

International Conference on **COPD AND ASTHMA**

March 07, 2022 | Webinar

**Evaluation of Respiratory functions and quality of life in a Child diagnosed with iron deficiency Anemia****Ozden Gokcek<sup>1</sup>, Cigdem El<sup>2</sup>**<sup>1</sup>Ege University, Turkey<sup>2</sup>Hatay Mustafa Kemal University, Turkey

Childhood iron deficiency is one of the most common nutritional disorders worldwide. One of the effective factors of iron deficiency anemia is inflammation. In iron deficiency anemia, the symptoms of fatigue, respiratory distress, and weakness, which are the same as the symptoms seen in other types of anemia, significantly reduce the quality of life. Since hemoglobin carries oxygen to the tissues, respiratory functions are inevitably affected by anemia. A thirteen-year-old girl presented fatigue in daily life, especially in walking activity. Our cases were recorded demographic information and hemogram values. Pulmonary function test, respiratory muscle strength measurement with a mouth pressure measuring device, and a 6-minute walking test were performed to evaluate functional capacity. PedsQL questionnaire was used to measure the quality of life and the leisure activity questionnaire was used to evaluate physical activity. According to the evaluations in our case, the pulmonary function test was low and the respiratory muscle strength value was found to be much lower than expected. In the 6-minute walking test, it was observed that she walked 570.6 m, and was lower than the expected value. In terms of quality of life, it was observed that especially physical, school success, and emotional impact were high. As a result of the children's leisure time activity survey which we used to evaluate the activity level, it was determined that she had a moderate MET/hour value of , 28,5, moderate-intensive value 44,5, intensive value 16. It is known that exercise provides an increase in hemoglobin level, has an anti-inflammatory effect, and increases functional capacity and quality of life. With this case report, it is thought that children diagnosed with iron deficiency anemia should be evaluated in terms of physiotherapy along with medical treatment and included in the treatment program in physiotherapy treatment.

**Biography**

As a physiotherapist, Ozden Gokcek has a passion for improving children's health and well-being. It aims to explain the health improvement mechanism of physiotherapy applications and investigates its role in the treatment process. He has experienced the positive effects of physiotherapy applications on patients both in the clinic for many years. She has been serving in this field for 9 years in educational institutions. She is of the opinion that a multidisciplinary and interdisciplinary approach to health should be integrated.

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**Systemic inflammation induced by exacerbation of COPD or pneumonia in patients with COPD cardiac troponin elevation****Vidar Soyseth***Akershus University Hospital, Norway*

**Statement of the Problem:** Troponin is a biomarker of myocardial injury. In chronic obstructive pulmonary disease (COPD), troponin is an important determinant of mortality after acute exacerbation. Whether acute exacerbation of COPD (AECOPD) causes troponin elevation is not known. Here, we investigated whether troponin is increased in AECOPD compared to stable COPD.

**Methodology & Theoretical Orientation:** We included 320 COPD patients in the stable state and 63 random individuals from Akershus University hospital's catchment area. All participants were  $\geq 40$  years old (mean 65.1 years, SD 7.6) and 176 (46%) were females. The geometric mean of high-sensitivity cardiac troponin T (hs-cTnT) was 6.9 ng/L (geometric-SD 2.6). They were followed regarding hospital admission for the subsequent five years.

**Findings:** During the 5-year follow-up, we noted 474 hospitalisations: Totally, 150 and 80 admissions were due to AECOPD or pneumonia, respectively. The geometric mean ratio with geometric standard error (gse) between cTnT at admission and stable state in AECOPD and pneumonia was 1.27 (gse = 1.11,  $p = 0.023$ ) and 1.28 (gse = 1.14,  $p = 0.054$ ), respectively. After inclusion of blood leucocyte count and CRP at hospitalisation, these ratios attenuated to zero. However, we estimated an indirect of AECOPD and pneumonia on the ratio between hs-cTnT at admission and the stable state to 1.16 ( $p = 0.022$ ) and 1.22 ( $p = 0.008$ ), representing 91 (82 – 100) and 95 (83 – 100) % of the total effects, respectively (95 % confidence intervals in parentheses).

**Conclusion & Significance:** AECOPD and pneumonia in COPD patients is associated with higher cardiac troponin T levels. This association appears to be mediated by systemic inflammation.

**Biography**

Vidar Soyseth is a professor, affiliated with the Akershus University Hospital, University of Oslo Norway. He is a Norwegian Physician, researcher, Member of European Respiratory Society, Norwegian Medical Association and his research interests are Pulmonary Medicine.

