were excluded. 11 healthy volunteers acted as a control group. Serum levels of interleukin (IL) 1ß, 2, 4, 6, 8, 10, 12p70 and tumour necrosis factor α (TNFα) were measured using a multiplex analyser. A Wilcoxon matched pairs test was used to compare differences between baseline and follow-up samples.

Results Patient and control demographics are presented in the Abstract P3 table 1. Levels of IL 2,6,8,10 and TNFα were elevated in the patient group compared to controls. Average time to repeat patient sampling was 4.7±1.8 months. There were no significant differences between the levels of any cytokines between baseline and follow-up in the patients and no overall correlation existed between change in 6MWT and change in any cytokine. When patients who had improved their 6MWT on follow-up were analysed as a separate group, there was a significant (p=0.0068) drop in IL6 levels on follow-up.

Abstract P3 Table 1 Patient and control demographics

	Patients	Controls
N	19	11
Male	6 (32%)	3 (27%)
Age (years) (Mean ± SD)	58±14	43±8
NYHA Class (III/IV)	15 (79%)/4 (21%)	
Aetiology of PH		
IPAH	7 (37%)	
Connective tissue disease	5 (26%) (3× SSc, 2× MCTD)	
Congenital heart disease	3 (16%) (2× Unrepaired, 1× Repaired)	
Distal CTEPH	3 (16%)	
Veno-occlusive disease	1 (5%)	
Medication on follow-up		
Endothelin antagonist	8 (43%)	
Prostanoid (Nebulised)	1 (5%)	
Sildenafil	9 (47%)	
Combination	1 (5%)	
mPAP (mm Hg)(Mean ± SD)	51.8±13.5	
CI (L/min/m^2)(Mean ± SD)	1.89±0.62	

CI, Cardiac index; MCTD, Mixed connective tissue disease; mPAP, Mean pulmonary artery pressure; SSC, Scleroderma.

Conclusions Several cytokines were elevated in these patients with pulmonary hypertension. The role of cytokines in disease monitoring requires further study and there seems to be little relation with serum cytokines, except IL6, and a change in a patient's clinical state as measured by 6MWT. Replication of results from this pilot study and examination of longitudinal trends in cytokine levels are warranted.

P4 THE USE OF SILDENAFIL TO TREAT PULMONARY HYPERTENSION ASSOCIATED WITH SARCOIDOSIS

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Introduction and Objectives Pulmonary hypertension (PH) is increasingly recognised as a major contributor to poorer outcomes in pulmonary sarcoidosis. Current treatment guidelines for sarcoidosis associated pulmonary hypertension (SAPH) recommend the optimisation of underlying lung disease and use of oxygen when hypoxaemia is present,[1] however there is insufficient evidence to recommend advanced therapies. Limited data suggest a benefit following treatment with sildenafil in SAPH, although no randomised controlled treatment trials have been reported. We report our experience of patients with SAPH treated with sildenafil.

Methods We reviewed 22 patients (mean age 56±12 years; 10 men) with SAPH (mean pulmonary artery pressure 46±9 mm Hg) who received treatment with sildenafil. Haemodynamic measurements were evaluated by right heart catheterisation in all patients. Serial measurements of brain natriuretic peptide (BNP), pulmonary function testing and functional status were collected.

Results The mean duration of follow-up after the commencement of sildenafil was 21±14 months. Six patients died during follow-up, and four patients required the addition of an endothelin receptor antagonist due to worsening pulmonary hypertension. Patients were dichotomised into responders (those maintained on sildenafil monotherapy; n=12) or non-responders (those who died or required and escalation of PH therapy during follow-up; n=10). Compared to responders, non-responders had a significantly lower % predicted DLco (20% vs 34%; p=0.03) and % predicted Kco (46% vs 62%; p=0.05) at commencement of sildenafil (Abstract P4 table 1). Within 6 months of commencement of sildenafil, non-responders had a 21% increase in median BNP levels, while responders had a 51% reduction in BNP levels. Sildenafil was ceased in one patient after the development of ocular side-effects attributed to the drug.

Abstract P4 Table 1 Demographic and baseline clinical characteristics of patients with sarcoidosis associated pulmonary hypertension receiving treatment with sildenafil*

	Non-responders (n=10)	Responders (n=12)	p Value
Age (years)	53.5 (14)	58.5 (10)	NS
Gender, M:F	5:5	5:7	
Follow-up (months)	23 (16)	20 (12)	NS
Baseline			
mPAP (mm Hg)	50 (10)	43 (8)	NS
PaO_2 (kPa)	7.8	8.2	NS
DLco % predicted	20	34	0.03
Kco % predicted	46	62	0.05
Δ BNP at 6 months	+21%	−51%	

*Values are given as mean (SD) or median (range).

Conclusions Our observations suggest that sildenafil is safe and well tolerated in SAPH. At commencement of sildenafil, a lower % predicted DLco and Kco, and an increasing BNP level within the first 6 months of therapy, was associated with increased risk of death or requirement for additional PH therapy. Controlled trials are warranted before therapeutic recommendations can be made.

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P5 PULMONARY HYPERTENSION IN A MOUSE MODEL WITH REDUCED MACROPHAGE NUMBER (MACLOW)

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Introduction and Objectives Pulmonary arterial hypertension (PAH) is a devastating condition with high morbidity and poor life expectancy. Pathologically PAH is characterised by the medial thickening of the small distal pulmonary arteries. Early endothelial cell (EC) dysfunction and apoptosis, and the subsequent abnormal proliferation and migration of pulmonary artery smooth muscle cells are thought to be a major contributing factor. Macrophages are proposed to play an important role in regulating these processes and are recruited to remodelled pulmonary arteries but the exact role of