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# Alveolitis Due to Hair-Spray

## Ultrastructural Observations in Two Patients and the Results of Experimental Investigations

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**Summary.** Observation of two patients with hair-spray induced lung disease have prompted us to study the ultrastructure of the lung lesion. We have compared the results with experimental lesions in animals injected with hair-spray extracts and with human monocyte cell cultures exposed to hair-spray.

The lungs show a chronic alveolitis with a striking granulomatous reaction including macrophages and multinucleated giant cells of the foreign body type. The intraalveolar and interstitial macrophages and the giant cells all contain PAS-positive material. Ultrastructurally distinct lamellar inclusions are found in the secondary lysosomes of the macrophages and giant cells. Identical structures can be produced in animals injected with hair-spray extracts and with polyvinyl-pyrrolidone and -acetate (PVP/PVA), which are regular ingredients of hair-sprays. Large, presumeably polymeric particles (PVP/PVA) are ingested by giant cells. This "gigantophagocytosis" is associated with the fusion of mononuclear phagocytes and leads to the genesis of giant cells. In cell cultures of human blood monocytes hair-spray extracts and PVP/PVA induce maturation and aggregation of these cells, with PAS-positive cytoplasmatic inclusions. The development of multinuclear giant cells in these monocyte cell cultures is also seen.

These observations suggest that hair-spray induced lung disease is caused by the prolonged and extensive body response of the local mononuclear phagocyte system (MPS). Overstimulation of the MPS leads to a quantitative and qualitative change which is followed by a partial blockade of this system. The alveolitis is a consequence of the foreign body response to inhaled hair-spray substances.

**Key words:** Alveolitis – Mononuclear phagocyte system – Macrophages – Granulomas – Giant cells – Foam cells – Hair-spray – Polyvinyl pyrrolidone.

* Dedicated to Professor Dr. Wilhelm Doerr on the occasion of his 65. birthday

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## Introduction

Whether or not hair-spray causes lung disease is a matter of dispute. A recent review of earlier studies on accumulation of hair-spray components in lung tissue ("thesaurosis") concluded that the existence of hair-spray induced storage disease of the lungs has yet to be proved (Gowdy and Wagstaff, 1972). However, Bergmann (1973) reviewing his own and other studies, came to the conclusion that there is much evidence of the association between hair-spray use and lung disease. Pulmonary function tests have shown that hair-spray inhalation is followed by an acute, reversible narrowing of small air-ways (Zuskin and Bouhuys, 1974; Cohen, 1977). The relativly small number of reports describing the association between hair-spray exposure and lung disease (review: Gebbers et al., 1979) contrasts with the very common use of hair-spray. It is usually very difficult to prove a causative relationship between hair-spray use and lung disease. Only a comprehensive clinical examination (roentgenology, lung functions tests, demonstration of a significant hair-spray exposure) combined with histopathological observations allow the diagnosis of "hair-spray" lung. We recently published a report of two patients with severe lung disease due to an excessive hair-spray exposure (Gebbers et al., 1979). The results of the electron microscopic investigation of the biopsies from these two patients and the details of animal experiments and recations of human monocyte cell cultures exposed to hair-spray extracts, are presented in this paper.

## Material and Methods

*Case Report.* This study is based on clinical and histological observations in two patients with a severe disease of the lung parenchyma (Gebbers et al., 1979). Both patients are non-smokers and had no other exposures to inhaled dusts or gases. One patient is a 48 year old housewife, the other is a 60 year old hairdresser. In both cases significant discrepancy was found between the roentgenological findings of the lung and considerably decreased respiratory function. X-ray pictures of the lungs showed only a milky and reticular cloudiness, while functional studies showed reduction of the vital capacity and compliance to half of normal values.

An open lung biopsy was performed to establish the diagnosis in both cases. On the basis of histopathological findings exogeneous thesaurosis of the lung was considered to be the cause of the disease and a "hair-spray lung" was diagnosed. Considerable hair-spray exposure could be established in both cases. After withdrawal of hair-spray a significant improvement occurred within half a year in both patients (for further clinical data see Gebbers et al., 1979).

*Histology.* The biopsy speciemens, which were obtained by thoracotomy, were injected with Bouin's solution and fixed for 5 h. The paraffin sections were stained with HE, PAS, Masson-Goldner, elastic-van Gieson and for iron.

*Electron Microscopy.* 1 $mm^3$ small tissue specimens were fixed immediately in Dalton's solution (Dalton, 1955) for 2 h. After dehydration in a graded series of alcohols, the specimens were embedded in Epon 812. Semithin sections were stained toluidin blue and ultrathin sections were stained with uranyl acetate and lead citrate. Ultrathin sections were studied with a Zeiss Elektronenmikroskop EM 9A and a Siemens Elmiskop 1A.

*Animal Experiments.* Extracts of complete hair-spray, which had been used by one of our patients for 10 years, were dissolved in sterile isotonic saline and injected subcutaneously between the

shoulder-blades of adult mice, rats and guinea pigs according to the method described by Bergmann et al. (1958). Parallel polyvinyl pyrrolidone and polyvinyl acetate in saline were injected under the same conditions in mice, rats and guinea pigs. Saline alone was injected into control animals. The animals were killed after 4, 10 and 30 days and the subcutaneous tissue at the site of the injection, and parts of the liver, spleen and kidney were prepared for electron microscopy.

*Cell Cultures.* Mononuclear cell fractions were prepared from 20 ml of heparinized peripheral blood obtained from a healthy volunteer as described by Zucker-Franklin et al. (1978). The blood was centrifuged at 250 × g for 7 min after which the buffy coat layers were harvested by Ficoll-Paque gradient centrifugation. The band containing monocytes and lymphocytes was washed once in Hank's solution and placed on glass slides in Petri dishes. The cells were cultured in a medium described by Cohn and Benson (1965). The medium was changed after 3 h, in the course of which the lymphocytes were washed away while the monocytes adhared to the glass slides. At that time hair-spray extract of PVP/PVA were added to the culture. After 24 h the glass slides with adherent monocytes were covered with Bouin's solution for 10 min and the PAS-reaction was performed.