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**Two-Stage Bronchoscopic Endobronchial valve treatment can lead to progressive Lung volume reduction and may decrease Pneumothorax risk****Thomas Egenod***Dupuytren University Hospital Limoges, France*

**Background:** Since successful development of endobronchial valves (EBV) as treatment for severe emphysema, its main complication, pneumothorax, remains an important concern.

**Objective:** We hypothesized that a two-step EBV implantation, during two distinct iterative procedures could lead to a more progressive target lobe volume reduction (TLVR) and thus ipsilateral lobe re-expansion, resulting in a significant decrease in the pneumothorax rate.

**Methods:** This retrospective bi-center study carried out by Limoges and Toulouse University Hospitals included patients following the inclusion criteria established by the BLVR expert panel. All patients were treated by two distinct procedures: first, EBVs were placed in all but the most proximal segment or sub-segment. The remaining segment was treated subsequently. All patients had a complete evaluation before treatment, and 3 months after the second procedure.

**Results:** Out of 58 patients included, only 4 pneumothoraxes (7%) occurred during the study. The other complications were pneumonia and severe COPD exacerbation (8.6% and 13.7% of patients, respectively). Significant improvement was found for FEV1 (+19.6 ± 25%), RV (-468 ± 960mL), 6MWD (30 ± 85m), BODE Index (-1.4 ± 1.8 point) and TLVR (50.6 ± 35.1%). Significant TLVR (MCID) was obtained in 74.1% of patients (43/58).

**Conclusion:** This new approach using EBV could reduce the incidence of pneumothorax without increasing other complication rates. Clinical and physiological outcomes are similar to those reported in studies using the conventional single-step treatment.

**Biography**

Thomas Egenod is head of the interventional pulmonology service at the Limoges University Hospital. An active member of the French Endoscopy Group, he is known in his country as an expert in endoscopic lung volume reduction. He is also the one who designed this two-step approach for endoscopic lung volume reduction with valves and disseminated it to other French centers which use it widely.

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### **Unexpected cause of tracheal obstruction: Pleomorphic adenoma with Lipometaplasia**

**Maria de los Milagros Lewkowicz<sup>1</sup>, Florencia Von Stecher<sup>1</sup>, Agustina Bruno<sup>1</sup>, Alejandro Damonte<sup>1</sup>, Alejandra Avagnina<sup>1</sup>, Joaquín García Moratto<sup>1</sup>**

*<sup>1</sup>Hospital de Clínicas José de San Martín, Argentina*

Pleomorphic adenoma of the trachea is a benign tumour, extremely rare, that generates obstructive symptoms and sometimes is confused with asthma, which delays its diagnosis. We present the case of a 40-year-old woman with history of asthma and airway obstruction, of 8-months duration. Fibro bronchoscopy showed polypoid, pedunculated lesion in first tracheal ring which occluded 80% of the lumen that was resected. The histopathological diagnosis was pleomorphic adenoma with lipometaplasia. Treatment of these tumours consists in complete resection of the lesion and long-term follow-up due to low probability of recurrence, malignancy and metastasis.

#### **Biography**

Maria de los Milagros Lewkowicz is a medical doctor who studied and graduated in UBA (University of Buenos Aires). Currently in the 4th year of her residency in pathology at a University Hospital called Hospital de Clínicas "José de San Martín" in Argentina. She will present an extremely strange and clinically enriching case, studied as a team by Pathologist and Surgeons of the hospital. This clinical case has been recently published in the most important magazine in the country called Medicine.

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**Development of a small mtDNA-encoded regulatory non-coding RNA region as a potential therapeutic agent to modulate mitochondrial bioenergetics****Anna Blumental Perry***University at Buffalo, USA*

**Statement of the Problem:** Mitochondrial malfunction is a hallmark of COPD. We identified an mtDNA-encoded small ncRNA, mito-ncR-805, that is upregulated in a cell-type specific manner in alveolar epithelial type II (AETII) cells during the stress of smoking. Mito-ncRNA-805 transcript functions as a retrograde signaling molecule between mitochondria and nucleus, with enhanced function during the adaptive stress response to smoking.

**The purpose of this study:** To study evolutionary conservation of mito-ncR-805, and interrogate the hypothesis that forced expression of synthetic oligos identical to evolutionary Conserved Region (CR) of mito-ncR-805 improves mitochondrial function.

