The cone-in-cone structure is seen in the calcareous sandstone/shale succession exposed on the slopes, northeast of Sancha Malla. In this succession it occurs in lenticular concretions and is made up of nested and interfering cones (Figs. 1–3). The individual cone ranges in height from 3 to 6 cm and the apical angle varies from 30° to 50°. The base of the cone is circular to elliptical. A thin clay film commonly separates the individual cones. The axes of the cones are more or less parallel to each other. Their apices point either in the same direction or in opposite direction.

Figs. 1–3. Figs. 1 and 2. Cone-in-cone structure in the Upper Flysch Series, Sancha Malla, Pithoragarh district, U.P. Mark is equal to 1 cm. Fig. 3. Basal section of the cone-in-cone structure, Upper Flysch Series, Sancha Malla, Pithoragarh district, U.P. Mark is equal to 1 cm.

In thin section the cone-in-cone structure is made up of fibrous calcite. The calcite fibers are 0.08 mm in diameter and up to about 1 cm in length.

The Upper Flysch Series of Sancha Malla is a deep sea deposit (Heim and Gansser)². It appears that the cone-in-cone structure was formed during diagenesis of the sediments under some special conditions which favoured precipitation of fibrous calcite.

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TRANSPERATION IN RUST INFECTED LEAVES OF GROUNDNUT

The rust (Puccinia arachidis Sp.) of groundnut (Arachis hypogaea Linn.) is a very destructive parasite and a menace to groundnut crop in many countries. The disease has been reported from almost all groundnut growing areas of India. Rust infection is generally known to be accompanied by an increase in transpiration. The present investigation deals with the effect of rust infection on the rate of transpiration in groundnut.

Groundnut plants (cv. TMV 2) were inoculated with freshly collected urediospores as described earlier. The fourth leaf (counted from the tip), having uniformly distributed uredosori was sampled at different stages of disease development as outlined below:

Stage 1: Four days after inoculation; no visible symptoms on leaves.
Stage 2: Eight days after inoculation; small and circular white flecks on abaxial surface of the leaves.
Stage 3: Ten days after inoculation; the pustules were orange red with mature uredospores; epidermis on the sorus is unruptured.
Stage 4: Twelve days after inoculation; pustules ruptured and brown in colour.
Stage 5: Fifteen days after inoculation; pustules turn dark brown in colour.

The transpiration rate was determined by the method described by Padhi and Aruna Misra for Jatropha. The rate of transpiration at each stage of disease was determined and the results are expressed as water loss in mg per sq. cm of leaf area per hour.

After determining the water loss at every stage, the epidermal peelings of upper and lower surfaces of both healthy and infected leaves were fixed and mounted in Heath's reagent and the perimeter of stomata was calculated from the measurements of length and breadth of the stomata.

In another experiment, the relation between the intensity of the disease and the rate of transpiration was measured. Ten days after inoculation, leaves showing 4, 8, 15, 20, 25 and 30 uredosori per sq. cm area were collected from the infected plants and their rates of transpiration were determined.

Water loss from infected leaves was significantly lower than that from controls during early stages of the disease (Fig. 1). The rate of water loss increased very rapidly from the third stage onwards, when the pustules were well developed and the host epidermis was ruptured. Durbin reported that water loss from rust infected bean plants was significantly lower than that from healthy plants until the time of sporulation.

FIG. 1. Rate of transpiration of rust infected leaves of groundnut at different stages of infection.

when the epidermis was ruptured. From this point water loss from infected plants increased compared to healthy plants. Padhi and Aruna Mista reported similar results in the case of Justicia gendarussa infected by Puccinia thebaeica. The rupture of epidermis by the developing uredia provides breaks through which water can pass. The low rate of water loss during early stages of disease development may be due to the closure of stomatal aperture or decrease in the size of the stomatal openings. From Table I, it is evident that the average perimeter of stomata of both the upper and the lower epidermal surfaces of healthy leaves was greater than those of infected leaves during the first and second stages of disease development. From the third stage onwards, the perimeter of stomata of infected leaves was greater than that of healthy leaves. These results reveal that the changes in water loss as a result of infection may be due to changes in stomatal movements. Such changes in turn are known to be due to changes in stomatal movements. Such changes in turn are known to be due to changes in cell permeability caused by the pathogen or by its metabolites. The decreased transpiration during daylight caused by bean rust infection before the pustules open was also attributed to closure of stomata. Reduction in transpiration may also be due to presence of necrotic spots or dying off of a part of foliage at later stages of disease. The decrease in the rate of transpiration during early stages of disease development may cause a decrease in the activity of glycolic acid oxidase which in turn influences the stomatal movements.

