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Contributors are requested to be clear and concise. Manuscripts should not exceed 8,000 words and should be in a final form for the press. Each paper should start with a short summary which should be an abstract of the whole paper, complete and clear in itself, and not over 3 per cent. of the length of the paper. The introduction and reviews of literature should be restricted to closely pertinent papers.

The manuscript should be typewritten on one side of the paper only, with wide margins and be double spaced throughout including titles, footnotes, literature citations and legends. Symbols, formulae and equations must be written clearly and with great care. Scientific names of genera and species are printed in italics and should be underlined in the typescript. Too many tables, graphs, etc. should be avoided. Each table should be typed on a separate sheet with its proper position marked in the text in pencil.

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References:

Raman, C. V. (1949) The theory of the Christiansen experiment. *Proc. Indian Acad. Sci.*, A, 29: 381-90.

Sahni, B. (1936a) Wegener's theory of continental drift in the light of Palaeobotanical evidence. *J. Indian bot. Soc.*, 15: 31-32.

Sahni, B. (1936b) The Karewas of Kashmir. *Curr. Sci.*, 5: 10-16.

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Semiothisa Pervolgata wlk., A Semilooper Pest on Sesbania Aculeata (Dhaincha)*

BY

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(Received for publication, August 29, 1957)

Introduction :

Sesbania aculeata is one of the popular and important green manure crops grown largely in Madras State. It is subjected to the attack of number of pests. Ramakrishna Iyer (1940) noted the following pests :—

Azygophleps scalaris F., *Prodenia litura* F., *Alcidodes bubo* F., *Laphygma exigua* H., *Pericyma glaucinans* Hb., *Terias hecabe* L., *Catopsilia pyranthe* L., *Metripes* G., *Coptosoma cribraria* F., *Brachyplatis vahlii* F., and *Semiothisa pervolgata* Wlk. as pests of *Sesbania aculeata*. Among them *Semiothisa pervolgata* Wlk. is a serious pest belonging to the family Geometridae of the order Lepidoptera.

Distribution :

Semiothisa pervolgata Wlk. is distributed in Bengal, Poona and in Southern India (Hampson, 1895). In the Madras State it was noted in Coimbatore in 1936 doing severe damage to Dhaincha for the first time. Soon, an attempt was made to study the pest by Cherian and Rangiah Pillai (1938) and they have given broad outlines on its life history and methods of control. The present contribution gives a more detailed knowledge on its biology, bionomics and latest control measures.

Nature, symptoms of damage and alternate host plants :

It has no alternate host plants and is deemed to be a specific pest of Dhaincha, appearing in a pest form during the months

* Formed part of M.Sc. thesis submitted to Madras University in 1956.

of February and March, June to November when the crop is on the field. It assumes a characteristic position on the leaflets in that it clings to the edge of the blade in a looping fashion (Plate I, Fig. 1). When the attack is severe, it completely defoliates the entire plant leaving only the mid-rib and stem resulting in loss of all green matter.

Life history, habits and description of stages.

Copulation and oviposition :

Two days after the emergence of adults, copulation between female and male moths take place. The female moth after copulation rests on the branches of the side shoots and lays eggs. The eggs are laid singly on the leaves and tender shoots. A single female moth has been found to lay as many as 290 eggs at a time.

Egg : (Plate I, Fig. 2). The eggs are blue green in colour and oval in outline with a depression in the middle and measuring 0·577 mm. The egg period varies from 3 to 4 days.

Larva :

1st Stage : The newly hatched caterpillar is studded with tiny hairs all over the body and measures 1·5 m.m. in length. The head capsule is light brown or orange yellow and the thorax and abdomen dark green in colour. On the second abdominal segment, two black dots are very prominently noted. The prolegs are borne on the sixth and last abdominal segments. There are four moults during the larval stage before it undergoes pupation at its fifth moult.

2nd Stage: The larva after first moult measures about 5 to 6 m.m. and is light green in colour. The head region is globular with black dots. The thoracic legs are black in colour. Prolegs are slightly pinkish in tinge.

3rd Stage : After the second moult, the larva measures 1 cm. and turns ashy green with violet coloured intersegmental areas. There are a number of white parallel lines running on the dorsal aspect of the body. Head shield at this stage has a number of black lines and dots and the spiracle is circular with black dots.

PLATE I

Stages of *Semiothisa pervolgata* Wlk.

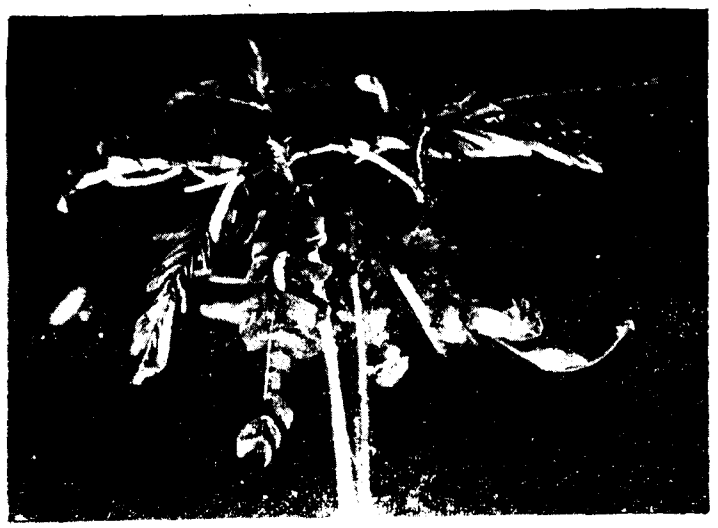


FIG. 1. *Larva on Sesbania aculeate* plant.

FIG. 2. Egg.

4th Stage: The larvae in this stage attains a length of 1·8 cm. and has a dark green body with parallel white lines more patent on the dorsal aspect of the body. The black dots and lines become darker in colour on the head region. There is distinct yellow line flanking the sides of the larvae. Minute short hairs are seen arising on either side of the mid-dorsal line. Small blackish hairs arise on the lateral sides of the head region.

5th Stage: The larva is pink and measures 2·7 cm. in length. There are horizontal bands of violet patches confined to the dorsal aspect of each segment and the head is bigger and mottled. Body is smooth and wrinkled in appearance with isolated and obscure short hairs. Spiracles are large, oval with pale centre and black rims. Prolegs are stout, fleshy and strong and occur as two pairs.

Pupa (Plate ··, Fig. 3(. The full grown larva enters the soil for pupation and has larval period of 10 to 12 days. The pupa is cylindrical, reddish brown and measuring 1·3 cm. In fields where the soil is hard, naked pupae have been collected from crackes and crevices. The pupal period is 6 to 8 days.

Adult (Plate II, Fig. 4). A detailed description of the moth of *Semiothisa pervolgata* Wlk. belonging to the sub-family Boarmiinae is described by Hampson (1895), as follows: "Whitish, slightly suffused with brown and striated with fuscous. Fore-wing with obliquely waved ante-medial, medial and post medial dark lines angled below the costa; a black spot at the end of cell; some fuscous suffusion beyond the post medial line; a dark patch on costa and spot below vein 5; a series of marginal black specks. Hind-wing with black spot at the end of cell; ante and post medial waved lines, the latter with fuscous suffusion beyond it and a black spot or spots at vein 2; a marginal crenulate black line. Underside the marginal area largely suffused with rufous and with white patches on it."

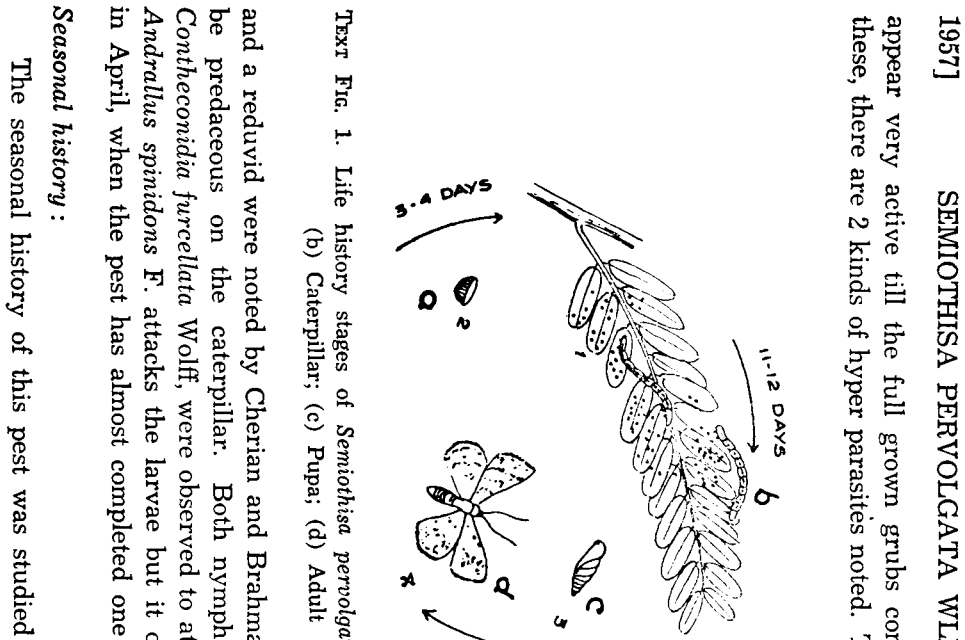
Total life cycle. Text Fig. 1. The total life cycle is about 20 to 23 days, the egg, the larval and pupal periods being 3 to 4, 10 to 12 and 6 to 8 days (Table I).

Natural enemies: In nature, the pest is controlled to a larger extent by a braconid, *Apanteles hypsipylae,* Wlk. In June they appear in large numbers and attack the caterpillar in all stages of growth. Medium sized caterpillars are highly susceptible. The caterpillars attain a greenish yellow colour and for all purposes

TABLE 1

Summary of life history of *Semiothisa pervolgata* Wlk. during 1953 to 1955.

S. No.	Date of egg laying.	Date of hatching.	Incubation period.	Date of pupation.	Larval period.	Adult emerged on.	Pupal period.	Total in days.
1.	2— 2—53	5— 2—53	3	15— 2—53	11	22— 2—53	8	22
2.	9— 7—53	12— 7—53	3	24— 7—53	12	31— 7—53	8	23
3.	20— 7—53	24— 7—53	4	3— 8—53	11	9— 8—53	7	22
4.	18— 8—53	21— 8—53	3	31— 8 53	10	6— 9—53	7	20
5.	12— 9—53	16— 9—53	4	25— 9—53	10	30— 9—53	6	20
6.	18—12—53	22—12—53	4	2— 1—54	11	8— 1—54	7	22
7.	7— 2—54	10— 2—54	4	21— 2—54	10	25— 2—54	6	20
8.	16— 7—54	19— 7—54	3	30— 7—54	11	6— 8—54	8	22
9.	14— 8—54	17— 8—54	3	28— 8—54	11	2— 9—54	6	20
10.	10— 9—54	13— 9—54	3	24— 9—54	11	30— 9—54	7	21
11.	18—12—54	22—12—54	4	11—12—54	10	6— 1—55	6	20
12.	4— 8—55	8— 8—55	4	19— 8—55	11	25— 8—55	7	22
	Maximum		4		12		8	23
	Minimum		3		10		6	20
	Average		3·6		10·8		6·9	21·2



TEXT FIG. 1. Life history stages of *Semiothisa pervolgata* Wlk. (a) Egg; (b) Caterpillar; (c) Pupa; (d) Adult

and a reduvid were noted by Cherian and Brahmachari (1941) to be predaceous on the caterpillar. Both nymphs and adults of *Contheconidia furcellata* Wolff, were observed to attack the larvae. *Andrallus spinidons* F. attacks the larvae but it occurs very late in April, when the pest has almost completed one generation.

Seasonal history :

The seasonal history of this pest was studied in Coimbatore. It is noted during the months of February to April and from June to November. About four broods were observed during the duration of the crop in the field.

Control measures :

Light trap was found to be effective, as it gave encouraging results. Calcium arsenate mixed with lime (1 : 6) applied to the plants on a small scale, both as dust and spray is not very effective and economical to use in the long run. In the present studies the trial on *Semiothisa pervolgata* Wlk. was conducted in the Central Farm Wetlands. Of the recently introduced insecticides the following chemicals DDT, BHC, Aldrin and Dieldrin in varying proportions were tried for each experiment. For each treatment 5 cent plots were taken and initial count of the population on 30 plants at random were taken prior to treatments. The plots for

different treatments were selected at different corners so as to avoid the effect of each chemical on one and the same plot. The following treatments were tried. (1) BHC 5%. (2) D.D.T. 5%. (3) BHC. 0·1%. (4) DDT 0·1%. (5) Aldrin 0·1%. (6) Dieldrin 0·1% and (7) Control. One 5 cent plot was set apart as control and this was located as distant from treated plots as possible. Mortality counts were taken 24 and 48 hours after treatment. The results have been presented in Table 2. It will be observed from the table that BHC 0·1% and Dieldrin 0·1% gave cent percent kill. Next in descending order come BHC 5%, DDT 5%, and DDT 0·1%. In the control there was slight reduction probably due to the effect of parasites on the medium sized caterpillars. The results were put to statistical interpretation as shown in Table 3. It is seen from the table that BHC 0·1%, Aldrin 0·1% and Dieldrin 0·1% were not significantly better than DDT 5% and 0·1%.

TABLE 2

Control trials on *Semiothisa pervolgata* Wlk.

S. No.	Treatments.	Initial Counts.	Counts after 24 hours.	Counts after 48 hours.	% of reduction in population after 48 hours.
1.	BHC 5%	42	5	3	93
2.	DDT 5%	35	12	7	80
3.	BHC ·1%	28	2	Nil.	100
4.	DDT ·1%	20	14	3	80
5.	Aldrin ·1%	30	6	Nil.	100
6.	Dieldrin ·1%	40	8	Nil.	100
7.	Control	45	30	25	

TABLE 3

Statistical analysis of the control trials on *Semiothisa pervolgata Wlk.*

S. No.	Treatments.	Difference in proportion.	Standard error.	Significance.
1.	DDT 5% / BHC ·1%	0·20	·079	No.
2.	DDT 5% / Aldrin ·1%	0·20	·078	No.
3.	DDT 5% / Dieldrin ·1%	0·20	·069	No.

PLATE I

Stages of *Semiothisa pervolgata Wlk.*

FIG. 3. Pupa.

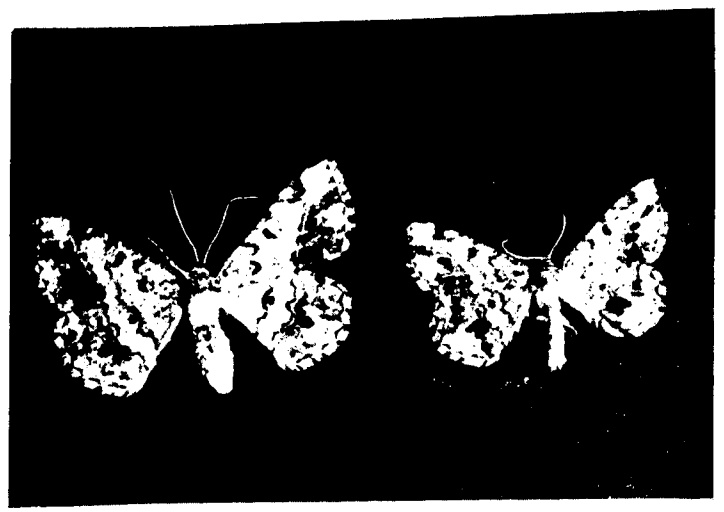


FIG. 4. Adult.

Summary :

Among the cultivated green manure crops in Southern India, *Sesbania aculeata* is frequently affected by a Geometrid pest, *Semiothisa pervolgata* Wlk. It was noted for the first time in Coimbatore during the year 1936 doing extensive damage to Daincha. It attacks the leaflets of the plant and in severe attack only naked stems are left behind, devoid of any green matter.

The moth lays eggs on the tips of leaflets and on buds. A single female is capable of laying upto 290 eggs. The egg period lasts from 3 to 4 days. The caterpillar undergoes 4 moults and at its fifth moult it pupates in the soil after a larval period of 10 to 12 days. The adult moth emerges after a pupal period of 6 to 8 days. The entire life cycle of the pest from egg to adult is completed in about 20 to 23 days. In nature, the pest is controlled to a large extent by a braconid *Apanteles hypsipylae* Wlk., but two kinds of hyper parasites are noted. It can be controlled to some extent by light traps by attracting gravid females. Calcium arsenate dust mixed with lime in proportion of 1 : 6 is also somewhat effective. Application of 5% BHC dust or spray of 0·1% Wettable BHC, Aldrin and Dieldrin completely controls the pest.

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On Strictly Convex Spaces

BY

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(Received for publication, September 16, 1957)

In this paper a new definition of strict convexity (s.c.) of a normed linear space is given, and in theorem I it is proved to be equivalent to the definition used by Clarkson (1). In theorem II I have obtained a necessary and sufficient condition that a seminorm is s.c. which is different from the one given by Smiley (2) in that the "betweenness" has been replaced by a single point namely the "mid-point". In theorem III the necessary and sufficient condition that F-space may be normed by a s.c. norm is obtained.

Section 1. Let B be a linear space; if it is normed (seminormed) the norm (seminorm) is denoted by $||\ ||$ ($|\ |$). Let $a, b, c, \ldots\ldots x, y, z, \ldots\ldots$ be the elements of B and θ be the null element.

Def. 1. We define the $||\ ||$ to be s.c. if $||x|| = ||y|| = 1$, $||\ x+y\ || = ||\ x\ || + ||\ y\ ||$ together imply $y = x$.

Def. 2. Clarkson (1) defines the $||\ ||$ to be s.c. if $||x+y|| = ||x|| + ||y||$, implies $y = \lambda x$ $(\lambda > 0)$.

These definitions can be extended to seminorms also.

Theorem 1. Definitions 1 and 2 are equivalent.

Let $||\ ||$ satisfy conditions of def. 1 and let a, b, ε B such that $||\ a+b\ || = ||\ a\ || + ||\ b\ ||$ ..... (1)

If possible let $b \neq \lambda\ a$; and $c = a + b$. Then

$$|| \frac{c}{||c||} + \frac{a}{||a||} || \leqslant || \frac{c}{||c||} || + || \frac{a}{||a||} ||$$

$$\leqslant 2 \qquad \ldots\ldots\ldots\ldots\ (2)$$

The equality sign in (2) may be dropped for in that case by def. 1 the result follows:

From inequality (2) $\| c \| a \| + a \| c \| \| < 2 \| a \| \| c \|$

Similarly $\| c \| b \| + b \| c \| \| < 2 \| b \| \| c \|$

Hence $\| c (\| a \| + \| b \|) + \| c \| (a + b) \|$

$< 2 \| c \| (\| a \| + \| b \|)$

i.e. $2 \| c \|^2 < 2 \| c \|^2$ which is a contradiction.

Hence $b = \lambda a$. Substituting λa for b in equation (1) λ is seen to be positive.

Next we shall prove that def. 2 implies def. 1.

Let $\| \ \|$ satisfy def. 2, and let

$\| a + b \| = \| a \| + \| b \|$, so that $b = \lambda a \quad (\lambda > 0)$,

If further $\| a \| = \| b \| = 1$, then it follows $\lambda = 1$ or $a = b$.

Section 2. If $a, b, c, \ldots\ldots$ be elements of a linear space. then b is said to be the algebraic mid-point of a, c if $b = \frac{1}{2} (a + c)$, and is a metric mid-point of a, c, if $d(a, b) = d(b, c) = \frac{1}{2} d(a, c)$.

Lemma: In an F-space the algebraic mid-point is also a metric mid-point.

For $d(a, b) = d(a - b, \theta)$ where d is the F-metric.

Theorem 2. The necessary and sufficient condition that a seminorm $(| \ |)$ is s.c. is that any pair of elements has a unique metric mid-point.

Proof: Suppose that the seminorm, $| \ |$ is s.c. and let 'b' be the unique metric mid-point of a, c.

i.e. $| a - b | = | b - c | = \frac{1}{2} | a - c | = t$.

Hence $| a/t - b/t | = | b/t - c/t | = \frac{1}{2} | a/t - c/t | = 1$

and $| a/t - b/t | + | b/t - c/t | = | a/t - c/t |$

Since $| \ |$ is s.c. by def. 1 it immediately follows that b is the algebraic mid-point of a, c, which is unique. Hence the condition is necessary. It is also sufficient. For the metric mid point being

unique coincides with the algebraic mid-point. $| x | = | y | = 1$, and $| x + y | = | x | + | y |$. Hence,

$$d(x, \theta) = d(\theta, -y) = \tfrac{1}{2} d(x, -y) = 1$$

i.e. $\theta = \frac{1}{2} (x - y)$ or $x = y$.

Section 3. If $a, b, c, \ldots\ldots$ are elements of B, b is said to be algebraically between a, c if

$$b = ta + (1 - t)c, \ 0 \leqslant t \leqslant 1,$$

and metrically between a, c if

$$d(a, b) + d(b, c) = d(a, c).$$

It is obvious that any point algebraically between x, x is x. In what follows $d(x, y)$ is a metric of the F-type over a linear space.

Theorem 3. The necessary and sufficient condition that $d(x, \theta)$ becomes a s.c. norm is that metric betweenness is identical with algebraic betweenness.

Proof: Let $\| x \| = d(x, \theta) = d(\theta, x)$. We shall show $\| x \|$ thus defined is a norm.

If $\| x \| = 0$ then $d(x, \theta) + d(\theta, x) = d(x, x) = 0$

Hence θ is algebraically between x, x. i.e. $x = \theta$.

Since metric betweenness coincides with algebraic betweenness and d is of the F-type for λ, $+ve$ integral

$$\| \lambda x \| = d(\lambda x, \theta) = \sum_{p=0}^{\lambda - 1} d(\overline{\lambda - p}x - \overline{\lambda - p - 1}x, \theta)$$

$$= \lambda d(x, \theta)$$

If λ is $a - ve$ integer, let $\lambda = -k$,

$$\| \lambda x \| = d(-kx, \theta) = kd(-x, \theta) = | \lambda | d(x, \theta).$$

If λ is rational equal to p/q, q positive,

$$q \| \frac{p}{q} x \| = \| px \| = | p | \| x \|.$$

Hence $||\frac{p}{q}x|| = |\frac{p}{q}|\,||x||$.

Next λ be irrational, and let $\frac{p_n}{q_n}$ be a sequence of rationals such that $\lim_{n\to\infty}\frac{p_n}{q_n} = \lambda$.

Since $d(\frac{p_n}{q_n}x,\ \theta) = |\frac{p_n}{q_n}|\,d(x,\ \theta)$ by the continuity of multiplication of the F-metric it follows that $d(\lambda x, \theta) = |\lambda|\,d(x, \theta)$ when λ is irrational. Thus for real λ,

$$||\lambda x|| = |\lambda|\,||x||.$$

It is easily verified that $||x||$ satisfies the other postulates of the norm. Hence the condition is sufficient. The condition is also necessary for if $||x|| = d(x, \theta)$ is a s.c. norm the metric betweenness coincides with the algebraic betweenness.

In conclusion I wish to thank Dr. M. V. Subba Rao for his help in the preparation of the paper.

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J. Madras Univ., B, XXVII, Nos. 2 & 3, pages 299-304, 1957

A Note on Some Properties of the Power Function of Chisquare*

BY

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(Received for publication, September 21, 1957)

SUMMARY

Some properties of the power function of the chisquare distribution are considered in this paper. It is noted that when the non-central distribution is used as an alternate test criterion for hypothesis H_1 against chisquare itself as H_0, the power is seen to increase with increase in degrees of freedom and decrease with increase in the the value of the parameter λ. When both vary the power decreases if the ratio $\frac{\lambda}{n}$ is greater than 0·2.

Let ξ_i, $i = 1, 2, \ldots n$ be 'n' normal variates with zero mean and unit variance. Then the sum $\sum_1^n \xi_i^2$ follows the well known χ^2-distribution with 'n' degrees of freedom. If, however, the ξ_i's are normal variates with unit variance, but with different means a_i $(i = 1, 2, \ldots n)$, Fisher (1920) derived the distribution of $\sum_1^n \xi_i^2$ as a particular case of multiple correlation coefficient. Later Tang (1938) gave an analytical proof for the same distribution which is known as non-central χ^2 and is usually denoted by χ'^2. This distribution is given by

$$p(\chi'^2) = e^{-\lambda/2}\sum_{j=0}^{\infty}(\frac{\lambda}{2})^j\frac{1}{j!}\frac{e^{-\chi'^2/2}(\frac{\chi'^2}{2})^{\frac{n}{2}+j-1}}{2^{\frac{n}{2}+j}\,\Gamma(\frac{n}{2}+j)} \qquad \ldots (1)$$

with 'n' degrees of freedom, where 'λ' is the non-central parameter. If the variance is σ^2, then $\sum_1^n \xi_i^2$ is distributed as $\sigma^2\chi'^2$.

* Some of these results are included in a thesis accepted for the M.Sc. Degree by the University of Madras.

Let the test criterion for a hypothesis 'H' depend on the statistic $\chi^2 = \sum_1^n \xi_i^2$ where ξ's are normal variates with zeromean and unit standard deviation and let χ'^2 be the criterion for the hypothesis H_1, with n-degrees of freedom, and a non-central parameter λ. Then the power of the test w.r.t. an alternative hypothesis, H_1 is $\beta(n, \lambda, \alpha) = \int_{\chi_\alpha^2}^{\infty} p_n(\chi'^2/\lambda)\, d\chi'^2$ where χ_α^2 is the 100 α% point of the χ^2-distribution with 'n' degrees of freedom, and this is given by

$$\beta(n, \lambda, \alpha) = \sum_{j=0}^{\infty} \frac{e^{-(\lambda/2)}(\lambda/2)^j}{j!\,\Gamma((n/2)+j)} \int_{\chi_\alpha^2}^{\infty} e^{-(\chi'^2/2)}(\chi'^2/2)^{(n/2)+j-1} d(\chi'^2/2) \quad .. \quad (2)$$

since term by term integration is justified on account of uniform convergence of the series.

From (2) it can be seen that the power function depends on $\lambda = \sum_{i=1}^{\infty} a_i^2$, the overall non-centrality so that the power, remains unaltered for any set of a_i's that will give the same λ.

Power as a decreasing function of the degrees of freedom.

Let the significance level α be kept constant. Then (2) can be written as

$$\beta(m, \lambda) = \sum_{j=0}^{\infty} \frac{e^{-(\lambda/2)}(\lambda/2)^j}{j!\,\Gamma(m+j)} \int_{x_a}^{\infty} e^{-x} x^{m+j-1}\, dx \quad .. \quad (3)$$

where $\frac{\chi'^2}{2} = x$, $\frac{\chi_\alpha^2}{2} = x_a$ and $\frac{n}{2} = m$.

