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Americas association of sarcoidosis and other granulomatous disorders 2024 conference highlights

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Abstract. The Americas Association of Sarcoidosis and Other Granulomatous Disorders (AASOG) 2024 conference, held in Baltimore, Maryland, leveraged a multidisciplinary approach to disseminating and addressing the latest updates, challenges and opportunities in multisystemic sarcoidosis. The conference, aptly titled "The Art of Working Together for Progress," featured insights from diverse perspectives in sarcoidosis both nationally and internationally. This review summarizes the key takeaways from the six conference sessions: I. Sarcoidosis Multidisciplinary Care, II. Health Disparities in Sarcoidosis, III. The Search for Precision in Sarcoidosis, IV. Clinical Outcomes in Sarcoidosis, V. Clinical Trials in Sarcoidosis, and VI. Advanced Disease in Sarcoidosis.

Key words: sarcoidosis, multidisciplinary approach, conference report, health disparities, precision medicine, biomarkers, fibrosis, cardiomyopathy, granuloma

From August 15 to 16, 2024, over 200 registrants representing 59 institutions attended the Americas Association of Sarcoidosis and Other Granulomatous Disorders (AASOG) 2024 Conference, titled: "The Art of Working Together for Progress." The conference was chaired by Dr. Michelle Sharp, Dr. Nisha Gilotra, and Dr. Carlos A. Pardo, who work together in the Johns Hopkins Multidisciplinary Sarcoidosis Center of Excellence. With an emphasis on the integration of basic, translational, and clinical approaches, the AASOG 2024 Scientific Conference was focused on a central question: *how can multidisciplinary care, research, and education in sarcoidosis improve patient outcomes, reduce health disparities, and advance disease understanding?*

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This review highlights the key topics discussed at the AASOG 2024 conference, subdivided into the six sessions of the program.

Session 1: Teamwork makes the dreamwork: Aarcoidosis multidisciplinary teams

Session 1 was moderated by Dr. Ogugua Obi (East Carolina University, USA) and Dr. Brian Houston (Medical University of South Carolina, USA). After a welcome by AASOG president Dr. Alicia Gerke (University of Iowa Hospitals & Clinics, USA), the session was opened by AASOG Conference Co-Chair Dr. Nisha Gilotra (Johns Hopkins University School of Medicine, USA). Dr. Gilotra outlined the two primary goals of the conference: 1) To highlight research related to the pathogenesis and treatment of sarcoidosis and 2) To foster multidisciplinary collaboration and inspire future partnerships aimed at addressing gaps in sarcoidosis care.

Dr. Athol Wells (Imperial College London, United Kingdom), president of WASOG, subsequently spoke about the significance of multidisciplinary care and how to cultivate it in sarcoidosis. He mentioned that discussions about multidisciplinary care in sarcoidosis have historically focused on leveraging it to improve diagnosis and management of disease. The concept of holistic multidisciplinary care, where a diverse care team addresses all aspects of a patient's well-being—such as physical, emotional, and psychosocial factors—is less frequently discussed. Dr. Wells highlighted the challenges of validating multidisciplinary care compared to other care models in sarcoidosis but praised its ability to address nuances in disease diagnosis and management. He noted that multidisciplinary care should seek to better categorize disease phenotypes and provide clarity in cases of diagnostic uncertainty.

Dr. Laura Hinkle (Indiana University, USA) subsequently spoke about training physicians who specialize in sarcoidosis. She discussed the "dry pipeline" of academic faculty who have expertise in sarcoidosis. Issues that prevent training of physicians who specialize in sarcoidosis include decreased awareness of disease, limited support from funding agencies, lack of protected time, a paucity of available mentorship, and salary discrepancies between academic and private practice (1). She discussed ways to engage trainees and junior faculty to specialize in sarcoidosis. These include cultivating intellectual curiosity, protecting time, encouraging collaboration with patient action groups, and building networks for mentees to collaborate with others in the field. She ended her session by emphasizing the importance of prioritizing diversity of thought and backgrounds when building the pipeline of future sarcoidosis providers.

Following Dr. Hinkle was a panel discussion moderated by Dr. Kristen Mathias (Johns Hopkins University School of Medicine, USA) and Dr. Michelle Sharp (Johns Hopkins University School of Medicine, USA). Together with panelists Dr. Laura Koth (University of California San Francisco, USA), Dr. David Moller (Johns Hopkins University School of Medicine, USA), Dr. Lisa Maier (National Jewish Health, USA), Dr. Daniel Culver (Cleveland Clinic, USA), and Dr. Wonder Drake (University of Maryland School of Medicine, USA), they discussed pearls and pitfalls of navigating mentorship and a research career in sarcoidosis. The importance of cultivating a research niche, nesting work in developed research cohorts, collaborating with multicenter sarcoidosis researchers, and maintaining rigor in research methods were all emphasized. Additionally, the panel stressed the need to advocate for sarcoidosis research within journal editorial boards and in study sections for grants.

