characterization of circulation trends over time and by geographic region, and improved understanding of the contribution of HCoV to the winter respiratory virus season.

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1032. Human Coronavirus (HCoV) Infection Among Adults in Cleveland, Ohio: An Increasingly Recognized Respiratory Pathogen
Anubhav Kanwar, MD; Suresh Selvaraju, PhD; and Frank Esper, MD; 1Medicine, Division of Infectious Diseases and HIV Medicine, Case Western Reserve University School of Medicine/UH Cleveland Medical Center, Cleveland, Ohio, 2Microbiology, MetroHealth Medical Center, Cleveland, Ohio, 3Rainbow Babies and Children's Hospital, Cleveland, Ohio
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Background. Human Coronaviruses (CoV) have been long recognized as a common cause of respiratory tract disease including severe respiratory tract illness, yet there are few recent studies characterizing disease among adults in the United States. Here, we describe CoV infections and clinical characteristics among adults (>18 years) presenting with respiratory illness in Cleveland, Ohio.

Methods. Between February 1, 2016 and April 30, 2017, 2949 nasopharyngeal swab specimens were analyzed by NxTAG Respiratory Pathogen Panel in adults presenting with respiratory illness at MetroHealth Medical Center. Clinical data were collected on adults whose samples screened positive for CoV-HKU1, CoV-OC43, CoV-229E or CoV-NL63.

Results. Coronaviruses were detected in 192 (6.5%) adults including 105 (3.5%) OC43, 67 (2.3%) 229E, 13 (0.4%) HKU1 and 7 (0.2%) NL63. The majority of adults with coronavirus infection were females (66.2%) with a median age of 53 years. Common comorbidities included smoking (40.0%), asthma (38.0%), COPD (35.4%), and inhaled corticosteroid use (28.6%). Eighty-five (46.4%) required admission to the hospital. Common presenting symptoms included shortness of breath (42.7%) and cough (31.0%) whereas fever was uncommon (12.5%). Gastrointestinal symptoms were more common in HKU1 and NL63 infected adults. Seventy-three percent of coronavirus disease occurred between the months of January and March. Despite the recognition of coronavirus infection, 70 (36.5%) received antibiotics for their disease.

Conclusion. This study provides needed insight into clinical characteristics and severity associated with coronavirus infection in adults. Coronavirus infection should be considered in differential diagnosis of respiratory tract illness in adults including those that require hospitalization, have a history of smoking and have pulmonary comorbidities.

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1033. Evaluation of Serum TNF-alpha, IL-6, IL-10, and IFN-gamma Levels in Patients with Crimean–Congo Hemorrhagic Fever
Hulya Vural, MD; Griedal Sekaraya, PhD; Ugur Kostakoglu, Assistant professor2; Mustafa Arslan, MD; Suleyman Caner Karahan, Professor3 and Iftihar Koksal, Professor3; 1Department of Medical Biochemistry, Health Sciences University, Kanuni Training and Research Hospital, Trabzon, Turkey, 2Department of Infectious Diseases and Clinical Microbiology, Karadeniz Technical University, Medical Faculty, Trabzon, 3Microbiology, Kanuni Training and Research Hospital, Trabzon, Turkey
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Background. Human coronavirus OC43 (HCoV-OC43) causes common cold, and is associated with severe respiratory symptoms in infants, elderly and immuno-compromised patients. HCoV-OC43 is a member of Betacoronavirus genus that includes also the Severe Acute Respiratory Syndrome (SARS) and the Middle East Respiratory Syndrome (MERS) coronaviruses. Both SARS-CoV and MERS-CoV were shown to express proteins with the potential to evade early innate immune responses. However, the ability of HCoV-OC43 to antagonise the intracellular antiviral defences has not yet been investigated. The objective of this study was to investigate the role of HCoV-OC43 structural (membrane and nucleocapsid) and accessory (ns5a and ns2a) proteins in the modulation of antiviral gene expression profile in human embryonic kidney 293 (HEK-293) cells using PCR array analysis.

Methods. HCoV-OC43 membrane (M), nucleocapsid (N), ns5a and ns2a mRNA were amplified and cloned into the pAgFPNI expression vector (Clontech), followed by transfection in HEK-293 cells. Expression of M, N, ns5a and ns2a proteins were confirmed by indirect immunofluorescence test. Three days post-transfection, the cells were challenged by Sendai virus. The Human Antiviral Response PCR array system (Qiagen) was used to profile the antiviral gene expression in HEK-293 cells, using the fold regulation comparison and the manual normalisation methods.