Also from the definition of x_a

$$\alpha = \frac{1}{\Gamma(m)} \int_{x_a}^{\infty} e^{-x} x^{m-1}\, dx \quad .. \quad (4)$$

If λ is fixed, and m increases to $(m+1)$ let $\chi_\alpha'^2$ be the 100 α% point of the χ^2-distribution with $2(m+1)$ degrees of freedom. Thus we have corresponding to (3) and (4), the relations

$$\beta(m+1, \lambda) = \sum_{j=0}^{\infty} \frac{e^{-(\lambda/2)}(\lambda/2)^j}{j!\,\Gamma(m+j+1)} \int_{x'_a}^{\infty} e^{-x} x^{m+j} dx \quad .. \quad (5)$$

$$\text{and } \alpha = \frac{1}{\Gamma(m+1)} \int_{x'_a}^{\infty} e^{-x} x^m dx \quad .. \quad (6)$$

Integrating by parts the above integral, (6) becomes

$$\alpha = \frac{1}{\Gamma(m+1)} \left[m \int_{x'_a}^{\infty} e^{-x} x^{m-1}\, dx + e^{-x'_a} x_a'^m \right]$$

substituting the value of α given by (4), it follows

$$\int_{x_a}^{x'_a} e^{-x} x^{m-1}\, dx = (e^{-x'_a} x_a'^m)/m \quad .. \quad (7)$$

Since the expression on the right of (7) is positive, m being greater than zero, the integral on the left is positive. Hence it follows that

$$x'_a > x_a.$$

The inequality $\beta(m+1, \lambda) < \beta(m, \lambda)$ follows if

$$\frac{1}{\Gamma(m+j+1)} \int_{x'_a}^{\infty} e^{-x} x^{m+j}\, dx < \frac{1}{\Gamma(m+j)} \int_{x_a}^{\infty} e^{-x} x^{m+j-1}\, dx \quad .. \quad (8)$$

is true. Integrating by parts the left side of (8). and simplifying (8) becomes

$$\frac{e^{-x_a'} x_a'^{m+j}}{(m+j)} < \int_{x_a}^{x'_a} e^{-x} x^{m+j-1}\, dx \quad .. \quad (9)$$

By using equation (7) the inequality (9) reduces to

$$\frac{m x_a'^j}{(m+j)} \int_{x_a}^{x'_a} e^{-x} x^{m-1}\, dx < \int_{x_a}^{x'_a} e^{-x} x^{m+j-1}\, dx.$$

Since $e^{-x} > 0$, the above inequality holds if

$$x'_a{}^j\,(x'_a{}^m - x_a{}^m) < (x''_a{}^{m+j} - x_a{}^{m+j})$$

i.e., if $x'_a{}^j > x_a{}^j$ which is true as already noted. Hence the inequality (8) is true for every j. So $\beta(m+1, \lambda) < \beta(m, \lambda)$. From this result it can be noted that when n increases by 2 or multiples of 2 the power decreases. Since both χ^2-curve and corresponding χ'^2-curve move steadily to the right we infer that the power function must steadily decrease when 'n' increases.

Power as an increasing function of the non-central parameter λ.

Write (2) as a series of integrals so that

$$\beta(m, \lambda) = \sum_{j=0}^{\infty} \frac{e^{-(\lambda/2)}(\lambda/2)^j}{j!} \int_{x_a}^{\infty} \frac{e^{-x}x^{m+j-1}}{\Gamma(m+j)}dx. \qquad .. \quad (10)$$

Differentiating (10) w.r.t. λ and arranging the result by combining the part obtained by differentiating $(\lambda/2)^j$ in the $(j+1)^{\text{th}}$ term with the part obtained by differentiating $e^{-(\lambda/2)}$ in the j^{th} term, gives

$$\frac{\partial\beta}{\partial\lambda} = \sum_{j=0}^{\infty} I_j.$$

$$\text{where } I_j = \frac{e^{-(\lambda/2)}(\lambda/2)^j}{2 \cdot j!}\left[\int_{x_a}^{\infty} \frac{e^{-x}x^{m+j}}{\Gamma(m+j+1)} - \int_{x_a}^{\infty} \frac{e^{-x}x^{m+j-1}}{\Gamma(m+j)}dx\right] \qquad .. \quad (11)$$

The term by term differentiation is justified since the series (10) is uniformly convergent for $\lambda \geqslant 0$. Integrating the first integral in equation (11) by parts and simplifying we get

$$I_j = \frac{e^{-(\lambda/2)}(\lambda/2)^j}{2 \cdot j!}\left[\frac{e^{-x_a}x_a{}^{m+j}}{\Gamma(m+j+1)}\right]$$

which is always positive for all j. (x_a and λ being always positive).

Hence, $\frac{\partial\beta}{\partial\lambda}$ is positive so that $\beta(m, \lambda)$ is an increasing function of λ.

Changes in Power when n and λ *both vary.*

Next we shall consider the power when n and λ both vary. Consider when $\frac{\lambda}{n} = \text{const.}$, say c^2. Let the increase in n be $\triangle n$ and the corresponding increase in λ and β be $\triangle\lambda$ and $\triangle\beta$ respectively.

Omitting differences in derivatives of higher order than one we have

$$\frac{\triangle\beta}{\triangle n} \simeq \frac{\partial\beta}{\partial\lambda}\frac{\triangle\lambda}{\triangle n} + \frac{\beta(n+\triangle n, \lambda) - \beta(n, \lambda)}{\triangle n}$$

$$\simeq \frac{\partial\beta}{\partial\lambda}c^2 + \left[\frac{\beta(n+\triangle n, \lambda)\,\beta(n, \lambda)}{\triangle n}\right] \qquad .. \quad (12)$$

It has been shown in the preceding section that $\frac{\partial\beta}{\partial\lambda}$ is positive, and the expression in the square bracket is negative. Hence the sign of $\frac{\triangle\beta}{\triangle n}$ will depend on the relative magnitudes of $\frac{\partial\beta}{\partial\lambda}c^2$ and the expression in square brackets. Hence it is clear that $\frac{\triangle\beta}{\triangle n}$ will be positive if c^2 exceeds some particular value. Let this value of c^2 be equal to $c_o{}^2$. It is seen from the tables of power function of χ^2-test that the value of $c_o{}^2 = 1/5$. Hence as 'n' and 'λ' both vary the power increases as long as $\frac{\lambda}{n} > 1/5$. Since $\frac{\lambda}{n} = \frac{(\Sigma a i^2)}{n}$ the above result implies that the power of χ^2-test of H_o against H_1 increases when more observations having the same non-centrality c are included. This is true even when the expectation of all the x_i's under H_1 are not same, but are such that $\frac{\lambda}{n} = c^2$. This is in conformity with the general observation that where the χ^2-test is relevent, the sensivity of the test increases with the size of the sample.

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Theory of Demi-group Structures—I Axioms and Closure Operations for D-structures

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Introduction

In this and subsequent papers we shall be considering commutative demi-groups with unit element over which further structures are imposed. Such structures based on a demi-group we shall call d-structures. The conditions that these satisfy for the main forms of extra structure on the demi-group give the suggestion for the axioms stated for d-structures in this I Part. Then, in a manner very similar to, and in some sense dual to, the development of a theory of operations for c-structures presented in an earlier paper entitled 'Closure operations on c-structures' (Proc. Dutch Acad. Sci., Ser A, 56, No. 4, pp. 317-329), the operational structure for the d-structures is discussed in this part. As a concrete example of such d-structure one may consider demi-groups with neutral elements with a partial order monotone under the operation of the demi-group, or with a uniform structure determined by nuclii around the neutral element with the demi-group operation uniformly continuous from the squared space to the original. In these cases the a-homomorphisms are order-preserving or index-lowering as the case may be; where the index of a point of a demi-group in a nucleus is defined as by S. Kaplan in his paper on 'Extension of Pontrajagin duality' I, (Duke math. jour., 15, ('48) 649-58).

These examples and questions of duality will be treated in greater detail in the II Part of this paper.

1. *Axioms for d-structures.*

The primitive concepts are: the d-structure, the a-structure, the d-homomorphism, and the a-homomorphism. Before listing the

axioms that these satisfy, let us define some preliminary notions that are also necessary.

Def. A set D closed for a binary associative operation (written here as $+$) is called a demigroup. An element O of D is a neutral element for the demigroup if $a + O = O + a = a$, for all a from D; such an element is surely unique when it exists. The demigroup is a semigroup, if the two cancellation laws hold: $a + x = b + x$ implies $a = b$, and $x + a = x + b$ implies $a = b$. If the demigroup has a neutral element and an inverse $-a$ for each element a (such that $a + (-a) = (-a) + a = O$) then we get a group.

Def. Given a class of demigroups D_i with neutral elements O_i, their direct sum $D(= S(D_i))$ is a demigroup whose elements are collections (a_i) of one element from each D_i, with $a_i = O_i$ for all but an atmost finite number of the i; the sum of two such elements (a_i) and (b_i) being computed componentwise, i.e., it is (c_i), where $c_i = a_i + b_i$. The (1-1) mapping of D_i in D defined by: $\delta_i(a_i) =$ the element (a_i) of D with $a_j = O_j$ except for $j = i$ when a_j is the element a_i taken from D_i, is called the ith injection in D. Clearly then any element α of D can be written as $\Sigma\, \delta_i\ (\alpha_i)$.

We may observe that D has $O = (O_i)$ as neutral element, and that when all the demigroups D_i are (i) commutative or (ii) are semi-groups, or (ii) are groups, then so is D.

We start with the axioms now.

B1. To each d-structure **A** is associated a unique a-structure A, called the a-base of **A**; each a-structure is the a-base of some d-structure. To each a-structure A is associated a unique commutative demigroup α, called the D-base of A. The elements of α are also called the elements of A, while if A is the a-base of **A**, and α the D-base of A, then α is also called the D-base of **A**, and its elements are called elements of **A** also.

B2. If **A**, **A′** are d-structures and f is a d-homomorphism of **A** in **A′**, then f is also a a-homomorphism of the a-base of **A** in the a-base of A′. If A, A′ are a-structures and f is a a-homomorphism of A in A′ then it is a demigroup homomorphism of the D-base of A in the D-base of A′.

It is a consequence of B2 that the d-homomorphisms and a-homomorphisms are special instances of mappings of the elements of the one in the other.

Def. A (1-1) mapping f of the D-base of **A** in that of **A′** (of the D-base of A in that of A′) is called a d-isomorphism (a a-isomorphism) if both f and f^{-1} are d-(a-) homomorphisms.

B3. Two d-(a-)structures are identified (considered indistinguishable) if they have the same D-base and the identity mapping of this base in itself is a d-(a-)isomorphism. The composition of two successive d-(a-)homomorphisms is also such a homomorphism.

B4. If f is a a-homomorphism of a a-structure A *on* each of two a-structures B′, B″ having the same D-base, then B′ and B″ are identical. If α, β are demigroups and f is an isomorphism of α on β, then if A is a a-structure having α as D-base, a a-structure B with β as D-base can be found such that f is a a-isomorphism of A on B. If f is a a-homomorphism of A in B and $f(\alpha) = \gamma =$ a subset of β, where α, β are the D-bases of A, B, then a a-structure C with γ as D-base can be found such that f is a a-homomorphism of A on C and the identity mapping of γ in β is a a-homomorphism of C in B.

Def. If E is a congruence relation on the commutative demigroup α (that is an equivalence relation such that aEb implies $(c + a)E(c + b)$, for all a, b, c, from α), and β is the quotient demigroup α/E (whose elements are the equivalence classes $E(a)$, a in α, with $E(a) + E(b) = E(a+b)$) a a-structure B over β (i.e., with β as D-base) is called a 'quotient' of a a-structure A over α by E if the canonical mapping (a in α mapped on $E(a)$ in β) of α in β is a a-homomorphism of A in B; by the first part of B4 it follows that if a quotient of A by E exists it must be unique. We denote it by A/E, when it exists.

Def. Two d-structures with the same a-base are said to be comparable; when **A**, **A′** are comparable d-structures (over the a-base A) we say **A** is finer than A′ (in symbols $\mathbf{A} < \mathbf{A'}$) if the identity mapping of the D-base of A in itself is a d-homomorphism of **A** in **A′**.

B5. The d-structures comparable with a given d-structure **A** (over the a-base A) form a class **C** (A); given a d-homomorphism f of a d-structure **A** in a d-structure **B** from the class **C** (B), there exists a finest among the d-structures **B′** of **C** (B) for which f is a d-homomorphism of **A** in **B′**; we denote this d-structure by f(**A**).

B6. If f, g are a-homomorphisms of A in B and of B in C, where A, B, C are a-structures, and if **A**, **C** are d-structures on

A, C such that gf is a d-homomorphism of **A** in **C**, then a d-structure **B** on B can be found such that f, g are d-homomorphisms of **A** in **B** and of **B** in **C** respectively.

Def. When α is a commutative demigroup, and $\beta = \alpha/\mathbf{E}$ is a quotient demigroup, then a d-structure **B** over β is called a quotient d-structure of **A** over α by E if the canonical mapping f of α on β is a d-homomorphism of **A** on **B**, and further $\mathbf{B} = f(\mathbf{A})$.

Again the quotient structure if it exists is unique and is denoted by **A**/E; the a-base of **A**/E is the quotient A/E where A is the a-base of **A**.

Def. For a class of d-structures $\mathbf{A}_i$ we define the d-structure **A** to be a direct sum of the class, if the D-base of **A** is the direct sum of the D-bases of the $\mathbf{A}_i$, if the injections δ_i are all d-homomorphisms of the $\mathbf{A}_i$ in **A**, and finally whenever there is a family of d-homomorphisms φ_i of the $\mathbf{A}_i$ in some d-structure **B**, then the mapping φ of **A** in **B**, defined by $\varphi(a) = \varphi((a_i)) = \Sigma\varphi_i(a_i)$, for any $a = (a_i)$ in A, is also a d-homomorphism.

From the definition follows the uniqueness of the direct sum of a class of d-structures, when it exists. We denote the sum of the $\mathbf{A}_i$ by $\mathrm{S}(\mathbf{A}_i)$.

B7. For any class of d-structures there exists a direct sum.

Def. If the D-base α of a d-structure **A** is contained in the D-base β of a d-structure **B**, and the identity mapping g of α in β is not only a d-homomorphism of **A** in **B**, but even $\mathbf{B} = g(\mathbf{A})$, then **B** is called an extension of **A**. If there is a d-homomorphism g of a d-structure **A** in a d-structure **B**, and $\mathbf{B} = g(\mathbf{A})$, then **B** is called an envelope of **A**, and g is referred to as a map enveloping **A** in **B**.

B8. If, for a class of d-structures $\mathbf{A}_i$, $\mathbf{B}_i$ is an extension of $\mathbf{A}_i$ for each i, then $\mathrm{S}(\mathbf{B}_i)$ is an extension of $\mathrm{S}(\mathbf{A}_i)$.

2. *First consequences from the axioms.*

Lemma 1. (i) If the D-base β of a d-structure **B** is isomorphic with a demigroup α, then there is a d-structure **A** with D-base α such that the isomorphism between the D-bases is also d-isomorphism between **B** and **A**.

(ii) If f is a d-homomorphism of a d-structure A in a d-structure **B**, and E is the equivalence determined by f for the D-base

of α, then f can be extended to a d-homomorphism of the quotient **A**/E in **B**.

Proof. The axioms B4 and B6 give (i), while (ii) follows from B4, B5 and B6.

Lemma 2. (i) If f envelopes **A** in **B** and g envelopes **B** in **C**, then gf envelopes **A** in **C**; (ii) **B** is an envelope of **A**, if, and only if, **B** is an extension of an isomorph of a quotient d-structure of **A**.

Proof. (i) That gf is a d-homomorphism is clear; if **C**′ is comparable with **C**, and gf is d-homomorphism of **A** in **C**′, then as f, g are a-homomorphisms of the a-base of **A** in **B** and of **B** in **C**′, by B6 there is a d-structure **B**′ comparable with **B** such that f, g are d-homomorphisms of **A** in **B**′ and of **B**′ in **C**′. Hence as f, g are enveloping mappings, **B** is finer than **B**′, g is a d-homomorphism of **B** in **C**′ and **C** is finer than **C**′. So gf envelopes **A** in **C**.

(ii) An isomorphic mapping, an extension mapping and the canonical mapping on a quotient structure are all enveloping mappings, and so, by (i), an extension of an isomorph of a quotient d-structure is an envelope. While if f envelopes **A** in **B**, and **E** is the equivalence relation on the D-base of **A** defined by f, then it follows from lemma 1, (ii), that f can be considered as a d-homomorphism of the quotient d-structure **A**/E in **B**. By B4 it can be seen that f is then a a-isomorphism between the a-base of **A**/E and a a-structure C with D-base $\gamma = f(\alpha)$, α being the D-base of **A**, and the identity mapping of γ in β (the D-base of **B**) is an a-homomorphism of C in B. As in the proof of (i) above, using B6 we can then see that there is a d-structure **C** with C as a-base such that **A**/E is isomorphic with it, and **B** is an extension of it.

Lemma 3. If **A** is the direct sum of a class of d-structures $\mathbf{A}_i$, and for each i, $\mathbf{A}_i^*$ is a quotient d-structure of $\mathbf{A}_i$ by E_i, then the direct sum **A***of the $\mathbf{A}_i^*$ is d-isomorphic with the quotient d-structure of **A** by an equivalence $\mathrm{E} = \mathrm{S}\mathrm{E}_i$ defined on it by: $(a_i)\ \mathrm{E}\ (a_i') <=>$ for all i, $a_i \mathrm{E}_i a_i'$:.

Proof. Let δ_i, δ_i^* denote the injective mappings into the sums **A**, **A*** respectively. Then if a_i^* is the class under E_i containing a_i of $\mathbf{A}_i$, from the fact that the mappings of $a_i \to a_i^*$, and $a_i^* \to \delta_i^*(a_i^*)$ are d-homomorphisms it follows, from the definition of $\mathbf{A} = \mathrm{S}\mathbf{A}_i$, that the mapping $(a_i) \to (a_i^*)$ from **A** to **A*** is a d-homomorphism. As this mapping determines on **A** just

the equivalence E by axiom B5 and the definition of the quotient structure it follows that this homomorphism splits up into the canonical one into $\mathbf{A}/\mathrm{E}$ followed by a 1-1 d-homomorphism on $\mathbf{A}^*$. That the inverse of this 1-1 mapping is also a d-homomorphism is deduced from the fact that the mappings $a_i^* \rightarrow$ the class under E containing $\delta_i(a_i)$, where a_i is any element of a_i^*, are d-homomorphisms.

Corollaries. The direct sum of $\mathbf{B}_i$ is d-isomorphic with (an envelope of) the direct sum of the $\mathbf{A}_i$, if, for each i, $\mathbf{B}_i$ is d-isomorphic with (an envelope of) $\mathbf{A}_i$.

The isomorphism part is evident; the other then follows from B8, lemma 2 (ii) and the above lemma.

3. *The Lattice of comparable d-structures.*

The relation $<$ of 'being finer than' is a partial ordering relation in the class $\mathbf{C}$ (A) of all comparable d-structures with the common a-base A. We examine this partially ordered set further now.

Theorem 1. The $p.o$ set $\mathbf{C}(\mathrm{A})$ is a conditionally complete lattice; for a family $\mathbf{A}_i$ of its elements that are bounded above the lattice sum is d-isomorphic with the quotient of the direct sum of the $\mathbf{A}_i$ by an equivalence relation E.

Proof. Define on the D-base of the direct sum $\mathrm{S}(\mathbf{A}_i)$ the equivalence relation E by: $(a_i)\mathrm{E}(a_i')$ if $\Sigma a_i = \Sigma a_i'$, these being finite sums in the commutative demigroup A. Then the correspondence f: taking the class under E containing (a_i) to Σa_i in A is a demigroup isomorphism between the quotient demigroup of the D-base of $\mathrm{S}(\mathbf{A}_i)$ by E and the D-base α of A. If the quotient d-structure of $\mathrm{S}(\mathbf{A}_i)$ by E exists then f gives a d-isomorph over A which is $>$ all the $\mathbf{A}_i$; while, conversely, if an upper bound $\mathbf{A}'$ exists for the α_i, f^{-1} gives over the base of $\mathrm{S}(\mathbf{A}_i)/\mathrm{E}$ a d-isomorph, which implies then that the quotient structure $\mathrm{S}(\mathbf{A}_i)/\mathrm{E}$ exists, and is $>$ than this isomorph. Thus it follows that the existence of an upper bound to the $\mathbf{A}_i$ is equivalent to the existence of the quotient structure, and when this exists, from the quotient structure f gives rise to a d-isomorphic structure on A which is the lattice sum, of the $\mathbf{A}_i$. Since every sub-family of $\mathbf{C}(\mathrm{A})$ bounded above has a lattice sum, every subfamily bounded below will also have a lattice product (namely the lattice sum of

the family of lower bounds). So the ordered set $\mathbf{C}(\mathrm{A})$ is a conditionally complete lattice.

Lemma 4. (i) If $\mathbf{A} = \mathrm{S}(\mathbf{A}_i)$, then $\mathbf{A} = \mathrm{V}(\delta_i(\mathbf{A}_i))$, V denoting the lattice sum. (ii) If f envelopes $\mathrm{V}(\mathbf{A}_i)$ in $\mathbf{B}$, then $\mathbf{B} = \mathrm{V}(f(\mathbf{A}_i))$.

Proof. (i) As δ_i is a d-homomorphism of $\mathbf{A}_i$ in $\mathbf{A}$, so $\delta_i(\mathbf{A}_i)$ is $< \mathbf{A}$, for each i. If $\delta_i(\mathbf{A}_i)$ is $< \mathbf{A}'$, for a d-structure comparable with $\mathbf{A}$, for each i, then from the definition of the direct sum it follows that the identity mapping of the D-Base of $\mathbf{A}$ on itself is a d-homomorphism of $\mathbf{A}$ in $\mathbf{A}'$. Thus $\mathbf{A} = (\delta_i\ (\mathbf{A}_i))$ is proved.

(ii) if α, β denote the D-bases of $\mathbf{A}$ and $\mathbf{B}$, and the identity mappings of α, β in themselves are denoted by α', β' then, $f = f.\alpha' = \beta'.f$. Hence as α' from $\mathbf{A}_i$ to $\mathbf{A}$, and f from $\mathbf{A}$ to $\mathbf{B}$ are d-homomorphisms, f is a d-homomorphism from $\mathbf{A}_i$ to $\mathbf{B}$, thus $f(\mathbf{A}_i) = \mathbf{B}_i$ exists and it is $< \mathbf{B}$ for each i. If each $\mathbf{B}_i$ is also $<$ some $\mathbf{B}^*$ comparable with $\mathbf{B}$, then f from $\mathbf{A}_i$ to $\mathbf{B}_i$ and β' from $\mathbf{B}_i$ to $\mathbf{B}^*$ being d-homomorphism f is a d-homomorphism of $\mathbf{A}_i$ in $\mathbf{B}'$ for each i; it follows that the mapping $(a_i) \rightarrow \Sigma f(a_i)$ is a d-homomorphism of $\mathrm{S}(\mathbf{A}_i)$ in $\mathbf{B}^*$. Since $\Sigma a_i = \Sigma a_i'$ implies that $\Sigma f(a_i) = \Sigma f(a_i')$, from theorem 1 we see that f is a d-homomorphism of $\mathrm{V}(\mathbf{B}_i)$ in $\mathbf{B}^*$; it follows that $\mathbf{B}$ is $< \mathbf{B}^*$, and the lemma is proved completely.

4. *Direct classes of d-structures and their limits.*

Def. A class $\mathbf{B}_d$ of d-structures indexed by a directed set $(\mathrm{D}, >)$ is called a directed class of d-structures if for each pair of indices d, d' with $d < d'$ there is a d-homomorphism $\lambda(d, d')$ of $\mathbf{B}_d$ in $\mathbf{B}_{d'}$, such that when $d < d' < d''$ then $\lambda(d', d'')$. $\lambda(d, d') = \lambda(d, d'')$.

Def. If $\mathbf{B}_d$ is a directed class of d-structures, a d-structure $\mathbf{B}$ is called the limit of the directed class if it is the quotient d-structure of the direct sum $\mathrm{S}(\mathbf{B}_d)$ by the equivalence E defined by: $(b_d)\mathrm{E}(b_{d'})$ for (b_d), $(b_{d'})$ from the D-base of $\mathrm{S}(\mathbf{B}_d)$, if there is a $d'' >$ all the d's for which b_d or $b_{d'}$ is not equal to O_d, such that $\Sigma\lambda(d, d'')(b_d)$ and $\Sigma\lambda(d, d'')(\mathrm{b}_{d'})$ (summed over the $d < d''$) are equal in the D-base of $\mathbf{B}_{d''}$.

Evidently E is an equivalence relation as D is directed by $>$. So the limit is unique if it exists.

Theorem 2. (i) If $\mathbf{A} = \mathrm{S}(\mathbf{A}_i)$, i in a set I, then $\mathbf{A}$ is isomorphic to the limit of the directed class $\mathbf{B}_d$, where d ranges over the family D of finite subsets of I directed by $\supset$, where $\mathbf{B}_d$ is taken as the direct sum $\mathrm{S}(\mathbf{A}_i)$, i in d, and $\lambda\ (d, d')$ for $d' \supset d$ is the mapping taking an element (a_i) of $\mathbf{B}_d$ to (a_j') of $\mathbf{B}_{d'}$, with $a_j' = a_i$ for $j' = i$ in d, and $a_j' = \mathrm{O}_j'$ otherwise.

(i) If $\mathbf{A} = \mathrm{V}(\mathbf{A}_i)$, i in I, then $\mathbf{A}$ is isomorphic to the limit of the directed class of d-structures $\mathbf{B}_d$, where d is taken from the family D of finite subsets of I directed by $\supset$, $\mathbf{B}_d$, is $\mathrm{V}(\mathbf{A}_i)$ for i in d, and $\lambda(d, d')$ for $d' \supset d$ is the identity mapping of the D-base of $\mathbf{A}$ in itself.

