(Figure 3). Large number of acute inflammatory cells were present in the lamina propria and submucosa. Muscular layers presented normal appearance. Vascular congestion was severe. No proliferative activity of the epithelial mucosa was observed.

Benzanthrone caused significant decrease of ascorbic acid level in blood and other body organs[6]. PANDYA[7] noted the presence of benzanthrone or its possible metabolites in guinea-pig urine after i.p. administration of benzanthrone. Lowered body ascorbic acid level combined with the effect of benzanthrone or its possible metabolites excreted in the urine may be responsible for epithelial damage. Mucosal lesion was not discernible on dermal application or oral administration of benzanthrone[8].

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Effect of Lindane[1] on the Skin of Albino Rats

Lindane is being used extensively as dusts, emulsions and vapours to control insects under different conditions[2,3] Occupational poisoning among workers engaged in the synthesis, formulation and application of lindane is also reported by different workers[4–6]. Literature on the histopathological changes in the skin of individuals repeatedly exposed to lindane is not adequate. Physicochemical factors such as particle size, the period of exposure and the vehicles are known to influence the degree of toxicity. In the light of such variations, dermal application of lindane under tropical conditions, as in this country, merits further study. This report, which is the continuation of our earlier observations[7,8], deals with the histopathological changes in the skin of albino rats after they are exposed directly to the action of lindane.

Materials and Methods. 50 female albino rats of I.T.R.C. stock with an average body weight of 80 g were used in the experiment. The lateroabdominal area measuring approximately 4×4 cm was previously made ready by hair clipping for lindane painting. Lindane (98% purity) was used with propylene glycol (BDH Analar) as the vehicle. 1 ml of the solution which contained 14.4 mg of lindane (this dose is 5 times less than the acute dermal LD_{50} values for the female rats; dermal LD_{50} of lindane for female rats is 900 mg/kg)[9] was slowly transferred on the specified area of the skin with the help of a graduated pipette attached to a vaqupette. 30 animals were treated with lindane daily for a period of 25 days (total number of skin paintings 25). 20 animals of the control group were similarly treated with 1 ml of propylene glycol alone. The animals were killed at intervals of 24 h, 5, 10, 15, 20 and 25 days after treatment. The skin tissue was fixed in Bouin's fluid and paraffin cut sections were stained with haematoxylin and eosin for histopathological observations.

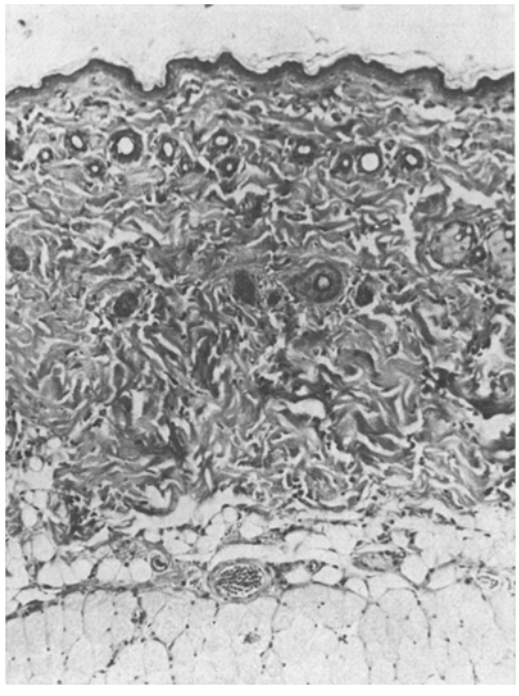

Fig. 1. Normal picture of the skin. Control (25 paintings). ×100.

Results and Discussions. Macroscopic examinations of the skin of experimental animals showed mild dermatitis in 3 animals after 15 paintings. This condition continued up to 25 paintings. In comparison, the skin of control animals did not show any such change.

The normal structure of the skin of the control rat is given in Figure 1. In contrast, microscopic study of the lindane-painted skin revealed various pathological changes. Hyperkeratinization and the migration of inflammatory cells through epidermis was observed in the animals painted for 20 days. Further application of lindane caused formation of abscess, filled in with polymorphonuclear cells. Mild acanthosis was seen with elongated rete ridges and cells of epidermis at some places appeared necrosed (Figure 2). Another skin area of the same animal showed both hyperkeratinization and abscess formation. Exposure of the skin to lindane for a period of 10, 15, 20 and