The increase in the rate of transpiration of diseased leaves, after the rupture of pustules is known to be due to changes in stomatal movement besides the exposure of mesophyll cells to the atmosphere. There was a statistically significant increase in the perimeter of stomata of infected plants in the 3rd, 4th and final stages of pustule development. The rate of water loss in diseased leaves increased concomitantly with the

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<td>μ</td>
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<tr>
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* Signifies n.s. at 1% level. ** Signifies r.t. 5% level. NS—Not significant.

increase in the number of pustules per unit area (Fig. 2).

FIG. 2. Rate of transpiration of rust infected leaves of groundnut at different levels of disease intensity.

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INFECTIOUS VARIEGATION—A NEW VIRUS DISEASE OF COTTON IN INDIA

The survey, carried out in different localities of Madras, showed an infectious variegation on *Gossypium hirsutum* cv. Laxmi. MCU-5, CBS-136, Acala glandless, H-4 [G-67 (*G. hirsutum*) × American Nectarless (*G. hirsutum*)], Varlaxmi (SB-29 (*G. barbadense*) × Laxmi (*G. hirsutum*)) and Buri Nectarless with an incidence of 1 to 5%. The disease was characterized by the symptoms of irregular or sectorial patches of yellow or white discoloration reminiscent of chimeric breakdown on the leaves of *G. hirsutum* cultivars. The yellowish white coloured patches often turned pinkish and necrotic with age and the leaves dried and consequently abscessed. No stunting or other ill-effects of the disease were evident on cotton plants.

For graft transmission, the buds of field infected plants were taken from Laxmi cultivar and were grafted on healthy Laxmi cotton seedlings grown from healthy seeds in earthen pots containing steam sterilized compost and soil (1:1 proportion) mixture under insect-free glass-house. The characteristic symptoms of infectious variegation were evident on Laxmi budlings after 8 weeks of bud inoculations thereby indicating the viral nature of the disease.

Since this disease resembled cotton yellow mosaic caused by *Abutilon* infectious variegation virus in symptomatology, Costa1, it was attempted to transmit it by white fly insect (*Bemisia tabaci* Gen.) naturally occurring on cotton. White-flies collected from the field were enclosed on diseased plants of *G. hirsutum* cv. Laxmi with the help of polyethylene tubes, 9" × 3" (Muniyappa et al.5). The white-flies were given an acquisition feeding period of two days and then they were enclosed on healthy 7 day old seedlings of cotton and *Abutilon indicum* Sweet. for two days. Five white-flies were used per test plant. All the 20 plants inoculated took infection. The symptoms appeared in two weeks after inoculation and typical symptoms of infectious variegation were produced on cotton seedlings. On *A. indicum* dendrite yellow spots which spread into veins forming yellow net veils, typical of *Abutilon* infectious variegation on *A. sivatam*, Noordam4 were produced. The mechanical transmission of cotton infectious variegation by using 0.1 M phosphate buffer with reducing agents *viz.*, 2-mercaptoethanol (0.02 M) or DIECA (0.01 M) or Na2S2O3 (0.1%) was negative. The results on mechanical transmission for the present disease are similar to the one reported from Brazil, Costa1 for cotton yellow mosaic.

For seed transmission, seeds from infected Laxmi cotton and *A. indicum* were grown in earthen pots under insect free glass-house and seed transmission was determined by growing on test. About 600 seedlings of cotton and 140 from *A. indicum* germinated but none displayed the symptoms of infectious variegation. These results indicate that cotton infectious variegation was not transmissible through the seeds of cotton. The results on seed transmission through cotton seeds for infectious variegation are in concurrence with the results of Costa1 for cotton yellow mosaic. Keur2 has reported the transmission of *Abutilon* infectious variegation through the seeds of certain species, notably some hybrids of *Abutilon* species. However, the virus of cotton infectious variegation was not transmissible through the seeds of *A. indicum*.

On the basis of symptomatology, transmission and host-range studies, the disease *per se* is similar to cotton yellow mosaic caused by *Abutilon* infectious variegation virus from Brazil. This is the first record...