In the final talk of Session 1, Dr. Barney Stern (Johns Hopkins University School of Medicine, USA), Dr. Andrew Rosenbaum (Mayo Clinic, USA), Dr. Ennis James (Medical University of South Carolina, USA), Dr. Nadera Sweiss (University of Illinois Chicago), Victoria Wotorson, CRNP (Johns Hopkins University School of Medicine, USA), and Dr. Bryn Burkholder (Johns Hopkins University School of Medicine, USA) had a multidisciplinary discussion about therapeutics for sarcoidosis. Dr. Stern discussed treatment of patients with significant neurologic manifestations of sarcoidosis, defined as inflammation of the brain and/or spinal cord, hydrocephalus, and cranial neuropathies, highlighting approaches to corticosteroid dosing. Dr. Rosenbaum discussed the approach of concurrent initiation of corticosteroid and corticosteroid sparing therapies to minimize prednisone exposure, as well as the role of biologic agents in cardiac sarcoidosis. Ms. Wotorson emphasized the importance of patient education and the complexity of care coordination in treatment of multisystemic sarcoidosis. This is exacerbated by the fact that few therapies are approved for sarcoidosis by the United States Food and Drug Administration.

Session 2: Mind the gap: Addressing health disparities

Session 2 was moderated by Dr. John Odackal (The Ohio State University, USA) and Dr. Wonder Drake (University of Maryland, USA). The first speaker, AASOG Conference Co-Chair Dr. Michelle Sharp, provided an overview of disparities in sarcoidosis. She discussed socioeconomic disparities in sarcoidosis, highlighting that low-income patients are more likely to report lower health related quality of life (HRQoL), report barriers to treatment, and to be hospitalized. Dr. Sharp highlighted racial disparities in sarcoidosis, specifically that Black individuals are more likely to have multiorgan disease, have higher sarcoidosis-associated mortality and admission rates, and delayed access to multidisciplinary care compared to non-Hispanic White individuals (2). Finally,

Dr. Sharp touched upon gender disparities in sarcoidosis, noting that females have more lost workdays, lower HRQoL, lower lung function, and higher rates of hospitalizations compared to males (3–5).

The next speaker, Dr. Yvette Cozier (Boston University, USA) discussed gene-environment interactions with sarcoidosis and their interplay with socioeconomic factors. She discussed the complexities of classifying phenotypes of sarcoidosis and emphasized the significant genetic foundation. While numerous genetic loci have been identified in individuals with sarcoidosis, it remains unclear to what extent these genetic variants directly drive disease pathogenesis or interact with other factors to modulate the risk of developing disease. Dr. Cozier highlighted studies demonstrating how the interaction between HLA haplotypes, gene polymorphisms and environmental exposures, which are influenced by various social determinants of health, can impact the risk of developing sarcoidosis (6–8). Dr. Cozier discussed the idea of epigenetic changes–defined as potentially heritable changes in gene expression that are not result of changes to the underlying DNA–modulating sarcoidosis risk and briefly highlighted pertinent studies of DNA methylation patterns (9,10).

Dr. Logan Harper (Cleveland Clinic, USA) subsequently proposed ways to mitigate disparities in sarcoidosis. He discussed the generalized model in health promotion that consists of assessing needs, setting goals and objectives, developing and implementing an intervention, and evaluating the results. Dr. Harper described multileveled nonpharmacological interventions to improve quality of life in low-income Black individuals with sarcoidosis. He emphasized the role of a community advisory board, which facilitates creation of inclusive programs that educate, guide, and support patients, families, and the medical community on the effects of sarcoidosis, promoting personalized solutions and overall well-being.

Dr. Catherine Bonham (University of Virginia, USA) discussed ways to address health disparities from bench to bedside. She reviewed the progress in understanding sarcoidosis pathobiology and identifying contributors to health disparities and outcomes. Nevertheless, there remain a paucity of interventional studies addressing health disparities. Additionally, there is suboptimal diversity in clinical research and funding to support equitable design and analysis. Dr. Bonham outlined ways in which translational science could help understand the biology of chronic stress, environmental exposures, and disease progression in sarcoidosis. She discussed that gaps in access to technology can stymie equitable implementation of internet-based services and research. Dr. Bonham concluded by stressing that achieving health equity in sarcoidosis requires the integration of health equity research with healthcare delivery and policy.

In the final talk of the session, Dr. Sumita Khatri (National Heart, Lung, and Blood Institute, National Institutes of Health, USA) addressed improving diversity in sarcoidosis clinical trials. She outlined multifaceted barriers including individual time and resource constraints, participant mistrust of research and academic institutions, a paucity of funding for health equity research, and limited support for investigators who are Black, Indigenous and/or People of Color. Ways to mitigate these barriers include providing monetary and non-monetary incentives for research participants, making recruitment and research materials more understandable and accessible to patients, building research teams that are well versed on structural racism and implicit bias, including community members in research endeavors, prioritizing funding that includes minority participants, and increasing loan repayment and diversity supplement programs. Dr. Khatri ended by discussing the resources to help investigators improve inclusivity in clinical research.