## Results

*Histological Findings.* In both patients similar alterations of the lung parenchyma were found. There is active chronic inflammation with numerous macrophagocytic granulomas containing multinucleated giant cells of the foreign body type and few lymphocytes and granulocytes (Fig. 1a). These granulomas are situated in the interstices of the alveolar septa and in the peribronchiolar tissue. No plasma cells, mast cells, eosinophils, giant cells of the Langerhans type or areas of necrosis are detectable. The lung parenchyma shows interstitial oedema and disseminated foci of fibrosis. The pneumocytes are often swollen and desquamated, there is marked "cuboidal transformation" of alveolar lining cells (Fig. 2a). The alveolar lumina contain desquamated lining cells, numerous large macrophages, "foam cells", multinucleated giant cells and some granulocytes and lymphocytes (Fig. 2). The macrophages and giant cells in the interstitial tissues and in the alveolar lumina show remarkable PAS-positive cytoplasmatic inclusions (Fig. 1a), which are seen to be granular in the semithin sections (Fig. 2a). The diagnosis in both cases is *granulomatous fibrosing alveolitis.*

*Electron Microscopic Findings.* The interstitial and intraalveolar macrophages show all degrees of maturation; most cells appear to be highly stimulated with euchromatic nuclei, abundant cytoplasm with many primary and secondary lysosomes and prominent ruffles and pseudopodia of the outer cell membrane (Figs. 2b, 3–6). A lamellar material is often attached to the cell membrane of the alveolar macrophages (Fig. 3a) and this material can also be seen in secondary lysosomes (Fig. 3a, b). Mononuclear phagocytes ingesting larger particles fuse to form multinucleated giant cells (Fig. 4a, b). During this process deep interdigitations of the cellular membranes of the phagocytic cells can be seen, which interlock in zipper-like arrays (Fig. 4a, b). It is noteworthy that often no limiting membrane can be detected around the ingested material (Fig. 4b). Rather, special differentiation of the cytoplasm surrounding the ingested material is observed, with the formation of a clear, finely granular,

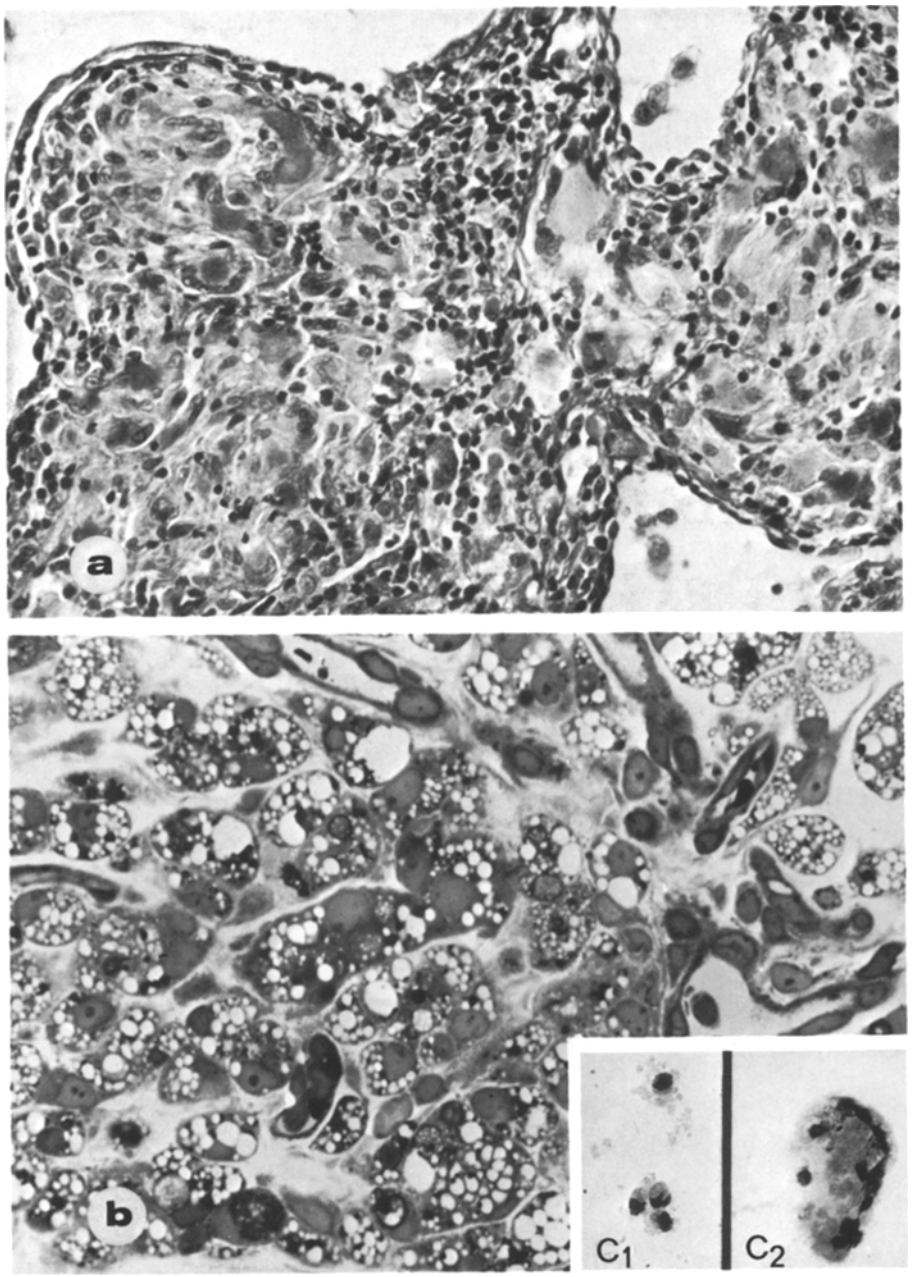


**Fig. 1a–c.** Hair-spray lung, 48 years, ♀. **a** Chronic inflammation with macrophage granulomas and multinucleated giant cells of the foreign body type. PAS-positive cytoplasmic inclusions in macrophages and giant cells. PAS, ×300. **b** Guinea pig, 4 days after subcutaneous injection of PVP/PVA. Subcutaneous tissue with foreign body response. Semithin, toluidin blue, ×480. **c** Cell cultures of human blood monocytes: $c_1$ control after 24 h incubation with small disseminated and immature monocytes. $c_2$ 21 h after exposure to PVP/PVA: Aggregation and maturation of monocytes with increased cytoplasm containing PAS-postive material and development of multinucleated giant cells. PAS, ×250