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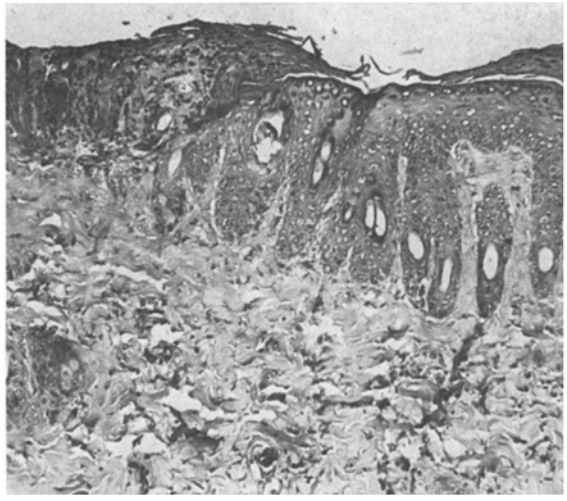

Fig. 2. Intra-epidermal as well as dermal abscess filled in with polymorphonuclear cells. Mild acanthosis seen with elongated rete ridges and cells of epidermis at some places appeared necrosed (25 paintings) × 100.

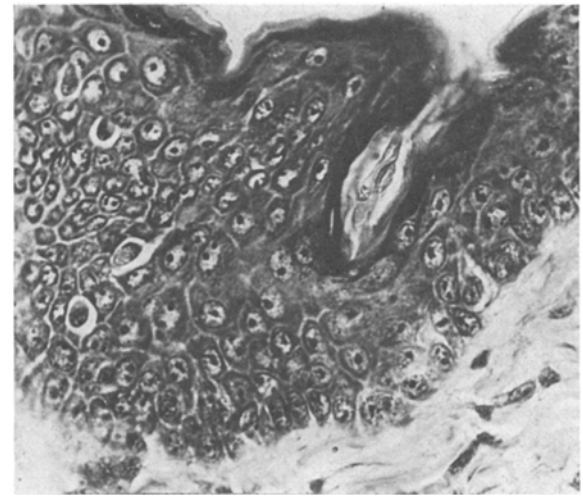

Fig. 3. Vacuolization and multinucleate condition of the cells of malphighian layer (25 paintings). × 400.

25 days has shown vacuolization and multinucleate condition of the cells of malpighian layer (Figure 3).

The danger of absorption of lindane is increased with prolonged handling of powders and emulsions. Higher dermal toxicity of lindane in lipoid solvents is especially noteworthy[2]. Tolerance to progressively increased concentrations of lindane has not been observed. Reports are available both for and against the safety of lindane. The comparatively quick excretion of the compound by the renal system acts as a protective measure against the cumulative effects of the compound, particularly after repeated exposure. Cases of dermatitis and urticaria in humans are also recorded in relation to lindane exposures. These symptoms of allergy are, however, manifested in individuals who are susceptible to this compound[3].

Findings reported here are of interest because of their possible relationship to the cases of occupational poisoning, due to such exposure under tropical conditions. These abnormalities are not reported here as an indication that the picture in man would be similar, but the information gathered from these studies indicates the possible types of skin damage due to insecticides[10].

Zusammenfassung. Langfristig wiederholte Lindan-Applikation ruft bei der Abinoratte histopathologische Hautveränderungen hervor: Hyperkeratinisation, intraepidermale und dermale Abszesse, Infiltration polymorphkerniger Zellen sowie Vakuolisation und Vielkernigkeit in der Malpighi-Schicht.

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[10] Authors are grateful to Dr. S. H. Zaidi, Director of the Centre for his keen interest and constant encouragement. They also express their grateful thanks to Plant Protection Ltd., Research Department, Yalding for sending the gift sample of lindane through ICI (India) Pvt. Ltd., New Delhi. Thanks are also due to Mr. J. Prasad for technical assistance and to Mr. M. Ahmad for photomicrography.

The Effect of Spironolactone on Acute Toxicity and Liver Damage in Mice Induced by Cerium Chloride

Cerium belongs to the light lantanons. The greater part of i.v. administered cerium quickly accumulates in the hepatic tissue. The maximal concentration, about 70% of the administered dose, is obtained a few hours after administration[1]. In large doses, cerium causes fatty infiltration and necrosis in the liver. The structure of hepatic endoplasmic reticulum is changed[2], and an inhibition of drug metabolism in rat liver has been established[3]. Arvela and Kärki[4] found that phenobarbital impairs the inhibitory effect of cerium on drug metabolism. We were interested in finding out if and to what extent the spironolactone pre-treatment, which induces drug metabolism in the liver[5], prevents the acute toxicity and liver damage induced by cerium chloride ($CeCl_3 \cdot 6H_2O$).

Material and methods. Altogether, 134 female NMRI mice were used in the experiment. The initial weight of the animals was about 30 g (ranging between 26–36 g), and the average age was about 3 months. During the experiment, the animals received normal laboratory pellet food and water ad libitum; the temperature varied be-

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