Proof. (i) It is easy to see that the $\mathbf{B}_d$ do form a directed class of d-structures. The mapping f_d taking (a_i) of $\mathbf{B}_d$ to the element of $\mathbf{A}$ having the same nonzero terms at the same places, is a d-homomorphism. Hence the mapping f taking (b_d) of $\mathrm{S}(\mathbf{B}_d)$ to $\Sigma f_d(b_d)$ in $\mathbf{A}$ is a d-homomorphism and this defines on $\mathrm{S}(\mathbf{B}_d)$ the equivalence E under which $(b_d)\mathrm{E}(b_{d'})$ if and only if $\Sigma\lambda(d, d'')\,(b_d) = \Sigma\lambda(d, d'')\,(b_{d'})$, for d'' equal to or containing the union of the d's for which the b_d, $b_{d'}$ are nonzero. Thus, from the definition of the limit and lemma 1 (ii), it follows that the limit of the directed class $\mathbf{B}_d$ exists and f is a 1-1 d-homomorphism of the limit on $\mathbf{A}$. That the reverse of this 1-1 mapping is a d-homomorphism follows from the fact that each of the mappings $\varphi_i: a_i \to$ the class under $\mathbf{B}$ containing (b_d), where b_d is O_d except for $d =$ the one element set (i) for which it is a_i itself, is a d-homomorphism of $\mathbf{A}_i$ in the quotient structure of $\mathrm{S}(\mathbf{B}_d)$ by E. Then from the definition of $\mathrm{S}(\mathbf{A}_i)$ it follows that the inverse of f from $\mathbf{A}$ to the limit structure is a d-homomorphism. Thus $\mathbf{A}$ is isomorphic with the limit, which exists, of the directed class of the $\mathbf{B}_d$.

(ii) That the $\mathbf{B}_d$ form a directed class is easily verified. For each d $\mathbf{B}_d$ is comparable with $\mathbf{A}$ and $< \mathbf{A}$; hence the identity mapping I of the D-base α of A in itself is a d-homomorphism of $\mathbf{B}_d$ in$\mathbf{A}$. It follows that the mapping f taking $(\mathbf{B}_d)$ of $\mathrm{S}(\mathbf{B}_d)$ to the element Σb_d of $\mathbf{A}$ is a d-homomorphism, defining the equivalence E under which $(b_d)\mathrm{E}(b_{d'})$ means that $\Sigma\, b_d = \Sigma\, b_{d'}$ in α, that is to say that $\Sigma\lambda(d, d'')\,(b_d) = \Sigma\lambda(d, d'')\,(b_{d'})$ when d'' contains the union of the d's for which b_d, $b_{d'}$ in the two elements given are nonzero. Thus as before the limit of the $\mathbf{B}_d$ exists and there is a 1-1 d-homomorphism of the limit on $\mathbf{A}$. To prove that the inverse of this map is also a d-homomorphism it is enough to

note that the mappings $\varphi_i: a_i \to$ the class under E containing the (b_d) with zero terms except for $d =$ (i) for which $b_d = a_i$, are d-homomorphisms from the $\mathbf{A}_i$ to the limit.

5. *Operations on types of d-structures.*

By adding extra restrictions we get types of d-structures. Of two types t, t' we say t is stronger, and write $t' => t$, if every d-structure of type t' is also of type t. If $t => t'$ and $t' => t$ (or as we also write it $t <= t'$) then the two types t, t' are called equivalent, and we write $t <=> t'$.

From a binary relation R defined for ordered pairs of d-structures, or an operation θ defined for certain classes of d-structures, we can derive operations, also denoted by R or θ, on types of d-structures: $\mathrm{R}(t)$ is the type of d-structure $\mathbf{A}$ for which a d-structure $\mathbf{B}$ of type t can be found such that $\mathbf{ARB}$ is true; $\theta(t)$ is the type of d-structure $\mathbf{A}$ for which a class of d-structures $\mathbf{A}_i$ of type t can be found such that $\mathbf{A} = \theta(\mathbf{A}_i)$. Evidently if $t => t'$, then $\mathrm{R}(t) => \mathrm{R}(t')$ and $\theta(t) => \theta(t')$, for these operations on types. When φ is an operation on types (i.e., associates to each type t another $t' = \varphi(t)$) which satisfies such a monotonicity condition w.r.t. $=>$, then a type t is said to be closed for φ if $\varphi(t) => t$. If t' is a type closed for φ stronger than a given type t and feebler than any type t'' closed for φ stronger than t, then t' is called a φ-closure of t; it is unique upto equivalence of types if it exists. The operation on types φ is a 'closure operation on types' if, for any type of d-structure t, $\varphi(t)$ is a φ-closure of t; if φ is monotone w.r.t. $=>$, a sufficient condition that φ be a closure operation on types is that, for any type t, one has $t => \varphi(t)$ and $\varphi\varphi(t) => \varphi(t)$. Of two operations on types θ, φ we say θ is stronger if, for any type t, $\theta(t)$ is stronger than $\varphi(t)$.

We consider now the interrelations between the various operations on types defined by the following relations and operations on d-structures:

1. $\mathbf{AiB}$ is the relation $\mathbf{A}$ is d-isomorphic with $\mathbf{B}$.
2. $\mathbf{AqB}$ is the relation that $\mathbf{A}$ is the quotient d-structure of $\mathbf{B}$ by an equivalence $\mathbf{E}$.
3. $\mathbf{AxB}$ is the relation that $\mathbf{A}$ is an extension of $\mathbf{B}$ by a mapping f.

4. $\mathbf{A}e\mathbf{B}$ is the relation that $\mathbf{A}$ is the envelope of $\mathbf{B}$ by a mapping f.

5. $\mathbf{A} = \mathrm{S}(\mathbf{A}_i)$, $[\mathbf{A} = s(\mathbf{A}_i)]$ stand for the operations of taking the direct sum of an arbitrary [of a finite] class of d-structures.

6. $\mathbf{A} = \mathrm{V}(\mathbf{A}_i)$, $[\mathbf{A} = v(\mathbf{A}_i)]$ stand for the operations of taking the lattice sum of an arbitrary [of a finite] class of comparable d-structures.

7. $\mathbf{A} = \mathrm{L}(\mathbf{A}_d)$, d in $(\mathrm{D},>)$, stand for taking the limit of a directed class of d-structures $\mathbf{A}_d$.

The results of the earlier section then lead to the following results:

Lemma 5. For any given type of d-structures t, we have

(i) $t => \varphi(t)$, if φ is any one of the operations, i, iq, x, e, S, s, V, v, iL; $i.\ i(t) <=> i(t)$, $i.x(t) <=> x.i(t)$, $i.q(t) <=> q.i(t)$, $e.e(t) <=> e(t)$; $e(t) <=> x.i.q(t)$, $\mathrm{S\,S}(t) => i.\mathrm{S}(\mathrm{T})$, $s.s.(t) => i.s(t)$, $\mathrm{VV}(t) <=> \mathrm{V}(t)$, $v.v.(t) <=> v(t)$, $\mathrm{S}(t) => \mathrm{V}.x(t)$, $s(t) => v.x(t)$. $\mathrm{V}(t) => i.q.\mathrm{S}(\mathrm{T})$, $v(t) => i.q.s(t)$;

(ii) if φ denotes any one of the operations, i, iq, x or e, then $\mathrm{S}.\varphi(t) => \varphi.\mathrm{S}(t)$, $s.\varphi(t) => \varphi.s(t)$, $\varphi.\mathrm{V}(t) => \mathrm{V}\varphi(t)$, $\varphi.v(t) => v.\varphi(t)$;

(iii) $\mathrm{S}(t) => i.\mathrm{L}.p(t)$, $V(t) => i.\mathrm{L}v(t)$, $\mathrm{L}(t) => q.\mathrm{S}(t)$, $L(t) => \mathrm{V}.e(t)$.

Proof. (i) The first two sets of assertions are easily deducible from the definitions involved. Lemma 2 (ii) gives $e(t) <=> x.i.q(t)$; Theorem 1 and lemma 4 (i) take care of the last four assertions, while the others are merely forms of associativity of the direct sum or lattice sum, allowance being given to distinguish isomorphic structures.

(ii) Lemma 3 and its corollary prove the first results, while lemma 4 (ii) gives the results $e.\mathrm{V}(t) => \mathrm{V}.e(t)$ and $e.v(t) => v.e(t)$, while similar proofs can be given to prove the corresponding results $\varphi.\mathrm{V}(t) => \mathrm{V}.\varphi(t)$, $\varphi.v(t) => v.\varphi(t)$, for $\varphi = i, i.q.$ or x.

(iii) Theorem 2 (i) and (ii) give the first two results; the definition of the limit implies that $\mathrm{L}(t) => q.\mathrm{S}(t)$, while this with $\mathrm{S}(t) => \mathrm{V}.x(t)$ leads to $\mathrm{L}(t) => q.\mathrm{V}.x(t) => i.\mathrm{V}.q.x(t) => \mathrm{V}.i.q.x(t) => \mathrm{V}.e(t)$, as each of $i(t)$, $q(t)$ and $x(t)$ implies $e(t)$.

From the above results we deduce the main structure theorem:

Theorem 3. (i) The following are closure operations on types of d-structures: φ, $\varphi.\mathrm{S}$, $\varphi.s$, $\mathrm{V}.\varphi$ and $v.\varphi$, where φ stands for any one of the operations i, $i.q$, x or e.

(ii) There is a closure operation on types stronger than all combinations of the basic operations i, q, x, e, S, s, V, v and L, and this is equivalent to each of the following: $e.\mathrm{S}$, $\mathrm{V}.e$, $e.\mathrm{L}s$, $i.\mathrm{L}.v.e$ and $i.q.\mathrm{S}.e$.

Proof. (i) The lemma 5 proves that the operations mentioned are all closure operations on types.

(ii) $e.\mathrm{S} => e.\mathrm{V}.x => \mathrm{V}.e.x => \mathrm{V}.e => i.q.\mathrm{S}.e => q.e.\mathrm{S} => e.\mathrm{S}$
$e.\mathrm{S} => e.i.\mathrm{L}.s => e.\mathrm{L}.s => e.q.\mathrm{S}.s => e.\mathrm{S}$
$\mathrm{V}.e => i.\mathrm{L}.v.e => i.\mathrm{V}.e.v.e => i.\mathrm{V}.v.e => i.\mathrm{V}.e =>$
$\mathrm{V}.i.e => \mathrm{V}.e$
$\mathrm{V}.e => i.q.\mathrm{S}.e => i.q.e.\mathrm{S} => e.\mathrm{S}$

So the five operations given are all equivalent, and as one of them $e.\mathrm{S}$ is a closure operation so are all the others. And any one of the single basic operations and hence also all combinations of them imply one of these five and so all of them.

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Solvent Extraction of South Arcot Lignite

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ABSTRACT

Solvent Extraction formed part of the series of investigations taken up on the study of the Utilisation of the South Arcot Lignite. A number of solvents were used in order to establish the best solvent that gives an appreciable quantity of good quality extractive. With one of the solvents, viz., Xylol, a study was made of the effect of temperature and pressure on the yield of extractive, and correlations have been obtained between the yield on the one hand and the time and temperature of extraction on the other. The properties of the various extractives obtained were determined and a sample of refined montan wax from the Xylol extract, was prepared.

Introduction :

Solvent Extraction of coals, lignites and peats has been an attractive line of investigation pursued with the object of either studying the coal constitution or isolating technically important extractives. In this laboratory a sample of the South Arcot Lignite was subjected to solvent extraction with a number of solvents with a view to preparing some commercially important resins and waxes and the results are discussed in this paper.

The extraction of coals with organic solvents involves not only a physical solution of the material but also a chemical decomposition or depolymerisation. Reference has also been made to the possibility of chemical combination of the solvent and the coal substance (Lowry, Vol. I). Hence at each temperature the amount of the extractive obtained is governed by the solubilising power of the solvent at that temperature and the degree of decomposition and the consequent losses due to gasification of the extracted product.

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The extractives obtained are composed of the original coal substance extracted by true solution as well as those resulting from the thermal depolymerisation of the coal substance. Besides, in the case of certain solvents like pyridine a process of peptisation or colloidal dispersion of the coal substance is brought about. Quite a variety of organic substances ranging from more or less familiar low molecular weight compounds to highly complex molecules and colloids are therefore met with in the extractives. The extractive obtained from peats, lignites and semi-bituminous coals are commonly termed Montan Wax, although strictly speaking the term must refer to the refined wax obtained from the Crude extractive. The montan wax as obtained from lignite is essentially a mixture of waxy constituents consisting of monohydric alcoholic esters and high molecular weight acids, along with some resinous and asphaltic materials (Warth). The composition of the crude montan wax is given as: ester of waxy acids 58-59%, free wax acids 17-19%, free monohydric alcohols 3-4% (primary), 1% (secondary), resins 10-12%, and ketones less than 10%.

Montan wax has been of great industrial importance in its applications for electrical insulation, for wax coatings on leather, for polishes, for brewer's pitch, for phonograph records, etc. Crude montan wax may be advantageously used to lower the pour points of oils.

The suitability of the South Arcot Lignite for the production of montan wax and resins and the properties of the waxes obtained under various conditions were thoroughly investigated. Soxhlet extraction of the lignite was conducted with a number of solvents and the best solvent which gives a good quantity of the wax having the desired properties was established. The effect of various conditions like moisture content and particle size of the lignite on the yield on extraction for a particular solvent was also studied. This was followed by a series extractions conducted under pressure. Attempts were made to refine the crude wax.

Experimental: Extraction of South Arcot Lignite at atmospheric pressure.

An air-dry sample of −30, +60 mesh size was used in the extractions. 10 gms. of the sample were taken in a Whatman extraction thimble and extractions were conducted in soxhlet apparatus at atmospheric pressure. A variety of solvents like Benzol, Xylol,

Tetralin, etc., were tried, in addition to a mixed solvent, ethanol-benzene. In each case the extraction was allowed to proceed until the siphoned solution was colourless, thereby ensuring complete extraction of all the soluble components. The yield of extractives was determined by distilling off the solvent under reduced pressure. The last traces of the solvent were removed by placing it in an air oven at a temperature of 105-110°C for several hours until constant weight was recorded. The melting point of each extractive was determined by the open capillary tube method. Table I gives the results.

TABLE I

YIELDS ON EXTRACTION WITH SEVERAL SOLVENTS

No.	Solvent.	No. of siphonings.	% Yield, (on air-dry lignite).	m.pt. of extractive.	Appearance and Colour.
1.	Benzol	16	9·1	78°C	Dark brown, brittle waxy.
2.	Xylol	18	11·8	80	Dark brown, resinlike structure.
3.	Tetralin	16	11·6	95	-do-
4.	Tetrachloroethylene	26	7·7	84	Light brown, waxy.
5.	Chlorobenzene	30	8·1	96	-do-
6.	Pyridine	25	14·8	140	Black, hard and brittle.
7.	Ethanol-benzene 1:3	20	13·7	117	Dark brown resinlike.

Results:

The largest quantity of extractive is given by Pyridine and it is due to its peptising power. This is closely followed by the benzene-alcohol mixture. But these extractives have high melting points indicating a high C/H ratio in the waxes present and as such are somewhat limited in their uses. Tetralin and Xylol give the next largest amount of extractive and the latter gives a product having melting point quite close to that of standard commercial samples. Hence it was concluded that among the solvents tried, Xylol was the best in respect of yield consistent with quality.

The effects of particle size and moisture content of the lignite were studied for some of the solvents. The results show that, as a rule, the yield increases with the fineness of the particle size. But in practice, there was some difficulty due to choking in the case of very fine lignite. This was remedied to some extent by admixing the lignite with clean washed sand.

TABLE II

EFFECT OF THE CONDITION OF THE LIGNITE ON THE YIELD OF EXTRACTIVES

No.	Solvent.	Condition of lignite. Size.	Condition of lignite. Moisture content.	No. of Extractions.	% Yield Extractive on air-dry lignite).
1.	Rectified Spirit and Benzol, 1:3	-30, 60	Air-dry	20	13·7
2.	Absolute alcohol and Benzol, 1:3	-30, 60	Oven-dry	23	12·5
3.	-do-	-30, 60	Oven-dry 10·4% moisture added	25	13·6
4.	-do-	-30, 60	Oven-dry 15% moisture added	28	13·5
5.	Rectified Spirit and Benzol, 1:3	-4, 6	Air-dry	25	11·5
6.	-do-	-6, 10	-do-	26	12·9
7.	-do-	-80, 120	-do-	30	15·5
8.	Pyridine	-4, 6	-do-	20	12·5
9.	-do-	-6, 10	-do-	20	13·8
10.	-do-	-30, 60	-do-	24	14·5

The moisture content seems to play an important role in controlling the process of extraction. Oven-dry lignite extracted with a mixture of absolute alcohol and benzol yielded a definitely lesser amount of the crude extractive than the air-dry lignite (moisture 10·4 %). Remarkably enough, the yield improved and reached the original value when the oven-dry lignite was moistened with the requisite amount of water to bring the moisture content to that of the air-dry sample. However, no further improvement

was obtained when more water was added to the lignite. These experiments indicate that a small amount of moisture facilitates extraction, although there is no water soluble constituent in the extractives obtained. The exact mechanism of the phenomenon is not definitely known. The results are given in Table II.

Extraction of the lignite under pressure:

Experiments were conducted to determine the yield on equilibrium extraction of South Arcot lignite under various pressures at high temperatures and to study the effect of the variables on the yield. Commercial xylol was used as the solvent. The correlations obtained are sure to be of interest in designing large scale extraction plants.

The pressure extraction was conducted in a one-litre Chas. W. Cook's Tilting type stainless steel autoclave with a rated pressure of 350 atm. and a rated temperature of 350°C. Heating was done by a wire-wound electric furnace surrounding the autoclave. The temperature was controlled to $\pm$ 2°C for any length of time by means of a Wheelco Rheotrol Input Controller.

The autoclave was charged with a weighed amount of the air-dry lignite (50 gms) and 500 cc of Xylol. The mixture was kept agitated by rocking the autoclave and then it was heated. After attaining the desired temperature the mixture was maintained at the same temperature for a measured interval of time. Then the mixture was cooled, filtered and washed once with fresh solvent. The extractives were separated by distilling off the solvent under vacuum. The weights of the solvent-free residue and the extractive were determined. Extractions were done at various temperatures for a period of 2 hours. Also a series of runs were taken all at 200°C for periods ranging from $\frac{1}{2}$ hour to 5 hours. The results are tabulated in Table III.

Results:

The weight of the dried extractive was reported as % on the moisture-free lignite. The yields were also calculated following the method of Parr and Hadley by subtracting the weight of the dried residue and the weight of the moisture from the original weight of the lignite. The difference between the computed and experimental values for the yields, though small, increases with temperature because of the possible decomposition at higher

temperatures. This was evident in the runs conducted above 270°C, in which on opening the cooled autoclave, crude gas was found to escape from the autoclave indicating decomposition at higher temperatures.

TABLE III

PRESSURE EXTRACTION OF SOUTH ARCOT LIGNITE*

No.	Extraction Temp. °C.	Time of Extraction hrs.	Wt. of Residue gms.	Moisture-free basis			
				Extract calculated		Extract obtained	
				gms.	%	gms.	%
1.	30	2	41·42	3·68	8·25	3·54	7·8
2.	140	2	39·98	5·12	11·4	5·00	11·1
3.	170	2	38·72	6·38	14·2	6·32	14·0
4.	200	2	38·25	6·85	15·2	6·72	14·9
5.	240	2	36·24	8·86	19·8	8·51	18·9
6.	270	2	34·94	10·16	22·5	9·86	21·8
7.	300	2	34·97	10·33	22·9	9·91	22·0
8.	325	2	33·96	11·14	24·7	10·53	23·4
9.	200	½	40·94	4·16	9·2	4·05	8·9
10.	200	1	39·19	5·91	13·1	5·73	12·6
11.	200	3	38·10	7·00	15·5	6·82	15·1
12.	200	4	37·80	7·30	16·2	7·09	15·5

*Note: 50 gms. of air-dry (9·8% moisture) lignite was taken for each run.

The influence of temperature on yield is represented graphically (Fig. 2). The yield increases with temperature, but not proportionally. Since greater and greater decomposition was observed at higher temperatures, there is reason to believe that the yield may reach a maximum around 400 or 450° C. But owing to the limitation on the maximum permissible temperature of the autoclave used, a direct determination at a higher temperature was not possible.

The yield increases with time, but the graph (Fig. I) indicates that most of the extractives go into solution in the first hour after which there is very little increase.

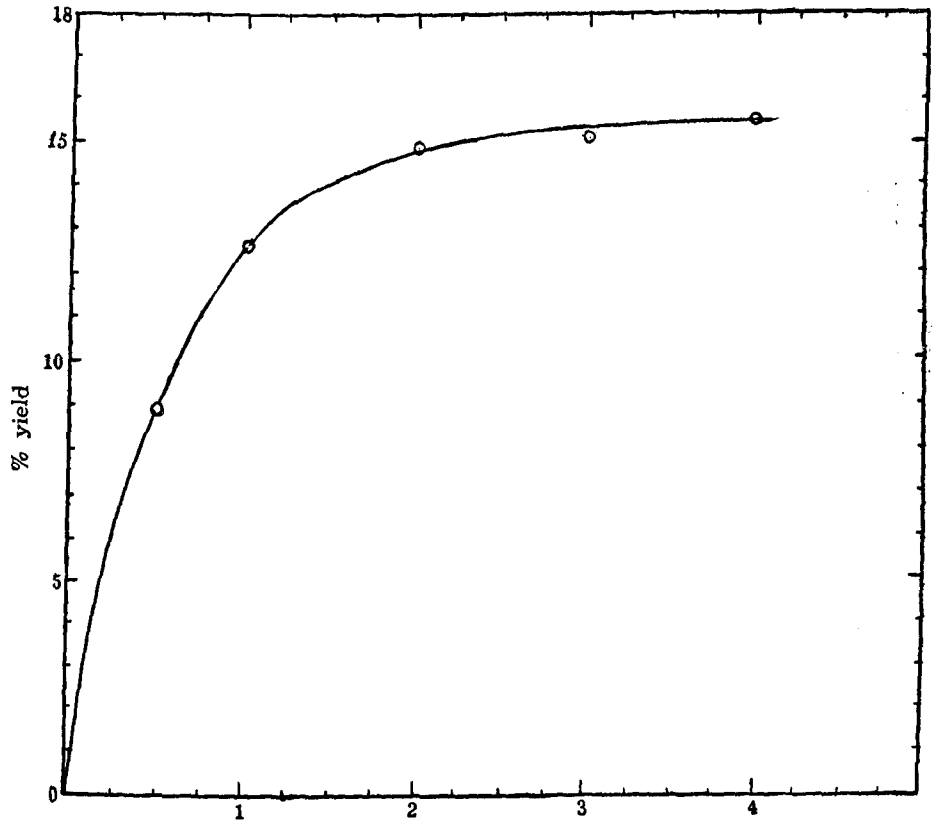

Fig. 1 Solvent Extration under Pressure (Yield *vs.* Time)

Properties of the Extractives:

The extractives as obtained or the crude montan wax as it is often called, is a dark brown solid with a varying degree of hardness, having a conchoidal fracture. It is a mixture of montan ester-waxes, resins and bitumens. The quantity of wax in the extractives was determined by dissolving away the resins in ether at room temperature in which the wax is insoluble. In order to compare the quality of the waxes obtained, their properties like the Acid Value, Saponification Value, Ester Value, etc. were determined. The procedure followed was according to Warth, p. 322. In the estimation of the saponification value the titration

of the dark coloured solution was done by conductometry. The values are given in Table IV.

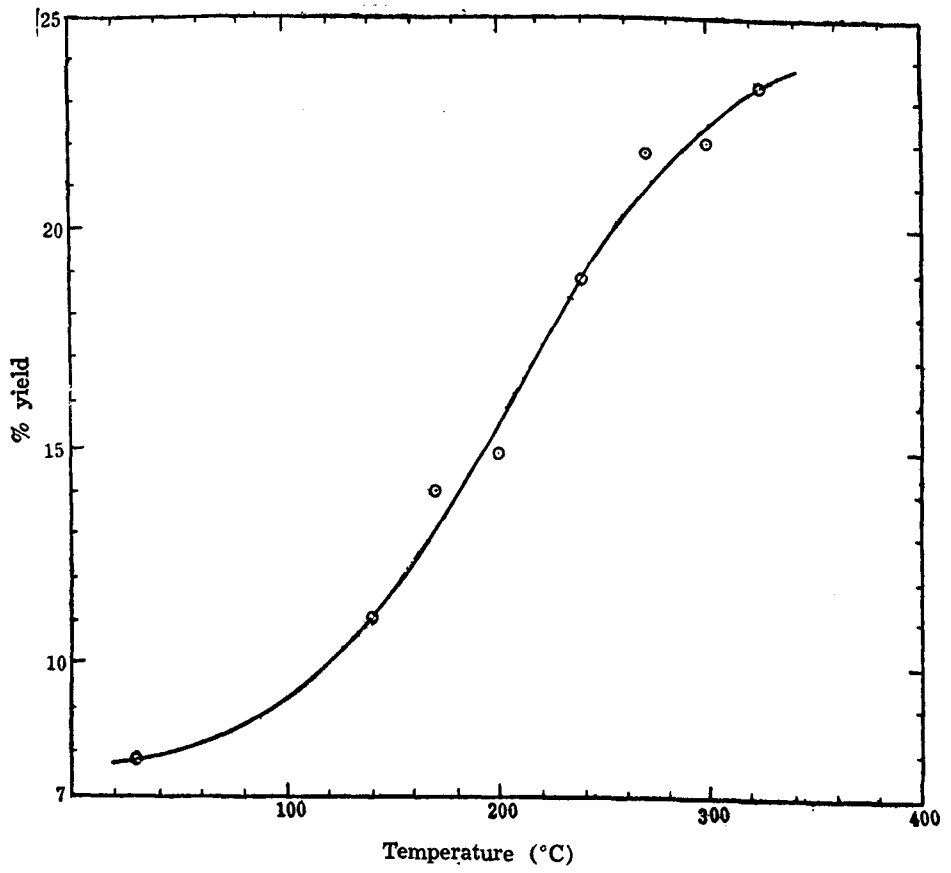

Fig. 2. Solvent Extraction under Pressure (Yield *vs.* Temperature)

A study of the results gives rise to the following conclusions. Larger yields are obtainable on extraction with Pyridine and with mixed solvents like benzene-alcohol. But in these cases the crude waxes have an appreciable resin content with a high melting point. The extractives with Tetrachloro-ethylene and chlorobenzene are comparatively rich in their wax content although the total yield is poor. The Xylol extraction conducted at various temperatures has indicated that although the total quantity of extractives obtained increases with temperature, much of the increase is due to the formation of larger quantities of resins resulting from the thermal depolymerisation rather than to the production of any wax.