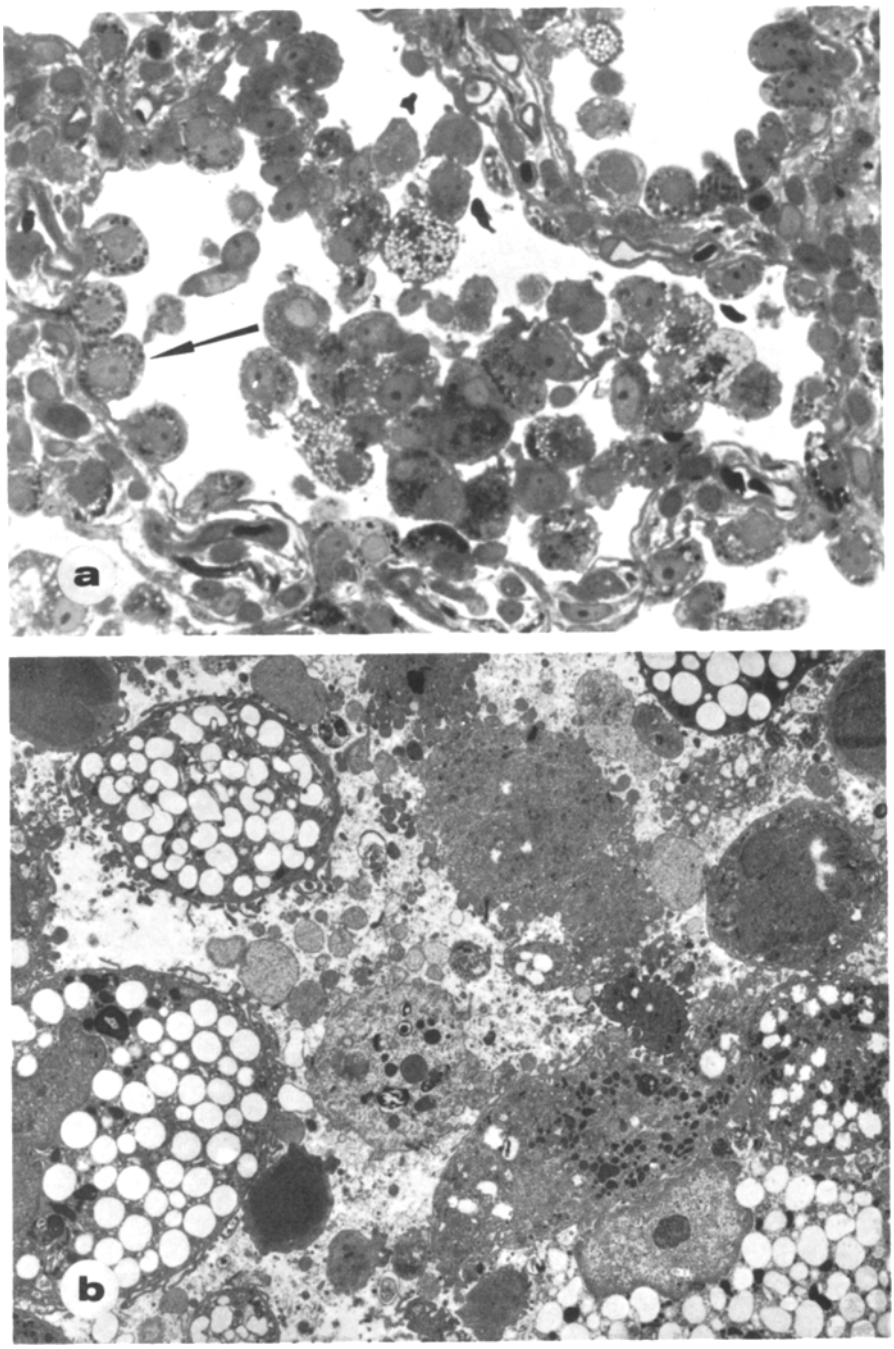


**Fig. 2. a** Hair-spray lung, 48 years, ♀. Acute alveolitis with desquamated alveolar lining cells, numerous alveolar macrophages with granular inclusions and foam cells. Reactive proliferation of cuboidal type II pneumocytes (*arrow*). Semithin, toluidin blue, ×480. **b** Hair-spray lung, 60 years, ♂. Intra alveolar foam cells and macrophages. ×2,500

