SARCOIDOSIS VASCULITIS AND DIFFUSE LUNG DISEASES 2024; 41 (3): e2024046 DOI: 10.36141/svdld.v41i3.16196 © Mattioli 1885

WASOG 2023 conference highlights

Olga D. Chuquimia, Natalia V. Rivera
Division of Respiratory Medicine, Department of Medicine Solna, Karolinska Institutet, Karolinska University Hospital 171 77 Stockholm, Sweden

Abstract. The World Association of Sarcoidosis and Other Granulomatous Diseases (WASOG) 2023 conference held in Stockholm, Sweden, focused on disseminating the science and updating the interstitial lung diseases (ILDs) community with the latest science and concepts in precision medicine. This article summarizes the first four sessions of the conference that focused on disease and phenotype characterization, genetics in population cohorts, uses of biobanks and big data, epidemiology in diverse racial and ethnic groups, and data-driven applications that can be applicable to move the ILD field toward precision medicine.

Key words: diagnosis; sarcoidosis; pulmonary fibrosis; biomarkers; GWAS; genetics; biobanks; big data; precision medicine

Conference summary for sessions focused on translation medicine in ILDs

Last June 19-21, 2023, more than 300 delegates from more than 45 different countries representing over 130 institutions from different continents of the world gathered at Aula Medica, Karolinska Institutet in Stockholm, Sweden, for the World Association of Sarcoidosis and other Granulomatous Disorders (WASOG) 2023 international conference with the theme "Big data - How to move the field towards precision medicine in Interstitial Lung Diseases (ILDs)"

The conference chair carefully drafted the 3-day intense WASOG 2023 scientific program, Dr. Natalia Rivera, Principal Investigator at Karolinska Institutet. Dr. Rivera, together with the local and international committees, provided all the delegates with the perfect platform to discuss the latest advances in the research of ILDs, highlighting topics related to clinical updates and treatment options, big data, biobanks, and single-cell approaches, artificial intelligence applications toward precision medicine, new concepts on disease characterization, phenotypes, and socioeconomic determinants, and immunological approaches and methods. The conference also offered patient education and serial industry-sponsored sessions.

This article summarizes key topics discussed at the WASOG 2023 conference, covered by insightful oral and poster presentations.

1. New concepts on disease characterization and phenotypes in ILDs

Session 1 was kick-off by Dr. Jan Grutters (St. Antonius Ziekenhuis, The Netherlands), who introduced new concepts and approaches to define phenotypes and endotypes (shared pathogenic mechanisms - validated biomarkers to target) in ILDs apart of the classical established phenotypes in ILDs such as Löfgren's syndrome in sarcoidosis (a distinct clinical phenotype of a subgroup of patients). He cited the short telomere syndrome phenotype(1) as the deteriorated prognosis of IPF and its endotype form could play a more prominent role

Received: 11 June 2024
Accepted: 11 July 2024
Correspondence: Natalia V. Rivera, PhD
Karolinska Institutet
Respiratory Medicine Unit
Department of Medicine Solna
171 77 Stockholm, Sweden
E-mail: natalia.rivera@ki.se

in the monitoring of sarcoidosis prognosis by multi-omics derived signatures in bronchoalveolar lavage (BAL) as well as the four imaging "PET types" generated by F-FDG PET/CT cluster analysis (2) that are relevant to the prognosis of sarcoidosis. He also introduced newly established pertinent criteria for the clinical practice of PPF (progressive pulmonary fibrosis) phenotype treatment, concluding that disease phenotype and endotype identification could establish precision medicine in ILDs. A recent review addresses the challenges of phenotype characterization in sarcoidosis (3).

Dr. Wonder Drake (University of Maryland School of Medicine, USA) discussed the role of dysbiosis gut microbiota on ILDs. The studies of microbial diversity conducted in mice showed that gut microbiota diversity promotes less severe lung disease and suggests that the severity of lung fibrosis under gut dysbiosis is greater than estrogen. There is an intricate relationship between dysbiosis and the immune system (4) and chronic lung inflammation (5).

Dr. Stephen Humphries (National Jewish Health, USA) introduced the latest artificial intelligence (AI) approaches that enable quantitative CT as a key biomarker of deep learning for objective detection and quantification of IPF phenotypes. Artificial intelligence is a booming field and will play a significant role in future developments for personalized medicine.