TABLE IV

PROPERTIES OF THE EXTRACTIVES FROM SOUTH ARCOT LIGNITE

No.	Solvent Used.	Sap. Value.	Acid Value.	Ester Value.	Iodine Value.	Resin % Ether sol.	Wax % Petr. Ether sol.	M. P. of Wax °C
1.	Benzol	93·6	27·6	66·1	12·9	54·3	74·1	78-80
2.	Xylol	89·4	28·1	61·1	11·4	56·8	66·2	85-86
3.	Tetralin	98·5	32·1	66·4	14·8	45·8	49·5	93-95
4.	Tetrachloro-ethylene	99·0	31·4	67·6	10·7	36·1	69·3	85-89
5.	Chlorobenzene	102·0	24·3	67·7	12·5	31·8	68·4	79·82
6.	Pyridine	184·0	44·8	139·2	19·2	15·8	64·5	120-130
7.	Ethanol-Benzene, 1:3	126·0	36·1	89·9	12·1	42·8	59·3	74·80
8.	Xylol (200°C)	82·3	25·4	56·9	14·7	60·7	56·4	68·70
9.	Xylol (325°C)	78·8	24·5	54·3	15·1	63·4	47·5	66·68

Purification of Montan Wax :

The montan wax as separated from the crude extractives by a subsequent ether extraction of the resins is light brown to black in colour and for certain uses it needs further refining. The crude wax obtained by Xylol extraction under pressure at 325°C was purified by the following procedure. About 10 gms of the extractive was extracted repeatedly with petroleum ether at room temperature until the solution was practically colourless. The residue (resin) left behind was estimated. The Petroleum ether solutions were added together and treated with concentrated sulphuric acid in a separating funnel, and the acid layer run off. The treatment was continued until the wax solution was colourless, after which it was collected in a weighed china dish, the solvent evaporated, and the purified wax weighed. About 30 cc of the acid is used for the purification.

It was possible to prepare a refined montan wax, pale white in colour, amounting to 47·5% of the deresinified wax or 21·1% of the crude extractive. This works out to nearly 4·5% of the

weight of lignite. The properties of the wax are comparable to those varieties available commercially (Warth) although there is still scope for improving the colour. (Table V).

TABLE V

PROPERTIES OF MONTAN WAX OBTAINED FROM SEVERAL SOURCES

No.	Source	Sap. value.	Acid value.	Ester value.	Ether soluble.	M.P. °C.	Remarks.	Ref.
1.	South Arcot Lignite	84	36	48	20	73-75	Brownish white.	Authors
2.	German Lignite	76	33	43	14	74-82	Dark brown.	Cawley & King.
3.	Devon Lignite	75	30	45	40	73-83	-do-	-do-
4.	Standard Montan Wax	105	100	5	15	75	——	Berle & Lunge.
5.	Double Bleached Montan Wax	105	100	5	15	76	——	-do-
6.	Eisenreich	95	83	2	—	77	Yellowish brown.	Warth.

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On a Function Connected with $\varphi(n)$

BY

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1. Let $\varphi(n)$ denote Euler's function representing the number of numbers less than and prime to n. Let $\varphi_1(n) = \varphi(n)$; $\varphi_r(n) = \varphi(\varphi_{r-1}(n))$, $r = 2, 3, \ldots$. For a given n, $\varphi_r(n)$ decreases as r increases, and hence there is a least value of r, say r_1, such that

$$\varphi_r(n) = \varphi_{r+1}(n) = \ldots = 1.$$

S. S. Pillai proved that

$$\left[\frac{\log\ (n/2)}{\log\ 3}\right] + 1 \leqslant R(n) \leqslant \frac{\log\ n}{\log\ 2} + 1.$$

where $r_1 = R(n)$. In this paper I consider an analogous function $S(n)$ given by

$$S(n) = \varphi_1(n) + \varphi_2(n) + \ldots + \varphi_{r1}(n).$$

It is shown here that

$$R(n) \leqslant \frac{\log\ n}{\log\ 2} \text{ if } n \text{ is even}$$

and $$\leqslant \frac{\log\ (n-1)}{\log\ 2} + 1 \text{ if } n \text{ is odd,}$$

which is an improvement over Pillai's result;

Also

$$S(n) \leqslant n - 1 \text{ if } n \text{ is even}$$

$$\leqslant 2n - 3 \quad \text{,,} \quad \text{odd,}$$

while $S(n) \geqslant 2^{[\log\ (n/2)/\log 3]+1} - 1$.

I also consider the solutions of the equation $S(n) = n$, and show that each one of the following values of n provides a solution:

$$n = n_1,\ 3n_2,\ 3n_3,\ \ldots.$$

where $n_1 = 3^k,\ k = 0, 1, 2, \ldots.$

$n_2 = 1 + 4n_1$

$n_3 = 1 + 12n_2$

$n_4 = 1 + 12n_3, \ldots.$

provided these are primes. The question whether these are the only primes remains open.

2. *Theorem* 1. If n is even,

$$R(n) \leqslant \frac{\log n}{\log 2};\ S(n) \leqslant n - 1.$$

If n is odd, $R(n) \leqslant \dfrac{\log (n-1)}{\log 2} + 1$

$$S(n) \leqslant 2n - 3.$$

Proof: When n is even, $\varphi_1(n) \leqslant (n/2)$;

$$\varphi_2(n) \leqslant \frac{1}{2}\varphi_1(n) \leqslant \frac{1}{4}n; \ldots.$$

$$\varphi_r(n) \leqslant \frac{1}{2^r}n.$$

If $r = r_1 = R(n)$ we get

$$1 \leqslant \frac{1}{2^{r_1}}n,$$

$$r_1 \leqslant \frac{\log n}{\log 2} \qquad \ldots \quad (1)$$

If n is a power of 2, r_1 is actually $\left(\dfrac{\log n}{\log 2}\right)$

Again $S(n) = \varphi_1(n) + \varphi_2(n) + \ldots + \varphi_{r1}(n)$

$$\leqslant \frac{1}{2}n + \frac{1}{4}n + \ldots + \frac{1}{2^{r_1}}n;$$

$$= n\left(1 - \frac{1}{2^{r_1}}\right)$$

$$\leqslant n\left(1 - \frac{1}{n}\right), \text{ by (1)}$$

Hence $S(n) \leqslant n - 1$.

If n is a power of 2, it is easily seen that $S(n) = n - 1$.

Let n be odd. Then $\varphi_2(n) \leqslant \frac{1}{2}\varphi_1(n)$;

$$\varphi_3(n) \leqslant \frac{1}{2}\varphi_2(n) \leqslant \frac{1}{2^2}\varphi_1(n); \ldots.$$

$$\varphi_r(n) \leqslant \frac{1}{2^{r-1}}\varphi_1(n)$$

Hence $1 \leqslant \dfrac{1}{2^{r_1-1}}\varphi(n)$,

$$r_1 \leqslant \frac{\log \varphi(n)}{\log 2} + 1 \leqslant \frac{\log (n-1)}{\log 2} + 1 \qquad \ldots \quad (2)$$

If n is a prime of the form $2^k + 1$, we get the equality sign. Again

$$S(n) \leqslant \varphi(n)\,(1 + \tfrac{1}{2} + \ldots + (1/2^{r_1-1}))$$

$$= 2\varphi(n)\,(1 - (1/2^{r_1}))$$

$$\leqslant 2(n-1)\,(1 - (1/2^{r_1}))$$

$$\leqslant 2(n-1)\,(1 - (1/2n-1)), \text{ by (2)}$$

$$= 2n - 3.$$

Actually $S(n)$ attains this value if n is a prime of the form $2^k + 1$.

3. Before considering the lower bounds of $R(n)$ and $S(n)$ we prove the following:

Theorem 2.* For a given value of $R(n)$, the maximum value of n is $2 \cdot 3^{R(n)-1}$.

The proof of this depends on the

Lemma. $R(pn) > R(n) + (\log n/\log p)$, if p is a prime > 3. If $p = 3$ it becomes an equality.

Proof by induction: Suppose the result is true for all primes $q < p$ so that

$$R(qn) \geqslant R(n) + \frac{\log q}{\log 3} \qquad \ldots \quad (3)$$

* This is equivalent to Pillai's theorem II; but the proof by induction given here is different from Pillai's.

for all $n > 0$ and all $q < p$. We will prove the same for p. Let $p - 1 = 2^a q_1^{b_1} q_2^{b_2} \ldots$.

Then $$R(n) = 1 + R[\varphi(n)] \quad \ldots (4)$$

assuming as we may that $n > 2$; for if $n = 2$, the result of the lemma is obvious.

Also $$R(pn) = 1 + R(\varphi(pn)).$$

Assume that p is prime to n as a first case. Then

$$\begin{aligned} R(pn) &= 1 + R[(p-1)\varphi(n)] \\ &= 1 + R[2^a q_1^{b_1} q_2^{b_2} \ldots . \varphi(n)] \\ &= 1 + R[q_1^{b_1} q_2^{b_2} \ldots . \varphi(n)] + a, \end{aligned}$$

since $\varphi(n) \geqslant 2$ and $R(2m) = R(m) + 1$ if m is even;

Hence $$R(pn) \geqslant 1 + R[\varphi(n)] + a + \Sigma \left(\frac{\log qi}{\log 3}\right) bi,$$

by using (3) repeatedly,

$$\begin{aligned} &= R(n) + a + \left\{\frac{\log (p-1)/2^a}{\log 3}\right\} \\ &= R(n) + \frac{\log (3/2)^a (p-1)}{\log 3} \\ &\geqslant R(n) + \left(\frac{\log p}{\log 3}\right) \end{aligned}$$

if $(3/2)^a (p-1) \geqslant p$ which is true since $a \geqslant 1$, and $p \geqslant 3$. Since the result is true for $p = 3$, the lemma follows in the case when p is prime to n. Next let $(p, n) \neq 1$. Then in each of the pairs $(\varphi_1(pn); \varphi_1(n)); (\varphi_2(pn); \varphi_2(n)); \ldots .$ p occurs upto a stage, say in the first k pairs, and in the pair $(\varphi_{k+1}(pn); \varphi_{k+1}(n))$ the first member contains p while the second does not.

Let us call $\varphi_{k+1}(np) = pu$; $\varphi_{k+1}(n) = u$; then $(p, u) = 1$. Hence by the above result,

$$R(pu) > R(u) + \left(\frac{\log p}{\log 3}\right) \quad \ldots (4)$$

But

$$\begin{aligned} R(pn) &= k + 1 + R(pu) \\ R(n) &= k + 1 + R(u) \end{aligned}$$

Hence adding $k + 1$ to both sides of (A) we get

$$R(pn) > R(n) + \left(\frac{\log p}{\log 3}\right), \quad p > 3,$$

so that the lemma follows in this case also. To prove Theorem 2, let $R(n) = t$ (fixed), and consider all possible values of n satisfying this. The greatest of such n's, say n_1, must not contain a prime factor p greater than 3, for then

$$\begin{aligned} R(n_1) = R\left(\frac{n_1}{p} \cdot p\right) &\geqslant R\left(\frac{n_1}{p}\right) + \left(\frac{\log p}{\log 3}\right) \\ &\geqslant R\left(\frac{n_1}{p}\right) + \left[\frac{\log p}{\log 3}\right] + 1 \\ &= R\left(\frac{n_1}{p} \cdot 3^k\right), \end{aligned}$$

where $K = \left[\frac{\log p}{\log 3}\right] + 1$, since $R(3u) = R(u) + 1$ for all u.

But $\frac{n_1}{p} \cdot 3^k > n_1$. Hence n_1 is not the largest solution.

Let now $n_1 = 3u$; then $t = R(3u) = R(u) + 1$ so that $R(u) = t - 1$. Assuming that the theorem is true for all values of $R(n) < t$, it follows that the maximum value of u is 2.3^{t-2}. Hence $3u = 2.3^{t-1}$ and the theorem follows by induction. From the theorem it follows, as proved by Pillai, that

$$R(n) \geqslant \left[\frac{\log n - \log 2}{\log 3}\right] + 1. \quad \ldots (5)$$

Theorem 3. $S(n) \geqslant [\log (n/2)/\log 3] + 1$.

For $S(n) = \varphi_1(n) + \varphi_2(n) + \ldots + \varphi_t(n)$; $t = R(n)$

Now $\varphi_t(n) = 1$; $\varphi_{t-1}(n) = 2$; $\varphi_{t-2}(n) \geqslant 2^2$; $\varphi_{t-3}(n) \geqslant 2^3$; $\ldots$

Hence $$\begin{aligned} S(n) &\geqslant 2^{t-1} + 2^{t-2} + \ldots + 2 + 1 \\ &= 2^t - 1. \end{aligned}$$

The theorem now follows by using (5).

(4) *$S(n)$ as a function of $R(n)$.* We will show that

$$2^{R(n)}-1 \leqslant S(n) \leqslant 3^{R(n)}.$$

For a given $R(n)=t$, proceeding as above we get $S(n) \geqslant 2^t - 1 = 2^{R(n)} - 1$. When n is a power of 2, this becomes an equality. Again, by Theorem 2,

$$\varphi_t(n)=1;\ \varphi_{t-1}(n)=2;\ \varphi_{t-2}(n) \leqslant 2\cdot 3;\ \varphi_{t-3}(n) \leqslant 2\cdot 3^2 \ldots.$$

$$\varphi_1(n) \leqslant 2\cdot 3^{t-1}$$

Hence for a given t,

$$S(n) \leqslant 2\cdot 3^{t-1}+2\cdot 3^{t-2}+\ldots+2\cdot 3+2+1$$
$$=3^t.$$

$S(n)$ actually reaches this value when $n=2\cdot 3^{t-1}$.

4. Let us finally consider the solutions of the equation $S(n)=n$. By Theorem 1, n can only be odd. It is easily verified that two sets of solutions are $n=3^k$ $(k=0,\ 1,\ldots)$ and $n=3p$ where p is a prime $=4$. 3^k+1, $k=1,\ 2,\ldots$; we will prove

Teorem 4. If $S(3p)=3p$ then $S((p-1)/4)=((p-1)/4)$, where p is a prime >3.

Proof. $3p=S(3p)=2(p-1)+S(2(p-1))$

$$=2(p-1)+2S(p-1)+1,$$

using $\cdot S(2u)=2S(u)+1$ if u is even

$$=S(u) \qquad \text{if } u \text{ is odd.}$$

Hence $S(p-1)=\dfrac{p+1}{2}$

Since $p>3$, $\dfrac{p-1}{2}$ is even,

Hence $S\left(\dfrac{p-1}{2}\right)=\left(\dfrac{p-1}{4}\right)$,

Since $S(n)$ is always odd, $\dfrac{p-1}{4}$ must be odd, $p=4k+1$ (k odd);

$\dfrac{p-1}{2}=2k$, k odd $S\left(\dfrac{p-1}{2}\right)=S\left(2\dfrac{p-1}{4}\right)=S.\left(\dfrac{p-1}{4}\right)$

$=S$ since $\dfrac{p-1}{4}$ is odd.

Hence the theorem follows.

With the help of this result, we can derive an infinite sequence of classes of solutions of $S(n)=n$.

One is $n=3p$, where $\dfrac{p-1}{4}=3^a$ or $p=4\cdot 3^a+1=p_1$ say.

Another is $n=3\ p_2$ where p_2 is a prime given by $\dfrac{p_2-1}{4}=3p_1$

or $p_2=12p_1+1$. .... ..

This sequence of solutions can be continued indefinitely. It is conjectured that this sequence exhausts all solutions.

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Structure and Physiology of the Timber Borer *Bankia Indica* with Special Reference to Boring Habit [1]

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ABSTRACT

The role played by the shell, the adductor muscle and the foot in the boring process of *Bankia indica* is described after giving an account of their minute structure. A detailed account of the structure of the mantle and gills together with a description of the ciliary currents is given. The alimentary canal is described in detail with notes on the histology of the different regions so as to throw light on the adaptations to the peculiar diet to which the borer is committed. The course of the food particles through the alimentary canal is described. The results are discussed in relation to the findings of other workers.

1. *Introduction*

The Teredinidae as a group of molluscs has not only specialised towards a timber-boring habit but also has adapted their physiology to the exploitation of wood into which they bore as an important source of nourishment. These features have been attended with modifications in the structure of the organism to suit the new conditions of habitat and food. The present study is an attempt to elucidate several points in their anatomy which have a direct bearing on the boring habits of this bivalve. Such a study is an essential prerequisite for the formulation of the means for the control of these pests.

2. *Previous Work*

Historical: The most notable work on teredine anatomy is that of Sigerfoos (1908) who studied *Bankia gouldi, Teredo dilatata* and *Teredo navalis;* though studies on the anatomy have been

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made by earlier authors like Deshayes (1848), Quatrefages (1849), Grobben (1888), Menegaux (1890), Pelseneer (1891), and Beauk (1899). Important among the recent contributions on the anatomy of ship-worms are those of Potts (1923), on the structure and functions of the liver, Lazier (1924), on the morphology of the digestive tract, Yonge (1926 d), on the digestive diverticula, Miller (1922, 1924), on the shell and the mechanism of boring, Coe (1933, 1933a, 1934, 1935, 1936, 1943) on the sexual phases, and Purchon (1941) on the ciliary mechanism.

3. *Material and Methods*

The material for the following study was obtained from the wooden floats used for fishing off the Madras coast and also from test planks. The course of the ciliary currents in live specimens was studied with the help of current indicators like mercuric sulphide, powdered carmine and carborundum dust of various grades. Bouin-Dubosq was the fixative chiefly used, but Zenker's fluid, corrosive sublimate-acetic, osmic fluids and formaldehyde were also employed. Paraffin sections were cut at a thickness of from 4 to 10 microns and were stained with either Delafield's or Iron-Haematoxylin and counter stained with eosin or orange G. Mucicarmine was handy for the study of mucous glands.

4. *General Organisation of the Body*

Like other teredines, *Bankia indica* has a long, slender, cylindrical and soft body (Plate I). Covering only a small portion of the anterior end of the visceral mass, the irregularly shaped shell functions as the main organ for the excavation of the burrow and accommodates within it only the discoidal foot, the anterior part of the alimentary canal and the adductor muscles. The shell valves when in contact gape widely in front and behind for the projection of the foot and extension of the body respectively. The edges of the shell are not brought together because of the modified contact on the dorsal and ventral articulations (*vide* shell).

Fig. 1 shows the general arrangement of the organs in *B. indica* as seen from the right side when the shell and mantle are removed. Due to the lengthening of the body, the visceral mass occupies approximately a third of the body while the ribbon-like posterior part of the ctenidium (G) extends from the visceral mass to the base of the siphons (ES, IS). Most of the organs lie behind the posterior adductor muscle (PA) instead of anterior to

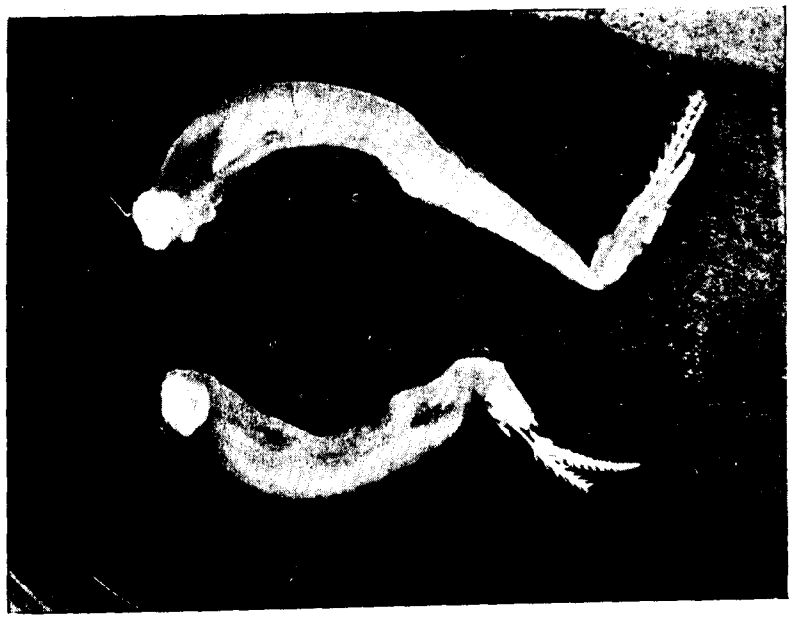
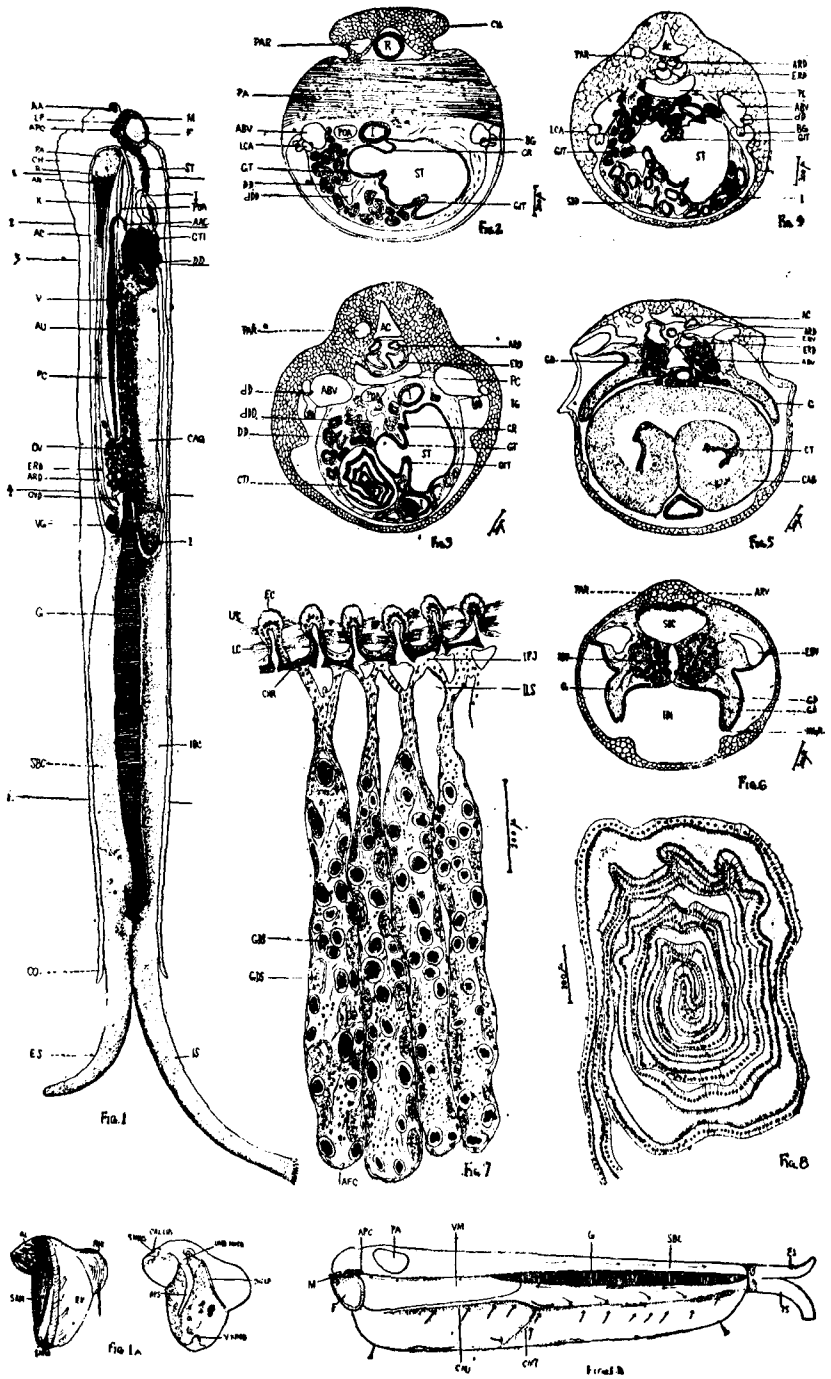


PLATE 1

Two entire Shipworms (*Bankia indica*)

it as in most lamellibranchs. The discoidal foot (F) occupies the anterior most portion in relation to its use as an organ holding the shell in position while boring. The pericardial cavity (PC) with its contained heart has not only considerably elongated but also has undergone rotation around the posterior adductor with the result it occupies a position above the intestine. Associated with these the relation of the various parts of the circulatory system has been changed. The posterior aorta (POA) continues in front of the ventricle while the anterior aorta (AAC) proceeds backwards. The kidney (K) and ureters come to lie on the upper side of the pericardial cavity and ventral to the anal canal (AC). The gonads are creamy white conspicuous bodies occupying the dorso-median and lateral portions. The renal and reproductive openings have been shifted behind as also has the visceral ganglion (VG). The digestive organs have not been rotated like the pericardium. The cylindrical stomach (ST) is surrounded ventrally and posteriorly by the irregular tubuless of the digestive diverticula (DD), the capacity of the stomach being greatly increased by the long cylindrical caecum (CAG), and the intestine (I) extendnig posteriorly around the caecum as a long loop. The mouth (M) and the anus (AN) retain their usual positions, the former between the anterior adductor and foot, and the latter on the dorsal side of the posterior adductor. The mantle cavity extends behind as a long cylindrical canal accommodating the ctenidia.

The shell: Fig. 1a. In an animal 18 cm. long the shell measures only 6·6 mm. in length and 7·6 mm. in height and therefore covers only a small front region of the body (Plate I). The valves are equal, each being inequilateral with a distinct anterior lobe, a median lobe, and a posterior lobe called the auricle. In outline the anterior lobe resembles a triangle which joins the median lobe by one of its long sides. The free angle of the lobe points forwards as a beak. At the dorsal free margin the outline is interrupted by a deep sinus from which a smooth callus is reflected over its surface. The outer surface of this lobe is marked by forty dental ridges, parallel to the ventral free margin, extending from the callus on the dorsal margin to the median lobe, where they meet those borne by the anterior region of the latter almost at right angles. The denticulated ridges of the median lobe run parallel to the anterior free margin and bear stouter denticles and are more numerous than those on the anterior lobe. Some of the dental ridges in the umbonal area appear worn out by friction. The dental ridges are turned upwards and con-



Figures 1—8

tinued into the middle region where the ridges become smooth and low and extend into the posterior region where they finally cease. The middle region of the median lobe is further marked by a shallow groove running down from the umbonal region widening as it reaches the ventral margin of the shell. The posterior region of the middle lobe is broadest, thick and convex. Posteriodorsally it extends into the auricle (Posterior lobe). The surface of the latter is marked by curved lines of growth which run parallel to its margin. This region of the shell is thin at the edges and slightly reflected outwards at the margin.

When viewed from within, the middle lobe appears to be pushed to the outside forming a trough in which is lodged a part of the anterior region of the animal. The front edge of the posterior lobe forms a shelf inside the shell projecting inwards into the hollow of the median lobe. Towards the extreme ventral edge of this lobe which is slightly bent inwards appears a sperical knob-like thickening of such a form as to permit a movable articulation with a knob borne by the opposite valve. This articulation is a feature peculiar to teredines and some pholads and marks the specialisation of the shell. Dorsally the margin of the middle lobe curves inwards to form a pronounced umbonal region, the tip of this region, the umbonal knob is of such a shape as to permit a freely movable articulation with the similar process of the opposite valve. The elastic ligament is reduced to a small squarish structure, since the two valves do not open and close. The movement of the shell is around the axis formed by the umbonal knob in the dorsal side and the ventral knob.