Session 3

Part 1: The search for precision in sarcoidosis

The first part of Session 3 was moderated by Dr. Natalia Rivera (Karolinska Institutet, Sweden) and Dr. Nicholas Arger (University of California San Francisco, USA). Dr. Edward Chen (Johns Hopkins University, USA) opened by providing an overview of the current understanding of sarcoidosis pathobiology. He summarized that sarcoidosis was first recognized as a TH1-polarized immune response with dominant expression of interferon-gamma and ambient expression of other proinflammatory cytokines including IL2, TNF-alpha, IL1-beta, and IL6 that reflects how the immune system is primed to react in sarcoidosis (11). Historically, the Kveim-Siltzbach reaction provided evidence that this cytokine milieu in sarcoidosis was, in part, driven by an

adaptive response to environmental antigen(s) (12). More recently, the majority of lymphocytes from sarcoidosis patients expressing interferon-gamma show signs of Th17 lineage and are thereby dubbed Th17.1 cells (13). Human studies have identified TLR2 and mTOR as innate pathways involved in sarcoidosis and could promote Th17.1 differentiation. Serum amyloid A, an endogenous innate ligand, is upregulated in sarcoidosis also interacts with TLR2, and recent results from pre-clinical animal models show that the formation and maintenance of experimental granulomatous inflammation is controlled, in part, through TLR2 involving SAA and Th17.1 differentiation. Other animal models have demonstrated an important role for mTOR in Th17.1 differentiation and granuloma proliferation (14,15). Future translational studies are needed to further define the interactions between adaptive and innate immune responses that result in granulomatous inflammation seen in sarcoidosis.

Dr. Landon Locke (The Ohio State University, USA) subsequently discussed in-vitro models of granuloma and pathogen response, emphasizing the need for accurate models of sarcoidosis granulomas to improve clinical outcomes. He and his team have developed in vitro models of sarcoidosis using patient-derived peripheral blood mononuclear cells (PBMCs) to study underlying signaling and molecular events. The advantages of this approach are that it accounts for the genetic complexity, captures immune cells involved in granuloma formation, and allows for potential future discovery of biomarkers, antigens, and therapeutic testing. Their group has previously shown that PBMCs from patients with sarcoidosis form granuloma-like structures when exposed to tuberculosis (TB) antigens and have enhanced antimicrobial responses as well as divergent gene expression profiles compared to latent TB infection donors. Dr. Locke additionally discussed the validation of the in-vitro granuloma model using RNA-sequencing data that has shown similarities between the in-vitro model and diseased lung and lymph node tissues with respect to pathways related to TH2 activation, antigen presentation, and phagosome formation.

Dr. Alejandro Pezzulo (University of Iowa, USA) next spoke about the role of airway epithelial innate immunity in sarcoidosis. He discussed how airway epithelial cells are first-line sensors for pathogens and modulate immune cell activation, acting as key players in innate immune responses. Through this mechanism, he proposed that abnormal airway epithelial responses determine susceptibility to sarcoidosis and its progression. He described his group's ongoing efforts to investigate this hypothesis prospectively in patients with biopsy-confirmed sarcoidosis, with serial epithelial cell collection. The goal is to perform transcriptomics and targeted secreted protein assays on participant samples after exposure to triggers that simulate the granulomatous inflammation of sarcoidosis, hypothesizing that sarcoidosis epithelial cells have abnormal cytokine responses and upregulation of unique immune pathways after exposure to these triggers. In the long-term, they hope that this work can lead to better understanding of sarcoidosis mechanisms and identify therapeutic targets that block abnormal epithelial responses seen in the disease.

Dr. Erin McCaffrey (National Institute of Allergy and Infectious Diseases, USA) continued by discussing advances in molecular studies of granulomas in tuberculosis (TB). She highlighted the paradoxical nature in which TB granulomas in some cases facilitate sterilization of the microbe but in others facilitate dissemination of the microbe and immune system activation. These disparate outcomes can occur within a single individual, highlighting that the mechanisms of immune response regulation seem to be tightly controlled within the granuloma itself. Dr. McCaffrey and her group also aim to understand the granuloma at the 3-dimensional microenvironment level using single cell imaging, post-translational glycan imaging, and spatial transcriptomics. She discussed her group's recent work showing that the myeloid core of the TB granuloma is a microenvironment defined by expression of IDO1 and PD-L1, actively proliferating T-regulatory infiltrates, and production of TGF-β, not IFN-γ. Additionally, myeloid regulation and depleted lymphocyte activation were found emblematic of active TB in tissue and blood. Dr. McCaffrey ended by comparing TB and sarcoidosis granulomas, showing that the latter were strikingly enriched for CD4+ T-cells. Additionally, sarcoidosis granulomas express PD-L1, but not IDO1.