Dr. Anna-Maria Hoffmann-Vold (Oslo University Hospital, Norway) discussed data on the relationship between rheumatoid arthritis (RA) and ILD progression that predicts mortality and the criteria and strategy for treating patients before progression. The definition of ILD progression will majorly impact the mortality of the different RMDs. Dr. Mridu Gulati (Yale School of Medicine, USA) concluded the session by stating the need to approach new concepts on disease characterization and phenotypes in ILDs.

2. Progress in complex phenotype prediction in ILDs across ancestries

Session 2 was introduced by Dr. Amke Caliebe (Christian–Albrechts–Universität zu Kiel, Germany) on genetic epidemiology studies and their importance on novel discoveries, the search for different genetic backgrounds (different allele frequencies and linkage-disequilibrium (LD) structure in populations), the role of different environments on phenotypes and their gene-environment interaction and the importance of equitable health care to improve the outcome across ancestries in diseases such as sarcoidosis. Study design and interpretation of data can be affected by ancestry (based on genes of the biological ancestors) and ethnicity (self-declared social group identity) concepts. Over time, the participation by ancestry in genome-wide association studies (GWAs) has not improved for multiple reasons. To improve future genetic studies, more actions need to be placed, such as recruiting more individuals of different ancestries, making available resources such as omics data, developing new statistical methods to help with studies with diverse populations, addressing misconceptions in human genetics, and interpreting the results with different ancestries.

Dr. Yvette C Cozier (Boston University School of Public Health, USA) presented an overview of race and ethnicity in epidemiology studies of sarcoidosis, showing the difference between the incidence of disease in white and black populations in the past and more recent (including more ethnic and representative populations) studies in the USA. African American populations have the most considerable disease risk and mortality, and this questioned how biology or sociology associated with race influences health inequality. The impact of social determinants on the health of communities with high deprivation index as high sarcoidosis risk and mortality in the USA and other countries highlights that black and other non-white individuals are more likely to reside in these communities. Dr. Cozier concluded that clinicians and researchers should focus on the role of sarcoidosis genetics and should consider routinely including patients' social contexts to avoid health disparities in disease studies.

Dr. Michelle Sharp (Johns Hopkins University School of Medicine, USA) discussed the health disparities in sarcoidosis, such as socioeconomic (high cost of sarcoidosis diagnosis), gender (females with sarcoidosis), and race (higher risk of disease for African American population) and how we can achieve equity. Data shows the association of air pollution with lower health-related quality of life. Therefore, there is a need to implement environmental policies that promote environmental justice against environmental exposures and health disparities in respiratory diseases such as sarcoidosis. Also, policies that

contribute to institutional and structural racism need to be confronted to achieve health equity.

Additionally, Dr. Sharp showed that studies in ILDs have less research investment and innovation than other respiratory diseases (e.g., COPD or asthma) and that policies should be implemented to improve minority participation in clinical research. The research on big data and phenotyping should consider the outcome to be measured and the need for multi-level approaches to ensure patient support and access to care for all patients.

Dr. Logan Harper (Cleveland Clinic, USA) took us through the history of how race has implications for differences we consider between white and black patients in sarcoidosis. He stressed that we study the impact of structural racism when we study differences related to race without using genetic markers. Through surveys of 2318 sarcoidosis patients in the USA, income is associated with clinical outcomes in these patients, and the black race is highly correlated with low income, demonstrating persistent effects of structural racism. This has degraded the care and mistrust of patients with sarcoidosis. Therefore, the history and social implications of race should be identified while using race as a variable in big-data interpretation of data. The inclusion of patients and researchers from under-represented racial groups will improve the quality of research by avoiding and understanding past mistakes and barriers and mitigating bias.

Dr. Marc Judson (Albany Medical Center, USA) summarized and emphasized considering race as a variable to define phenotypes in ILDs apart from most of the variables such as genetics, socioeconomics, and environmental variables that can affect the care of patients as well as the research on ILDs such as sarcoidosis.