Hanging from the umbonal knob on the inner side of the shell is a broad sickle-shaped blade-like process, the apophysis accommodated within the concavity of the middle lobe. This plate serves for the origin of the pedal retractor group of muscles.

The three layers, periostracum, prismatic layer and the inner nacreous layer though present, differ from those of other bivalves in being relatively thickened and in the penetration of the prismatic layers by laminae of nacre. Over the dental ridges the periostracum is extremely thin, evidently worn out by friction in burrowing. It is evident that the shell of *Bankia indica* is highly specialised for performing boring movements.

The pallets: These peculiar calcareous structures which occur at the base of the siphons on either side beneath the muscular

collar of all the Teredinidae have been shown (Bartsch, 1922) to have a toxonomic value. The two pallets are pressed against each other so that the straight inner sides become apposed and the semicircular outer surfaces form a complete conical plug effectively closing the circular opening when the siphons are retracted within the burrow. If the salinity of the sea water should greatly change, or if poisons are introduced or the timber is taken out of water the pallets are thurst into the opening, closing the burrow as a shield protecting the soft animal inside. The retraction and protrusion of the pallets are brought about by a set of retractor and protractor muscles respectively. The adductors of the pallets facilitate the separation of the right and left blades of the pallet before the retraction of the pallets to enable the siphons to extend. The pallet consists of a series of upto 19 funnel-shaped, cone-in-cone structures which constitute its blade when the overall pallet length is 16·8 mm., which have been secreted and cemented in succession to a calcareous cylindrical handle. The cone-in-cone units of the blade are not of the same size, the largest one being found towards the base and the smallest at the distal tip, hence the blade tapers from base to tip. They are clearly separated, their distal margins covered with yellowish brown periostracum which is entire and separated from those of its neighbours, and drawn out into pointed processes laterally. The pallets are formed as a result of the secretory activity of the inner epithelium of the mantle at the posterior region and are formed in special pockets of the mantle. The handle is the first to be secreted and this is followed by the formation of the cone-in-cone units which are added on at the base as the animal grows. Hence the distal one is the oldest, since it is the earliest to be formed while the younger and larger ones are added in a series at the proximal part.

5. *The Internal Anatomy*

1. *The adductor muscles.* In sections the posterior adductor is about eleven times larger in area than the anterior adductor. Below the posterior adductor runs the terminal part of the intestines which bends over it in front, to terminate at the anus above it. Applied to the posterior face of the adductor lies the kidney. The adductor, unlike those of *Ostrea* and *Pecten* is homogeneous and consists of coarse muscle strands reddish in colour, stretching transversely across the anterior part of the body, from valve to valve. The muscle bundles are attached to nearly the entire inner surface of the outwardly turned auricles of the

shell and near its attachment to the shell the muscle becomes spread out so that it is thicker there than elsewhere. The anterior adductor is comparatively small and is attached to the outwardly turned anterior edge of the shell-valves just in front of the dorsal knob. This is also a compact bundle of similar striated fibres.

From their position of attachment on the valves and their size it is evident that these two muscles instead of contracting simultaneously as in typical dimyarians do so alternately drawing together the front and hind parts of the shell-valves causing the two valves to swing upon each other back and forth on the pivot formed by the dorsal and ventral knobs. This type of movement effects in boring. The large size and mode of attachment of the posterior adductor suggest that the valves can be brought together more vigorously in the posterior region than in the anterior and that this vigorous adduction is responsible for the powerful outward thrust of valves at the anterior part. The divarication of the front ends of the shell is of significance in the boring operations, the relatively feeble adduction through the contraction of the anterior adductor being sufficient to bring back the shell to the normal position (Miller, 1924).

1. *The foot*: The short, cylindrical foot of *Bankia indica* is situated at the extreme anterior end of the visceral mass ventral to the mouth (Fig. IF). This organ serves to grip at the burrowing end while the shell is rotated round this point of attachment during the drilling process. The front surface of the foot which is thus applied to the wood is cordate in outline, with a smooth cetral disc-like area or 'sole' (SO) bounded by elevated, wrinkled, peripheral, glandular margins (PR) which are ciliated and thrown into folds. This shape and structure are quite in keeping with its habit of using this organ for attachment. The median notch, marking the divisions between the muscle insertions of the right and left halves of the foot of *Teredo navalis* is absent in *Bankia indica*.

The distal end of the crystalline style-sac with the contained style lies close behind the epithelium of the central disc (Fig. 19) and extends backwards through the length of the foot. The anterior extensions of the digestive diverticula surround the sac of the crystalline style. The rest of the interior of the foot is packed with loosely arranged connective tissue containing muscle strands and blood spaces.

Histology: The folded rim of the foot is covered externally by an epithelium composed of very closely set columnar ciliated cells containing large nuclei. Large numbers of club-shaped vesicular glands sometimes grouped in bunches of varying sizes which are sub-epithelial open on the surface of the foot by slender ducts (Fig. 13) through small crypts. Basic stains act quickly on the granular cytoplasm of the gland. A deeply staining oval nucleus containing dark chromatin masses is present in each cell. Vacuolated cells also occur among the epithelial cells with large vacuoles surrounded by a thin layer of cytoplasm. The central disc-like 'sole' is formed of an outer layer of regularly arranged columnar non-ciliated epithelial cells with large round nuclei (Fig. 22). Beneath the outer epithelium of the foot, forming the main bulk of the organ lies loose connective tissue through which strands of muscles run in various directions. The histology of the connective tissue is not unlike that of turgescent tissues of other protractile organs. The cells are irregular with large intercellular lacunar spaces which can be filled with blood.

The musculature of the foot: The typical lamellibranch plan consisting of anterior and posterior retractors, as well as the protractors is modified in *Bankia indica*. The two anterior retractors run on either side of the foot from their origin on the anterior umbonal region of the right and left valves to their insertion on the peripheral ridge just beneath the ciliated epithelium (Fig. 19). The posterior pedal retractors are semicircular bands springing from the whole length of the apophyses and ending along the margins of the foot. The fibres are arranged all round the foot in such a way that their contraction must result in lessening the diameter of the foot. The protractors are comparatively smaller than the retractors and are attached to the anterior umbonal region close to the origin of the anterior retractor.

The Movements of the foot: are mainly two-fold, extension and retraction. The retraction may result in the withdrawal of the foot as a whole or of the 'sole' of the foot alone, producing a cupping action and facilitating attachments to the end of the burrow. The extension of the foot is mainly brought about by the flow of blood into the pedal sinuses and consequent turgescence. The blood flow is brought about by the contraction of the protractor muscle fibres which compresses the anterior part of the body driving the blood into the lacular spaces of the connective tissue of the foot. Due to the relaxation of the retractor muscles the

opening into the pedal sinus gets closed. Now the small fibres of the intrinsic muscles through their contraction diminish the diameter of the foot creating an area of increased pressure which tends to distend the foot sometimes 3 to 4 mm. beyond the anterior margins of the shell. The retraction of the foot is brought about by the contraction of the posterior retractor which increases the diameter of the foot, which thereby opens up the passage of blood into the foot and permits the blood to flow out. This is also aided by the rhythmic contractions of both the retractors which drive away the blood from the lacuna of the pedal mass due to general contraction of the foot. The constant changes of the contour and the thickness of each of the folds of the epithelium forming the rim of the foot are caused by the intrinsic muscle fibres in those regions.

3. *The mantle.* This 'fold of the integument of the dorsal part of the molluscan body' (Cooke, 1895) consisting of two membraneous flaps lining the two valves of a typical bivalve, has in *Bankia indica* and other ship-worms fused along its edges resulting in the formation of a long and delicate tube open in front for the protrusion of the foot and produced into long siphons behind for the ingress and egress of the water. The mantle is thin, soft, translucent, and not of uniform thickness. Anteriorly, the mantle is in close relation with the shell, it extends underneath the shell and ends in a slightly thickened edge below the front edges of the shell. On the hind edges of the shell the mantle is raised up and extends over the umbonal region as a thickened muscular ridge. This duplication of the mantle, called the 'cephalic hood' by Quatrefages (1849) was considered by him as the organ directly concerned with boring into wood. Sigerfoos (1908) and Kofoid (1921) were of opinion that it fitted into the burrow like a washer around the 'head' preventing fine particles of wood from entering the burrow. In all the animals examined the 'cephalic hood' (Figs. 1 & 2 CH.) appeared very calloused suggesting frequent rubbing against the walls of the burrow. The rest of the mantle except the siphons is thin and the organs of the pallial cavity are seen through it in the fresh animal and it is responsible for the secretion of the nacreous layers of the shell, the calcareous protective tubing, the apophyses and the dorsal and ventral knobs. Since, the entire surface of the mantle is thus capable of deposition of calcium, the opening area of the burrow, the oldest region, occupied longest by the animal has received the maximum deposition of calcium.

In cross sections of the animal, the mantle, on either side of the median line ventrally shows two prominent longitudinal thickenings (Figs. 3, 4, 5, & 6). The exact significance of these two ridges which are absent in *Teredo navalis* and *Teredo megotara* (Purchon, 1941) is not clear. It is probable that these folds from incomplete longitudinal partitions of the mantle chamber helping to some extent in regulating the movement of sediments accumulating in the mantle cavity.

The anterior edge of the mantle is thickened and consists of three lobes typical of lamellibranchs (Fig. 12). The outer lobe (OM), formed by the turning upwards of the mantle is nearest the shell while the inner lobe (IM) formed by the extension of the inner epithelium of the mantle projects forwards away from the shell. Between these two the edge forms the middle lobe (MM). Between the middle lobe and the outer lobe runs the periostracal groove (PEG) which is shallow in *Bankia indica*. This groove is lined by small flat secretory cells with deeply staining contents, which become continuous with those of the middle lobe and the tall and columnar cells of the outer lobe lying adjacent to the shell. The epithelium of the inner lobe consists of cells about half as high as those in the middle lobe. The middle lobe which is primarily sensory in function and bear tentacles and even eyes in forms like *Pecten* and *Spondylus* is devoid of any special sensory structures except at the tips of the siphons where sensory papillae are found. That part of the middle lobe forming the periostracal groove and its duplication over the shell are secretory. The fold of the outer lobe over the shell is responsible for the ridges and denticles resulting in the growth of the shell by marginal increment. Strands of periostracum (PE) are formed from the secreting cells at the bottom of the periostracal groove, where the epithelium is thicker than on the sides. Several such fibres coalesce to form thick strands which pass over the edge of the shell to the outer surface. The outer epithelium of the outer lobe lying below the shell secretes the prismatic part of the shell.

The Siphons: The Siphons are formed by the fusion of the mantle edges at the posterior end. The inner lobe of the mantle takes part in the formation of the siphons. The inhalant and exhalant siphons are joined together for about half their lengths, their openings fringed with sensory tentacles and papillae which perceive even suspended particles. Since the animal is buried inside the wood the siphons are the only visible soft parts of the

body in the natural habitat. The surface of the inhalant and exhalant siphons are mottled with small scarlet or brown spots. The interior of the former is uniformly of a light brownish hue except for six slightly raised white longitudinal patches running lengthwise. The inhalant siphon is, in normal life extended to about 5 cms. beyond the opening in an animal whose burrow measured 12 cms., the exhalant siphon is shorter, thinner and bears sensory papillae which are absent in *B. gouldi*. The withdrawal of the siphons into the burrow is brought about by the contraction of the siphonal retractors while their extension is through the relaxation of these muscles and by the flow of blood into the sinuses. Due to the arrangement of their intrinsic muscles the siphons can also shorten their length and diameter and thus increase or decrease the force of the current passing through them. As the inhalant siphon is wider than the exhalant, the inflowing current is less powerful in this form than in deposit-feeding forms like *Abra* and *Scorbicularia* (Yonge, 1949). In this respect the present form resembles forms like *Gari* and *Donax* (Yonge, 1949). The exhalant siphon is capable of bending and twisting movements. When faecal filaments are forced out, the exhalant extends sometimes even beyond the inhalant siphon and the excurrent water is ejected so forcibly and in such a swirling manner as to prevent the contamination of the neighbourhood. Both inhalant and exhalant are capable of constricting their distal end, and are sensitive to touch, light and salinity of the medium.

Structure of the Siphonal walls: (Fig. 11). In cross sections the siphons show a musculature far more specialised than the rest of the mantle. There are four layers of circular fibres separated by three layers of longitudinal fibres. Of the three longitudinal fibres the middle one is very thick and of the circular layers the outer is thinnest being two fibre thick. In the disposition of these muscle layers the present form resembles *Donax* (Yonge, 1949) except in the relative thickness of the various layers.

The histology of the mantle: The mantle consists of three layers. The outer layer is epithelial in character. It becomes thicker anteriorly and gets folded in the region of the cephalic hood where the cells are distinctly tall and columnar. Iron haematoxyln reveals large nuclei with prominent nucleoli arranged towards the base of the granular cells (Fig. 20). The rest of the epithelium is of secretory cells responsible for the formation of the calcareous lining of the burrow (Fig. 14). The inner epithelium is composed

of very flat cells the boundaries of which are not discernible. The protoplasm shows a faint reticular structure. On the side facing the mantle cavity the cells bear cilia which are especially long along the lateral margins of the mantle opposite the marginal and branchial grooves. The cells along these tracts of the mantle are tall, columnar and resemble those of the outer epithelium. The middle layer is of loose connective tissue. It is filled with numerous blood spaces and traversed by muscle bundles and nerve fibres. Longitudinal and circular muscle bundles can be made out beneath the outer epithelium while oblique fibres run between the two epithelia. Among the cells of the connective tissue of a piece of mantle freshly removed are spherical nodular bodies. Deshayes (1948) thought them to be non-nucleated mucous cells. Hancock (1845), on the other hand, believed them to be silicious particles responsible for the formation of the burrow. Sigerfoos determined them as deposits of calcium used for the formation and thickening of the calcareous tube which lines the burrow. These being opaque in transmitted light and white in reflected light and they being soluble in water together with their becoming russet when treated with iodine suggest their being of the nature of glycogen. They are probably reserve food stored in the mantle as glycogen. Alcohol in which specimens have been preserved turns turbid due to the re-precipitation of this glycogen.

Glands of the mantle : Numerous mucous glands are found in the anterior region of the inner layer of the mantle. These mucous cells appear distended with mucous and bulge slightly beyond the general epithelial surface. They bear short cilia at their free ends and have their nuclei on the proximal part of the cell. Large numbers of them occur crowded together along two lateral tracts which unite into a median tract behind the visceral mass and extend to the base of the inhalant siphon. Mucous glands are also concentrated along the dorso-median line of the roof of the supra-branchial cavity (Fig. 6). The composite gland composed of numerous vesicular acini lined by flattened, non-ciliated slightly granular cells described by Sigerfoos in *Bankia gouldi,* is absent in *Bankia indica.*

The ciliation of the mantle and course of water-currents in the mantle cavity. Tests with carmine particles and carborundum powder showed the course of water currents set up by the different groups of cilia. The cilia borne by scattered groups of cells on the inner mantle surface beat in such a manner that

particles falling on them are swept towards two distinct ciliated tracts which lie close to the branchial grooves in the anterior region and thence running backwards as a single tract behind the visceral mass. The cilia along these tracts beat vigorously backwards pushing the particles entangled in the mucus to the base of the inhalant siphon. Of the particles brought into the body by the inhalant siphon the coarse ones sink down by gravity over the gill-surface to the floor of the mantle cavity. The small particles impinging on the mantle surface are swept and fed on to the current caused by the ciliated tracts and sent to the base of the inhalant siphon and collected in a small space close to the base of the latter. When a sufficient mass of these is thus accumulated the particles are forcibly ejected by the contraction of the mantle as 'Pseudo-faces'. The finer particles form a slender rope with the mucous and are carried along the marginal and branchial groove towards the mouth. In the region of the labial palps the scattered groups of cilia function in a different manner. They beat forwards instead of downwards and backwards as those of the inner face of the mantle and drive forwards particles. In this way the particles of wood scattered during boring are prevented from passing backwards and clogging the burrow. The epithelium of the visceral mass of *Bankia indica* is non-ciliated unlike that of *Teredo norvegica* (Purchon, 1941) and hence there are no water currents over the visceral mass.

6. *The Alimentary Canal.*

The morphology of the digestive tract of teredines have been studied by Deshayes (1848), Beauk (1899), Sigerfoos (1908) and Lazier (1924) while Potts (1923), and Yonge (1926) have described and commented on the digestive diverticula of *Teredo Sp.* and *T. norvegica* respectively. In furnishing a complete account of the anatomy of this form *B. indica* hitherto not studied, it is hoped that comparisons with other forms will be facilitated.

The alimentary canal of *Bankia indica* measures 10 cm. in a specimen having a length of 11 cms. and is similar to those of *Bankia gouldi* and *Teredo navalis* in its general course. (Fig. I). The *mouth opening* is a medium transverse crescentic slit (M) whose concavity is dorsal, located below the anterior adductor muscle and above the foot and bounded laterally by the labial palps (LP). The labial palps in *B. indica* are small but distinct structures, the outer ones being larger than the inner ones. The

epithelium lining the mouth opening is a continuation of the epithelium of the inner surface of the labial palp and is composed of ciliated columnar cells among which occur large numbers of mucous glands and wandering phagocytes. Some of the epithelial cells have vacuolated cytoplasm containing prominent nuclei which vary from a round to an elliptical shape and are scattered through the centre and base of the epithelium. The cells covering the inner surface of the palp are tall while those of the outer surface are flat and non-ciliated with no mucous glands. The epithelium is underlined by a well defined basement membrane and the surrounding connecting tissue consists of large irregular cells with a few strands of circular and longitudinal muscle fibres lying immediately below the basement membrane. The mouth leads into the *oesophagus* which is a dorso-ventrally flattened ciliated tube measuring 1·1 mm. This narrows posteriorly and has its walls thrown into longitudinal folds of which a dorso-median ridge divides the tube imperfectly into a right and left portions. *The oesophagus* has a wall composed of three layers (Fig. 16) continuous through the whole length of the gut, an outer layer of loose connective tissue, a basement membrane and a columnar ciliated epithelium. The connective tissue is composed of loose irregular cells between which pass strands of muscle fibres and blood sinuses. The epithelium has a border cuticle and interspersed among the cells are many mucous glands as well as phagocytes some of which appear to migrate through the basement membrane. The oesophagus leads into a spacious, long, almost cylindrical *stomach* (ST) whose posterior part is slightly dilated. The stomach is divisible into a proximal part which lies high in the visceral mass receiving the oesophagus anteriorly and a distal part which lies lower in the visceral mass behind the posterior adductor communicating with the caecum and intestine.

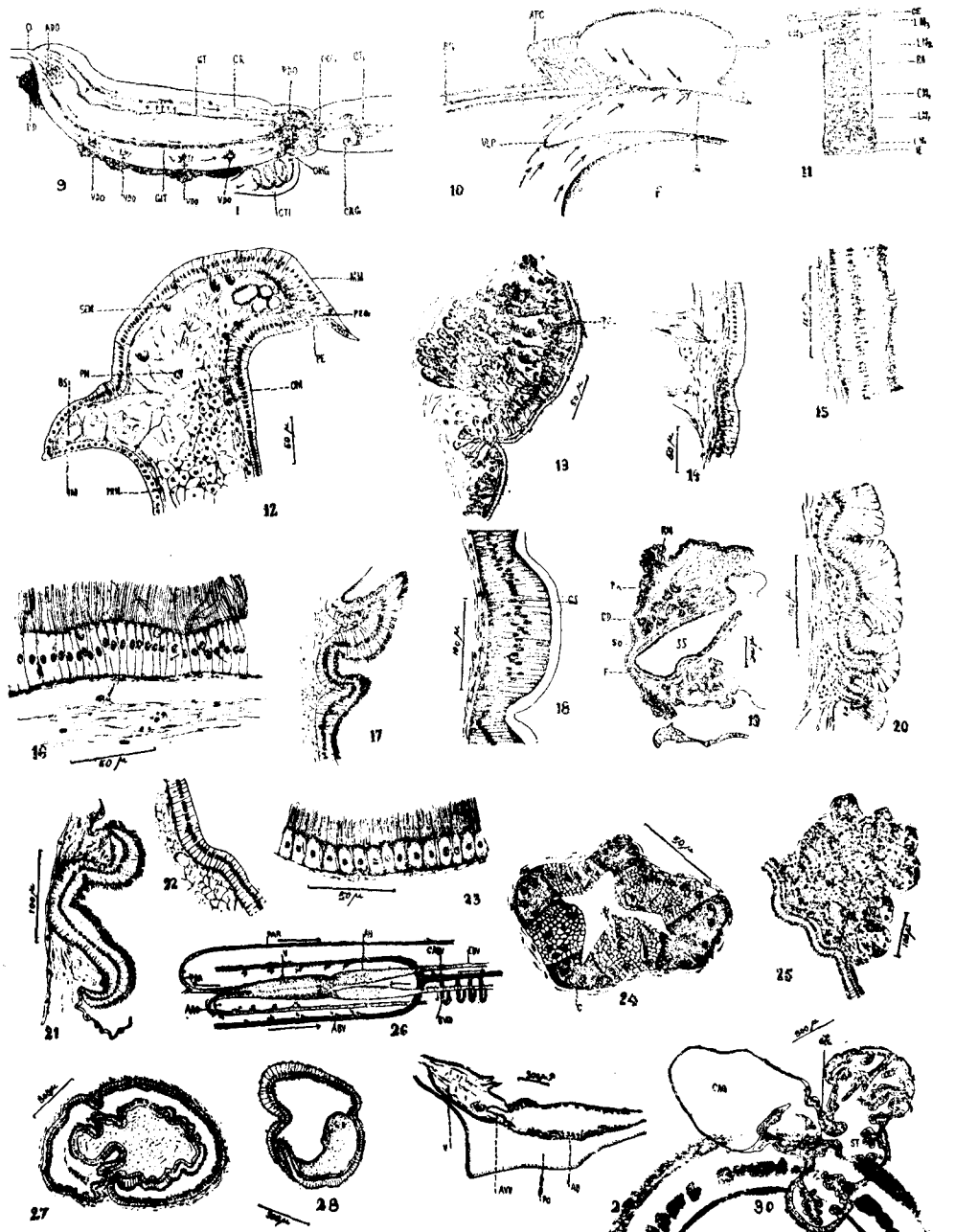
When casts of the stomach were made, the presence of three longitudinal ridges on the ventral wall extending almost the whole length of the stomach was revealed. Lazier (1924) has named these ridges in *Teredo navalis* as (1) *gastro-intestinal typhlosole* (2) *gastric typhlosole* and (3) *ciliated ridge*. 1. The *gastro-intestinal typhlosole* (GIT) starts in the lateral pouch (*vide infra*) and passes across the stomach ventral to the oesophageal opening and runs posteriorly along the ventral wall towards the right side of the stomach and continues into the posterior opening of the digestive diverticula and encircles the lumen of the posterior lobe of the digestive diverticula. This typhlosole emerges at the dorsal

edge of the hepatic opening and then passes posteriorly and ventrally into the intestine where it enlarges and coils forming a large coiled typhlosole of the intestine. (Fig. 3 CTI and Fig. 8). It is continued further as a low ridge and eventually disappears at the place where the intestine takes the bend at the posterior end of the caecum. From here to the anus there is no typhlosole for the midgut and hindgut. It is interesting to note that in *Bankia gouldi*, a small typhlosole extends throughout the whole intestine. Deshayes (1848) and Quatrefages (1849) on the other hand, describe the intestine of the forms they studied as being of uniform diameter indicating the absence of a coiled typhlosole. 2. The *gastric typhlosole* (Figs. 2, 9, GT) originates in the dorsal caecum and passing dorsal to the oesophageal opening runs side by side with the gastro-intestinal typhlosole and extends up to the posterior hepatic opening. 3. The *ciliated ridge* (CR) runs parallel to the free edge of the gastric typhlosole and ends on the right side of the caecal orifice. The cells of the gastro-intestinal typhlosole, gastric typhlosole and ciliated ridge are columnar, and vacuolated bearing long cilia. Their nuclei are prominent and sub-spherical.

Besides these three major folds is a row of transverse folds between the ciliated ridge and the gastro-intestinal typhlosole extending from the posterior end of the gastric typhlosole to the right fold in the caecal orifice. Another row of folds and furrows starts from the left fold in the caecal orifice and takes a spiral course to the right fold. These are visible through the surface of the stomach. Lazier (1924) believes that these irregular corrugations serve to increase the surface or the capacity of the stomach. But observations on living animals, however, suggest that this elaborate mechanism found especially towards the opening of the caecum at the junction of the latter with the stomach is only for sorting and guiding the food particles into the three channels namely, the caecum, the intestine and the openings of the digestive diverticula. The stomach is lined by an epithelium whose cells are not of uniform height, those on the roof being shorter when compared to the tall and columnar cells lining the floor. The whole epithelium is ciliated except for that region covered by the gastric shield. The ciliation is especially dense on the typhlosoles and the ciliated ridge. The nuclei are oval and prominent and are orientated along the central part of the epithelium (Fig. 15). The cells beneath the gastric shield do not bear any cilia and (Fig. 18) the nuclei which they bear vary from a round to an elliptical shape and are orientated at different levels, but the majority occur

towards the central part of the epithelium. Mucous glands do not occur but wandering phagocytes have been found both among the epithelial cells as well as among the connective tissue.