Finally, Dr. Antje Prasse (University of Basel, Switzerland) ended the first part of the session by discussing the role of Th17.1 cells and their interplay with macrophages in sarcoidosis. Th17 cells differentiate into Th17.1 cells that produce IFN-γ, TNF-α, IL-21, and GM-CSF but can also differentiate into

Tr1 cells that have similar features to T regulatory cells. Th17.1 cells are increased in lymph nodes and bronchoalveolar lavage fluid of individuals with pulmonary sarcoidosis. Moreover IFN-γ and TNF-α induce granuloma formation (13, 16). Using receptor-ligand analysis, Dr. Prasse's group has found that monocyte-derived macrophages communicate directly with Th17.1 cells. Therapeutic modulation of Th17.1 cells may thus be an attractive target in sarcoidosis.

Part 2: Is there an "Omics" definition of sarcoidosis

The second part of Session 3 was moderated by Dr. Skip Garcia (University of Florida Health, USA), Dr. Daniela Cihakova (Johns Hopkins, USA) and Dr. Antje Prasse (University of Basel, Switzerland). Dr. Courtney Montgomery (Oklahoma Medical Research Foundation, USA) started the "Omics" discussion by reminding the audience that two-thirds of US FDA-approved drugs resulted from the integration of multiple layers of genetic and functional genomics data (17). Within sarcoidosis, there is a need for clinically relevant biomarkers and clinical trial endpoints, and Dr. Montgomery explained how genetics could play a role in this goal. She discussed the evolution of SNP genotyping technology to DNA sequencing including exome, targeted, and finally whole genome sequencing. She talked about how genetic data is still leading to novel discoveries including a genome wide association study that described fibrosis in African American patients with sarcoidosis (18). Dr. Montgomery also reviewed the study showing common HLA pathways between patients who had resolved sarcoidosis and patients who were resistant to tuberculosis (19). Additionally, Dr. Montgomery discussed how genetic data can guide other omics studies.

Dr. Lisa Maier (National Jewish Health, USA) gave a talk titled "Transcriptomics in Sarcoidosis: What have we learned?" She reviewed that one of the early studies utilizing microarrays showed elevated gene density in sarcoidosis tissue including CXCL9, high expression of which was associated with more severe disease longitudinally (20, 21). Studies using microarray data developed a prognosticator using PCR genes for interferon-gamma, CXCL9, and TCR (22). Looking forward, Dr. Maier recommended looking at specific cell components important in granuloma formation. She also discussed using single cell sequencing to identify targets. Lastly, Dr. Maier explained that to better categorize cells she is using cellular indexing of transcriptomics and epitopes by sequencing.

Dr. Ivana Yang (University of Colorado Anschutz Medical Campus, USA) presented a talk on epigenetics. She described how there is an association between the epigenome and gene expression, disease risk and disease progression. By using epigenetics in sarcoidosis there has been identification of novel targets for understanding disease biology and activity. Additionally, epigenetic methods can aid in identifying potential biomarkers of disease and disease progression. In the future, it would be helpful to understand how the environment, genetics, and social determinants of health impact epigenetics.

Dr. Manesh Bhargava (University of Minnesota, USA) began a talk on proteomics by outlining the gaps in the field of sarcoidosis, including that it is a disease of exclusion, lack of biomarkers, and mechanistic uncertainty of various disease phenotypes. Proteins are commonly the targets of drugs and can likely serve as surrogate end points of disease activity. Proteomic studies can be leveraged to gain a deeper understanding of disease mechanisms. In a study by Dr. Bhargava, differential protein expression was found using quantitative proteomics between control and sarcoidosis cells. These proteins were then mapped to several pathways, finding the importance of the aldosterone pathway in granulomatous inflammation (23, 24). Dr. Bhargava also discussed examples of how proteomics has been used to discriminate sarcoidosis and subtypes. Dr. Bhargava recommended using proteomics in future clinical trials, being thoughtful about controls, power, and treatment status.

Dr. Jonas Schupp (Yale School of Medicine, USA) discussed spatial transcriptomics in sarcoidosis. Using transcriptomics Dr. Schupp described how the center of granulomas contains SPP1+ profibrotic macrophages which was previously described in IPF (25). Additionally, using transcriptomics, it was found that the central niches also contain proinflammatory macrophages. Another example of the utility of transcriptomics includes understanding that granuloma fibroblasts are derived from adventitial space and not from alveoli. Spatial transcriptomics data also enables spatial ligand-receptor analysis. Moving forward, one aim Dr. Schupp mentioned is to determine the micro-architecture of sarcoid granulomas by using spatial transcriptomics.

To wrap up the session, there was an engaging round table discussion about how to integrate the different "Omics" to create precision medicine in sarcoidosis. The group focused on the importance of data integration from these multimodalities to move the field forward.

Session 4: Outcomes in sarcoidosis: To what end?