3. Big data, biobanks, and genetic approaches in ILDs

Dr. Lisa Maier (University of Colorado, USA) presented an overview of sarcoidosis as a heterogeneous systemic disease with disparities other than those mentioned in the previous sessions. Varying clinical courses, especially organ involvement, are important for biobanks, big data, and genetics. In biobanking, cluster analysis on organ phenotypes in sarcoidosis cohorts does not apply to all populations, even if the number of participants is large. Clinical and demographic variables such as race, sex, age of diagnosis, and time of treatment since diagnosis are associated with several sarcoidosis phenotypes. Such variables shall be considered while working with big data sources. Genetics and environment are important in sarcoidosis studies, and human leukocyte antigen (HLA) genes are associated with different phenotypes, ethnicities, and prognoses of sarcoidosis. The only genetic predictor available at the clinic is the *HLA-DRB1*03* for Löfgren's syndrome cases and disease resolution versus the non-*HLA-DRB1*03*; however, this allele occurs in specific populations of European ancestry than in black populations. Similarly, genome-wide association studies (GWAs) are performed predominantly in European background populations, except for some GWAs on black populations, which confirm some of the findings identified on chromosome 6 associations. Genetic factors differ by phenotype, as shown by Rivera et al. (6). Here, the authors describe 170 genome-wide loci associated with the Löfgren's syndrome phenotype, which is a very homogenous phenotype with a specific architecture. However, this may not be assessed within more heterogeneous populations. Omics predict disease and severe phenotypes, and big data can predict biological foundations for clinical data. Therefore, multiple factors must be considered to balance study design, population, and data to address big data and Biobanks.

Dr. Courtney Montgomery (University of Oklahoma Health Sciences Center, USA) talked about immunogenetics on sarcoidosis since it is established by GWAs, linkage, epidemiologic, and even Single-cell transcriptomic studies that sarcoidosis is genetically influenced and ancestry specific. However, the mechanisms underlying the disease are still unknown, and there is no gold standard biomarker for diagnosis or prognosis. A case and control study on an active recruiting cohort in the USA with European and African backgrounds showed that sarcoidosis patients shared a cytokine profile that varies by the clinical outcome and/or ancestry (unique and significant association in more homogenous populations). One can capture the clinical and serological differences in the transcriptome using patient stratification and pathway analysis. Ancestry and sex differences were also found in the transcriptome, although many genes were overlapping across all cell types. A prediction model showed promising results based on the data from cytokine profile and single cell analysis that includes ancestry, establishing how big data and

bioinformatic tools could be used to help sarcoidosis patients for identification of biomarkers for diagnosis, prognosis, and/or personalized treatments.

Moreover, the generated data may give insights into the mechanism of sarcoidosis onset or progression, as was shown in the analysis of circulating cytokine levels that translated to gene expression (significant differential gene expression on immune cell types).

The ILD Biobank- Beyond just collecting samples talk was presented by the coordinator of the ILD Biobank of the St. Annalis Wind (St. Antonius Ziekenhuis, NL), who gave an overview on the ILD Biobank at St. Antonius Ziekenhuis in Netherlands that follows the guidelines of the statement of Biobank definition by the European Commission as well as all the laws and regulations regarding the medical research and ethics. The St. Antonius Ziekenhuis ILD Biobank saves and stores several patient materials like blood, tissue, and BAL-fluid (over 30,000 samples) and data from more than 10,000 Dutch patients diagnosed with different ILD diseases, including the ultra-rare. The access to biological material and data from this ILD Biobank is not only for projects of Dutch researchers but also for collaborators worldwide from over 200 centers in 28 countries. This biobank is a clinical/research biobank focused on specific diseases (based on participants) that works with retrospective and prospective materials (based on approach). The aims are not only the storage of representative cohorts with large numbers but also to register all available information (triggers, pathogenesis, and hereditary causes of lung diseases) of patients with specific ILDs to accelerate advances in personalized medicine (improvement of health care) and to simplify future research on ILDs. The ILD Biobank at St. Antonius Ziekenhuis is considered a center of excellence and welcomes requests for access to materials and data from collaborators worldwide.