Diverticula of stomach: The stomach bears five distinct kinds of diverticula, namely 1. *the caecum* (CAG), 2. *the sac of the crystalline* style (Fig. 19 SS), 3. the *dorsal caecum*, 4. the *lateral* pouch, and 5. the *digestive diverticula*. (1) The *caecum* is a long cylindrical blind tube extending from the stomach to the posterior end of the visceral mass and forms the largest part of the alimentary canal. The intestine makes a long loop around it (See Fig. 1.). Dorsally it is covered by the gonads and dorsolaterally by the ctenidia and branchial groove. This organ is usually filled with wood particles. The ventral wall of the caecum is unfolded to form a two coiled typhlosole extending through the entire length of the caecum (Fig. 5, CT). This fold is of independent origin and not homologous with the fold of the intestine and increases the surface area of the caecum. The surface of the typhlosole is further thrown into many smaller folds resulting in the formation of additional longitudinal folds. The free edge of the typhlosole is further rolled up, considerably increasing the internal surface area of the caecum. The wood particles held between the folds of this typhlosole are found in a state of dissolution suggesting that a certain amount of digestion of wood takes place in the caecum. In *B. gouldi* the interior of the caecum is lined by a ciliated mucous membrane (Sigerfoos, 1908). However, in *B. indica* such a ciliated membrane appears absent as in *T. navalis* and there is no sign of ciliary movements in the living animal. The gastro-caecal orifice (Figs. 2, 9, 30, OCG) is guarded by two lateral folds of the wall, that on the right side being continuous with the caecal typhlosole. In transverse sections through this orifice a series of vertical incomplete partitions are seen and there is considerable development of musculature, both longitudinal and circular around the orifice. A transverse valvular fold also is present opening towards the caecum orientated towards the left side. The lateral folds divide the orifice incompletely so that two separate openings are provided probably one for the entrance and the other for the exit of wood particles in and out of the caecum. The cavity of the caecum is lined by non-ciliated flat epithelial cells the cell walls of which are very difficult to distinguish giving the appearance of a syncitium. The nuclei of the cells are spindle shaped and lie flat in the cells. The cells are so flat that they are hardly higher than the diameter of their nuclei. The cell boundaries are not perceivable



Figures 9—30

but probably exist. The distal tip of the folded typhlosole is, however, different in the histological details. The cells in this region are columnar and broad with vacuolated cytoplasm. In between the two folds of the typhlosole there is a considerable amount of connective tissue composed of cells bearing deeply staining nuclei. These cells become shorter and shorter and eventually become flat towards the proximal part of the typhlosole. 2. The *sac of the crystalline style* (Fig. 19, SS) is pyriform and oval in cross section. It lies in the lower side of the cavity of the foot with its broad end very close to the epithelium of the central disc of the foot. In live specimens when the foot is turgid, the style could be seen through the epithelium of the foot and could easily be squeezed out by making a puncture in the 'sole' of the foot. The style-sac opens into the stomach at its left side on the anterior end. At the anterior end of the stomach where the style sac arises the epithelium of the stomach is raised into a ring which protects the style from the action of the fluid in the stomach which is more alkaline. An epithelial fold overhangs the opening of the sac into the stomach preventing the particles in the stomach from getting into the style sac. The epithelium of the style sac is composed of a single layer of ciliated cells with clear cytoplasm containing large and oval nuclei, situated almost towards the centre or slightly disposed towards the distal side of the cell and contain one or two nucleoli. From the distal end of each of the epithelial cells projects a number of large bristle-like cilia. Yonge (1926) has shown in *Ostrea edulis* that the cilia covering the epithelium are continued into the body of the epithelial cells where they form 'an internal-fibrillar apparatus'. These fibres are greatly thickened below the nucleus and form "a bundle of thick rod-like bodies". A series of intra-epithelial canals also have been described at the base of the epithelial cells, the function of which is to give additional strength to the epithelium. In *Bankia indica* these structures seem to be absent probably due to the relative smallness of the style in proportion to the stomach as compared with *Ostrea*. The epithelial cells rest on an underlying basement membrane surrounded by a few strands of circular and longitudinal muscles, which, in turn, is enveloped by the connective tissue. A small groove runs through the wall extending from the mouth of the sac to its distal end to join the *appendix* of the style sac. This appendix of the style sac is a small tubular extension of the blind end of the style sac. The cells composing the groove are different from the rest in histological details but identical to those of the

non-ciliated granular cells lining the appendix of the style sac. The cytoplasm is granular with their nuclei disposed towards the base. The wall of the appendix has a lining epithelium closely resembling that of the groove. The cells forming it are not of uniform thickness those arranged on its roof being very thin and flat. The granular, nonciliated nature of the cells suggest that they are secretory and contribute to the formation of the style. The *crystalline style* is a gelatinous flexible body of a glassy transparency shaped like a club. Its widest part is near the central disc of the foot while the tapering part projects across the cavity of the stomach. Styles carefully dissected out showed spiral markings suggesting the rotation of the style by the cilia lining the sac as observed by Nelson (1918). The style in *B. indica* is smaller than those of *Martesia* and *Mya*. Since the style matter is continuously added on, the style was found retaining its bulk in all the specimens assisting both in digestion by the liberation of enzymes as well as in the mechanical process of pushing the particles of wood in the stomach. Transverse section of the *crystalline style* shows that it is composed of a homogeneous substance. The inner core of food materials reported for forms like *Anodonta and Lampsilis* (Nelson, 1918) is absent in *Bankia*. The *gastric shield* (Fig. 18) which in other bivalves functions as a protective shield against the abrasive action of the tip of the rotating style covers only a very small area of the stomach wall in *B. indica*. In freshly opened specimens the distal end of the style was found to be drawn out as a long flabby thread and it is difficult to conceive that the head of the style in a state of dissolution is pressed against the gastric shield as in other forms. This shield is closely applied to the gastric epithelium and can be easily dissected out of the animal. In sections the gastric shield is seen as a cuticular laminated structure in close relation with the underlying epithelium. According to Gutheil (1912) this structure is secreted by the epithelial cells that lie below. Pelseneer (1906), described it as the cuticular lining of the stomach and Johnstone (1899), called it as a gelatinous layer. Nelson (1918), who considered it to be chondrin named it as the gastric shield. Yonge (1926), is of opinion that the gastric shield is not a secretion but 'formed by the fusion of cilia originally in response to the irritation caused by the head of the style'. Berkely (1935), found the gastric shield composed of chitin. 3. The *dorsal caecum* is an outpocketing of the anterior, dorsal wall of the left side of the stomach. The epithelium of this region is folded and ciliated

internally. Since this sac also is filled with particles of wood it is probable that it serves as a receptacle of large particles of wood too many of which would block the proximal region of the stomach and impede normal functioning. 4. The *lateral pouch* is a distinct outpocketing of the stomach wall in between the dorsal caecum and the sac of the crystalline style. Since the wall of this pouch shows several muscle fibres it is probable that this sac by its pulsations aid in the movement of food in the stomach. A double fold of epithelium overhangs this pouch. The pouch is lined by tall non-ciliated columnar cells, which are not of uniform thickness. The epithelial lining is folded and rest on a thin basement membrane. Longitudinal and circular muscles and irregular connective tissue cells surround the epithelium.

5. The *digestive diverticula*: Yonge (1926 a) to whom we owe our knowledge of the assimilative function of these organs designated the term digestive diverticula. Since either 'liver' or hepatopancreas implies an assumption regarding its homology and since these diverticula of the stomach are accepted as digestive in function they are referred to as digestive diverticula.

These digestive diverticula in *Bankia indica* are composed of numerous small blind tubules, and occupy the ventral region and posterior side of the stomach and also extend into the foot and communicate with the stomach by ducts (See Figs. 1, 2, 3, 4, 9, 24, 25 and Ph.M. 1). The diverticula are distinguishable into three groups according to their distribution around the stomach. Most of the anterior diverticula occupy the interior of the foot forming a system of blind tubules, opening into the anterior part of the stomach by a number of opening (Fig. 9 ADO) on the right wall and by a single large opening in the anterior wall of the lateral pouch. Below the stomach closely applied to its wall and opening into it by four orifices form the second group (VDO). The posterior mass which is by far the largest is distinguishable into a large more dorsal right half and a small left half. The right part is brownish green and lies on the right side of the posterior part of the stomach while the left part is fawn coloured composed of few tubules and containing particles of wood. Although these two regions of the digestive diverticula differ in structural details they open into the stomach by a single large circular opening (PDO) on its ventral wall posteriorly. Neither Sigerfoos (1908) nor Lazier (1924) have described the histological structure of the *digestive diverticula* in detail. Our present knowledge is due to

PHOTOMICROGRAPH 1

Section through the foot showing the glandular development

the contributions of Potts (1923) and Yonge (1926 d). As in other teredines the unspecialised diverticula are composed of thick-walled columnar cells forming the tubules considered by Potts (1923) as excretory, whereas the specialised diverticula consist of thin walled wide tubules which are specially designed for the reception and intra-cellular digestion of wood. The lumen of the former is not of uniform diameter and in cross sections they appear as cruciform or triradiate (Figs. 4, 25, and Ph. M. 2). The cells forming these diverticula are irregular in shape and of varying heights with indistinct cell outlines. Two types of cells are distinguished in the epithelium of the tubule, those occupying the extremities of the arms (Fig. 24 c) of the lumen stain deeply and contain roundish nuclei. The others are lightly-staining larger cells with large vacuoles, dirty brown or yellowish pigment bodies, refringent granules, rod shaped as well as irregularly shaped bodies. Since fragments of these larger cells are found free in the lumen of the organ it is possible that they are cast out of the epithelium when loaded with the refractile and pigmented bodies. The darkly staining small cells found at the bases of the larger cells and at the extremities of the arms are considered (Yonge, 1926 a) as young cells which by dividing are able to make good the loss resulting from the casting off of larger cells. This suggestion is supported by the fact that division stages can be observed in the small cells and that this fact has been recorded by Gutheil (1912) in *Anodonta* and Yonge (1923) in *Mya arenaria*. The *specialised digestive diverticula* are different in histological structure. The epithelium is formed of circular, oval or irregular cells whose boundaries are indistinct or ill-defined. They contain clear vacuoles and circular nuclei containing darker nucleoli. Many cells with wood fibres within them are found loose or in groups within the cavity of the diverticula. These cells are amoeboid in character and movements. Ciliated columnar epithelium consisting of granular cells with basal nuclei line the duct and ductules of the digestive diverticula. They are placed over a thin basement membrane supported by a layer of connective tissue (Fig. 25). Enveloping this is a layer of circular muscle strands which pass round the duct.

The digestive diverticula have a sheath of muscle fibres which may aid in the circulation of food through the lumen. The ducts being lined by ciliated epithelium help the movement of particles. These facts and the presence of wood particles in the fawn

coloured region lead to the inference that this region of the digestive diverticula serve as a place where wood can be received and acted upon by enzymes (*vide* physiology of digestion). The occurrence of two functionally different kinds of diverticula has been reported in other ship-worms as well and Sigerfoos (1908), Potts (1923) and Yonge (1926, a) considered that these diverticula are specialised for the digestion of wood.

The *intestine* is dilated into a bulb (Fig. 1. CTI) at the place of its origin at the postero-lateral part of the stomach, runs a short course anteriorly, and then makes a sharp backward turn upon itself and approaches the median ventral part of the body and proceeds backward beneath the caecum, bends over it at its posterior end as a loop and continues forwards through the gonadal tissue and passes below the posterior adductor muscle and then bends over it in front and turns back to open into the anal canal at the anus.

Sections through the intestine show the presence of a *typhlosole*. The anterior bulbous part contains a typhlosole which is very much coiled (Fig. 8) which becomes a simple fold along the ventro lateral side of the intestine (Fig. 27) where the intestine takes a sharp backward turn and continues as a low ridge (Fig. 28) along the inner wall of the intestine till it makes the loop around the posterior end of the caecum. From that point to the anus there is no trace of a typhlosole and the midgut leads indistinguishably into the hindgut. It is noteworthy that in *Bankia gouldi* Sigerfoos has described a typhlosole extending throughout the intestine. On the other hand in *T-navalis* (Lazier, 1924), typhlosole is present only in the anterior portion of the intestine while Deshayes (1848) and Quartrefages (1849) describe an intestine in the species they studied as having uniform diameter thereby indicating the absence of a typhlosole. The intestine is lined by an epithelium of columnar ciliated cells whose oval nuclei are basal in location. The epithelial cells lining the wall of the hindgut are columnar and ciliated. There appears to be no muscle fibres on its wall.

It has been shown that there is a definite correlation between the length and also the function of the midgut and position where the faeces are extruded. Graham (1932) has observed that the very long coiled gut in *Patella vulgaris* is probably concerned exclusively with the formation of firm faeces. It is necessary that an animal which discharges its faeces into the mantle

cavity should avoid the fouling which would ensue, were these extruded in a loose or particulate condition. As absorption does not take place in the midgut, probably this region is mainly concerned with the preliminary moulding of the faeces.

The course of food particles through the alimentary canal

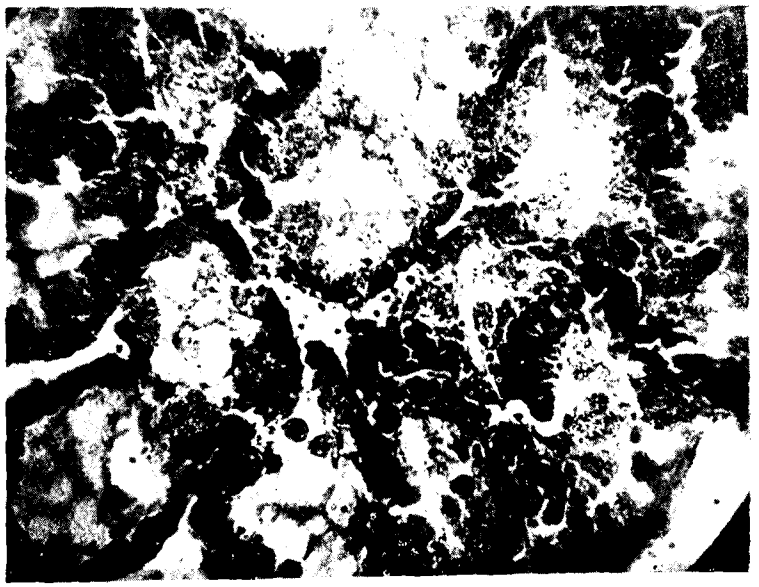
A study of the alimentary tract of living animals removed from the wood and examined under a binocular microscope with current indicators like carmine and carborundum revealed that besides ciliary action muscular movements comparable to peristalsis also aid in the progress of food materials. The lateral pouch on the proximal part of the stomach has muscular walls and their rhythmic bellowing action and elasticity help in driving the food particles with force towards the distal part of the stomach. The stomach is not ciliated throughout, while the typhlosole and the ciliated ridge are covered with long cilia. The typhlosole contains besides the ciliated cells numerous mucus-secreting glands which exude large quantities of mucus. The particles entering the stomach come under the influence of the rows of cilia (Fig. 9). The major part of it is carried away right into the caecum by the strong currents set up by the cilia on the typhlosole and the ciliated ridge. The smaller particles are found freely entering the ducts of the digestive diverticula where whirl-pools of ciliary currents are noticed. The lighter and finer particles are directed towards their openings. At the distal end of the stomach the coarser and heavier particles come in contact with the transverse folds between the ciliated ridge and the gastro-intestinal typhlosole and a major part of the food-mass is separated and sent towards the caecum. Another strong current is developed towards the large posterior hepatic orifice where particles are seen streaming in. At the region of the folds and furrows a section of the mass especially those comprising the larger particles are separated from the main food stream and is carried into the intestinal opening and finally into the midgut.

Besides these movements facilitated by ciliary action the ingested materials are subjected to the mechanical and chemical action of the rotating style and mixed with the enzymes liberated during the dissolution of the style. The ducts of the digestive diverticula bear abundant cilia and an internal investment of long cilia on the thin walls of the diverticula (Potts, 1923). Besides these the digestive diverticula are provided with muscle fibres so

that alternating contractions and expansions not always simultaneous in the different regions maintain a rapid circulation of food during and after the meal. In addition to all these it is to be presumed that the twisting and turning movements of the body while boring considerably augment the movement of particles in the stomach and to some extent in the caecum and prevent chocking of the regions with wood particles. The caecum is not ciliated in *B. indica* and feeble waves of muscular movements are noticeable both on the walls as well as on the typhlosoles. These muscular movements resembling peristalsis is evidently more suited for this storage organ which is usually found so full of wood chips that the cilia cannot efficiently function. The movements of materials in the intestine are obviously by cilia and the currents move towards the anus at an average rate of 1 mm. per two minutes. They are steadily propelled out of the hind-gut into the anal canal where they come under the influence of the exhalant current and are flushed out through the exhalant siphon by the contraction of the mantle which presumably exerts a squeezing action on the suprabranchial cavity, thereby bringing about the expulsion of the faecal matter in the form of long filaments some distance clear of the opening of the burrow. Observations on living animals inside the opened burrow sealed with a glass slide revealed that after burrowing for sometime the animal contracted the anterior ⅓ of the body. This contraction of the anterior end would naturally bring the head of the crystalline style far into the distal end of the stomach and even right into the caecal opening. This probably helps the animal not only to push in the particles of wood into the caecum preventing blockage of the gastrocaecal orifice, but also in conveying the style ferments into the caecum.

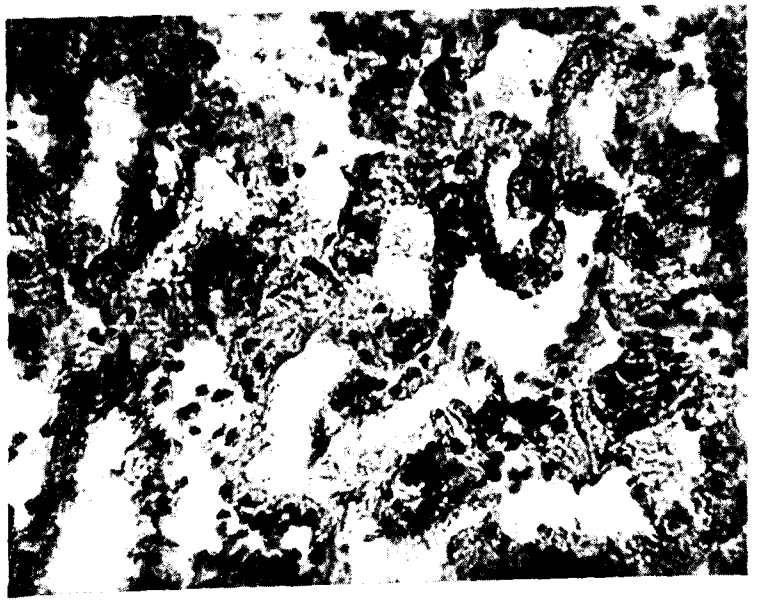
6. *The Gills*

Ridewood (1903), Sigerfoos (1908), and Purchon (1939, 1941) have described the ctenidia (Gills) of *Teredo navalis, Bankia gouldi* and *Teredo norvegica,* while the mechanism and function of the ciliary currents of the lamellibranch ctenidium have been elucidated by Wallengren, (1905), Kellog (1915), Yonge (1923, 1926, 1928) and Atkins (1936, 1937, 1937 a, 1937 b, 1938, 1938 a), further, the significance of the muscular activity in the ctenidia has been described by Setna (1930) and more recently by Atkins (1943).



PHOTOMICROGRAPH 2

Section through the digestive diverticula



PHOTOMICROGRAPH 3

Section through the specialised region of the digestive diverticula showing the particles of wood inside the cells

The ctenidia are outer demibranchs (Fig. 1. G), creamy white in colour, ribbon-like in form and conspicuous structures extending from the labial palps to the base of the siphons, measuring 9·5 cms., in a specimen 10 cms. long. Of this length nearly 2·9 cms. is occupied by only a groove, the branchial groove where no branchial filaments occur with the result that the anterior nine filaments (APC) are separated from the major part of the ctenidium situated towards the posterior part of the body. All the posterior filaments are of the usual 'V' shape found in demibranchs with a small groove at the apex of the 'V'. The grooves of the adjoining filaments form a long marginal groove for the demibranch, which anteriorly becomes continuous with the branchial groove thereby establishing connection with the anterior nine filaments on each side of the body. Sigerfoos (1908) considers this groove as formed by the great broadening of the 10th or 11th filament in *B. gouldi* during the post-larval development. The anterior nine filaments are disposed in such an oblique manner as to cover the base of the sickle-shaped apophysis situated behind the base of the labial palps at the sides of the foot dorsally, towards which the minute food particles are propelled along the marginal and branchial grooves. The major part of the ctenidium measuring 5·1 cms. lies behind the visceral mass embracing the posterior tip of the cylindrical caecum to some extent. The ctenidial axes of the right and left demibranchs converge behind the caecum into a singal common dorso-median axis bearing the filaments of the demibranch of the two sides.

Vertical sections taken through the entire gill across the axis bring out differences in the shape of the demibranchs between this species and *Bankia gouldi*. In this form the lamellae of the demibranchs pass laterally first and then vertically downwards unlike those in *Bankia gouldi* and *T.megotara* where they pass vertically downwards. The thickness of the visceral mass as well as the location of the gill-axis are probably responsible for this difference. However, this feature is likely to be of taxonomic importance since the section passing through *Teredo navalis* commented on by Ridewood (1903) also is strikingly different.

Each ctenidium consists of homorhabdic branchial filaments inserted uniformly on a vascular axis of loose connective tissue traversed by muscle fibres. They descend into the mantle cavity forming the lamella and the distal portions of the filaments folded upon themselves outwards forming the reflected limb of the

lamella. The interfilamentar junctions are organic indicating a synaptorhabdic condition and the intrafilamentar junctions are vascular representing a typical eulamellibranch condition.

The suprabranchial chamber is a long cylindrical canal disposed dorsally in the median line behind the visceral mass. Anteriorly, where the gills diverge and cease they become narrowed into slender canals lying above the branchial groove (Fig. 2. LCA) on the outer surface of the branchial vein in the substance of the mantle.

The *microscopic structure* of each branchial filament shows that its epithelium consists of three kinds of cells.

1. *The ciliated cells.* The frontal edge of the filament is occupied by five large cells bearing the shortest and weakest cilia (Fig. 7. FC). Two cells in the latero-frontal regions bear long and fine cilia in such a way that they interdigitate with their fellows of the adjacent filaments (LFC). These cells are tall and thin containing darkly staining nuclei placed towards the proximal part of the cell. The third type of cells occupy the lateral regions of the filaments. They are, however, separated from the latero-frontal cells by a non-ciliated region. The cilia are stiff and dense (LC) though not so long as those of the latero-frontal cells, the cells themselves are short and cubical in form containing large oval nuclei with scattered deeply staining reticular chromatin. Near the interlamellar regions on the hind end, the cells are short and bear thin short cilia (AFC) as in *Teredo* (Ridewood, 1903). In the filaments of the anterior portion of the ctenidium the cilia are not distinguishable into the four types so characteristic of the posterior part of the ctenidium. Further, the blood space of the filament is much more spacious in these than those in the posterior ones. In this respect it differs from *Bankia gouldi.* There are five cells in the frontal regions of the anterior filaments just as in the posterior filaments in the present form. It may be noted that in *B. gouldi* the frontal cells of the anterior filaments are more numerous, the central ones of which do not bear any cilia.

2. The non-ciliated cells form a single row one cell thick between the latero-frontals and the lateral cells in the filaments of the posterior part of the ctenidium. These cells differ from the rest in that they are short and contain lightly staining small nuclei.

3. The *secretory cells* are typical mucous cells found here and there in the free external borders of the epithelium. They are so distended with mucus that their nuclei are pushed towards the side. These glands are more abundant in the epithelium of the filaments of the anterior part of the ctenidium.

Internally the filament is filled with phagocytes, connective tissue where blood freely flows, and also by a glandular tissue known as the glands of Deshayes (Fig. 7 GDS).

A chitinous skeleton (CHR) is seen best developed in the posterior part of the gill and consists of thin chitinous rods which are thickened at the filamentar junctions.

The *glands of Deshayes*: (Fig. 7 GD) These glandular tissue occurs in the gill laminae as two distinct types of structures composed of two types of cells differing in histological details. A highly irregular ramification of filamentous structures lie in the epithelial walls of the gill laminae. They contain numerous deeply staining nuclei strewn indiscriminately in a mesh of granular cytoplasm which does not show any cell boundaries. The second type differs markedly from these in that they are composed of spherical bodies which take a deep stain with Delafields haematoxylin. The spherical bodies occur in groups of two, three or four and are distributed irregularly in the internal space of the gill lamina. The details of their distribution are shown in the figure. In *Bankia indica* these glandular structures are developed only in the gills. The umbonal portion of the gland found in *Teredo dilatata* and *Bankia gouldi* is not met with. From these glands located in the gills on either side a narrow cylindrical duct starts and passes forwards by the side of the branchial groove; (Fig. 4. dD); its lumen containing granular cells which take stain deeply and open on either side of the mouth. The arrangement of the different cellular elements of the glands and their disposition in the gill lamina differ considerably in *Bankia indica* when compared with that in *Bankia gouldi* where it is described fully by Sigerfoos (1908).

8. *The Ciliary Action of the Gills*

The part played by the vertical rows of four different types of cilia borne by the gill filaments in the collection and disposal of particles suspended in water accumulating in the mantle chamber was studied by using carmine particles and carborundum powder.

Large sized particles too heavy for ciliary action were dropped down to the ventral regions of the mantle cavity. Medium sized and fine particles are, however, swept over the surface of the ctenidium by the action of the cilia. The ciliary action of the gills is two-fold, since the four rows on each filament are responsible for the circulation of sea water through the body and also responsible for the collection of particles of food from the sea water and passing them on to the mouth. Since the former is primary it may be described first. As Wallengren (1905), and Orton (1912) have shown, the lateral cilia are mainly responsible for the in-going water current. The lateral cilia play upwards in a rhythmic manner making the water flow from infrabranchial cavity to suprabranchial cavity from where it finds exit through the exhalant siphon. Thus the water which enters the body through the inhalant siphon is lifted up into the suprabranchial cavity before it reaches the anterior end of the animal. Here, in the suprabranchial cavity, the water flows backwards aided by the backwardly beating cilia on the roof of the cavity and the pulsations of the heart, in a direction opposite to that of the incurrent water, till it reaches the exhalant siphon at the hind end, from where it is expelled out of the mantle chamber by muscular contractions of the mantle. During the course of this circulation inside the mantle cavity, the food particles suspended in it impinge on the surface of the gill filaments. On each filament long fine cilia belonging to the latero-frontal band beat slowly with effective strokes in a direction towards the frontal face of each filament. The movements of these cilia are not so rapid but they are vigorous. These cilia strain the particles of food and even push them by contact. To a slight extent water will be displaced out of the infra-branchial chamber in the process of straining movements of the latero-frontal cilia. The particles which are prevented from passing between the filaments are pushed from either side of the filament towards the frontal surface, get entangled in the mucous secreted in that region and are swept down along the filaments by the action of the frontal cilia beating downwards. At the ventral margin of the gills, the strong cilia borne by the marginal groove carry these particles forwards, along the edge of the gill. As a large number of mucous secreting cells occur in the groove a thick rope of mucus traps the medium and fine particles which alone are carried oralwards propelled by the forwardly beating rows of cilia. At the front edge of the posterior part of the ctenidium i.e., at the junction of the marginal groove with the branchial

groove large sections of the mucous thread weighed by the medium sized particles drop down to the floor of the mantle chamber due to the narrowness of the lumen of the branchial groove. Meanwhile the very fine particles which were not so stuck to the mucus are freely propelled oralwards by the cilia of the branchial grooves. These fine particles reach the mouth along the ciliated groove of the anterior gill filaments and between the outer and inner labial palps. It is noteworthy that only the finest particles of suspended matter are thus conveyed to the mouth. The medium sized particles though collected have been forced to be rejected due to the unusual lengthening of the body and development of the caecum both of which contributed to the narrowing of the branchial groove. The medium sized particles along with the heavy ones dropped down from the gills through sheer gravity are expelled from the mantle cavity, through the combined action of the cilia borne on the ciliated tracts and also by muscular contraction of the mantle as 'pseudo-faeces' through the inhalant siphon (*vide supra*). This peculiar type of selection of particles by the gill and subsequently by the branchial groove observed in *Bankia indica* is probably accounted for by the absence of the fine grooves which aid in selection of food particles in the palps of forms like *Ostrea*.