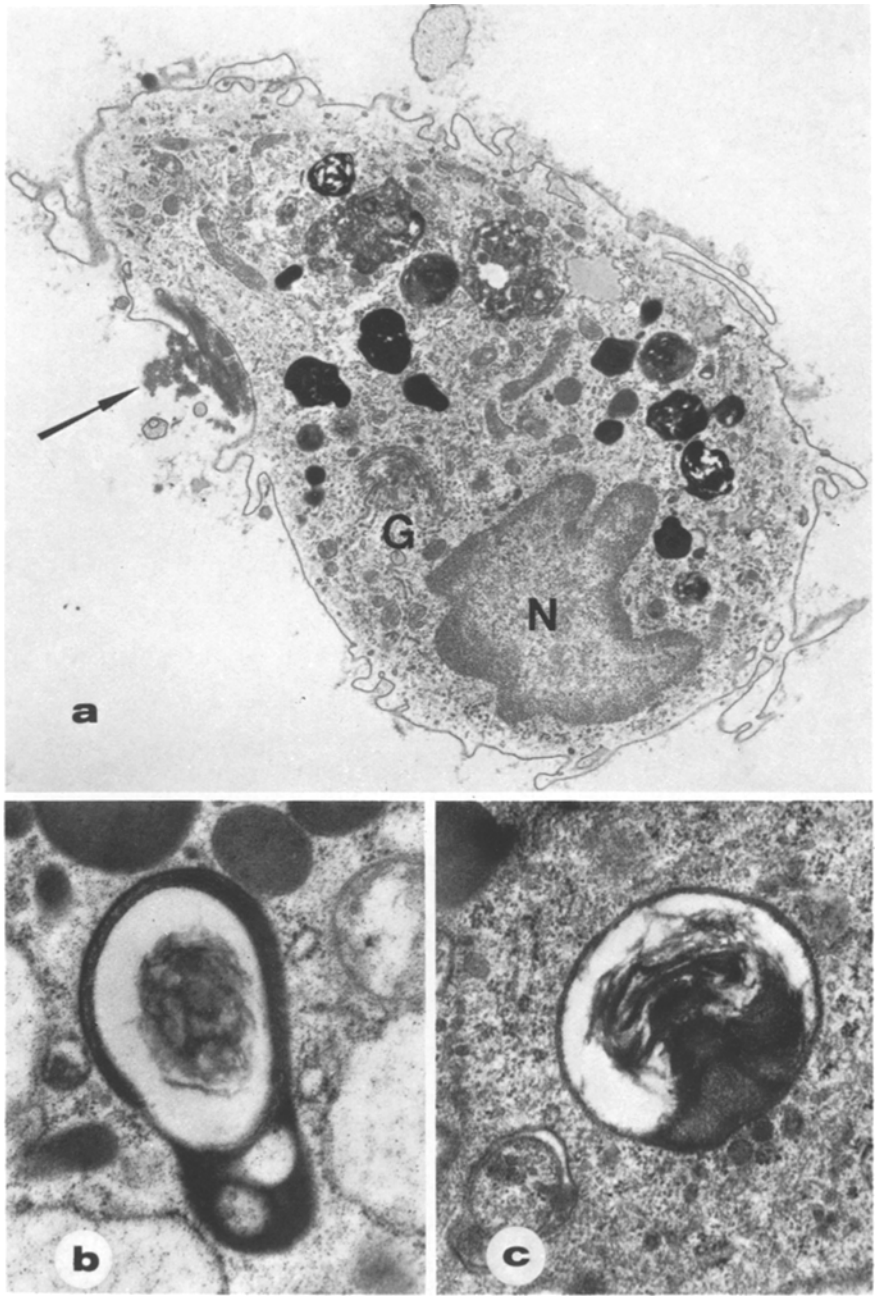


**Fig. 3.** **a** Hair-spray lung, 60 years, ♂. Stimulated mature alveolar macrophage with ruffeled cell membrane, large Golgi-field (*G*), euchromatic nucleus (*N*), and primary and secondary lysosomes. Lamellar material attached to the cell membrane (*arrow*), which is also found in the secondary lysosomes. ×6,100. **b** and **c**. Hair-spray lung, 48 years, ♀. Secondary lysosomes of alvelolar macrophages with a lamellar material. **b** ×27,300. **c** ×25,200