Dr. Natalia Rivera (Karolinska Institutet, Sweden) introduced the MESARGEN consortium and showed novel data in her presentation "Genetic architecture of sarcoidosis using Multi-ethnic cohorts in the MESARGEN consortium". As described previously, she emphasized the heterogeneity encountered in sarcoidosis phenotypes and cohorts and the repeated shortcomings in replicating gene associations in the past. These have been a significant limitation on genetic and molecular studies of sarcoidosis. However, GWAs have shown that HLA genes are constantly present across different populations of sarcoidosis patients. The HLA region located on chromosome 6 is one of the most complex genomic regions in the human genome, with genomic diversity, dynamic gene-gene and gene-environment interactions, and affected by evolution forces. About 28% of expressed genes in the HLA region are associated with different immune system functions, and 10% of the HLA region is associated with disease. Many studies in the past have described other genes associated with sarcoidosis; however, those findings could not be replicated in some cases. Despite this, the HLA region is considered the most important in the human genome as it has a significant role in the pathogenesis of sarcoidosis and autoimmune diseases. In order to understand the genetic architecture of sarcoidosis, it is first essential to understand the genetic variation of sarcoidosis phenotypes across ancestries. Thus, the MESARGEN consortium aims to catalog the genetic variation of sarcoidosis globally using clinically driven phenotype characterizations by dedicated specialists. MESARGEN has genotyped data from over 20,000 individuals from more than 10 European countries and has established collaborations with North America and Asia. MESARGEN includes many populations that have never been studied on a large scale for sarcoidosis genetics despite the prevalence of the disease in those countries. The clinical protocol that the MESARGEN consortium follows characterizes the population-based cohorts. The session was finalized with a thoughtful discussion about data protection of large cohorts, such as in the MESARGEN consortium, and how the definition of sarcoidosis phenotypes and endotypes impacts genetic association studies.

4. Translational Medicine and Data-driven Clinical Applications in ILDs

Natalia Rivera (Karolinska Institutet, Sweden) introduced the session on behalf of Martin Petřek (Palacký University of Olomouc, Czechia) regarding the aim of translational medicine in the ILD fields to identify the disease mechanisms and treatment by studying areas in the lung microbiome, epigenetic mechanisms, and genetic studies in disease susceptibility /development treatment responses as GWAs that are collaborative efforts among different groups.

Dr. Anna Duckworth (University of Exeter, United Kingdom) started the session by giving an overview of the impact of the IPF clinical data management of the UK biobank. The UK biobank has available data from over half a million people between the ages of 40 and 70 years old, with thousands of biomarkers data and follow-up data in hospital episodes statistics (HES) and GP records that can be derived from clinical cohorts such as IPF and sarcoidosis cohorts. In addition, the UK biobank also contains data on genetics, single nucleotide polymorphisms (SNPs) as common variance, whole exosome data, whole genome data, and more recently available telomere data. The availability of the UK biobank data allows the researchers to perform association, causality blood biomarkers, genetics, and health control with high-risk studies, to name a few. Dr. Duckworth showed data on association studies in IPF, COPD, and controls with rheumatoid arthritis, diabetes, coronary artery disease, and cancer, as well as primary and secondary mortality incidence in chronic disease. Causality was evaluated using Mendelian Randomization (MR) analysis using randomly assigned genetic variants like in a randomized controlled trial to address the question of whether telomere length is the cause of pulmonary fibrosis (PF) and/or idiopathic pulmonary fibrosis (IPF). Her work found that short telomeres have a causal effect on IPF but not on COPD (7), and smoking-associated variants cause COPD but not IPF. A very consistent association was found between short telomeres and depleted sex hormones and the established association with PF while evaluating traits known to affect sex hormones, particularly for current and former smokers in ILDs. Furthermore, high and low associations with short telomeres were found while evaluating other conditions, for instance, type 2 diabetes, rheumatoid arthritis, cancer, anemia, etc (7). To explore the clinical impact of the association of short telomeres with sex hormone depletion in PF, sex hormone therapy is currently being evaluated to elucidate its role in maintaining and restoring the telomere length and/or being protective in PF.

Tasha Fingerlin (National Jewish Health, USA) discussed the challenges in ILD prediction with or without Big Data for personalized medicine. Among other complex traits, sarcoidosis fits with the macro and microenvironment interaction paradigm with genetic sequences and epigenetic features to mediate the progression from health to disease. To tackle this paradigm, the need to intervene and make predictions must be implemented for personalized medicine. Identifying possible biomarkers that can be screened and used for the prevention of early diagnosis and determining new therapeutic targets and treatment strategies for individuals based on their molecular profile of the risk factors for disease are crucial components for this implementation. There are several challenges for personalized medicine, but it is essential to recognize that no two individuals have the same risk factors.

