sTable 1: Current Infectious and Environmental Factors Implicated in the Etiology of Sarcoidosis

Factors		Proof
1. **Occupational Hazards**		
Indoor air (molds, bacteria, microbial contaminant)	-	Epidemiologic and Immunologic proof[1]
Healthcare Workers	-	Epidemiological proof [2-4]
Inorganic dust exposure from construction work	-	Epidemiological proof[5, 6]
Metal dust, metalworking fluid, aerosols from Metal Industries	-	Epidemiological proof [7, 8]
Insecticides, Pesticides, Mold/ Mildew, Silicates exposure (Agricultural workers)	-	Epidemiological proof[9, 10]
Silicates from Mining	-	Epidemiological proof [11]
Organic Dust, Wood Dust, Metal Dust, Wood Burning (Naval and Military Personnel)	-	Epidemiological proof[12, 13]
Heavy dust exposure, Nanoparticles, carbon (Fire fighters)/Rescue workers	-	Epidemiological proof[14-16]
2. **Organic Particles**		
Brominated diphenyl ethers polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls, polychlorinated dibenzodioxins, polychlorinated dibenzofurans, pesticides, phthalate esters, and other hydrocarbons. From the explosion and collapse of the World Trade Center (WTC)	-	Analyses of total settled dust and smoke that settled immediately after the explosion and fire and the concurrent collapse of the two World Trade Center (WTC) structures[15]
3. **Inorganic Particles**		
Nanoparticles	-	Experimental Models of Sarcoidosis [16-18]
	-	Saroid-like granulomas developing after exposure to nanoparticles[6, 19-21]
	-	Immune Response [22]
Construction Materials, Soot, paint (leaded and unleaded), Fibers (e.g., mineral wool, fiberglass, asbestos, wood, paper, and cotton) Metals, Radionuclides, and Ionic species.	-	Data from World Trade Center [15]
4. **Infectious Agents**		
Bacteria, Fungi and Parasites		
• *Mycobacteria*	-	Nucleic acid[23-27]
	-	Cell Constituent [28]
	-	Immune Response and Circulating Antibodies [24, 27, 29-37]
	-	Success of Anti-mycobacterial therapy[35-37]
	-	Experimental Disease Models[38-47]
	-	Proteomics[48]
	-	Meta-analysis[29, 49]
	-	Similarities in Transcriptome Profile of Sarcoidosis to Tuberculosis infection[50-53]
• *Propionibacteria*	-	Success of Antibiotics[54-56]
	-	Experimental Disease Models[57-65]
	-	Meta-analysis [66]
	-	Bacterial Culture[67-69]
	-	Immune Response, Immunohistochemistry[60, 70-79]
	-	Microbial Components [80, 81]
	-	Nucleic Acid[65, 80-84]

• *Chlamydia pneumoniae*	- Immunological proof[85-87]
• *Rickettsia helvetica,*	- Nucleic acid[86, 88, 89]
• *Leishmania species*	- Nucleic acid[88, 90]
• *Molds*	- Immune response and elevated N-acetylhexosaminidase (NAHA) in homes of Sarcoidosis patients[88, 91-94]
• *Borrelia burgdorferi*	- Nucleic acid and Immunohistochemistry[95]
Viruses	
• *Epstein-Barr virus (EBV)*	- Antibodies, Association between Immunosuppression therapy and increased risk for the development of EBV in Sarcoidosis patients [96, 97]
• *Human herpesvirus 6 (HHV6)*	- Antibodies[98]
• *Human herpesvirus 8 (HHV8)*	- Antibodies[99]
• *Human T-lymphotropic virus 1 (HTLV1)*	- Antibodies[100]
• *Cytomegalovirus (CMV)*	- Antibodies[101, 102]

sTable 2: 2017 JMHW revised criteria to diagnose isolated cardiac sarcoid (ICS)[103, 104]

1. No clinical findings characteristics of sarcoidosis observed in any organs other than the heart
2. 67Ga scintigraphy or 18F-FDG PET without abnormal tracer accumulation in any organs other than the heart
3. A chest CT scan reveals revealing no shadowing along the lymphatic tracts in the lungs or no hilar and mediastinal lymphadenopathy (minor axis >10 mm)
4. Either a histological or a clinical diagnosis

sTable 3: Potentially Dangerous Clinical Scenarios Related to Pulmonary Sarcoidosis

Condition	Mechanism	Treatment	Percentage of pulmonary sarcoidosis patients
Fibrocystic sarcoidosis	The fibrosis is the result of granulomatous inflammation in a subset of sarcoidosis patients.	• No known specific treatment • Anti-granulomatous therapy may be beneficial in preventing further development of fibrosis	10 – 20%
Sarcoidosis-associated pulmonary hypertension	Predominantly from fibrotic distortion of the pulmonary vasculature. Other mechanisms include pulmonary venous hypertension from cardiac sarcoidosis or from corticosteroid-induced diabetic/atherosclerotic cardiomyopathy, pulmonary hypoxemia from parenchymal sarcoidosis, granulomatous inflammation of pulmonary arteries and veins.	• Pulmonary vasodilators, oxygen. • Anti-granulomatous therapy may be beneficial in a minority of patients.	5%, with most a subset of fibrocystic sarcoidosis patients
Bronchiectasis, severe airway distortion	Fibrotic distortion of airways	• Enhance mucociliary clearance • Intermittent appropriate antibiotics • Possibly consider roflumilast	5% - 10%, with most a subset of fibrocystic sarcoidosis patients
Mycetoma	Colonization of fungus in devitalized, fibrotic sarcoidosis lung	• Surgical excision • Anti-fungal agents - given systemically - Injected into mycetoma cavities	1%, with most a subset of fibrocystic sarcoidosis patients.