ANALYSIS OF BRONCHOALVEOLAR LAVAGE FLUID FROM PATIENTS WITH SARCOIDOSIS. E.A. van de Graaf, B.E.A. Hol, H.M. Jansen, T.A. Out, C. Alberts. Dept of Pulmonology and Clin Immunology Lab, AMC, Amsterdam, The Netherlands.

To obtain insight into the pathophysiology of pulmonary sarcoidosis we investigated local inflammation and production of immunoglobulins within the lung. In the bronchoalveolar lavage fluid (BALF) and in the blood from 19 patients with a biopsy proven sarcoidosis and from 9 healthy volunteers we measured albumin, alpha-2 macroglobulin (A2M), ceruloplasmin(CP), IgM, IgG, sIgA, and C3a by immuno assays.

Protein concentrations (mg/L) in BALF (median, range):

	controls		sarcoidosis	
albumin	31.4	22.5 - 50.2	109	28.2 - 608
CP	0.18	0.1 - 0.68	1.03	0.27- 6.39
A2M	0.14	0.05- 0.44	1.88	0.15- 16.3
IgG	6.95	4.13- 17.7	84.0	21.2 - 294
IgM	0.13	0.06- 1.05	2.52	0.19- 9.58
sIgA	0.50	0.28- 1.47	1.89	0.02- 4.72
C3a(nmol/L)	0.20	0.12- 0.31	1.30	0.10- 5.64

The IgG concentrations in the blood from the patients (18.0 g/L ± 5.2) were increased in contrast to other proteins.

The ratios of protein concentrations in the blood and the epithelial lining fluid (ELF, J Appl Physiol 1986; 60:532) were calculated (cQprotein). In the patients the cQalb, cQCP, cQA2M and cQC3a were increased (Mann-Whitney U-test, MWU, $p<0.05$). We also found a correlation between the cQA2M and cQC3a (Spearman's rank correlation: $r=0.84$, $p<0.01$). This indicated a correlation between increased exudation of plasma proteins in the lung and local complement activation.

The sIgA concentrations in ELF were higher in the patients (MWU, $p=0.001$) than in the controls, suggesting increased secretion of sIgA within the lung. Local production of IgM and IgG was investigated by comparing cQIgM and cQIgG to cQA2M and cQCP, respectively. We found indications for local production of IgG and IgM in several patients.

We conclude that in pulmonary sarcoidosis there is B-cell stimulation and inflammation within the lung. This inflammation possibly is caused by local complement activation.

CLINICAL INVESTIGATION OF THREE FORMS OF HYPERSENSITIVITY PNEUMONITIS (HP); JAPANESE SUMMER-TYPE HP, BIRD-FANCIER'S LUNG AND HUMIDIFIER LUNG. T.Shirai, A.Sato, T.Suda, H.Hayakawa, K.Chida. The Second Department of Internal Medicine, Hamamatsu University School of Medicine, Hamamatsu, Japan.

A variety of specific antigens are reported to cause various forms of HP. However, ouly limited information is available on whether there are differences in background and clinical characteristics of the forms. In this regard, we have compared the clinical features of Japanese summer-type HP (JST), bird-fancier's lung (BFL) and humidifier lung (HL). Subjects consisted of 28 cases of HP including 18 of JST, 5 of BFL and 5 of HL (5 males with mean age of 65 y.o.). It was noted that the number of female patients with JST or BFL were larger than that of males (F/M = 5/13, 1/4 in JST, BFL), whereas all of the HL cases were males. The mean age of the onset of the disease was different in three forms of HP (38 y.o in JST< 50 y.o. in BFL< 65 y.o. in HL). In the clinical courses, development of symptoms in BFL were subacute, and the values of acute phase reactants were lower and PaO2 was higher than those of other forms of HP. However, the period for the disappearance of abnormalities on the chest roentgenogram was prolonged in BFL, being 31.2 days, as compared with 21.3 days in JST and 17.3 days in HL. Examination of bronchoalveolar lavage (BAL) fluids revealed that the CD4/CD8 ratios of BAL lymphocytes were significantly different in these forms: the ratio was 0.59 in JST, 5.57 in BFL and 1.78 in HL. We also tested the presence of serum precipitins against 16 antigens. The data indicated that the mean number of the antigen causing positive reactions was 1.7, 1.0 or 2.8 in JST, BFL or HL, respectively. Precipitation reaction was positive to *Trichosporon cutaneum* in 90% cases of JST and Pigeon droppings in all cases of BFL. Each antigen was considered to the causative agent of the disease. Serum from HL patients reacted with several antigens such as *Cephalosporium acremonium*, *Aspergillus fumigatus* and *Candida albicans* without indicating causative antigens for the disease.