**Methodology and Findings:** mito-ncR-805 is a mouse-specific transcript. We identified a region of mito-ncR-805 which is conserved in mammalian mitochondrial genomes, and generated shorter versions of mouse and human transcripts (mmu-CR805, and hsa-CR805), which differ in a few nucleotides. We called these small transcripts "functional bit". Over-expression of mmu-CR805 in MLE12 cells, led to increase in Krebs cycle, and in the activities of OXPHOS, stabilized mitochondrial potential, faster resumption of cell division following stress, and lower predisposition to apoptosis. Similarly, forced expression of hsa-CR805 in Beas-2B cells instigated faster proliferation compared to control cells. Although beneficial effects of respective ortholog oligos are less prominent than that of the species-specific oligo, both confer cross-species rescue during severe stress.

**Conclusion & Significance:** Our data indicate a high degree of evolutionary conservation of retrograde signaling via a functional bit of mito-ncR-805 in mammals. This emphasizes the importance of the pathway, and suggests a potential for the development of the functional bit of mito-ncR-805 into therapeutic agent that restores mitochondrial bioenergetics. Caution and awareness for the potential differences that may exist between forced over-expression of mito-ncR-805 during acute and chronic stress should be further investigated.

**Biography**

Anna Blumental Perry is a lung researcher at University at Buffalo. The focus of her research has been to understand proteostasis imbalance in lung disease, with a focus on endoplasmic reticulum and mitochondria malfunction in smokers. She investigates alveolar epithelial type II cells responses to cigarette smoke, because those cells are local progenitors with the ability to repair the damage and replenish the loss of alveolar epithelial type I cells. Anna developed the fascination to delineate the molecular mechanisms that confer survival advantages to type II cells in response to stressors and specifically, cigarette smoke stress. The approach has been multidisciplinary including protein chemistry, cell and molecular biology, genetic engineering, and animal models of disease.

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**Metabolomic biomarker candidates for Skeletal Muscle loss in the Collagen-Induced Arthritis (CIA) model**

Paulo Vinicius Gil Alabarse<sup>1</sup>, Jordana M S Silva<sup>2</sup>, Rafaela C E Santo<sup>2</sup>, Marianne S Oliveira<sup>2</sup>, Andrelise S Almeida<sup>2</sup>, Mayara S de Oliveira<sup>2</sup>, Mônica L Immig<sup>2</sup>, Eduarda C Freitas<sup>2</sup>, Vivian O N Teixeira<sup>2</sup>, Claiton V Brenol<sup>2</sup>, Ricardo M Xavier<sup>2</sup>, Camilla L Bathurst<sup>3</sup>, Stephen P Young<sup>3</sup>, Lidiane I Filippin<sup>4</sup>, Priscila S Lora<sup>5</sup>

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**Statement of the Problem:** There is no consensus for diagnosis or treatment of RA muscle loss. We aimed to investigate metabolites in arthritic mice urine as biomarkers of muscle loss.

**Methodology & Theoretical Orientation:** DBA1/J mice comprised collagen-induced arthritis (CIA) and control (CO) groups. Urine samples were collected at 0, 18, 35, 45, 55, and 65 days of disease and subjected to nuclear magnetic resonance spectroscopy. Metabolites were identified using Chenomx and Birmingham Metabolite libraries. The statistical model used principal component analysis, partial least-squares discriminant analysis, and partial least-squares regression analysis. Linear regression and Fisher's exact test via the Metabo Analyst website were performed (VIP-score).

**Findings:** Nearly 100 identified metabolites had CIA vs. CO and disease time-dependent differences ( $p < 0.05$ ). Twenty-eight metabolites were muscle-associated: carnosine (VIPs  $2.8 \times 10^2$ ) and succinyl acetone (VIPs  $1.0 \times 10$ ) showed high importance in CIA vs. CO models at day 65; CIA pair analysis showed histidine (VIPs  $1.2 \times 10^2$ ) days 55 vs. 65, histamine (VIPs  $1.1 \times 10^2$ ) days 55 vs. 65, and L-methionine (VIPs  $1.1 \times 10^2$ ) days 0 vs. 18. Carnosine was fatigue- (0.039) related, creatine was food intake- (-0.177) and body weight- (-0.039) related, and both metabolites were clinical score- (0.093; 0.050) and paw edema- (0.125; 0.026) related.

**Conclusion & Significance:** Therefore, muscle metabolic alterations were detected in arthritic mice urine, enabling further validation in RA patient's urine, targeting prognosis, diagnosis, and monitoring of RA-mediated muscle loss.

**Biography**

Paulo Vinicius Gil Alabarse has his expertise in Rheumatoid Arthritis and related muscle loss, as well as osteoarthritis. His research focus on searching for novel metabolic biomarker of muscle loss targeting diagnosis, follow up, and treatment response in order to improve individual disease progress and treatment response.