Session 4 was moderated by Dr. Arthur Yee (Weill Cornell Medicine, USA) and Dr. Jan Griffin (Medical University of South Carolina, USA) and focused on defining and evaluating outcomes in sarcoidosis care and research. Through a combination of patient perspectives, expert talks, and panel discussion, the session examined how clinical, regulatory, and personal dimensions intersect in measuring what truly matters in sarcoidosis outcomes.

Kayla Nyakinye, CRNP (Johns Hopkins University, USA), moderated a roundtable discussion feature four patients living with sarcoidosis. They shared personal perspectives and priorities on care goals and outcomes. Several themes emerged: the importance of treatment that balances symptom control and quality of life, effective communication with providers, and access to care that respects patient preferences and emotional well-being. Patients discussed the frustrations of living with a chronic, unpredictable disease. Many cited fatigue, pain, and mental health tolls as major challenges, often exacerbated by administrative barriers such as insurance coverage or rigid provider assignment policies. The group emphasized that ideal care includes provider empathy, accessibility (e.g., through patient portals), and collaborative decision-making.

Dr. Daniel Culver (Cleveland Clinic, USA) next addressed the complexity of defining meaningful clinical outcomes in sarcoidosis. He emphasized the need for measurable surrogate endpoints that are mechanistically linked to meaningful patient benefit but also have undergone rigorous validation. He discussed the limitations of using longer-term outcomes such as mortality. He proposed composite outcome models that integrate patient-reported outcomes (PROs), physiologic function, and inflammatory markers (26). A promising example is the use of steroid withdrawal as a surrogate, which aligns with patient values and is being considered for regulatory approval. He concluded by underscoring that harmonizing clinical trial design with real-world priorities can accelerate progress in sarcoidosis therapeutics.

Dr. Lesley-Ann Saketkoo (Louisiana State University, USA) subsequently explored the evolving landscape of PROs and quality-of-life measurement in sarcoidosis. She described HRQoL as a complex interplay of symptoms, life disruption, emotional burden, and functional limitations—many of which remain unmeasured or dismissed in routine clinical care (27). She stressed the importance of validating subjective symptoms such as fatigue, pain, and brain fog, even in the absence of abnormal test results. Saketkoo advocated for clinicians to engage in "map-making" with patients—identifying priorities, preferences, and goals—rather than relying solely on biologic targets. She encouraged small, intentional acts of patient support: listening, acknowledging distress, and empowering patients to voice what outcomes matter to them most.

Dr. Lisa Shulman (University of Maryland, USA) wrapped up the session by discussing how innovation in outcome measurement can bridge gaps between patient experiences and clinical data. She began by highlighting barriers to reliable outcome assessment, including discrepancies between clinician observations and patient self-reports, and shifts in how patients recalibrate their expectations over time (the "response shift") (28). Shulman presented data showing that in neurological diseases such as Parkinson's, traditional measures often fail to capture quality of life or functional status. To address this, she recommended a mix of PROs, clinician-reported outcomes, and physical performance measures. She also highlighted the role of digital health technologies, such as wearable biosensors, in quantifying functional decline in more granular and continuous ways. Shulman further advocated for appropriately weighted composite endpoints that combine safety and efficacy signals.

Session 5: Clinical trials: Something old, something new, something borrowed, always purple

AASOG Conference Co-Chair Dr. Carlos Pardo (Johns Hopkins University, USA) started the session on clinical trials by discussing how current therapies for sarcoidosis are based on immunopathology studies of lymph node and pulmonary

sarcoidosis. However, future clinical trials should consider tissue-specific differences in pathology to elucidate effective therapies. Dr. Pardo emphasized that clinical trials should focus on improving the quality of life, which includes considerations of medication side effects, access, affordability, and alternatives for diverse patient populations.

Dr. Leslie Cooper Jr. (Mayo Clinic, USA) presented next and started off by commemorating Dr. Carol Johns, one of the first physicians to characterize treatment of cardiac sarcoidosis with glucocorticoids. Dr. Cooper recommended utilizing a classification schema related to disease activity in patients with cardiac sarcoidosis which is modeled after the heart failure classification schema. He started with at risk or "Stage A" patients which would have no clinical syndrome, abnormal biomarkers, or structural changes on imaging to the most advanced "Stage D" which would include patients unlikely to respond to medical intervention. Dr. Cooper reviewed the limited clinical trials in cardiac sarcoidosis including: the PRESTIGE study, a prospective analysis of methotrexate efficacy in patients who had a poor response or recurrent cardiac inflammation to corticosteroid therapy, the ongoing CHASM CS-RCT trial, a multicenter randomized controlled trial of prednisone monotherapy versus prednisone and methotrexate combination therapy designed to evaluate the optimal initial treatment strategy for active cardiac sarcoidosis, and the MAGiC-ART trial assessing interleukin-1 blockade therapy (29-31).