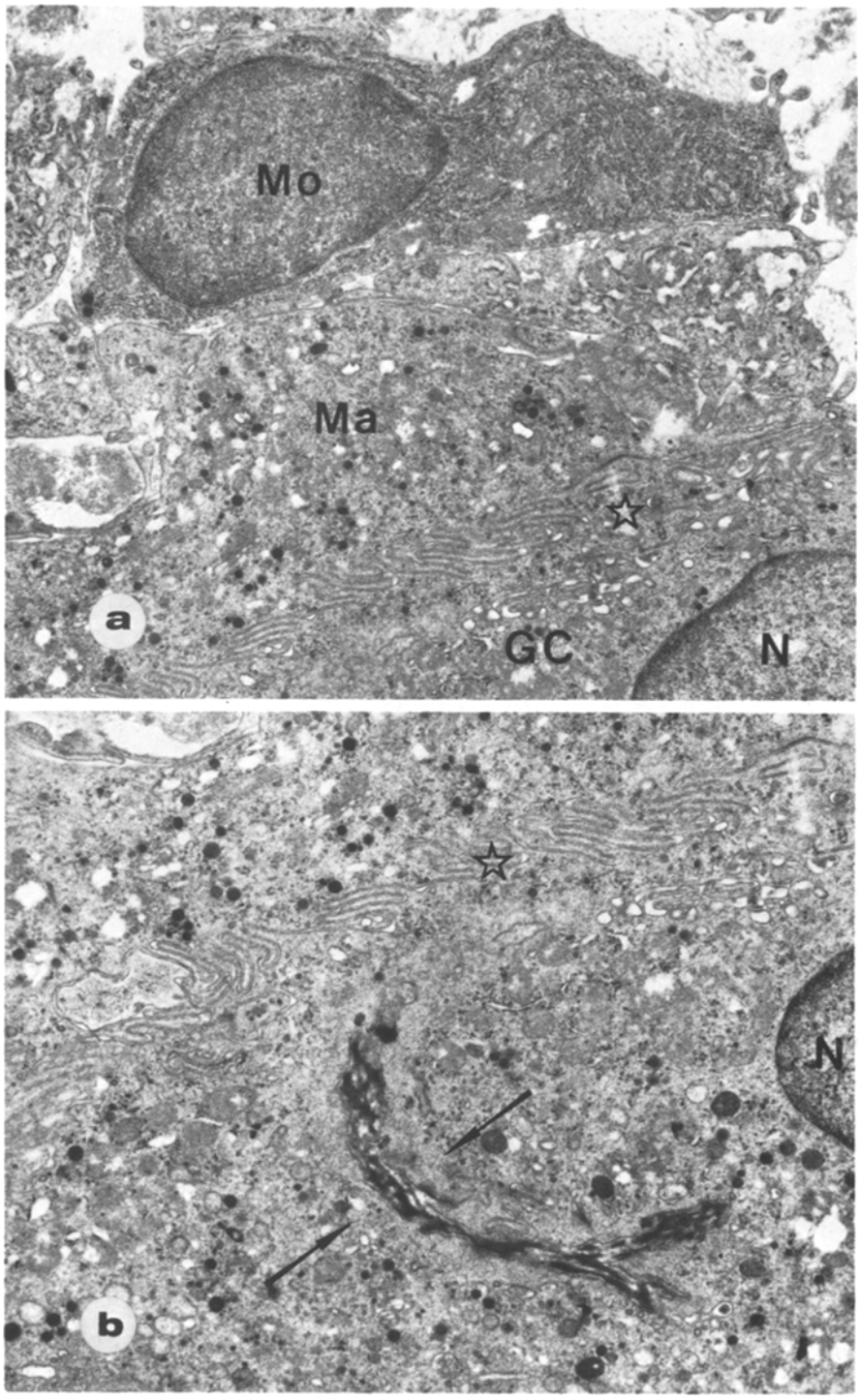


**Fig. 4a and b.** Hair-spray lung, 48 years, ♀. Multinucleated giant cell. **a** A monocyte (*Mo*) is attached to a macrophage (*Ma*). The cell membrane of the latter interlocks in a zipper-like pattern by deep and closely apposed interdigitations (*) with the cell membrane of a giant cell (*GC*). Nucleus of the giant cell (*N*). ×7,100. **b** Giant cell (*N*) with intimately associated macrophage. The cell membrane of both cells interlock in a zipper-like array (*). Lamellar material in the giant cell, which is enclosed by fusion of the giant cell and the associated macrophage. Note the clear zone surrounding the ingested material (*arrows*). No membrane, i.e. phagocytic vacuole, can be seen. ×8,500

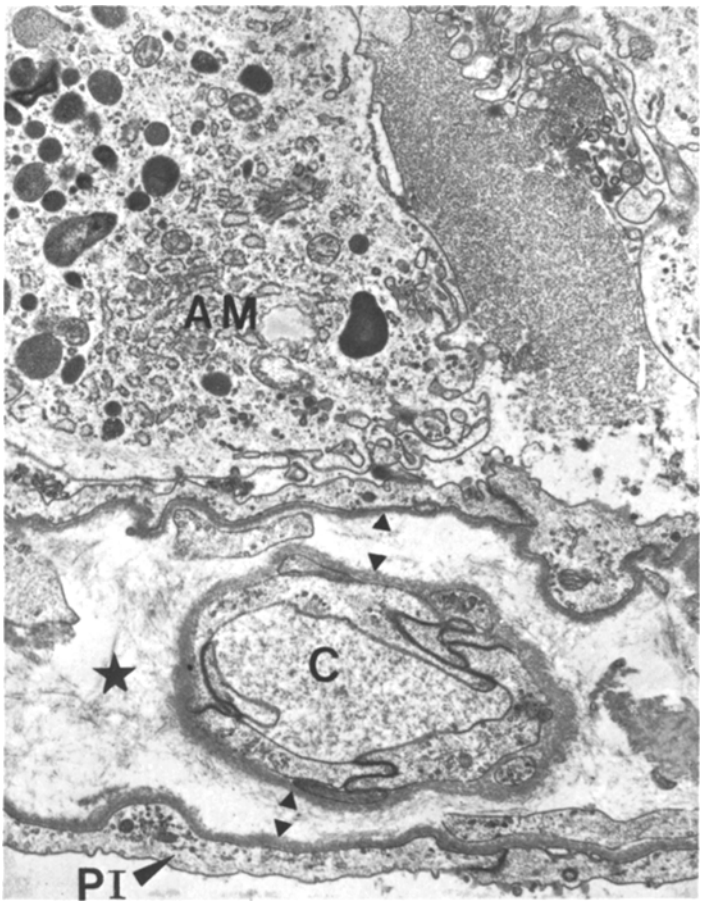


**Fig. 5.** Hair-spray lung, 60 years, ♂. Remarkable interstitial edema in the interalveolar septum (*) with separation of the common basement membrane of the type I pneumocyte (*PI*) and the capillary endothelial cell (*C*). Considerably increased alveolar-capillary diffusion distance of the respiratory membrane (*arrow heads*). Alveolar macrophage (*AM*). ×7,500

homogenous zone free of organelles (Fig. 4b). The cytoplasm of numerous alveolar macrophages is filled with "empty" vacuoles ("foam cells", Fig. 2b).

The interalveolar septa and the respiratory membrane are widened by marked oedema which causes an increase in the capillar-alveolar diffusion distance (Figs. 5, 6b). The oedema separates the formerly fused common basement membrane of the alveolar lining cells (pneumocytes type I) and the capillary endothelial cells (Figs. 5, 6b). At other sites more severe damage of the tissue is seen with rupture of the respiratory membrane and necrosis of pneumocytes (Fig. 6a). The necrotic type I pneumocytes are replaced by proliferating type II pneumocytes ("cuboidal transformation" of the alveolar lining cells, Fig. 6b). In the interstices of the interalveolar septa, fibrosis takes place.