Thus, even though the essential food collecting mechanism apparently is represented and functions as in typical lamellibranchs the enormous development of the caecum and elongation of the body have resulted in the attenuation of the branchial groove into a passage of such a length and narrow diameter that the food-particles that can travel along this to the mouth become limited in size and quantity. This involves such a rigorous elimination that the major part of the plankton and suspended particles conveyed by the water currents have to be rejected as 'pseudo faeces'. Thus, the function the gill discharges as a region of reception of food in other lamellibranchs is minimised owing to the high specialisation towards the exploitation of the wood which they bore into as food. This accounts for the absence of plankton and diatoms in the alimentary canal. As a direct consequence of the specialisation for a wood-boring habit and a diet of wood particles, this mollusc has become so incapable of making full use of the water-borne, planktonic food that when the wood supply is exhausted, it becomes helpless even though it is equipped with enzymes which can digest planktonic food (*vide*, physiology of digestion).

Rate of travel of particles: A few observations were made on the rate of travel of particles along the filaments towards the

marginal groove and also along the groove towards the mouth. These observations were of the speed of small particles conveyed along these paths. Rate of travel when examined with the help of thin bits of lead foil was found to be very regular as there are no conflicting currents. Most of the speeds recorded fell close to 0·3 mm./second along the marginal groove and 0·5 mm./second along the filaments at 30° C.

Branchial movement: The gills are capable of considerable muscular movements. When particles are deposited on the surface of the gills, writhing and swaying were observed. Such movements have been noticed by Kellog (1910) in *Pecten*. Touching a part of the gill with a needle causes contorsions for a distance along the lamellae which are mainly brought about by elongation followed by contraction. Transverse movements of the filaments were also noticeable. Probably this may be important for permitting large particles to fall off thereby removing more effectively the sediment falling on the gill-surface.

9. *Discussion*

The foregoing study of the anatomy of *Bankia indica* notably illustrates the remarkable co-ordination between structure and function. The foot of this lamellibranch illustrates how plastic the organic body proves when an animal adopts an entirely new habit. It is so specialised that it is just a sub-circular disc with pedal muscles of other lamellibranchs modified for assisting the boring into wood. Notwithstanding the correct suggestion of Home (1806), that the foot adheres to the wood acting as a centre-bit while the animal is boring with the shell, there has been from time to time repeated claims that the foot is directly concerned with the boring activities (Dall, 1895; Hedley, 1901; Kuhlman, 1914). These authors considered that the shell effects a grip on the sides of the burrow supporting the foot against the wood. The reason for this idea perhaps might be due to the fact that in other pelecypods such as *Mya*, *Solen*, and *Cardium*, the excavation is accomplished by the foot, the shell serving only for protection. A study of the histological structure of the organ in the present form, as well as the observation of this organ in living specimens lead the present author to conclude that its primary function is to effect a cupping action at the end of the burrow to hold the shell in position while boring. The peripheral wrinkled region with its ciliary covering helps to conduct the grated particles of wood to the mouth, since the labial

palps are reduced and the mucous secretion from this region assist in cementing the stray particles into a coherent mass. The unique shape of the shell, its elaborate denticulations, the development of additional faces such as the posterior lobe for the insertion of the posterior adductor muscle, the acquisition of secondary articulations to achieve a special type of movement, the loss of the hinge teeth, the reduction of the ligament and the histological peculiarities suggest that the shell in *Bankia* has a specific function to perform. When one considers that the shell in these forms is not only a boring organ but a feeding organ as well, Hedley's suggestion (1901), that the shell in Teredinidae is degenerate and that the next step would be its complete disappearance sounds strange. It is true that the shell has lost the protective character, but in this specialised lamellibranch in which protection to the naked body is afforded by nacre lined burrow, the shell is assigned the role of scraping off such fine particles of wood as will be easy to feed upon and the form, both of the foot and shell suggests a long specialised evolution. The pronounced development of the posterior adductor, its fibres being suited for powerful and frequent contractions, its insertions being adapted for facilitating powerful outward thrust of the denticulated regions of the valves indicate that this muscle is part of a mechanism which has been perfected as a whole. It is obvious, no one organ can be elaborated or simplified without the other organs associated with it undergoing appropriate changes. One is inclined to wonder whether such a complex mechanical pattern, can be produced by just an opportune gene pattern or by the gradual addition of details of structure.

Although the teredines have especially a basic palp structure consisting of an outer and inner one, they show various degrees of reduction, variation in size, shape and functional efficiency. While the outer and inner palps may be recognised in all the species studied, the relative proportions and the development of each of these vary considerably and the variations can be attributed to habit specialisations. The palps of *Teredo norvegica* are the least reduced and are reported as capable of exercising quantitative selection (Purchon, 1941), while those of *Teredo megotara* (Purchon, 1941), *T. navalis* (Lazier, 1924), *Bankia gouldi* (Sigerfoos, 1908) and *Bankia indica*, are greatly reduced and incapable of quantitative selection on the suspended food particles drawn towards the mouth. The presence of a long palp in *T. norvegica* is explained by its habitat in waters where turbidity is high whereas in others such a selection is unnecessary since they are found in

comparatively clear waters where turbidity is practically nil. Further the manipulation of the wood-chips during burrowing is mainly done by the peripheral folds of the foot. That the boring life is not a causative factor for the reduction of the palp is evident since other boring forms of the family *Pholadidae* do not, show reduction of the palps except *Xylophaga* (Purchon, 1941) which is also found in the offshore, less turbid waters.

The caecum of the *Teredinidae* is the enlarged 'dorsal pouch' of other eulamellibranchs evolved as a result of the response to the boring habit and utilisation of wood as food. The large quantities of saw-dust that accumulate while excavating has had to be stored so that no strain is placed upon the remainder of the alimentary canal. The structure of the gastro-caecal orifice indicates that there is provision for the simultaneous ingress and egress of wood-particles into and out of the caecum. The caecum helps to control the arrival of food particles like a safety valve. The development of a specialised region of the digestive diverticula for the intracellular digestion marks another adaptational feature evolved in the morphology of the teredines. Side by side with these unique specialisations there have been physiological adaptations as well, such as the relative dominance of the carbohydrase system correlated with a woody diet, and the elaboration of additional enzymes like cellulase and enzymes to deal with pentosans for the digestion of cellulose and other constituents of wood. (Nair, 1955.)

The gills of *Bankia* show considerable reduction in relation to its boring life, being represented only by a single demibranch. The orientation of the gill is noteworthy since nine filaments are placed far in front on either side of the labial palps and the main ribbon-shaped posterior part behind the visceral mass, the two being connected by a narrow branchial groove. Even though the food collecting mechanism is retained, the function of the gills as a region of reception of food particles has been subordinated since the amount of nourishment needed form the plankton is very little being a small amount of nannoplankton to suppliment the scarce nitrogenous constituents present in the wood.

However, this perfection towards the utilisation of the wood necessitated the growth and elongation of the body throughout life in various directions inside the wood. Even the major part of the planktonic food collected by the gills finds the small passages of the branchial groove too narrow in *Bankia indica* with the result a

good portion of the mucous rope with the cemented food has had to be rejected as pseudo-faeces. Thus, the dependence of these borers on wood has become so much that the borer community colonising a timber block will have to perish when the wood supply is exhausted. However, to some extent the dependence of these marine forms on wood which is a terrestrial product both for food and shelter, and the vicissitudes attending the free-swimming larvae in finding out the adult habitat have been compensated by a prolific fecundity, the development of sperm-conserving devices, and a protracted free-swimming period during which time dispersal is effectively accomplished.

10. *Acknowledgements*

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EXPLANATION OF FIGURES

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KEY TO LETTERING OF FIGURES

AA—Anterior adductor
AAC—Anterior *aorta*
ABV—Afferent branchial vein
AC—Anal canal
ADO—Anterior openings of the digestive diverticula
AFC—Abfrontal ciliated cells
AL—Anterior lobe of the shell
AM—Adductor Muscle
AN—Anus
APC—Anterior part of the ctenidium
APS—Apophysis
ARV—Afferent renal vein
ARD—Afferent renal duct
AU—Auricle of the heart
AUR—Auricle of the shell
AVV—Auriculo-ventricular valve
BC—Border cuticle
BG—Branchial groove
BM—Basement membrane
BS—Blood space
BV—Blood vessels
BVG—Blood vessels of the *gills*
BY—Byssus thread
C—Crypts of the darkly staining young cells
CA—Caecal artery
CAG—Caecum of the stomach
CABV—Common afferent branchial vein
CH—Cephalic hood
CHR—Chitinous supporting rod
CMT—Ciliated mantle tract
CM—Circular muscle
CO—Collar of the mantle
COT—Connective tissue
CR—Ciliated ridge
CT—Caecal typhlosole
CTI—Coiled typhlosole of the intestine
DD—Digestive diverticula

dD—Duct of the gland of Deshayes
dDD—Duct of the digestive diverticula
DLP—Outer labial palp
EBV—Efferent branchial vein
EN—Endodermal cells
ERD—Efferent renal duct
ES—Exhalant siphon
F—Foot
FC—Frontal cilia
FV—Food vacuoles
G—Gills
GA—Ascending limb of the gill
GD—Descending limb of the gill
GDS—Glands of Deshayes
GG—Groove at the ventral margin of the gill
GIT—Gastro-intestinal typhlosole
GS—Gastric shield
GT—Gastric Typhlosole
I—Intestine
IBV—Infra branchial cavity
IE—Inner epithelium
IFJ—Inter-filamentar junction
ILS—Inter-lamellar space
IM—Inner lobe of the mantle edge
IS—Inhalant siphon
K—Kidney
LC—Lateral cilia
LCA—Lateral canals
LFC—Latero-frontal cilia
LG—Lateral oral groove
LM—Longitudinal muscle
M—Mouth
MA—Mantle
MC—Mantle cavity
MES—Mesodermal cells
MF—Muscle fibres
MG—Mid gut
MGR—Margnial groove
MM—Middle lobe of the mantle
MUG—Mucous glands
N—Nuclei of the epithelial cells
O—Oesophagus
OCG—Gastro-caecal orifice
OD—Opening of the digestive diverticula
OE—Outer epithelium
OM—Outer lobe of the mantle edge
OMG—Opening of the stomach into the midgut
OV—Ovary
OVD—Oviduct
P—Phagocytes
PA—Posterior adductor
PAR—Pallial artery
PB—Pre-oral band of cilia
PC—Pericardium
PDO—Posterior opening of the digestive diverticula (posterior hepatic opening)
PEG—Periostracal groove
PGL—Gland cells of the foot
PN—Pallial nerve
POA—Posterior aorta
POM—Posterior median part of the shell
PR—Peripheral region of the foot
PRM—Pallial retractor muscle
R—Rectum
RA—Radial muscles
RM—Retractor muscles
SAM—Anterior median part of the shell
SBC—Supra branchial cavity
SDD—Specialised region of the digestive diverticula
SEM—Sub-epithelial mucous glands
SH—Shell
SHG—Shell-gland
SMM—Middle-median part of the shell
SOL—Non-ciliated central 'sole' of the foot
SS—Style sac
ST—Stomach
T—Typhlosole
Umb-knob—Umbonal knob
V—Ventricle
VDO—Ventral orifices of the digestiv diverticula
VG—Visceral ganglion
VLP—Inner labial palp
VM—Visceral mass.

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A Review of Plant Viroses in India

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Although much work has been done on fungal diseases of plants in India, similar work on plant virus diseases has been little attended to. The discovery of sandal spike, cotton stenosis and later on various growth abnormalities of potato, tobacco and other economically important crops, created a new field for plant pathologists and attention began to be focussed on plant virus diseases.

During the Forty-first Indian Science Congress, under the chairmanship of Dr. B. C. Kundu, many scientists expressed the need for a general survey of plant virus diseases in India, and urged for a greater interest in the field of plant virus research. The present work is aimed at such a need. In this review, since the identity of many viruses have not been established, the diseases have been divided based on the symptoms they produce, and the hosts described under each. Wherever identified, the synonyms of the virus based on Smith's and Holmes's systems of classification are given. At the end of this review is given a table of the physical properties of sap-transmissible viruses and a host index.

Most of the information presented here is gathered from the "Review of Applied Mycology", the rest from various Indian and a few foreign journals. In this review 70 diseases affecting 59 species belonging to 19 families of Angiospermae are reported.

MOSAIC DISEASES

'Mosaic' is a foliar symptom characterised by mottling of the leaves. Very often this main symptom is followed by other subsidiary symptoms like enation, distortion, vein-banding, sterility, and leaf-curl and accordingly several types of mosaic are recognised.

1. *Mosaic of Sugarcane* (*Saccharum officinarum* L.).

The virus : *Saccharum* Virus 1, Smith.

Marmor sacchari Holmes

The first record of the mosaic disease of sugarcane was made by Dastur (1923) during the year 1921 from Pusa. In 1925, typical infection was observed on the cane Co 213 at Pusa (Mc Rae, 1926a) and a search was made in other cane growing provinces of India. It was shown that the disease was prevalent in Assam, Bihar, Bombay, Central Provinces, Madras, Punjab and United Provinces (McRae, 1926a). During 1925, the disease was a serious epidemic throughout North India on the local variety Hemja, while in South, during 1925-29, Godavari district showed an average of 10% infection and Carnatic district 2%. Occasionally however, in these two districts the incidence was as high as 50% (McRae, 1926a; Sundararaman, 1928a). While it is difficult to establish the source of the disease in India, there is evidence to show that the disease was in existence for a long time in India among the thin varieties of cane (McRae, 1926a). The fact that mosaic of sugarcane was first observed in Java in 1892, and that Java varieties POJ 213 and Java 213 are highly susceptible to the disease, lends support to the view that the disease is imported from Java (Dastur, 1926; Barber, 1926). It is suspected that the disease at Pusa, was introduced from Lyallpur (Punjab) on Co 223 which in Punjab is reported to be highly susceptible (McRae and Subramaniam, 1928).

Symptoms :—

The characteristic primary symptom of the disease is the mottled appearance of the young leaves of the crown, with patches of light and dark green colour, alternating with each other in a definite manner. Mottling is most prominent on the first partly unfolded leaf of the crown, becoming faint in the older leaves below. The patches are of various sizes, but mostly linear and arranged parallel to the midrib of the leaf. The intensity of mottling varies with the cane variety and season, being prominent during the spring and summer (Chona, 1947). Some varieties do not manifest any signs of infection until late, while in others the symptoms disappear early (McRae, 1930). Eventhough the secondary symptoms are rare on the Indian varieties of sugarcane, they have been observed in a few cases. These include:— whitish or yellowish linear blotches on the internodes of the dark-rinded varieties, which gradually develop into narrow linear cankers resulting from the death of the rind tissue, shrinkage of the internode and in advanced cases stunting (Sundararaman, 1928a).

Histopathology :—

The pale areas of the infected leaf are thinner and the cells in such areas smaller than those in healthy parts. In the infected tissue, the chloroplasts are smaller, less regular in shape and fewer in number (Sundararaman, 1928a). Cytological studies revealed the presence of protozoan-like bodies in the infected canes in all parts of the plant body including the xylem (Dastur, 1926; 1933).

Transmission :—

Under experimental conditions, the disease has been found transmissible by sap inoculation, the sap from infected leaves when rubbed on to healthy leaf causing the disease. Injection of the infective juice into the stem using hypodermic needles and inserting plugs of infected tissue into the setts before planting were also successful (Sundararaman, 1928b).

Transmission of the disease through setts is very common, and this is perhaps one of the ways by which infection takes place in the field. The disease being systemic, the young shoots coming from the infected setts show mosaic symptoms. Seed transmission does not take place (Sundararaman, 1936; Chona, 1944) and the exact identity of the insect vector has not been established, although there are reports of the presence of *Aphis maidis* on the infected canes (Sundararaman, 1928a). Experiments at Pusa and Delhi reveal that infection is maximum in the months of May and June, hot weather favouring transmission of the disease and that the incubation period of the virus ranges from 7 to 10 days (Chona, 1944). The maximum spread of infection in the field is shown to be between the third and fifth months of growth (Sundararaman, 1928b). Secondary infection, if any, begins when the crop is three months old, and spreads rapidly during the next three months followed by a marked decrease (Sundararaman, 1928a).

Susceptible and Resistant varieties :—

While it is difficult to draw a distinction between the susceptible and the resistant varieties of sugarcane in India, it can be generally said that only a few varieties have been found to be resistant to the disease both under experimental and natural conditions. Forty-seven seedling canes, 9 crosses at the Coimbatore breeding station, 2 thin cane varieties and 38 tropical varieties are reported to be susceptible (McRae, 1928). The highly susceptible varieties include: A 2, B 147, B 208, B 254, B 688, B 6308, Chunnee, Co 203, Co 205, Co 210, Co 213, Co 223, Co 232, Co 281, Co 290, Chittal, D 625, D 1135, Fiji B, HM 313, HM 320, HM 332, HM 554, Java Hebbal, J 213, J 247, Khari, Local Reed, Local Striped, Mauritius 16, M 55, Purple Mauritius, Poovan, Q 813, Red Mauritius and Vellai (McRae, 1926b; 1928; Sundararaman, 1928a; 1929). *Saccharum spontaneum* varieties SES 320, Taiwan II, Thick gigas, and Tobange were found to contain sugarcane mosaic virus, though the symptoms were not very clear externally.

Among the resistant varieties, Co 214 is reported to be the only variety which has escaped infection in nature and also artificially (Chona, 1944). The POJ varieties are fairly resistant to the disease, and among the thin canes, Co 205, Co 214, Co 244 and Kassoer are resistant (Sundararaman, 1928b). POJ varieties 2714 and 2727 and a few Fiji varieties are also fairly resistant to the disease. Co 205, a highly susceptible variety at Pusa, is found to be resistant at Coimbatore (Sundararaman, 1931).

Many commercial varieties of sugarcane exhibit varied degree of susceptibility to the disease. Certain varieties which escape infection under one set of conditions succumb to the disease under a different set of conditions (Krishnaswami, 1936). The nature of epidermal structures on the leaf of sugarcane has been shown to determine the degree of resistance of the variety. Thus, the resistant Kassoer variety is shown to possess sharp, stiff unicellular bristles on the surface of the leaf which protect the stomata and the surrounding regions from insect invasion. On the other hand, a number of susceptible varieties like Poovan, Co 1, Red Mauritius, Purple Mauritius and Vellai are extremely soft leaved and lack the above said arrangement of the Kassoer variety, (Venkataraman and Thomas, 1936).

The varieties having the "*Saccharum spontaneum*" blood have been shown to be generally resistant to the disease, and some varieties are atleast tolerant. A few immune varieties exist, and they could be used as parents for the production of resistant hybrid canes (Dutt *et al.*, 1936; Krishnaswami, 1936).

Thomas (1931) applied a practical method of estimating the disease resistance of sugarcane to mosaic disease. This consisted of growing large number of varieties and strains under identical conditions of environment and exposure to identical conditions of infection to mosaic. This is followed by a close study of an equal number of individuals in each variety, a careful estimation of the incidence of infection and the evaluation of the extent of the disease and the loss due to it among the different varieties under trial.

Effect of the disease on host :—

Though sugarcane mosaic is said to be very destructive in other cane growing countries, in India, the disease is of little economic importance (Chona, 1947). Careful experiments at Pusa reveal, that at 100% infection, there is only 10 to 12% loss compared to 5 to 50% in other countries. The quality of the juice from infected canes is not much affected by the disease. Co 213, suffering from mosaic showed 11% less germination, 14·8% less in stripped cane, and 8·9% less in sucrose than healthy canes (McRae, 1932). The disease reduced the weight of the cane, but the quality of the juice, as measured by brix, glucose and sucrose remained unimpaired (McRae, 1932; McRae and Subramaniam 1933, 1934; Sarkar and Dutt, 1934; Sundararaman, 1933).

Physical properties and the strains of the Virus :—

The Indian strain of Sugarcane Mosaic Virus resembles strain I B of Summers, and is known to exist in three different strains designated X, Y, and Z (Chona and Rafay, 1950). The thermal death point of strain X in expressed juice is 60°C, that of Y 55°C and that of Z 45°C. X and Y are able to withstand a dilution of 1:100 and can remain viable for 6 hours at 30 to 32°C, whereas Z cannot remain infective at a dilution of 1: 50 and hardly survives for 2 hours at 30 to 32°C. These strains are not filterable through Chamberland L or Seitz filters and even the filterate through an ordinary filter paper is non-infectious. The virus

can be centrifuged for half an hour at 3000 r.p.m. without sustaining appreciable loss of virulence, though further centrifuging reduces its infectivity. *In vitro,* the virus loses viability in a few hours at room temperature, but at 5 to 6°C, it remains infective for about 15 days. Storage temperature is the most important factor in the *in vitro* longevity of the virus (Chona and Rafay, 1950).

Copper sulphate 1:1,500, hydrochloric acid 1:1,000, nitric acid 1:800, hydrogen sulphide 1:25 and formalin 1:25 inactivated the virus. Zinc powder has no effect on the virulence (Rafay, 1935).

The different strains readily cross-infected other hosts like *Sorghum,* maize and *Euchlaena mexicana.* The same strain produced widely different symptoms on different cane varieties.

Acquired Immunity :—

Immunity from the disease, after infection is reported for a few varieties. These varieties recover from the disease and the possibility of the plant acquiring a permanent immunity is postulated (Krishnaswamy, 1936; Sundararaman, 1936).

Control Measures :—

Selection of good, disease-free setts from tolerant varieties, regular and intensive roguing of clumps that may show mosaic and avoiding growing of jowar or maize near sugarcane plots, are some of the control measures recommended (Chona, 1947). Regular monthly inspections of the crop should be made especially between the second and sixth months of growth, and all suspected plants eradicated. When the incidence is very high, making roguing impossible, the crop should be allowed to mature, and then milled for the juice, care being taken not to use the setts of the diseased cane. Roguing has given excellent results at Coimbatore especially in reducing secondary infection (Sundararaman, 1928a).

Other Plants affected by Sugarcane Mosaic Virus :—

Maize, *Sorghum, Euchlaena mexicana* and *Eleusine coracana* have been shown to be susceptible to sugarcane mosaic virus (McRae and Subramaniam, 1928; Mitra, 1936; Venkatarayan, 1946). Often, these plants when grown near sugarcane plots, show mottling of the leaves, and this is also experimentally shown by inoculation of the infective sap from mosaic infected sugarcane into the leaves of these plants. The reverse infection from these plants back into healthy sugarcane is, however, seldom achieved (Mitra 1936).

2. *Mosaic of Potato* (*Solanum tuberosum* L.)

The Viruses:

1. *Solanum* Virus 1, Smith (Potato Virus X) *Marmor dubium* var. *annulus* Holmes.

2. *Solanum* Virus 2, Smith (Potato Virus Y) *Marmor cucumeris* var. *upsilon* Holmes.

3. *Solanum* Virus 3, Smith (Potato Virus A) *Marmor Solani* Holmes.

Potato is commonly affected with several types of mosaic, caused by one or more strains of the Potato Viruses. Mosaic of potato was first noted in North India in 1936 (Galloway, 1936), along with other types of virus diseases like leaf-roll. McRae (1923) reported its presence in Bombay Presidency. Annual inspection for seed certification showed, that in Kumaon hills, Chakratta hills and Simla hills, the percentage of infection with negligible and severe mosaics were respectively, 55 to 70 and 24·5 to 46, 24 to 37 and 2·5 to 23, 8 to 20 and upto 40 (Anonymous, 1948). Most of the Indian varieties and many foreign varieties suffer from various types of mottling which have been classified into, 'negligible mottle', 'mild mosaic', 'borderline mild mosaic', 'severe mosaic', and 'borderline severe mosaic' (Vasudeva, 1946). The incidence varied in different months, and masking of symptoms has been found to be very common. In North India the masking of symptoms is negligible in the first crop, whereas in the second crop, masking occurs during the second and third weeks of March, when the mosaic incidence is lowest (Vasudeva, 1946).

Potato Virus X :—

A relatively virulent strain of Potato Virus X (*Solanum* Virus 1) has been studied on Majestic potatoes in United Provinces (Vasudeva and Lal, 1945a). The infectivity of the virus is lost at a dilution of 1:100,000 and 10 minutes' exposure to 75°C. The virus is still active after passage through Chamberland filters

of grades L3 to L5 and after 202 days' storage, though its virulence is slightly affected after 234 days' storage (Vasudeva and Lal, 1945a).

A severe strain of ringspot virus has been recorded on potato plants of Gola variety (Sharma and Raychaudhuri, 1956). The virus is inactivated by extracts from chilli, spinach, and strawberry, the chilli extract being more inhibitory than the others. Growth products of *Aspergillus niger* grown in three different media and thiouracil at a concentration of 500 mg./litre inhibited the virus multiplication, and this aspect has been made use of in curing the mosaic of Aran Victory potato, by treating the plants with thiouracil at a concentration of 125 mg./litre for 7 days (25 c.c. per day per plant). Ultra violet light inhibited the virus, the rate of inhibition being dependent on the time of exposure. It is also shown that the UV light affects only the virus particles and not the host metabolism (Sharma and Raychaudhuri, 1956).

Potato Virus Y :—

Phulwa variety of potato, is known to suffer from mosaic by potato virus Y in North India (Pal, 1941, 1943) and the causative virus was first identified by Dr. R. N. Salaman. Observations at Delhi show that the disease can be controlled by roguing (Pal, 1941).

The disease caused by this strain is very serious and is characterized by reduction in size of leaves, stunting and dwarfing of the shoots, puckering of the leaves and production of necrotic spots on both sides of leaves. In the advanced cases, the lower leaves become brittle and yellow, and ultimately they fall off. The apical leaves remain highly mottled and curved ventrally. If the infection takes place when the plant is very young, no tubers are formed. In Craig's Defiance variety of potato the loss in yield is estimated as 77·5 to 87·5% (Khanna and Ganguly, 1953).

Vasudeva and Lal (1945b) isolated the pure strain of potato virus Y *Solanum* Virus 2) from Phulwa plants showing negligible mosaic and veinal necroses or severe mosaic only. The pure strain is transmissible to tobacco varieties Harrison's Special and White Burley, *Nicotiana glutinosa, N. sylvestris, N. rustica, Datura stramonium, Solanum nodiflorum, Petunia hybrida* and President potato variety. Potato Virus Y can be isolated from

a mixture containing viruses X, Y and A, by passage through *Petunia hybrida.*

The virus loses its activity after exposure to 50°C and at 54°C, it becomes innocuous. It is inactivated by storage of the standard extract for 24 hours at room temperature, and at a dilution of 1:1,000. The virus is held back when passed through Chamberland filters L to L5 (Vasudeva and Lal, 1945b).

