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## 474 Asthma Exacerbations and Intimate Partner Violence

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**RATIONALE:** The etiologies for difficult-to-control asthma are complex and incompletely understood. Intimate partner violence (IPV) is a pervasive issue and may be an important determinant of difficult-to-control asthma. IPV is associated with increased prevalence of asthma. There are no studies evaluating IPV's impact on adult asthma control and morbidity. This study hypothesized that IPV exposure is associated with asthma exacerbations among adults.

**METHODS:** Analyses are based on 1934 adults who participated in the 2005 Behavioral Risk Factor Surveillance System survey, reported active asthma, and completed the IPV questions. We used multivariate logistic regression to examine the association of IPV with asthma exacerbations within the last year while controlling for the following potential confounders: sex, race, education, smoking status, age, and self-assessment of health status.

**RESULTS:** The overall prevalence of IPV among asthmatics was 37.4%. 42.3% of women reported a history of IPV as compared to 22.6% of men. All of the potential confounders had a statistically significant association with IPV (i.e., all p-values were below .05). IPV was associated with increased odds of an asthma exacerbation in the last year (OR = 1.66, 95% CI: 1.35 – 2.04, $p < .0001$) while controlling for these potential confounders.

**CONCLUSIONS:** IPV is a prevalent and under-recognized determinant of exacerbations among adults with asthma, even after adjusting for key confounders. Further research is needed to more fully understand the effects of IPV on asthma.

## 475 Five-month Outcomes for Asthmatics with COVID-19 and Associations with Atopy and Inhaled Corticosteroids Use

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**RATIONALE:** COVID-19 viral infection has been associated with respiratory tract inflammation. Asthma exacerbations are frequently triggered by viral respiratory infections, which subsequently cause poor asthma control. However, the effect of COVID-19 on asthma exacerbations and outcomes beyond the initial COVID-19 infection is not well studied.

**METHODS:** Adult COVID-19-positive patients with history of asthma evaluated at a university medical center between February and April,2020 were entered in the study at the time of COVID-19 infection and followed for 4-6 months.

**RESULTS:** Seventy-six patients with asthma were followed for a 151-day-average after COVID-19 infection. At COVID-19 infection onset, 55.2% of subjects presented with symptoms suggestive of an asthma exacerbation. These patients experienced an average of 2.6-weeks of uncontrolled asthma and sought medical care for asthma symptoms at 1.9 mean provider visits. 9.2% of patients required step-up therapy; 23.6% received oral steroids post-COVID-19 infection. Stratified by asthma severity, 66.6% of subjects with intermittent, 50.0% with mild persistent and 68.4% with moderate/severe persistent asthma experienced exacerbations($p=0.78$). The asthma exacerbation rate in patients who took inhaled corticosteroids (ICS+LABA) (n=25, 53.3%) or ICS alone(n=13, 55.2%) did not differ from those who were not taking ICS (n=38, 52.6%), ($p=0.82$). 42.3% of asthma patients with a history of allergic rhinitis versus 64.5% of nonallergic patients experienced an exacerbation($p=0.086$).

**CONCLUSIONS:** Our data showed no effect of asthma severity nor ICS+/-LABA therapy on the rate of asthma exacerbations after COVID-19 infection in five-month follow up. Atopic patients had a trend towards protection against COVID-19-associated asthma exacerbations, which needs confirmation in larger studies.

## 476 Comorbid Asthma Within a Prospective Household Study of Respiratory Viral Infections

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**RATIONALE:** Viral upper respiratory infection is a common trigger of asthma exacerbations. We hypothesized that households with asthmatic members would have an overall higher burden of viral respiratory disease with longer duration of illness, more severe symptoms and greater transmission between household members in comparison to households without asthmatic members.

**METHODS:** 1171 subjects (119 with asthma) were enrolled by household (n=303) in a prospective longitudinal study of respiratory viral infections in Seattle, WA, USA from November 2019 through April 2020. Subjects self-reported sociodemographic data, symptoms, severity, and underlying comorbidities. At the onset of respiratory symptoms, individuals self-collected a mid-nasal swab and completed a symptom log. Swabs underwent RT-PCR testing for eight common respiratory viruses including RSV, rhinovirus, influenza and SARS-CoV-2.

**RESULTS:** 592 respiratory illness episodes (77 complicated by asthma) were reported with 40 secondary detections of virus in 33 of 207 households with illness. No significant difference was observed in secondary viral detection within household by asthma status, adjusted by household size (t-test). Individuals with asthma were more likely to report shortness of breath ($p=0.001$), without significantly increased overall illness duration or self-reported severity. Individuals with asthma had a lower rate of viral detection for symptomatic illness (34% versus 50%, $p=0.01$, $\chi^2$ test).

**CONCLUSIONS:** No significant difference in viral spread or duration of illness within households by asthma status was noted. Household members with asthma were more likely to have a symptomatic illness without an isolated viral pathogen, suggesting a different cause of acute illnesses.

## 477 Nociceptor Neurons Control Pollution-exacerbated Asthma

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**RATIONALE:** Half of the severe asthma patients suffer from uncontrolled exacerbations. Our work in neuro-immunology has shown that, in the context of asthma, vagal nociceptor neurons drive a feed-forward inflammatory loop with lung immune cells, and that silencing these neurons reverses allergic airway inflammation (AAI). Here, we aim to expand these findings to a clinically relevant model of pollution exacerbated asthma.

**METHODS:** Experimental allergen Ovalbumin (OVA) was co-exposed with fine particulate matter (FPM) to OVA-sensitized wild type or sensory neuron-ablated TRPV1-DTA mice. Cells in bronchoalveolar lavage fluid (BALF) and lung were immunophenotyped by flow cytometry. Gene expression in isolated alveolar macrophages and whole lung tissue was assessed by RT-qPCR.

**RESULTS:** We found that mice co-exposed to FPM and OVA show an aberrant bronchoalveolar lavage fluid immune profile characterized by a mixed infiltration of neutrophil and eosinophil as well as the expansion of lung γδ T cells. Along with these changes, we found that the neurotrophic factor artemin was increased in whole lung tissue as well as by FPM-stimulated alveolar macrophage. In addition, to these changes, we discovered that the genetic ablation of sensory neurons prevents the development of the pollution exacerbation of asthma.

**CONCLUSIONS:** In terms of inflammatory cell infiltration in BALF and lung γδ T cell expansion, co-exposure of FPM exacerbates OVA-induced AAI in a $TRPV1^+$ sensory neuron-dependent fashion. In parallel, artemin is induced by the exposure of FPM, which implies an artemin-related neuro-immune network in such pollution exacerbation of AAI.