In addition to mTOR and JAK/STAT, NLRP3 inflammasome is another key pathway activated in sarcoidosis

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Sarcoidosis is a rare "old disease" first described over a century ago, known as a multifaceted systemic multiorgan inflammatory granulomatosis characterised by the presence of non-necrotising epithelioid granulomas [1, 2]. In many ways sarcoidosis remains a mysterious disease with numerous unsolved knowledge gaps. The still unknown aetiology/aetiologies of sarcoidosis is one of these gaps, *i.e.* the disease develops in genetically predisposed individuals following as-yet-unknown antigen exposure. Various triggers or causes have been suspected, including bacterial agents (such as *Propionibacterium acnes* or mycobacterial antigens) or environmental particulates (such as crystalline silica), but their true identity and mechanisms of action are still not elucidated [1–3]. The second main gap regards the nature of the granuloma foundation, the hallmark of the disease, as its mechanisms related to complex immunopathogenesis are only partially understood [1–3]. Furthermore, even though the highly variable course of the disease is well documented, disease evolution is still quite unpredictable for a given patient. In many cases, tissue granulomas resolve spontaneously, while in others they persist or reoccur and require treatment; usually prednisone alone, or other drugs such as immunosuppressants [1, 2, 4]. The current therapeutic monitoring is hence mainly based on empirical knowledge and not on well-identified pathogenesis mechanisms. Therefore, a better knowledge of the intracellular pathways potentially activated is needed.