Consequently, a model such as a former statistical population-based study or years of clinical experience with a defined population has been used as a reference point to identify risk factors and estimate their effects to make good predictions for an individual. The challenge of defining the right reference group will differ depending on the context, for instance, the trait, age, or at what time window for prediction should be made. Also, considering that the same person will have different reference groups at a single time point and across the lifespan. For instance, genetic risk factors have effects in early life but have no effects later in life. Another challenge is identifying and collecting the correct information on the reference group and emerging deviations, such as treatment changes.

Dr. Fingerlin presented data on chronic beryllium disease (CBD) study on how the proper dataset should be collected. CBD is an interstitial lung disease that shares many characteristics with sarcoidosis. A risk factor for the disease has been associated with the genetic features of the *HLA-DPB1* gene and amino acid position 69 (glutamic acid, Glu^{69}), increasing the risk by 9-fold. However, a model described that depending on the prevalence of CBD in a screened population, the probability that an individual has CDB given at least one allele at residue 69 may be very low, meaning that it does not necessarily translate to a high likelihood of having CBD if this factor is found. This also showed that the lower the prevalence of the disease or trait to be screened in a population, the higher the probability of the positive predictor value for any test.

The role of exposure levels to beryllium in combination with carrying the E69 allele on the risk of disease was also explored in a model, and the risk estimates dramatically increase with exposure. However, due to a smaller sample size and other factors, this finding increases uncertainty on the estimates

and that reducing exposure is important across all the different groups. The probability of the disease of an individual having a specific genotype increases with exposure to several risk alleles. However, it is difficult to define an individual in a risk category and whether or not they will develop the disease (8). Applying the models described above is essential, especially in discovery and application, to maintain a balance between homogeneity and sample size and obtain sensitive and specific tests, although this is hard to maintain. Finally, the challenge to recognize that the prediction of events for an individual is not the same as predicting average risk has been addressed by a model that conceptionally has the desire for the predictions to have a high level of certainty and that the prediction interval for a person is not the same as the confidence interval for the average population.

Dr. Joanne Van Der Vis (St. Antonius Ziekenhuis, the Netherlands) presented the GAP +*MUC5B* model for IPF stages with a significantly more accurate prognosis in her talk "Incorporation of the *MUC5B* rs35705950 Genotype in the IPF gap". IPF is described as a progressive fibrotic diffuse parenchymal disease of unknown onset, associated with aging with a survival rate of 3-4 years following diagnosis. The disease is characterized by fibrosis on high-resolution computed tomography (HRCT). A well-established predictor (gender-age-physiology) GAP model has been used to predict IPF mortality, knowing that the risk factors for IPF mortality are male, older age, and pulmonary function associated with survival. In the GAP model, each variable is categorized and assigned points. The total point score classifies patients into 3 IPF stages; thus, the more points, the worse the outcome (9). Recently, the minor T-allele of the *MUC5B* rs35705950 promoter polymorphism was found to be associated as an independent predictor of better survival in 56-year-old and older IPF patients. This genetic variant was found to be suitable for adding value to the GAP model since *MUC5B* rs35705950 polymorphism impacts survival in the same range of the risk factors included in the GAP model (10). This was addressed in a European IPF population (1401) of 56 years and older, predominantly white male patients collecting retrospective data on demographic characteristics, lung function, transplant-free survival, and *MUC5B* rs35705950 genotype. Firstly, patients were classified into six stages by the additional approach, the GAP-*MUC5B* model, which calculates the original GAP stage. Each GAP stage was divided by *MUC5B* minor allele carriage. To apply this model, carrying the *MUC5B* minor allele was evaluated in the IPF cohort, and it was found that patients carrying the *MUC5B* minor allele had significantly better survival rates than non-carriers. Patients were also classified using the original IPF GAP model in three stages. Subsequently, the GAP+ *MUC5B* minor allele model was applied and found that in every stage (6), there was a significant difference between carriers and non-carriers in survival, and the significant difference was in stage 1 (22 months (about two years) difference) while the *MUC5B* minor allele carriers were the same in all stages. Evaluating the mortality rate of this model, we see that the mortality rate of stage 3 *MUC5B* minor allele carriers for two years is 55% (marking point for lung transplantation). Therefore, the GAP+ *MUC5B* minor allele model was found to be a refinement, particularly valuable for explaining the prognosis and stage of the disease. Secondly, like the GAP model, the GAMP model was performed to divide into three stages IPF patients to predict mortality for the incorporation approach – the GAMP model calculates extra points for *MUC5B* allele status in the GAP model. Both GAP and GAMP models were used to compare the mortality rate, and it found that the GAMP model can identify small high-risk groups and extensive low-risk groups. Under the GAMP model, stage II survival was significantly worse than the GAP model's. Data generated upon performing models like the GAP+ *MUC5B* minor allele model and GAMP model support the efforts to explore the translation of genetics into clinical care. The session was closed with a very productive discussion between all the invited speakers of this session.

