are many branches, which arise from the main stem at an angle of 50°. Leaves taste very bitter; the radical leaves form a rosette, and they are slightly petiolated, stiff and crisp, lyrate-pinnatisect, slightly lobed, with a serrate-dentate margin, sometimes sinuate, and hispid with white hairs. The lamina is uneven and dark green, and the terminal lobe is very large, forming about three quarters of the area of the leaf. The other lobes are smaller, and there are three or four of them. The main lobe is sparsely dotted with irregular protuberances on the upper surface. The cauline leaves are lyrate with a serrate-dentate margin, and are less hairy, while the upper leaves are lanceolate, entire and smooth. The inflorescence is a corymbose raceme. The flowers are bright yellow with petals separated by spaces. The anthers have introse dehiscence. Pods are smooth and form an angle of 40°–50° with the main axis, are 5 cm long, with a beak of 7·5 mm, and are torulose. There are many seeds, seventeen to twenty-eight seeds/pod; they are brown with reticulation on the surface.

The amphidiploid looks suitable for direct cultivation. It has a narrow flowering range, gives a high yield, is resistant to aphids in field conditions and bears 30 per cent oil in the seed. It has been named *Brassica amarifolia* because the leaves taste very bitter.

We thank M. S. Swaminathan, head of the Division of Botany, Indian Agricultural Research Institute, New Delhi, for advice.

ANUBHAVA NARAIN
SHYAM PRAKASH

Division of Botany,
Indian Agricultural Research Institute,
New Delhi 12, India.

[1] Olsson, G., *Hereditas*, **46**, 171 (1960).
[2] Srinivaschar, D., *Indian J. Genet.*, **25**, 71 (1965).
[3] Mohammad, A., and Sikka, S. M., *Curr. Sci.*, **9**, 280 (1940).
[4] Srinivaschar, D., *Curr. Sci.*, **33**, 497 (1964).
[5] Olsson, G., *Hereditas*, **40**, 398 (1954).
[6] Singh, S. R., *Indian Oilseeds J.*, **9**, 215 (1965).

PATHOLOGY

Atypical Reaction to Inhaled Silica

THE classical reaction of the lung to the deposition of airborne silica is the formation of discrete nodules, which in man exhibit a characteristic disposition of dust and connective tissue fibres[1,2]. Although the experimental lesion does not reproduce all the human features[3], I have confirmed that it remains focal and discrete[4,5]. In recent experiments on the disposal of inhaled particles within the lung parenchyma, specific pathogen-free (SPF) rats have been used in an attempt to eliminate the complication of pulmonary infection which not infrequently affects standard laboratory rats. The response of SPF rats differed greatly from that of the standard ones used in the earlier studies.

Some SPF rats inhaled Minusil quartz (particle size $< 5\mu$) and others Dörentrup quartz (particle size $< 3\mu$) for 600 and 1,200 h over periods of 6 and 12 weeks respectively. In the case of Minusil the mean atmospheric concentration of dust was approximately 40 mg/m³ and with Dörentrup 30 mg/m³. Rats have so far survived for up to a year after exposure. Each pair of lungs contained a mean of 20 mg Minusil or 11 mg Dörentrup quartz (courtesy of Dr. A. Critchlow).

Grossly the lungs in both groups were large and collapsed only partially on removal. They showed extensive and irregular yellowish-white consolidation but no typical silicotic nodules. Histologically, fibrosis was limited to a few small areas of irregular outline and tended to be intra-alveolar; it was usually associated with a peripheral accumulation of foamy histiocytes, which often contained periodic acid–Schiff positive (diastase resistant) granules and sometimes sudanophil fat. Fibrosis apparently started as an outgrowth from the walls of alveoli in which foamy macrophages had collected. Most of the remaining alveolar spaces were filled with eosinophilic granular material that also gave a positive periodic acid–Schiff reaction (diastase resistant). In this granular exudate birefringent particles of silica were identified in incinerated preparations. Rat lung thus consolidated bore a distinct resemblance to pulmonary alveolar proteinosis in man[6], a similarity which was increased by the presence in the rat exudate of acicular birefringent crystals, occurring singly or in bundles. As survival increased after dust exposure, the granular material tended to diminish in amount, scattered macrophages and polymorphs appeared, and it may have been removed via the bronchial tree. The alveolar walls for the most part persisted apparently unchanged, but occasionally a further unusual feature was seen in the form of localized areas of epithelialization of alveolar walls resembling adenomatosis, the lining cells being cuboidal, columnar or ciliated. There was no evidence of pulmonary infection.

The nature of this exceptional response to quartz has yet to be elucidated, but the alveolar exudate seems to be derived from disintegrated cells, possibly macrophages, as in alveolar proteinosis[6]. The failure to develop typical fibrotic nodules in the presence of silica is striking, as is the feeble aggregation of the dust into foci and its prolonged retention in large areas of the parenchyma. The fibrogenicity of the two samples of quartz was established by intraperitoneal injection into standard and SPF rats. The widespread deposition and retention of a relatively large amount of quartz in a period of 6 to 12 weeks might lead to necrosis of macrophages as quickly as they are produced and in some way interfere with fibrogenesis. It is not yet possible to say whether the atypical reaction to quartz is related to the use of SPF rats.

A. G. HEPPLESTON

Department of Pathology,
University of Newcastle upon Tyne.

[1] Simson, F. W., *J. Path. Bact.*, **40**, 37 (1935).
[2] Belt, T. H., *J. Path. Bact.*, **49**, 39 (1939).
[3] Belt, T. H., Ferris, A. A., and King, E. J., *J. Path. Bact.*, **51**. 263 (1940).
[4] Heppleston, A. G., *Amer. J. Path.*, **40**, 493 (1962).
[5] Heppleston, A. G., *Arch. Environ. Hlth.*, **7**, 548 (1963).
[6] Rosen, S. H., Castleman, B., and Liebow. A. A., *New England J. Med.*, **258**, 1123 (1958).

Effect of Chlordiazepoxide on Eosinopenia of Stress in Rabbits

CHLORDIAZEPOXIDE, a new psychotherapeutic drug, chemically unrelated to either phenothiazine derivatives or indole–alkaloid derivatives, has been reported to show a number of pharmacological activities[1]. A possible hypothalamic activity of chlordiazepoxide has been reported[2]. We have investigated the effect of this drug on a physiological activity controlled by the hypothalamus, that is, eosinopenia after emotional stress. The emotional stress was applied to rabbits weighing 1·5–2 kg by the method of O'Conor and Verney as adopted by Colfer *et al.*[3]. Blood samples were taken from the ear veins of rabbits once before and again 3·5 h after the emotional stress (electric shock) was administered. The strength of the stimulus given by an electronic stimulator was such that it produced signs of fright in the animal. A freshly prepared solution of chlordiazepoxide (50 mg/kg) was given intraperitoneally 0·5 h before the electric shock and by this time the animal was showing the tranquillizing effect of the drug. Electrical stimulus was not repeated for 2 days. After pretreatment with chlordiazepoxide the rabbits still showed signs of fright after a similar electric shock. The experiments were carried out on three rabbits, each animal acting as its own control. Average means of blood counts are as follows. Before administration of

© 1967 Nature Publishing Group