Dr. Clifton Bingham (Johns Hopkins University, USA) explored challenges and solutions of clinical trials in multisystem disease trials through a rheumatologic lens. He described core outcome sets as a collection of symptoms that need to be measured in every trial. This process involves identifying what needs to be measured, how it should be measured, and how it can be validated. For symptoms to be validated for example they must be truthful, able to discriminate between situations of interest, and feasible for inclusion in clinical trials. In development, it is beneficial to include all stakeholders including patients, providers, family members. Dr. Bingham related clinical trial challenges in sarcoidosis to similar challenges in rheumatologic diseases, such as lupus, both of which are complex, multi-system diseases. Lastly, Dr. Bingham discussed fatigue in rheumatic disease and how to measure meaningful change with treatment intervention.

Dr. Elizabeth Boulos (Food and Drug Administration, USA) joined the AASOG meeting to provide perspective from the Food and Drug Administration (FDA). She provided an overview of common challenges in rare disease drug development programs, including the lack of precedent for drug development, poorly understood natural history, small populations, significant phenotypic and genotypic diversity, and lack outcome measures. She highlighted the ARC program (Accelerating Rare disease Cures program), which provides a mechanism for sponsors to collaborate with the FDA throughout the efficacy endpoint development process. There is also the FDA rare disease innovation hub to leverage cross-agency expertise and enhance collaboration across centers. She also discussed the regulatory requirements and standards to establish "substantial evidence of effectiveness" which includes clinically meaningful effects and adequate and well-controlled investigations. For sarcoidosis specifically, she discussed how there are no established endpoints, and therefore biomarkers may be helpful for proof of concept and dose selection.

Dr. David Birnie (University of Ottawa, Canada) provided perspective on conducting a clinical trial in sarcoidosis. He started the talk by discussing how most studies are retrospective, single center, non-randomized, and with no blinded end point adjudication. There are many challenges to conducting randomized controlled trials in cardiac sarcoidosis, including unclear primary endpoint, lack of patients, and funding. One recommendation aligned with other talks was to extrapolate from "hard" clinical endpoints and use "soft" (or surrogate) endpoints when necessary. For example, in cardiac sarcoidosis a potential hard endpoint would be death or heart transplantation, whereas "soft" clinical endpoints could be ventricular tachycardia burden or heart failure hospitalization.

The session concluded with an engaging debate on the optimal approach to sarcoidosis clinical trial design regarding organ involvement (single vs. multiorgan). Dr. Brian Houston (Medical University of South Carolina, USA) was tasked with the supporting the stance that clinical trials in sarcoidosis should be approached in a single organ specific manner. He explained that specified single organ-system enrollment criteria and primary outcome measures will help identify organ-specific treatment effects, recognize that not every patient is affected multi-systemically,

and help trials answer specific questions. Dr. Lisa Maier (National Jewish Health, USA) then countered with why clinical trials in sarcoidosis should be multi-organ specific. She discussed that by not focusing on one organ clinical trials will be able to increase patient participation and improve inclusion of underrepresented groups. This would also be a more efficient enrollment and potentially less costly. Additionally, a multi-organ approach can address multiple outcomes as current treatment options are used for multiple organs.

Session 6: Addressing advanced disease in sarcoidosis

The final session of the conference focused on advanced organ manifestations and management in sarcoidosis. Attendees had the opportunity to join one of three breakout sessions focused on advanced pulmonary, end-stage cardiopulmonary or advanced neuro-ophthalmologic sarcoidosis.

The first group, moderated by Dr. Karen Patterson (University of Pennsylvania, USA) and Dr. Stephen Mathai (Johns Hopkins University School of Medicine, USA), addressed sarcoidosis-associated fibrotic lung disease and pulmonary hypertension. Dr. Marc Judson (Albany Medical Center, USA) discussed inflammation, fibrosis, and complications of pulmonary sarcoidosis. He highlighted that 10 to 20% of individuals with pulmonary sarcoidosis will develop fibrotic disease, with a higher prevalence in Black and male patients (32-34). Dr. Judson also emphasized that the presence of pulmonary fibrosis increases morbidity and mortality in individuals with sarcoidosis (35, 36). He debunked the misconception that fibrotic sarcoidosis is the result of "burnt out" disease, instead presenting compelling histopathologic, CT, and PET/CT images illustrating the role of active granulomatous inflammation in fibrosis. He discussed several potential biomarkers for fibrotic sarcoidosis, including single-nucleotide polymorphisms in GREM 1 and CARD15, as well as an allele of the TGF-β3 isoform. Dr. Judson concluded by highlighting several key unanswered questions including how to quantify the rate of fibrosis development, which anti-fibrotic agents are most effective, and how anti-granulomatous therapies influence the progression of pulmonary fibrosis.