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**The benefits vs harm reduction of “Alternative” vs “Dual” smoking of electronic cigarettes, and heated Tobacco products.****Ahmad Husari***University of California, USA*

The inhaled smoke from combustible cigarette smoke (CS) has numerous toxicants and carcinogens and is strongly associated with many cardiovascular and respiratory diseases and cancer. In response, Novel tobacco, and nicotine products, such as heated tobacco products (HTP), and electronic Cigarettes (ECIG) have been introduced into the global markets with a claim of reduced harm to the users when compared to conventional cigarettes. Influenced by such claims, we have witnessed conventional smokers adopting a “dual user” approach in which they attempt to partially substitute CS with ECIG or HTP, in an attempt to reduce harm and also to attempt at quitting smoking. Previously, we’ve showed that acute exposure to ECIG was less toxic on cultured A549 cells and the lungs in an in vivo animal model when compared to CS exposure only. The health effects of combining conventional CS and other products tobacco aerosols, however, are not well understood. In this presentation, will review the current research and our experiments on the effects of ECIG, HTP, the dual use of CS with either HTP or ECIG, as well as the dual use of non-combustible products.

**Biography**

Ahmad Husari MD, FCCP, D’ABSM is an associate professor at University of California at Riverside and a pulmonary and critical care consultant at Riverside Medical Clinics. Dr Husari graduated from the American University of Beirut and pursued additional training at prestigious universities in the United States (Johns Hopkins University, University of Maryland, and Stanford University). His basic science research and interests include the pathogenesis of lung injury and other organs injury secondary to the inhalation of harmful chemicals mainly hyperoxia and Tobacco smoke. Recently, Dr Husari documented the detrimental effects of combustible tobacco consumption, electronic cigarette, and heated tobacco products (HTP). His lecture will explore the role of electronic cigarettes, HTP and “alternative” Smoking as compared to traditional tobacco exposure.

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**PM2.5 induce NLRP3 inflammasome activation and Lung fibrosis****Tian Xia***University of California Los Angeles, USA*

Airborne fine particulate matter (PM2.5) is known to cause respiratory inflammation such as chronic obstructive pulmonary disease and lung fibrosis. NLRP3 inflammasome activation has been implicated in these diseases; however, due to the complexity in PM2.5 compositions, it is difficult to differentiate the roles of the components in triggering this pathway. We collected eight real-life PM2.5 samples, 4 samples each before and after the beginning of district/public heating in Zhengzhou, a big city in northern China. Northern Chinese cities operate with a centrally controlled district/public heating system, with most cities cranking up the heat, mostly by coal, in mid-November. Using these PM2.5 samples, we compared their effects on NLRP3 inflammasome activation in vitro and lung fibrosis in vivo. In vitro assays showed that although the PM2.5 particles did not induce significant cytotoxicity at the dose range of 12.5 to 100 µg/mL, they induced potent TNF-α and IL-1β production in PMA differentiated THP-1 human macrophages and TGF-β1 production in BEAS-2B human bronchial epithelial cells. PM2.5 triggers NLRP3 inflammasome activation by inducing lysosomal damage and cathepsin B release, leading to IL-1β production. This was confirmed by using NLRP3- and ASC-deficient cells as well as a cathepsin B inhibitor, ca-074 ME. Interestingly, samples collected during the public heating induced higher IL-1β production than that were collected before the beginning of district/public heating. Administration of PM2.5 via oropharyngeal aspiration at 2 mg/kg induced significant TGF-β1 production in the bronchoalveolar lavage fluid and collagen deposition in the lung at 21 days post-exposure, suggesting PM2.5 has the potential to induce pulmonary fibrosis. The ranking of in vitro IL-1β production for all samples correlates well with the in vivo total cell count, TGF-β1 production, and collagen deposition in the lung. In summary, we demonstrate that PM2.5 is capable of inducing NLRP3 inflammasome activation, which triggers a series of cellular responses in the lung to induce lung fibrosis in vivo.

**Biography**

Tian Xia is a professor in Division of Nano Medicine, Department of Medicine at UCLA. His main research area is on studying particulate matter collected from air pollution, wildfire, electronic cigarettes, and marijuana as well as engineered nanoparticles and their effects to the lung. Research findings on particle-induced toxicity have been used for the safer design of nanomaterials for biomedical applications including adjuvant, particles that can induce immune tolerance for allergy and autoimmune diseases, and antimicrobials based on these structure-activity relationships. He is an elected Councillor in the Southern California Chapter of Society of Toxicology and he is Associate Editor in Nano toxicology, a flagship journal in the nanomaterial field. He has published over 150 articles with total citation of over 50,000 and H factor of 75 in Google Scholar and he was named Highly Cited Researcher in Chemistry three times by Web of Science of Clarivate Analytics.