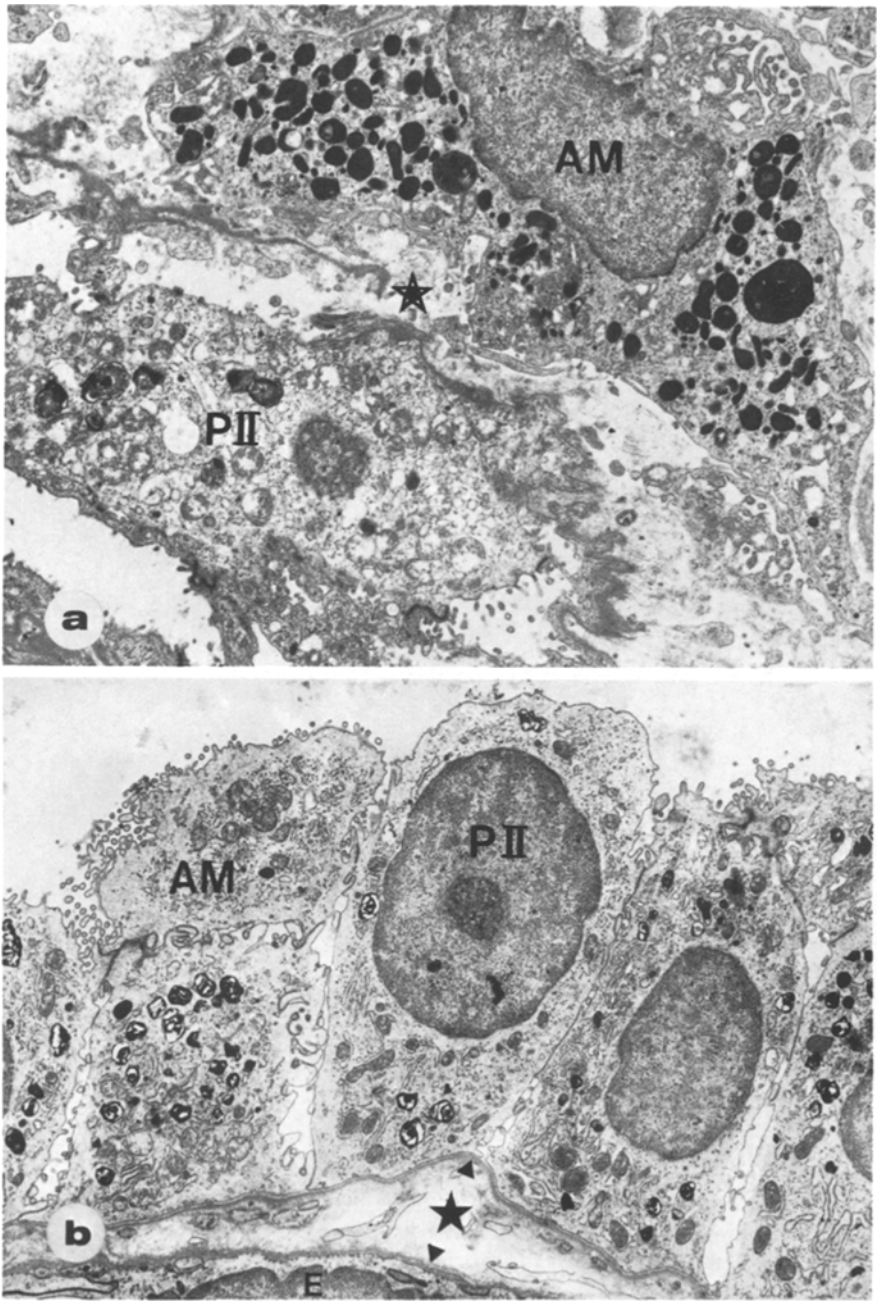


**Fig. 6. a** Hair-spray lung, 48 years, ♀. Alveolitis with marked alterations of the structural integrity. Rupture of the basement membrane of the respiratory membrane (*) with attached mature alveolar macrophage (*AM*). Necrobiotic desquamating type II pneumocyte (*P II*). ×4,800. **b** Hair-spray lung, 60 years ♂. Regenerative proliferation of type II pneumocyte (*P II*) ("cuboidal transformation" of the alveolar lining cells) replacing the desquamated type I pneumocytes. Marked interstitial oedema of the respiratory membrane (*) with distension of the epithelial and endothelial basement membrane (*arrow heads*). Capillary endothelium (*E*). Portion of an alveolar macrophage (*AM*) closely apposed to a type II pneumocyte. ×3,000

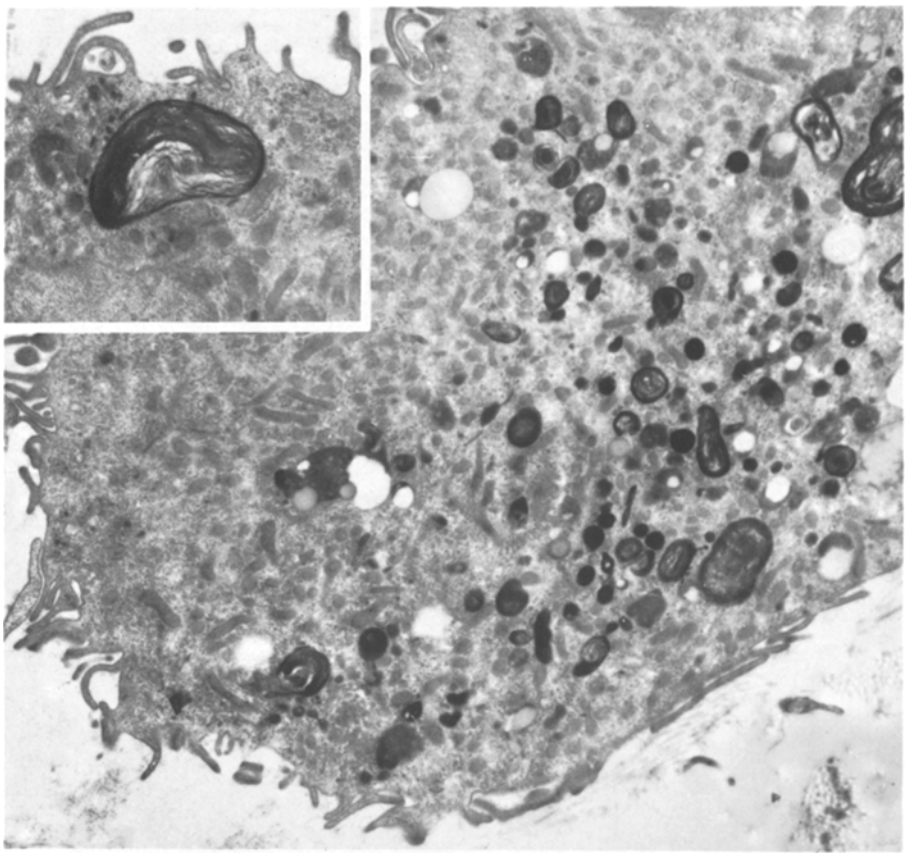

**Fig. 7.** Animal experiment, guinea pig, 10 days after subcutaneous injection of PVP/PVA. Stimulated macrophage with abundant cell organelles and secondary lysosomes containing a lamellar material. ×5,300. Inset: Enlarged view of a secondary lysosome with lamellar material. ×9,100

*Animal Experiments.* In all animals injected with complete hair-spray extracts and with PVP/PVA alone a marked subcutaneous foreign body reaction with granulomas was seen. This reaction could be detected between 4 and 10 days and vanished after 30 days. Control animals showed no changes.