Following Dr. Judson's talk was a debate regarding anti-fibrotic therapies for sarcoidosis related pulmonary fibrosis. Debating in favor of anti-fibrotic therapy was Dr. Catherine Bonham (University of Virginia, USA), while Dr. Karen Patterson provided the counterpoint. The arguments supporting anti-fibrotic use include its established efficacy in slowing the rate of forced vital capacity decline in multiple progressive fibrosing interstitial lung diseases, including sarcoidosis (37). Particularly severe sarcoidosis can clinically resemble idiopathic pulmonary fibrosis, and there is no evidence that suggests fibrotic sarcoidosis has a mechanism that would not respond to antifibrotic therapy. The arguments against anti-fibrotic use included the lack of compelling evidence that usual interstitial pneumonia is part of the spectrum of pulmonary sarcoidosis and that the biologic rationale for anti-fibrotic therapy in sarcoidosis is lacking. An argument was made that exploring this treatment in sarcoidosis distracts us from focusing on more relevant pathologic mechanisms to target in the disease. Prospective clinical trials will hopefully further elucidate their role in sarcoidosis.

Dr. Stephen Mathai concluded the first breakout group talks with a discussion on sarcoidosis-associated pulmonary hypertension (SAPH). He discussed how patients with sarcoidosis can develop pulmonary hypertension attributable to any of the five classes of pulmonary hypertension. SAPH is associated with reduced survival in sarcoidosis, particularly in those with post-capillary SAPH. Dr. Mathai discussed findings on initial workup that can alert one to the possibility of SAPH, as well as further evaluation that can help elucidate the contributors to pulmonary hypertension, including echocardiogram, ventilation perfusion scan, pulmonary function testing, arterial blood gas, overnight oximetry, polysomnography, and connective tissue and infection serologies. Right heart catheterization is key in confirming a diagnosis of SAPH and providing insight into a patient's overall hemodynamic profile and vasodilator response. Functional testing such as a 6-minute walk test and cardiopulmonary exercise testing can establish baseline prognosis. Future directions in SAPH include improved early detection, phenotyping beyond hemodynamics, and optimizing therapeutic management.

The second breakout session was moderated by Dr. Nisha Gilotra and Dr. Adam Morgenthau (Mount Sinai NY, USA) and covered end-stage pulmonary and cardiac sarcoidosis. Dr. Shambhu Aryal (Inova Health System, USA) discussed a holistic

approach to advanced pulmonary sarcoidosis and lung transplantation. He recommended considering factors such as support groups, pulmonary rehabilitation, and a multi-disciplinary approach in addition to pharmacologic treatment. Potential medications currently under investigation include efzofitimod for advanced pulmonary sarcoidosis. Dr. Aryal also discussed the importance of lung transplantation in quality of life and survival.

Dr. Jordana Kron (Virginia Commonwealth University, USA) presented management of advanced arrhythmias in sarcoidosis. She reviewed the risks, benefits, and limitations of various management approaches, including antiarrhythmic drugs, immunosuppression, ablation, sympathectomy, and radiation therapy.

Dr. Farooq Sheikh (Medstar Health – Georgetown University, USA) talked about advanced heart failure therapies in cardiac sarcoidosis. He reviewed studies finding favorable outcomes in patients with cardiac sarcoidosis receiving left ventricular assist devices or heart transplantation compared to other patients with heart failure, emphasizing that these therapies should be considered when appropriate (38-40). He highlighted the limitations of non-pathology confirmed, registry-based studies in heart transplantation and lack of consensus on treatment and monitoring of sarcoidosis post-surgery. Dr. Sheikh also discussed how cardiac sarcoidosis recurrence appears to be rare post-transplantation (41).

The third breakout session, moderated by Dr. Jinny Tavee (National Jewish Health, USA) and Dr. Barney Stern (Johns Hopkins University School of Medicine, USA), discussed management of high-risk neurologic and ocular sarcoidosis. Dr. Amanda Henderson (Johns Hopkins University School of Medicine, USA) spoke on the diverse presentations of optic neuropathy in sarcoidosis. She began by noting that neuro-ophthalmic involvement in sarcoidosis includes a spectrum of manifestations, ranging from optic neuropathy and orbital inflammation to cranial neuropathies, brainstem syndromes, and central visual field defects (42). Importantly, optic neuropathy in sarcoidosis may be the first sign of systemic disease, and up to 88% of patients lack a prior sarcoidosis diagnosis at presentation. Dr. Henderson emphasized that sarcoid optic neuropathy may arise from infiltrative, inflammatory, or compressive mechanisms, with MRI findings such as optic nerve enhancement or perineural thickening providing critical diagnostic clues. She presented compelling cases illustrating dramatic steroid responsiveness, steroid dependency, and misdiagnosed "meningiomas" later found to be sarcoid-related. The importance of cross-specialty collaboration, including full-body imaging and potential biopsy, was stressed for challenging cases. Her key message was that a high index of suspicion is vital.