These results suggest that specific population as well as presence of each causative antigen is responsible for the onset and different clinical features of JST, BFL, and HL.

SERUM PRECIPITINS AGAINST FARMER'S LUNG (FL) ANTIGENS IN THE FRENCH REGION OF THE DOUBS : PREVALENCE AND RELATION TO THE DISEASE. JC Dalphin, B Toson, E Monnet, D Pernet, A Dubiez, JM Aiache, A Depierre. Departments of Respiratory Disease and of Epidemiology, Besançon ; Department of Immunology, Clermont-Ferrand ; France.

FL precipitins give evidence of chronic exposure to FL antigens, but their exact relation to the disease remains unclear. In a region where FL is frequent (Thorax 1988;43 : 429-35) we evaluated the prevalence of FL precipitins in dairy farmers and analyzed the relation between the presence of FL precipitins and the probability of having the disease. All the exposed dairy farmers of boths sexes (n = 2555) from five districts of the Doubs region were asked to respond to a questionaire derived from the ATS questionaire. One thousand seven hundred and sixty three (69%) farmers agreed to participate in the study. Precipitin tests were conducted in 551 (31%) farmers who showed any respiratory symptoms and in a random sample of 100 asymptomatic farmers. Serum for each farmer was analyzed by both Double Diffusion (DD) and Immunoelectrophoresis (IE) ; detection antigens consisted of Micropolyspora faeni (MF) and three extracts of mouldy hay (HE) from the Doubs. The 651 farmers studied were divided into four groups with a respectively decreasing probability of having FL ; G1 : typical semi-delayed FL symptoms ; G2 : respiratory symptoms following exposure to FL antigens ; G3 : non-specific symptoms (chronic bronchitis and/or chronic dyspnea) ; G4 : asymptomatic farmers. Estimated prevalence (%) of precipitins in the total population and prevalence (%) of precipitins in each group are presented in the following table.

	Total n = 1763	G1 n = 35	G2 n = 140	G3 n = 376	G4 n = 100	p *	p**
DD:HE	83	83	91	81	82	NS	NS
MF	27	29	29	23	28	NS	NS
IE:HE	26	51	36	29	13	<0,0001	<0,0001
MF	19	26	12	10	10	<0,05	<0,05

* chi 2 test ; ** test of linear trend (Cochran)

We conclude that there is a close relation between the presence of precipitins and the probability of having FL. This is detected by the IE method but not by the DD method.

CHRONIC HYPERSENSITIVITY PNEUMONITIS CAUSED BY DIPHENYLMETHANE DIISOCYANATE FOLLOWED BY ACUTE HYPERSENSITIVITY PNEUMONITIS AFTER EXPOSURE TO A TOLUENE DIISOCYANATE ALKYD PAINT. C Ugolini, J Watkins, SM Hessl, J Coe, LC Grammer, P Orris. National Institute for Occupational Safety and Health, Morgantown, WV. Cook County Hospital, University of Ilinois, and Northwestern University, Chicago, IL.

Isocyanates are a common cause of occupational asthma and have also been associated with hypersensitivity pneumonitis (HP). We report a case of acute HP after a short exposure to TDI which was preceded by chronic HP from repeated exposures to an MDI foaming process. A 40 year old man, in April 1988, was employed to inject MDI polyurethane foam into automobile alarm boxes. Three weeks later he noted a nonproductive cough when working with MDI. This cough worsened and often woke him from sleep. His symptoms progressed over the next 1.5 years to include shortness of hreath and blood-streaked sputum at work. A distinctive odor was noted with the process, ventilation measures were poor, and no respiratory protection was worn. The worker was laid off in November 1989. Three weeks later he started a new job at an auto dealership where extensive maintenance painting of air ducts and pipes was in progress. The paint contained TDI. On the second day he became acutely ill and reported fever (105F), hemoptysis, shortness of breath, and general malaise. He was hospitalized and initial findings included an oral temperature of 101.6F, respirations of 24/minute, bibasilar inspiratory fine crackles, arterial blood gases with pH 7.47; pCO231.5; PaO2 54.3 mm Hg. IgE and IgG specific serum antibodies titers to MDI-Human Serum Albumin were 1:250 and 1:10,000 respectively. Three months later a methacholine challenge test was strongly positive. Presently, two years after discharge, he has a restrictive lung impairment and persistent bronchial hyperreactivity. This worker represents a case of HP caused by isocyanates with evidence of cross reactivity between MDI and TDI. The acute HP has been followed by bronchial hyperreactivity for two years without signs of improvement. This is also a possible second case of isocyanate hemorrhagic HP as previously described by Roy Patterson et al. (AM REV RESPIR DIS 1990;141:226-230)

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ABSTRACTS

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