Results. Around 50–60 genes were downregulated by HCoV-OC43 proteins, the most important downregulated genes being those critical for the activation of transcription factors involved in the antiviral response like interferon regulatory factors (IRFs) and activator protein 1 (AP-1). Among the most important downregulated genes were those coding for Interferons (IFNs) mitogen-activated protein kinases (MAPKs), pro-apoptotic and pyroptotic proteins (Caspases, cathepsins, tumour necrosis factor), pro-inflammatory cytokines (Interleukins), pattern recognition receptors (PRRs; toll-like receptors and NOD-like receptors) and their signaling transduction proteins (TICAM1, MAVS).

Conclusion. This study shows for the first time that similarly to SARS-CoV and MERS-CoV, HCoV-OC43 has the ability to downregulate the transcription of genes critical for the activation of different antiviral signaling pathways.

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Turkey, 3 Department of Infectious Diseases and Clinical Microbiology, Recep Tayyip Erdogan University, Rize, Turkey, 4 Department of Infectious Diseases and Clinical Microbiology, Amasya University Sabuncuoglu Serefeddin Training and Research Hospital, Amasya, Turkey, 5 Department of Medical Biochemistry, Karadeniz Technical University, Faculty of Medicine, Trabzon, Turkey.

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Background. Crimean–Congo hemorrhagic fever (CCHF) is a potentially fatal disease caused by a tick-borne virus from the Bunyaviridae family. Cytokines play an important role in the pathogenesis of viral, bacterial, and immunologic diseases. This study aimed to investigate the role of TNF-alpha, IL-6, IL-10, and IFN-gamma levels in the severity of infection and clinical outcome of patients with CCHF.

Methods. Patients with confirmed CCHF were divided into two groups (severe cases: Patients who exhibited hemorrhage during their hospital stay, and mild/moderate cases: Patients who displayed no hemorrhage during their hospital stay). Demographic characteristics, laboratory tests on admission of all patients with CCHF were investigated, and serum TNF-alpha, IL-6, IL-10, and IFN-gamma levels were measured.

Results. A total of 154 patients with confirmed CCHF were investigated. Forty-six (29.9%) of these patients were in the severe group. In patients with severe CCHF, significantly higher serum levels of TNF-alpha (68.2 ± 23.5; P = 0.006) and IL-6 (73.1 ± 41.6; P = 0.003) were detected, compared with cytokine levels in patients who were mild/moderate CCHF (Table 1). No differences in serum IL-6 and IFN-gamma levels between patients who severe CCHF and those who were mild/moderate CCHF were detected (P > 0.05).

Table 1: Cytokine levels, demographic and laboratory characteristics in patients with severe and mild/moderate cases with CCHF.

| Features            | Severe cases (n = 46) | Mild/moderate cases (n = 108) | P-value |
|---------------------|----------------------|-----------------------------|---------|
| Age                 | 50.6 ± 20.3          | 49.8 ± 21.0                 | 0.682   |
| Female gender, (%)  | 31 (67.4)            | 63 (58.3)                   | 0.291   |
| TNF (%)             | 68.2 ± 23.5          | 41.3 ± 17.4                 | 0.008   |
| IL-6 (%)            | 73.1 ± 41.6          | 38.0 ± 19.5                 | 0.003   |
| IL-10 (%)           | 6.2 ± 1.3            | 6.21 ± 1.4                  | 0.753   |
| IFN-gamma (%)       | 145 ± 96             | 126 ± 92                    | 0.664   |
| WBC/μL              | 3286 ± 5620          | 2.275 ± 1.286               | 0.280   |
| PLT                 | 53,564 ± 36,520      | 98,065 ± 42,768             | 0.001   |
| CRP                 | 3.2 ± 2.6            | 1.1 ± 1.4                   | 0.005   |
| ALT                 | 521 ± 482            | 208 ± 320                   | 0.044   |
| AST                 | 869 ± 112            | 296 ± 215                   | 0.016   |
| CPK                 | 1,138 ± 970          | 676 ± 835                   | 0.007   |
| LDH                 | 1,800 ± 1,254        | 598 ± 271                   | 0.002   |

Conclusion. Cytokines, chemokines, and other inflammatory mediators function in a manner, acting on many different cell types to regulate the host's immune response. When cytokines present in high concentrations, they might toxic or even lethal effects. In accordance with this view, we have detected increased serum TNF-alpha, IL-6 levels in the patients with severe CCHF.

Disclosures. All authors: No reported disclosures.

1034. Etiologic Involvement of Enterovirus and Human Bocavirus in Acute Flaccid Paralysis Cases in India
Manjari Baluni, Ph.D(Pursuing)1; Dharmavir Singh, Ph.D(Pursuing)1; Sneha Gahlidyal, PhD(Pursuing)1; Tanzeem Fatima, Ph.D (Pursuing)1; Amreen Zia, Ph.D(Pursuing)2; and Tapan Phole, M.D.1, 3Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, India, 1Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India, 2Microbiology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, India.