Dr. Jeffrey Gelfand (University of California San Francisco, USA) next presented on the management of high-risk neurosarcoidosis, focusing on patients with central nervous system (CNS) involvement and hydrocephalus. He reviewed data showing that neurosarcoidosis affects 5–10% of patients with sarcoidosis, though up to a third may have subclinical neurologic involvement. He emphasized the importance of early recognition and accurate diagnosis, utilizing clinical, imaging, and cerebrospinal fluid (CSF) findings, citing the 2018 Neurosarcoidosis Consortium consensus criteria (43). Dr. Gelfand discussed hydrocephalus as a life-threatening complication seen in up to 10% of neurosarcoidosis cases (44). He highlighted that timely immunosuppression—typically corticosteroids in combination with agents like methotrexate or infliximab—is essential for inducing and maintaining remission while minimizing corticosteroid toxicity. He cautioned against misdiagnosis when CSF glucose is severely low (<30 mg/dL), urging careful exclusion of infections such as histoplasmosis and cryptococcosis. His talk underscored that multidisciplinary care, serial imaging, and personalized therapy are essential pillars of management.

Dr. Carlos Pardo then presented on spinal cord involvement in neurosarcoidosis, drawing from the extensive longitudinal Johns Hopkins Neurosarcoidosis Registry. He highlighted that while meningeal and cranial neuropathy forms of neurosarcoidosis are commonly recognized, spinal cord involvement—termed "myelopathic neurosarcoidosis"—is both frequent and clinically significant. Among 260 patients in the registry, 25% had myelopathy at presentation, with diverse imaging patterns. Most cases followed a subacute or chronic course, presenting with gait disturbances, paraparesis, bladder dysfunction, and sensory abnormalities. Dr. Pardo emphasized that sarcoidosis-associated myelopathies can be the initial manifestation of systemic sarcoidosis and often necessitate differentiation from mimics, such as spondylotic myelopathy. He reported that patients with myelopathy had a lower rate of

relapse compared to those with encephalitic or cranial neuropathy phenotypes, and they also demonstrated greater improvement in functional outcomes. However, discontinuation or tapering of steroids was a key contributor to relapses across all phenotypes. These findings underscore the need for early recognition and careful long-term immunosuppressive management. Dr. Pardo concluded by advocating for increased attention to neurologic complications in sarcoidosis and highlighted the value of multidisciplinary collaboration through dedicated sarcoidosis clinics and research programs (45).

Dr. Paula Barreras (Cedars Sinai Medical Center, USA) subsequently presented on small fiber neuropathy (SFN), fatigue, and cognitive dysfunction in sarcoidosis. SFN affects 30–40% of sarcoidosis patients and often presents within three years of systemic diagnosis with symptoms like burning pain, paresthesia, and dysautonomia (46). Diagnosis relies on skin biopsy showing reduced intraepidermal nerve fiber density, as EMG is typically normal. Dr. Barreras emphasized that SFN in sarcoidosis is likely cytokine-mediated rather than granulomatous, citing elevated pro-inflammatory cytokines found in affected tissues (47, 48). Immune therapies including IVIG, TNF-α inhibitors, and tocilizumab have shown variable but encouraging results in retrospective studies and early cohort data (49, 50). She also discussed fatigue and "brain fog," common in sarcoidosis and often unrelated to disease activity or pulmonary function (51, 52). While evidence for treatment is limited, some studies suggest TNF-α inhibitors and cognitive behavioral therapy may offer benefit in select patients. Dr. Barreras highlighted the importance of recognizing and addressing these symptoms to improve quality of life.

To conclude the annual conference, early career research was highlighted from submitted abstracts and a panel engaged in a multidisciplinary case discussion. Dr. Wonder Drake presented Dr. Aisha Souquette's (University of Maryland, USA) work on "Gut Microbiota Induces Pulmonary CD4+ IL-6+ Expression Through PD-1/ HIF-1α Signaling in Mice." Dr. William Lippitt (University of Colorado, USA) then presented on "Chest Computed Tomography Contributes Information on the Extent of Physiologic Impairment Beyond Chest X-ray Alone in Pulmonary Sarcoidosis." Dr. Miles Hagner (University of Iowa, USA) presented work "Airway Epithelial Alarmin Responses to Cutibacterium acnes Identifies a Severe Cardiac Endotype of Sarcoidosis," and Dr. William Damsky (Yale School of Medicine, USA) presented "Spatial Transcriptomics Reveals Structurally Organized and Distinct Immune Polarization in Inflammatory Cutaneous Granulomatous Disorders". Dr. David Perlman (University of Minnesota, USA) then led an expert panel discussion around a case titled "Unmasking the Masquerade: Tuberculosis Infection Masquerading as Lofgren's Syndrome of Sarcoidosis" presented by Dr. Ifreah Usmaiel (SUNY Upstate Medical University, USA).

In summary, the AASOG 2024 Conference addressed the current state of the field of sarcoidosis and identified the future directions for multidisciplinary basic, translational, and clinical research in sarcoidosis. The meeting facilitated dissemination of knowledge as well as multicenter collaborations that are essential to progress in clinical and translational research in this rare disease.

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