The inflammatory infiltrate is characterized by many monocytes, large macrophages and multinucleated giant cells with PAS-positive inclusions. Numerous "foam cells" occur (Fig. 1 b). Electron-microscopically lamellar lysosomal inclusions can be seen in macrophages and giant cells (Fig. 7) after injection of both the complete hair-spray and of PVP/PVA. The lysosomal inclusions are similar in type to those found in the alveolar macrophages of the patients. Four weeks after the injection of the whole hair-spray extract of of PVP/PVA the Kupffer cells of the liver and the macrophages of the spleen contained PAS-positive cytoplasmatic inclusions.

*Cell Cultures.* The monocytes in the control cultures are disseminated and display small cytoplasmatic rims with bean-like nuclei (Fig. 1 $c_1$). After the addition

of complete hair-spray extract or of PVP/PVA to the liquid culture medium similar changes happen to the monocytes in the different cultures. Their cytoplasm increases in amount and the nuclei enlarge, PAS-positive material is stored in their cytoplasm, and they forme aggregates. Some aggregated cells in these cultures fuse to form multinucleated giant cells, which also contain PAS-positive material (Fig. $1c_2$).

## Discussion

The findings in these patients are in accordance with most of the previously established criteria for pulmonary thesaurosis due to hair-spray exposure. The patients were exposed regularly to hair-spray in considerable quantity for at least 10 years prior to the development of their lung disease. Also, as in the majority of previous cases, avoidance of hair-spray was followed by a significant relief of symptoms within half a year (Gebbers et al., 1979).

The histological findings in the open lung biopsies, which show similar alterations in both patients, are strongly suggestive of an exogeneous thesaurosis of the lung. The histopathology is characterized by a granulomatous fibrosing alveolitis with a striking foreign body reaction. In particular, there are PAS-positive cytoplasmatic inclusions of interstitial and intra-alveolar macrophages and multinucleated giant cells. These observations are in agreement with those made by other authors (Bergmann et al., 1958; 1962; Nevins et al., 1965; Ripe et al., 1969; Bergmann, 1973; Erhardt et al., 1973; Stringer et al., 1977).

The possibility of lung disease associated with hair-spray use has been discussed repeatedly and more than 30 cases of suspected "hair-spray lung" have been reported (review: Gebbers et al., 1979). The use of hair-sprays close to the nose and the mouth permits the inhalation of large quantities of the spray and may induce pulmonary disease.

The present investigations further support the assumption that hair-spray is a potentially injurious agent for the lung. The chemical nature of the active ingredient (or ingredients) in hair-sprays is not known. They contain polyvinyl pyrrolidone (PVP), polyvinyl acetate (PVA), alcoholic solutions of shellac, and numerous other substances, including ethylcellulose, carboxymethyl cellulose, acryl resins, alcohols, perfumes and fluorochlorohydrocarbon as propellant (Voigt and Bornschein, 1973).

Holographic particle analysis of sprays of various compositions demonstrates that the common cosmetic sprays produce a large amount of particles with a diameter around 5 μm (Groß and Peter, 1973). In a study of the distribution and the density of these particles in a model of the human respiratory system it has been established that these particles with a diameter of 5 μm reach the alveoli of the lung by inspiration (Groß and Gulden, 1974). Draize et al. (1959) showed that 65% of the sprayed particles are small enough to reach pulmonary alveoli. By analysis of room air they also determined that after a ten-second spray of a PVP type of preparation in an enclosed small space, 130 μg of the dry residue is available for inhalation in the next five minutes. Considering that the patients described in this report have used hair-spray several times a day, the possibility of inhalation of significant amounts of this material in the course of months and years seems to be well established. None of the contents of hair-sprays are readily identifiable in animal or human tissues by histochemical staining techniques (Bergmann, 1973; Erhardt et al., 1973).

Polyvinyl pyrrolidone (PVP) and the polyvinyl acetate (PVA) are of interest as potentially harmfull ingredients because of their high content in the spray and because they are capable of inducing granuloma formation. They are macromolecules, resulting from the polymerization of N-vinyl-pyrrolidone in variable sizes from 20–700 thousand molecular weight. Because of its chemical inertness PVP/PVA cannot be metabolized by the mammalian body and after intra-venous administration in man, has been shown to result in granulomatous lesions with giant cells and epitheloid cells in several organs (Brass, 1952; Fresen and Weese, 1952; Hüsselmann, 1952; Jeckeln, 1952; Leder and Lennert, 1972).

The mode of the intracellular accumulation of PVP/PVA is variable, possibly depending on the interval between exposure and examination and on the molecular size. Three different modes of macrophagocytic storage have been described (Leder and Lennert, 1972), which are consistent with our findings in hair-spray lungs: 1. foam-like material in large cells with peripheral nuclei ("foam cells"); 2. big opaque PAS-positive coacervates, grey-blue in the HE-stain (Hüsselmann, 1952) and 3. small, denser particles with a brown colour. It is assumed that the original achromatic PVP/PVA becomes brown within the organism after some time and aquires the characteristics of a pigment (Leder and Lennert, 1972).

Jeckeln (1952) assumed that PVP possesses antigenic properties, which were demonstrated by Andersson (1969). It is of interest that the immune response to PVP is T-cell independent as it is unaffected (Andersson and Blomgren, 1971) or enhanced (Kerbel and Eidinger, 1972) by procedures which deplete the T-cell population of the host. Although the exact role of T-cells in the regulation of this response is controversial (Price, 1978), it is evident that PVP can elicit IgM responses in the absence of T-cells. However, significant IgG production and immunological memory do not follow in response to PVP (Kerbel and Eidinger, 1972). It is remarkable, that tolerance can be induced with small quantities of PVP (Andersson, 1969).

In the two cases presented neither immuno-electrophoresis nor other immunologic-serological examination suggested involvement of the immune system. Histological findings do not correspond to the findings in a local immune response in the sense of an extrinsic allergic alveolitis (Seal, 1975; Wettengel, 1975).

Our findings indicate a response of the local mononuclear phagocyte system of the lung and are strongly suggestive of an overwhelming foreign body response to inhaled material. The granulomas, which are found in the lungs of our patients are compact (organized) collections of mature mononuclear phagocytes (Spector, 1974; Adams, 1976). They constitute a distinct response of the *m*ononuclear *p*hagocyte *s*ystem (MPS; van Furth et al., 1972), which is extensively developed in the lung.