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Background. Paralytic polio is an acute flaccid paralysis caused by enteroviruses that are transmitted through fecal-oral route. Enteroviruses, a genus of picornaviruses, are RNA viruses which are comprised of more than 100 serotypes that are expected for AFP . Molecular typing of these viruses is useful for characterizing emerging serotypes and their epidemiological investigation.

Methods. A total of 9 (1.5%) saffold viruses was detected and characterized by VP1 sequencing. Molecular and phylogenetic analysis showed 0.9 - 5.6% divergence at nucleotide level among HBoVs. Total 1.5% saffold viruses was detected and characterized by VP1 sequencing. Phylogenetic analysis showed 0.9 - 5.6% divergence at nucleotide level among HBoVs. Total 9 (1.5%) saffold viruses was detected and characterized by VP1 sequencing. Cytokine levels, demographic and laboratory characteristics in patients with severe and mild/moderate cases with CCHF

Table 1: Cytokine levels, demographic and laboratory characteristics in patients with severe and mild/moderate cases with CCHF.

| Features            | Severe cases (n = 46) | Mild/moderate cases (n = 108) | P-value |
|---------------------|----------------------|-----------------------------|---------|
| Age                 | 50.6 ± 20.3          | 49.8 ± 21.0                 | 0.682   |
| Female gender, (%)  | 31 (67.4)            | 63 (58.3)                   | 0.291   |
| TNF (%)             | 68.2 ± 23.5          | 41.3 ± 17.4                 | 0.008   |
| IL-6 (%)            | 73.1 ± 41.6          | 38.0 ± 19.5                 | 0.003   |
| IL-10 (%)           | 6.2 ± 1.3            | 6.21 ± 1.4                  | 0.753   |
| IFN-gamma (%)       | 145 ± 96             | 126 ± 92                    | 0.664   |
| WBC/μL              | 3286 ± 5620          | 2.275 ± 1.286               | 0.280   |
| PLT                 | 53,564 ± 36,520      | 98,065 ± 42,768             | 0.001   |
| CRP                 | 3.2 ± 2.6            | 1.1 ± 1.4                   | 0.005   |
| ALT                 | 521 ± 482            | 208 ± 320                   | 0.044   |
| AST                 | 869 ± 112            | 296 ± 215                   | 0.016   |
| CPK                 | 1,138 ± 970          | 676 ± 835                   | 0.007   |
| LDH                 | 1,800 ± 1,254        | 598 ± 271                   | 0.002   |

Conclusion. Cytokines, chemokines, and other inflammatory mediators function in a manner, acting on many different cell types to regulate the host's immune response. When cytokines present in high concentrations, they might toxic or even lethal effects. In accordance with this view, we have detected increased serum TNF-alpha, IL-6 levels in the patients with severe CCHF.

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1036. Risk Factors for Herpes Zoster: A Systematic Review and Meta-Analysis
Koushe Kawa, ScD7; Barbara P. Yawn, MD, MSc, MSHP, FAPAAP;1, 3Boston Children's Hospital and Harvard Medical School, Boston, Massachusetts, 2Department of Research, Olmsted Medical Center, Rochester, Minnesota; University of Minnesota, Minneapolis, Minnesota.

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Background. Well-recognized risk factors for herpes zoster (HZ), commonly known as shingles, are age and immunosuppression. Numerous studies have investigated other various risk factors for HZ in recent years. The objective of our study is to systematically review studies examining risk factors for HZ and discuss implications based on the updated evidence.

Methods. We performed a literature search using PubMed, Embase, and Web of Science and included studies that examined risk factors for HZ. Random effects model was used to summarize the risk ratio (RR) or odds ratio (OR) and 95% confidence interval (CI).

Results. Of the 3450 studies screened, we included 84 studies in the systematic review and conducted meta-analysis in 62 studies. Women are at increased risk of HZ with adjusted RR = 1.31, 95% CI 1.27, 1.34. Black individuals have almost half the risk of HZ than White individuals (pooled RR = 0.54; 95% CI: 0.47, 0.63). Family history was found to be a risk factor for HZ (pooled OR = 3.39; 95% CI: 2.39, 5.40). Autoimmune diseases, including rheumatoid arthritis (pooled RR = 1.67; 95% CI: 1.41, 1.99) and systemic lupus erythematosus (RR = 2.10; 95% CI: 1.40, 3.15), were associated with an elevated risk of HZ. Other comorbidities were associated with an increased risk of HZ, with the pooled RRs ranging from 1.25 (95% CI: 1.13, 1.39) for asthma to 1.30 (1.17, 1.45) for diabetes mellitus, and 1.31 (95% CI: 1.22, 1.41) for chronic obstructive pulmonary disease. Statin use was also