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**Existential suffering in the daily lives of those living with palliative care needs arising from chronic obstructive pulmonary disease (COPD)**Louise Elizabeth Bolton<sup>1</sup>, Claire Gardiner<sup>1</sup>, Jane Seymour<sup>1</sup><sup>1</sup>University of Sheffield, United Kingdom

**Statement of the problem:** There are an estimated 328 million cases of COPD worldwide (López-Campos, Tan and Soriano, 2016). It is likely to become the third biggest cause of death by 2030 (World Health Organisation, 2019). The impact of living with palliative care needs arising from COPD disrupts an individual's existential situation. Understandings of individual's existential situations within COPD are limited within the research literature and are rarely addressed within clinical practice, yet existential suffering has been linked to poor health-related quality of life for those living with other chronic conditions. The purpose of this integrative review is to provide a synthesis of existing evidence on existential suffering for those living with palliative care needs arising from COPD.

**Methods:** This is an integrative review undertaken in accordance with PRISMA guideline (Page et al., 2021). Nine electronic databases were searched from April 2019 to January 2021. 35 empirical research papers of both qualitative and quantitative methodologies, alongside systematic literature reviews were included. Data analysis was undertaken using an integrative thematic analysis approach.

**Findings:** Identified themes of existential suffering when living with palliative care needs arising from COPD are as follows: Liminality, Lamented Life, Loss of Personal Liberty, and Life Meaning and Existential isolation. The absence of life meaning and purpose was of most importance to patients.

**Conclusion and Significance:** This integrative review provides a synthesis of international evidence upon the presence of existential suffering. It is present and of significant impact within the daily lives of those living with palliative care needs arising from COPD. The absence of life meaning has the most significant impact, requiring further exploration of both its physical and psychological impact. Rediscovery of life meaning diminishes feelings of worthlessness and hopelessness in daily life and facilitates feelings of inner peace. For those with COPD living with such a relentless symptom burden, a positive existential situation is desirable. (Gold Standard Framework, 2011)(NICE, 2018)

**Biography**

Louise Elizabeth Bolton is PhD student at Sheffield University- Palliative Care COPD and Lecturer in Adult Nursing at University of Derby, England. She is acting as a peer reviewer and her research interests are Lung Diseases, Pulmonary Medicine, Airway Obstruction, Asthma and Chronic Obstructive Pulmonary Disease.

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**Coronavirus disease 2019 vaccine hypersensitivity evaluated with vaccine and excipient allergy skin testing****Pamela L Kwittken***Fair Haven Community Health Care, USA*

**Statement of the Problem:** Since COVID-19 mRNA vaccines became available in December 2019; there were early reports of rare cases of anaphylaxis. A selected group of excipients were believed to be the trigger for these reactions, namely polyethylene glycol (PEG), polysorbate 80, and polysorbate 20. The purpose of this study was to use the proposed methods for excipient testing as well as expand testing to include the mRNA vaccine in question as well as recommended options and outcomes for the second dose.

**Methodology & Theoretical Orientation:** Patients were referred to a private Allergy practice for evaluation of allergic reaction to the Pfizer or Moderna vaccines or who had complex underlying allergic disease and were felt to be at risk of allergic reactions to these vaccines. Testing was performed with the previously published protocol for COVID19 vaccine excipients as well as prick and intradermal skin testing to the Pfizer and Moderna vaccines (1:10 dilution and full strength). Recommendations regarding the second vaccine and premedication were based on the patient's medical history and skin test results. Patients were followed up via telephone. Demographic data, time to reaction, underlying medical conditions, and symptoms were also analyzed.

**Findings:** With rare exceptions, most patients were able to tolerate the second dose of the same mRNA vaccines with or without medication with mild or no symptoms.

**Conclusion & Significance:** While it is unclear whether the cause for allergic reactions to mRNA vaccines is IgE mediated, a complement mediated pseudo allergic reaction or another as of yet undefined mechanism, skin testing and premedication are useful to provide guidance for subsequent COVID-19 vaccine doses.

**Biography**

Pamela L Kwittken MD is board certified in Allergy & Immunology and Pediatrics. She splits her time between private practice in Allergy & Immunology in Milford CT and at a community health center based in New Haven CT. She continues to be a proponent for preventative health care for the underserved in both rural and urban populations.