Dr. Baughman presented the D. Geraint James Lecture during the Welcome Dinner. His lecture emphasized three main topics: updating the diagnosis of sarcoidosis, using placebo-controlled trials to illuminate the natural course of pulmonary sarcoidosis, and exploring multidisciplinary sarcoidosis clinic care using Centers of Excellence (11).

This summary addressed the first four sessions of the conference. The remaining sessions will be summarized in the following article, focusing on immunological approaches and omics applications toward precision medicine in ILDs.

Acknowledgments: The authors thank Professor Robert P. Baughman, Dr. Elyse Lower, and Dr. Daniel Culver for their discussions and feedback on this work.

Financial Support and Sponsorship: Financial support was received from the Swedish Heart-Lung Foundation, grant numbers 20200505 and 20200506.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. Alder JK, Chen JJ, Lancaster L, et al. Short telomeres are a risk factor for idiopathic pulmonary fibrosis. Proc Natl Acad Sci U S A. 2008; 105(35):13051-6. 20080827. DOI: 10.1073/pnas.0804280105.
2. Papiris SA, Georgakopoulos A, Papaioannou AI, et al. Emerging phenotypes of sarcoidosis based on 18F-FDG PET/CT: a hierarchical cluster analysis. Expert Rev Respir Med. 2020; 14(2):229-38. DOI: 10.1080/17476348.2020.1684902.
3. Grutters JC. Establishing a Diagnosis of Pulmonary Sarcoidosis. Journal of Clinical Medicine. 2023; 12(21):6898. DOI: doi:10.3390 /jcm12216898.
4. Levy M, Kolodziejczyk AA, Thaiss CA, Elinav E. Dysbiosis and the immune system. Nature Reviews Immunology. 2017; 17(4):219-32. DOI: 10.1038/nri.2017.7.
5. Saint-Criq V, Lugo-Villarino G, Thomas M. Dysbiosis, malnutrition and enhanced gut-lung axis contribute to age-related respiratory diseases. Ageing Research Reviews. 2021; 66:101235. DOI: 10.1016 /j.arr.2020.101235.
6. Rivera NV, Ronninger M, Shchetynsky K, et al. High-Density Genetic Mapping Identifies New Susceptibility Variants in Sarcoidosis Phenotypes and Shows Genomic-driven Phenotypic Differences. Am J Respir Crit Care Med. 2016; 193(9):1008-22. 2015/12 /15. DOI: 10.1164/rccm.201507-1372OC.
7. Duckworth A, Gibbons MA, Allen RJ, et al. Telomere length and risk of idiopathic pulmonary fibrosis and chronic obstructive pulmonary disease: a mendelian randomisation study. Lancet Respir Med. 2021; 9(3):285-94. 20201113. DOI: 10.1016/S2213-2600(20)30364-7.
8. Van Dyke MV, Martyny JW, Mroz MM, et al. Risk of chronic beryllium disease by HLA-DPB1 E69 genotype and beryllium exposure in nuclear workers. Am J Respir Crit Care Med. 2011; 183(12):1680-8. 20110311. DOI: 10.1164/rccm.201002-0254OC.
9. Ley B, Ryerson CJ, Vittinghoff E, et al. A multidimensional index and staging system for idiopathic pulmonary fibrosis. Ann Intern Med. 2012;156(10):684-91. DOI:10.7326/0003-4819-156-10-201205150 -00004.
10. van der Vis JJ, Prasse A, Renzoni EA, et al. MUC5B rs35705950 minor allele associates with older age and better survival in idiopathic pulmonary fibrosis. Respirology. 2023; 28(5):455-64. 20221226. DOI: 10.1111/resp.14440.
11. Baughman RP, Lower E. D. Geraint James Lecture: The sarcoidosis saga: what insights from the past will guide us in the future. Sarcoidosis Vasc Diffuse Lung Dis. 2023; 40(4):e2023057. 20231220. DOI: 10.36141/svdld.v40i4.15282.