Mononuclear phagocytes, unlike neutrophilic leukocytes, are not fully mature when released from the bone marrow (Gordon, 1977). Under the influence of yet unidentified stimuli, they mature into macrophages in the tissues (Adams, 1976; Gordon, 1977). This process can also be seen in the foreign body response in the hair-spray lung. Small, immature mononuclear phagocytes develop greater size and acquire large euchromatic nuclei, abundant cytoplasm, numerous Golgi profiles and lysosomes, stacks of smooth and rough endoplasmatic reticulum, and many mitochondria (Fig. 4).

It is established that morphological maturation of the mononuclear phagocytes has associated biochemical changes, increases in acid hydrolases are particularly impressive ranging up to fiftyfold (Cohn, 1968). The importance of the mononuclear phagocyte maturation lies in the direct relation to function; as the cells mature they attain increased functional capacity.

The accumulation and maturation of mononuclear phagocytes in granulomas and the marked alveolar macrophage reaction in hair-spray lung may be caused by functional limitation of the local MPS. Large quantities of hair-spray inhaled in al relatively short time may result in *a quantitative overstrain of the MPS.* It is known that a high local concentration of a given agent is more likely to evoke granulomas, which persist as long as the inciting agent (Spector, 1974). The foam cells which are of monocytic origin (Zucker-Franklin et al., 1978) may be a degenerative form of alveolar macrophage and would then be the morphological expression of functional "exhaustion" (Otto, 1978).

The formation of giant cells may be interpreted as *an additional qualitative overstrain of the MPS's capacity* indicating that larger particles have to be eliminated. In hair-spray lung these particles are presumably polymers. It can be demonstrated ultrastructurally that the giant cells in lung and in animal tissues arise from fusion of mature mononuclear phagocytes (Fig. 4) during the process of ingestion of large particles. This has been described under similar conditions by Spector (1974) and Burkhardt and Gebbers (1977). Through gigantophagocytosis (Burkhardt and Gebbers, 1977) the cells are able to ingest and digest large particles (Fig. 4).

This heavy quantitative-qualitative demand and possible functional exhaustion of the local MPS could lead to a partical blockade of the system. The resulting functional reduction may cause an increased susceptibility to infectious diseases of the lung.

The alveolitis and fibrosis may be viewed as the consequence of a permanent foreign body response to hair-spray material, leading to overstimulation of the local MPS. Although the fundamental physiological function of macrophages and granulomas is to rid the host of unwanted substances or to induce immunity, tissue damage and necrosis accompanies many granulomas (Dannenberg, 1968; Davies and Allison, 1975). A number of substances (endotoxins, certain bacteria, and antigen-antibody complexes) are not toxic by themselves but cause macrophages to release large quantities of acid hydrolases into the extracellular compartment (Davies and Allison, 1976). Lastly, the potent neutral proteases, elastases, and collagenases of mature macrophages are secreted extracellularly after complex triggering by ingested particles (Gordon, 1977; Gordon et al., 1975; Kapp, 1978). These mechanisms happen in alveolar macrophages (Horwitz and Crystal, 1976; Gordon, 1977; White et al., 1977) and may lead to an alteration of the structural integrity of the lung parenchyma (Fig. 7a). This causes further inflammatory reaction resulting in an alveolitis (Fig. 2) which is viewed as a nonspecific reaction of the lung to a multitude of injurious agents (Katzenstein et al., 1976; Gebbers et al., 1977). The early changes of this process are visible by electron microscopy (Figs. 5, 6). Increased damage of the lung parenchyma with necrosis of type I pneumocytes is associated with a greater proliferative response of type II pneumocytes, which are the primary source of repair (Fig. 6b) as established by sterologic examinations with electron microscopy of tissue

sections from rats exposed to $NO_2$ (Evans et al., 1973, 1978; Burkhardt et al., 1977; Gebbers et al., 1977). Evans and his colleages have suggested that the proliferative response of type II pneumocytes can be used as an indirect means to quantify acute damage of the alveolar epithelium (Evans et al., 1978). Activated macrophages release a factor, which produces fibroblast-stimulating activity (Leibovich and Ross, 1976), which may also help to explain the disseminated foci of fibrosis in hair-spray lung.

The histological and electron microscopical observations on the alterations in the lungs of our patients and in the animals injected with hair-spray show a macrophage and giant cell response of a similar nature. Both substances, the complete hair-spray extract and PVP/PVA, produce a striking subcutaneous foreign body reaction. Granulomata with macrophages, multinucleated giant cells aggregating PAS-positive material in their cytoplasm and foam cells are developing (Figs. 1 b, 7). The development of the granulomatous inflammation resembles that found by other authors (Adams, 1976). It can be demonstrated that the secondary lysosomes of the macrophages and giant cells in the animal experiment contain identical inclusions of a lamellar pattern to those visible in the lung macrophages and in the giant cells of our patients (compare Fig. 3 with Fig. 7). There is strong evidence that the phagocytosed material is PVP/PVA. The findings of our animal experiments are in accordance to the results of other authors (Bergman et al., 1958, 1962). Lowsmann et al. (1966) in experiments with rats by PVP inhalation, found that on initial exposure to PVP the lungs were PAS-negative. However, in about four months from the last exposure PVP had apparently induced a PAS-positive reaction. This is thought to be due to some unknown substances (proteins?) coating the PVP-particles extracellularly or within macrophages and giving rise to coacervates (Hüsselmann, 1952), which then stain with PAS.

Observations on the cell cultures of human blood monocytes establish that the complete hair-spray extract and PVP/PVA alone are ingested by monocytes, and that this is associated with maturation of these cells which increase in size and show PAS-positive material in their cytoplasm (Fig. 1 c). The maturation of monocytes promotes their aggregation (Adams, 1976) which is visible in the cell cultures. These aggregated cells give rise to multinucleated giant cells with PAS-positive inclusions (Fig. $1c_2$).

Observations on the experimental hair-spray granulomas and on the monocyte cell cultures amek it highly probable that hair-spray is the cause of the granulomatous alveolitis in our two patients. Apparently, a quantitative and qualitative overload of the pulmonary macrophage system causes a chronic inflammatory process, leading to severe and only partially reversible lung damage (hair-spray lung).

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