Potato Virus A :—

Phulwa and Dharjeeling Red Round potato varieties were found to be infected with Potato Virus A (*Solanum* Virus 3), when the former exhibited yellowing of leaf margins or a brilliant deep yellow mottle, and the latter extreme leaf crinkling (Vasudeva and Ramamoorthy, 1946). The mild mosaic caused by this virus, does not cause as high yield reductions as Potato virus Y (52% due to Potato virus A, compared to 72 to 92% due to Virus Y). Nevertheless, it is one of the most widely prevalent virus diseases of potatoes in India (Vasudeva and Azad, 1952).

Strain of Potato Virus X :—

Solanum virus 6, a strain of Potato virus X, producing necrotic spots on the leaves of Phulwa potato plants has been identified for the first time in India (Vasudeva and Gupta, 1946). The spots are of different sizes, and are found scattered both on young and old leaves, these being more on the lower leaflets. In extreme cases, the leaf-margins or even the entire leaflets may dry up. Transmission is achieved by sap inoculation using carborundum and also by grafting. The strain can infect tobacco variety White Burley, *Capsicum annuum, Datura stramonium,* potato variety President and Epicure.

Control Measures :—

Selection of good seed free from infection, timely sowing, and periodical roguing, often prove very useful in reducing the intensity of infection (Khanna and Ganguly, 1953; Pal, 1941).

3. *Mosaic of Sann hemp (Crotalaria juncea L.)*

Raychaudhuri (1947a) reported a mosaic disease of Sann hemp, *Crotalaria juncea* L. from Delhi.

Symptoms :—

Foliar mottling, stunting and in severe cases malformation, are the main symptoms of the disease. The dark green patches on the leaves alternate with light green ones and the upper side is often raised with corresponding depressions on the lower side. Anatomically, the mesophyll tissues are thinner and the intercellular spaces fewer than the healthy leaves. In advanced stages, the differentation into mesophyll and spongy tissues is lacking in the infected leaves. The cells are roughly isodiametric in transection and their chloroplast somewhat indistinct. The vascular tissues of the mottled leaf show hypertrophy of a few phloem cells.

Physical properties of the Virus :—

The virus has been purified using the method described by Bawden (1950) and a solution of very fine acicular crystals obtained.

The thermal death point of the virus lies between 68° to 70°C, has a longevity *in vitro* of 71 to 76 days and withstands a dilution of 1:1,000 to 1:5,000 (Raychaudhuri, 1947a, 1948). Das Gupta *et al.* (1951) showed that crystalline preparations under electron microscope revealed spherical nature of the particles composing the virus, with a diameter ranging from 26 to 40 mμ, while De (1951) reported a mean diameter of 37·4±6mμ. Ultra violet absorption spectra of Sann hemp mosaic virus and Cowpea mosaic virus reveal, that the two viruses are distinct strains with differences in the tyrosine-tryptophane components of the viruses and hence the inability of the two viruses to cross infect the hosts (Raychaudhuri and Ramamoorthy, 1949). The virus operates over a pH range of 1·55 to 9·87, and at a dilution of 1:10, it is inactivated by treatment with 0·5% copper sulphate (Anonymous, 1952a).

Transmission and Host Range :—

Sann hemp mosaic virus can easily be transmitted to healthy plants by sap-inoculation and the symptoms appear within 6 to 8 days. When the young leaves are punctured with insect needles dipped in inoculum, the symptoms appear within 3 to 4 days (Raychaudhuri, 1947a). No evidence of seed transmission is available. The virus does not infect cowpea (Raychaudhuri and Ramamoorthy, 1949) but can infect *Crotalaria striata* (Anonymous, 1947).

Southern Sann hemp mosaic :—

Capoor (1950) described a mosaic disease affecting *Crotalaria juncea* from Bombay, though the presence of the disease was noted as early as 1940 at Poona. The disease resembles the Delhi type described by Raychaudhuri (1947a) in general symptoms but differ greatly in host range and physical properties of the virus. It is, therefore, given a new name to distinguish it from the Delhi type, "Southern Sann hemp mosaic" (Capoor, 1950).

Symptoms :—

Like the Delhi type, the Southern sann hemp mosaic virus causes foliar stunting, slight malformation and puckering in the form of raised, blister-like green areas alternating with pale green ones. Occasionally however, thin elongated enations are seen on the ventral side of the leaves. The affected plants produce little seeds and are dwarfed (Capoor, 1950).

Transmission and Host Range :—

The virus can readily be transmitted by sap-inoculation and transmission by seed is not known. Besides *Crotalaria juncea*, the virus infects six others species of *Crotalaria, Lathyrus sativus, Phaseolus vulgaris, P. lunatus, Vigna unguiculata* and *V. sesquipedalis*. Local lesions develop on *Nicotiana glutinosa, N. sylvestris, Datura stramonium* and *Capsicum frutescens*. In tobacco, the virus is carried symptomlessly at low temperature, producing faint mottling at higher ones; tomato, *Solanum nigrum* and *S. nodiflorum* carry the virus symptomlessly (Capoor, 1950; Anonymous, 1954a).

Physical properties of the virus :—

The Southern sann hemp mosaic virus withstands a dilution of $1 : 10^{-5}$, survives after 10 minutes' exposure to 90°C, and the crude sap withstands 557 days' storage. The infectivity is not lost by treatment with 90% ethanol for 24 hours.

Mosaic of Crotalaria mucronata :—

Raychaudhuri and Pathanian (1950) reported a mosaic disease of *Crotalaria mucronata* from Delhi.

Symptoms :—

The symptoms first appear on young leaves in the form of pale areas. An occasional downward curling of the leaf-tips appear later, and typical mosaic mottling develops on the lamina, with a few pale interveinal areas on the older leaves. This disease, unlike the one reported on *Crotalaria juncea* (Raychaudhuri, 1947; Capoor, 1950), is rarely followed by foliar stunting and blistering.

Transmission and Host Range :—

The disease, like that of *Crotalaria juncea,* is sap-transmissible, and the virus infects besides *Crotalaria mucronata,* C. *juncea,* cowpea, *Phaseolus mungo* and *P. aureus.* Symptoms appear within 7 to 12 days after inoculation.

Physical properties of the Virus :—

The thermal death point of the virus lies between 80° and 85°C, and has a longevity *in virto* of 134 to 142 days at 12° to 15°C. It can withstand a dilution of 1:20,000 to 1:30,000 in crude sap.

4. *Mosaic of Cowpea* :—

(a) Mosaic of *Vigna catjang* Walp.

In 1942, cowpea plants (*Vigna catjang* Walp.) growing in the Agricultural College Farm, Poona, showed malformed leaves with dark green areas interspersed with light green ones. The chlorosis became severe at a later stage. The affected plants bore only a few small pods, with broad, light and dark green stripes on the surface, and contained only a few small seeds (Capoor, *et al.,* 1947).

Vasudeva (1942) reported a similar disease affecting Punjab 1 cowpeas, when the plants were inter-cropped with Desi cotton at Lyallpur. The affected plants showed three types of symptoms : — (i) Stunting especially of upper parts with thick foliage, showing vein-clearing, mottling and alternating areas of dark and light green colour, and sometimes raised blister-like undulations, (ii) Uniform foliar chlorosis without distortion, and (iii) Very conspecuous mottling with pale to vivid yellow areas, later turning brown to reddish brown spots.

Anatomically, the diseased leaf showed fusion of the epidermis with the cuticle at various places, paucity of palisade, containing few chloroplasts, discontinuous and irregular tissues, abnormal spongy parenchyma, thickening and enlarging of xylem vessels and sclerenchyma, and disarrangement of vascular bundles. In some cases, the spongy parenchyma was replaced by a large number of elongated cells (Vasudeva, 1942).

Transmission and Host Range :—

The virus is sap-transmissible and better infection is obtained by using 600 mesh carborundum. *Aphis medicaginis* caused 36% infection under glass-house conditions, and about 4% of the commercial cowpea seeds produced infected seedlings. The virus is found to infect besides cowpea, *Phaseolus lunatus* and *Canavelia ensiformis* (Capoor *et al.,* 1947).

Physical properties of the Virus :—

The virus withstands 10 minutes' exposure to 85°C, but at 90°C, it is inactivated. The dilution end point in the crude sap is between 1:50,000 to 1:10,000. Infectivity is lost after 15 days' storage at 24°C, but not after 9 days.

Affinities :—

The virus is considered to be a distinct one from cowpea and Asparagus bean mosaic viruses on account of its distinct physical properties.

(b) Mosaic of *Vigna cylindrica* Skeels :—

The virus: *Marmor vignae* var. *catjang*—Capoor and Varma, 1956.

Chavali cowpea (*Vigna cylindrica* Skeels) is reported to suffer from a mosaic disease. Symptoms of mosaic appear 7 to 19 days after carborundum-sap inoculation. Seeds from infected chavali cowpea carried 17·3 to 22·7% infection. *Aphis medicaginis* caused 25% infection while *Myzus persicae* and *Aphis gossypii* produced 12 and 4 percent infection respectively. The host range included *Crotalaria juncea* and soyabean. *Cyamopsis tetragonoloba* developed numerous necrotic lesions (Capoor and Varma, 1956).

Physical properties of the virus :—

The infectivity of the virus is retained for 19 days at 24°C in sap, but is lost from dried leaves in 6 days. The dilution end point of the virus is found to be between 1:50,000 and 1:1,00,000.

Affinities of the virus :—

The virus is distinguished from that described by Dale, and from Cucumber mosaic virus, by its inability to infect *Dolichos lablab* and cucurbits. It is not possible to decide its relation with the mosaic of cowpea described by Vasudeva (1942), and so a new name is proposed, "*Marmor vignae* var. *catjang*"—a variety of McLean's Cowpea mosaic virus (Capoor and Varma, 1956).

5. *Enation Mosaic of Dolichos lablab* L. :—

A disease of *Dolichos lablab* characterised by mosaic and chlorotic streaks on the leaves is reported from Poona. The size of the leaf blade is markedly reduced by the suppression of growth in the interveinal areas and enations are seen on the lower side (Capoor and Varma, 1948a).

Transmission and Host Range :—

Transmission of the disease is achieved by sap inoculation, and the virus is found to infect a large number of other leguminous plants including beans, *Phaseolus vulgaris* and White Burley tobacco. *Phaseolus vulgaris* reacts with a lethal systemic necrosis and White Burley tobacco shows primary lesions in the form of thin, white rings of necrotic tissue enclosing green areas, and a secondary systemic mottling, which develops 65 days after inoculation (Capoor and Varma, 1948a).

Physical properties of the Virus :—

The dilution end point of the crude sap lies between 5×10^{-6} and 3×10^{-6}. It can resist 10 minutes' heating at 90°C, but is inactivated at 95°C (Capoor and Varma, 1948a).

Affinities of the Virus :—

Though resembling the tobacco mosaic virus in its physical properties, the Enation mosaic virus of *Dolichos lablab* differs from it appreciably in its host range and by the type of symptom it

produces on tobacco. Cross-immunity tests with the two viruses reveal, that neither confers reciprocal protection, showing that the two are not related to each other (Capoor, 1954; Capoor and Varma, 1948a). The virus resembles Bean viruses 4 and 4A in thermal inactivation, and Pea Streak virus in dilution end point. Hence the virus is named "Dolichos enation Mosaic virus" (Capoor and Varma, 1948a).

6. *Yellow Mosaic of Dolichos lablab* L :—

During 1950 at Poona, and since then in many places like Deccan, Gujerat and Khandesh, a disease was observed on *Dolichos lablab*, characterised by broad yellow patches on the leaves (Capoor and Varma, 1950a).

Transmission :—

The disease is transmitted by the white-fly *Bemisia tabaci* Gen., and the virus has an incubation period of 14 to 20 days inside the vector. Juice inoculation and seed transmission gave negative results.

Affinities of the Virus :—

Phaseolus lunatus suffers from a similar disease, transmitted by the same vector, but several tests revealed the inability of the flies to transmit the *Dolichos lablab* virus to *Phaseolus lunatus*, though the yellow mosaic of *Phaseolus lunatus* was innocuous to *Dolichos lablab*. In its transmission, solely by means of the insect, it differs from the Enation mosaic of *Dolichos lablab* and hence a new name is proposed for the virus, "Yellow mosaic of *Dolichos lablab*" (Capoor and Varma, 1950a).

7. *Yellow Mosaic of Double-bean* (*Phaseolus lunatus* L.)

In 1939, a mosaic disease was observed affecting *Phaseolous lunatus*, in Poona. The disease is reported to be prevalent also in Delhi.

Symptoms :—

The disease is characterized by a mosaic pattern, which starts as faintly discoloured patches gradually becoming yellow, 20 days after infection (Capoor and Varma, 1948c).

Transmission and Host Range :—

Sap inoculation and seed transmission giving negative results, the disease is successfully transmitted by the white-fly *Bemisia tabaci* and by bud-grafting.

In addition to *Phaseolus lunatus*, the virus also infects *Phaseolus limensis, P. vulgaris, P. aureus, Dolichos biflorus* and *Canavelia ensiformis.*

Affinities of the Virus :—

The disease is shown to be similar to that described by Uppal as "Mosaic of *Dolichos biflorus*" and is named, "Double bean Yellow mosaic" (Capoor and Varma, 1948c).

8. *Mosaic of Phaseolus mungo* L. :—

A disease causing mosaic of *Phaseolus mungo* has been reported from New Delhi, (Anonymous, 1952a). The disease is sap transmissible and the virus loses its infectivity after 10 minutes' exposure to 60°C, and is inactivated by treatment with 50% alcohol, 0·5% copper sulphate, and 5% carbolic acid. It remains infective for 26 hours at 15° to 37°C (Anonymous, 1952a).

9. *Sterility Mosaic of Pigeon Pea* (*Cajanus cajan* L).

During 1946, 7% of pigeon pea (*Cajanus cajan* L.) in Bombay, and in 1947, 20% in Gujerat, were found to be affected by a disease named, "Pigeon pea sterility mosaic" (Capoor, 1952).

Symptoms :—

The affected plants are characterized by a general pallor, profuse upright vegetative growth, and an absence of flowering branches. The leaves of the affected plant are small, pale and often with a mosaic pattern.

Transmission :—

Out of 75 grafts with healthy plants, the disease appeared in 34 plants, the time taken for the appearance of the disease being 35 to 40 days. Out of 22 plants inoculated with the infective sap, only one showed symptoms after 81 days (Capoor, 1952).

10. *Yellow Vein Mosaic of Bhendi* (*Hibiscus esculentus* L; *Abelomoschus esculentus* Moench).

The Virus : *Hibiscus* Virus 1.

Ochrovena hibiscae n. gen. n. sp. (Capoor and Varma, 1950b).

Kulkarni (1924) reported the presence of a mosaic disease of bhendi (*Hibiscus esculentus* L) in Bombay, causing serious havoc to the cultivators. Subsequently it was found that the disease was widely prevalent in Bombay and Poona (Varma *et al.*, 1950; Uppal *et al.*, 1940).

Symptoms :—

The first visible symptom of the disease is the clearing of the small veins and then of the larger ones, the ill-defined yellowish green to pale green areas latter extending into the mesophyll. In severe infection, the young leaves develop a general chlorosis, rather than actual mosaic patterns. Growth after infection is stunted, the leaves are undersized, petioles short, the flowering sparse and the fruits few (Uppal *et al.*, 1940). It is shown that the appearance of the symptom could be delayed if the soil is depleted of nitrogen or phosphorous (Anonymous, 1942).

Transmission and Host Range :—

"Yellow vein mosaic of bhendi" is shown to be transmissible only by grafting and by the white-fly *Bemisia tabaci*, and is one of the diseases in which the vector-virus relationship has been studied in some great detail (Varma, 1952). It is shown, that to produce 100% infection at least 10 flies are necessary. A preliminary 4-hour fasting period, followed by a feed of one hour on diseased plants, makes the insect acquire the virus and improves their efficiency as vectors. A 30 minutes' feed on healthy plants is adequate for transmission. Post-acquisition fasting up to 4 hours is also effective, and the flies can transmit the disease after 15 minutes' feed on healthy plants. Flies fed on diseased plants for 12 to 24 hours remain infective for life. They can acquire the virus from the youngest leaf, a few days before the disease symptoms appear. The minimum incubation period of the virus inside the fly is 7 hours, but very few insects become infective at the end of this period.

The female flies are shown to be slightly more efficient as vectors than the males. The virus is shown not to pass to the eggs from viruliferous females. It is shown that the flies can carry the "Yellow vein mosaic of bhendi" and that of Pumpkin simultaneously. (Capoor and Varma, 1950b; Varma, 1955a, b).

"Yellow vein mosaic of Bhendi" is transmitted to a number of Malvaceae, like *Hibiscus tetraphyllus, H. manihot, H. cannabinus,* and *Abelomoschus moschatus* by grafting and by the white-fly, while *Hibiscus sabderifa* showed infection only by grafting (Anonymous, 1954d). Partial recovery from the disease is observed in *Hibiscus cannabinus* and *H. sabderifa* (Capoor and Varma, 1950b).

Changes in the physiology of the infected host :—

Govindjee *et al.* (1956) showed that the infected plants contained more leucine, phenylalanine, valine and methionine, asparaginic acid and asparagin than the healthy ones, and in addition, a nin-hydrin-positive substance.

Control Measures :—

Destroying all plants during April and May, growing none in June-July, and roguing the newly raised plants from August is a schedule for cultivation, reported to have given excellent results. Roguing should be done periodically at weekly intervals. Eradication of *Hibiscus tetraphyllus,* a common weed, often prove very useful in reducing the disease incidence. Spraying the crop with fish-oil-rosin soap to control the white-flies once every three weeks, and keeping the field clear of weeds are some more recommendations (Capoor and Varma, 1950b; Varma *et al.,* 1950).

11. *Mosaic of Soya bean* (*Soja max* or *Glycine soja* Sieb and Zucc).

Chattopadhyay and Das (1954) in their review of common virus diseases of crop plants in West Bengal, includes Soya bean as suffering from a mosaic disease.

12. *Mosaic of Cucurbits* :—

(a) *Mosaic of Bottlegourd* (*Lagenaria vulgaris* Ser).

The virus : *Cucumis* virus 2B, Smith

Marmor astrictum var. *lagenari* Holmes (Capoor and Varma, 1948e).

In 1939, a mosaic disease affecting *Lagenaria vulgaris* Ser. was first observed in the Agricultural College Farm, Poona and subsequently in Bombay Province. The disease is sap-transmissible and after 10 to 14 days of inoculation, pale and dark green patches appear on the leaves, which gradually become diffused. The affected leaves are much stunted, veins are thinner, dwarfed and weak. The fruits are small, very light, with some patchy discolouration (Capoor and Varma, 1948e).

Physical properties of the Virus :—

The virus withstands 10 minutes' heating at 95°C, but not at 98°C, and it retains its infectivity at a dilution of 1:1,000,000 and 308 days' storage in the laboratory. It resists 24 hours' treatment with 95% ethanol, and remains viable after dessication (Capoor and Varma, 1948e).

Host Range :—

The virus affects, besides *Lagenaria vulgaris,* cucumber, melon, watermelon, *Citrullus vulgaris* var. *fistullosus, Luffa acutangula* and *Trichsanthes anguina;* Vegetable marrow and *Momardica charantia* are symptomless carriers (Capoor and Varma, 1948e).

The virus is newly described from India, and is considered to be a strain of *Cucumis* Virus 2 (Cucumber Green Mottle mosaic and is named as "*Cucumis* Virus 2B" (Smiths' classification) and *Marmor astrictum* var. *lagenari* (Holmes's classification) by Capoor and Varma (1948e).

(b) *Mosaic of* Lagenaria leucantha *Rus.*

The virus :

Cucumis virus 2C, Smith.

Marmor astrictum var. *subobscurum Holmes.*

n. var. (Vasudeva *et al.,* 1950)

During 1947-'48, a new strain of *Cucumis* virus 2 (Cucumber Green Mottle mosaic virus) was reported from Delhi, on *Lagenaria leucantha,* by Vasudeva *et al.* (1950).

Symptoms : —

The first visible symptom of the disease is irregular light and dark green mottling, developing into mosaic. Vein-banding fol-

lows soon, and the young leaves show dark green blisters, on a crinkled pale green surface. The plants become stunted, and the flower and fruit production is reduced.

Transmission and Host Range : —

The virus is sap-transmissible and the symptoms appear 12 to 18 days (in winter) and 6 to 8 days (in summer) after inoculation. It produces visible symptoms on *Cucrbita moschata* and cucumber. On *C. moschata,* minute green spots appear 15 to 18 days after inoculation, followed by small, yellow circular spots along the veins. On cucumber, small yellow circular spots on young leaves are seen 7 to 10 days after inoculation, these spots later enlarging until the whole leaf appears pale. The virus is carried symptomlessly in *Datura stramonium, Luffa acutangula,* watermelon and *Momardica charantia* (Vasudeva *et al.,* 1950). Tobacco variety White Burley, *Solanum nodiflorum, S. nigrum* also suffer no visible infection, but yet with the virus in them, while the strain does not affect tomato or chilli (Vasudeva and Nariani, 1952).

Physical properties of the Virus :—

The virus has a thermal death point of 86° to 88°C, and dilution end point of 1: 1,000 in standard extract and 1: 10,000 in pure extract. It has a longevity *in vitro* for more than 90 days (Vasudeva *et al.,* 1950).

The inoculum is infective over a pH range of 1·88 to 9·96, though pH stability is influenced by temperature and storage. In standard extract the virus is resistant to 1-2 % nicotine sulphate, 40 % hydrogen sulphide, 0·5 % potasium permanganate, 0·5 % carbolic acid, 0·5 % silver nitrate, 0·1 0·25 % mercuric chloride, 50% alcohol, 50% glycerine and 50% acetone. It is inactivated by 24 hours' treatment with 50% hydrogen sulphide, 1% potassium permanganate, 1% carbolic acid, 1% silver nitrate, 0·5 % formalin and exposure to ultraviolet light for 2 hours, (Raychaudhuri *et al.,* 1951).

Leaf extracts of *Datura stramonium* and chilli, at a dilution of 1: 100, and those of *Solanum nigrum* and tomato at a dilution of 1: 1,000 inhibits the virus. The toxic principle of *Datura stramonium* is destroyed by heating the extract to boiling point and dilution with water, but not by dessication (Vasudeva and Nariani, 1952).

Affinities of the virus : —

The virus differs from "Cucumber Green Mottle mosaic virus" in its ability to cause visible symtoms on watermelon, and its ability to infect *Datura stramonium.* It also differs from Vasudeva and Lal's (1943) "Bottlegourd mosaic virus" in its greater longevity *in vitro,* higher thermal death point and tolerance to dilution. It also differs from "*Cucumis* virus 2B" of Capoor and Varma (1948e) in its higher thermal death point, dilution end point and host range. It is therefore given a new name, "*Cucumis* virus 2C" (Smith) or *Marmor astrictum* var. *subobscurum* (Holmes) n. var. (Vasudeva *et al.,* 1950).

(c) A similar disease, apparently caused by the same virus has been reported on *Lagenaria siceraria* Standl. (Vasudeva *et al.,* 1954).

(d) *Mosaic of bottlegourd* (*Lagenaria vulgaris* Ser.)

The virus :
(*Cucumis* virus 3, Smith. (Vasudeva *et al.,* 1943).

Symptoms :—

The plants affected with the disease, show chlorotic streaks, dark green blisters and wrinkling of the leaves with wavy margins. The older leaves shrivel and drop within 7 weeks; flowering and fruiting are sparse.

Transmission and Host Range : —

The virus is sap-transmissible, and infects besides *Lagenaria vulgaris,* cucumber, *Momardica charantia,* Melon, Watermelon and Vegetable marrow.

Physical properties of the virus : —

The virus is destroyed by 6 hours' storage at room temperature, and loses its infectivity at a dilution of 1: 500, and succumbs to 10 minutes' at 60°C. It does not traverse Chamberland filters L to L5, and its virulence is lost by passage through filter-papers. The virus is named, "*Cucumis* virus 3", (Vasudeva and Lal, 1943).

(e) *Mosaic of Melon* (*Cucumis melo* L.)

A suspected strain of "*Cucumis* virus 1", is reported to be capable of transmission through seed. When seeds of various

members of Cucurbitaceae were raised in sterilised soil, under insect proof conditions, one plant of *Cucumis melo* (melon) showed symptoms of virus infection, with puckering and interveinal mottling of the first and subsequent leaves. Further trials indicated that there was 56% seed transmission of the disease.

The disease has been found transmissible to *Cucumis sativus, Momardica charantia, Datura stramonium, Vigna sinensis,* and tobacco variety German Samson, by mechanical means. This is the first report of seed transmission of virus disease from India, (Vasudeva and Pavgi, 1943).

(f) *Yellow Vein Mosaic of Pumpkin* (*Cucurbita pepo* L.)

In 1951, a disease affecting pumpkin was observed in Poona (Anonymous, 1954c) and is named, "Yellow vein mosaic" of pumpkin.

Symptoms : —

The disease is characterized by pronounced vein-clearing, and irregular chlorotic patches on the lamina. Internodes turn yellow, and the under-sized fruits show yellow patches on the exterior (Varma, 1955b).

Transmission and Host Range : —

The virus is transmitted by the white-fly *Bemisia tabaci,* (Varma, 1955b) the vector needing 15 minutes to acquire the virus. The incubation period of the virus within the vector is 9 hours (Anonymous, 1954d). The insect is reported to be able to carry the viruses causing the Yellow vein mosaic of bhendi and pumpkin, simultaneously (Varma, 1955b).

The virus is transmissible to cucumber and Vegetable marrow, but does not affect any member of Malvaceae.

(g) *Mosaic of Cucumber* (*Cucumis sativus* L.)

The virus extract of Cucumber mosaic (strain not mentioned) is reported to lose its virulence when passed through sand and pulp filter or fuller's earth, (Uppal, 1934). Adsorption of the virus readily occur when Kaolin or fuller's earth, even in minute quantities is added to the virus extract. The virus is active between pH ranges 5 to 9, and inactive below 5. Adsorbed to fuller's earth,

the virus is freed *in vitro,* in an active state, by altering the pH from 6 to 6·7. It is concluded that the adsorption and inactivation of Cucumber mosaic virus are reversible phenomena (Uppal, 1934).

13. *Mosaic of Cardamom* (*Elettaria cardamomum* Maton)

Synonyms of the disease : "Marble" disease,
"Katte" disease.

Cardamom (*Elettaria cardamomum* Maton) is reported to suffer from a mosaic disease (otherwise called "Marble" or "Katte" disease) in Bombay (McRae, 1923), Delhi (Anonymous, 1955b), Kanara (Kulkarni, 1924), Madras (Anstead, 1924), Mysore and Travancore (Uppal *et al.,* 1945).

Symptoms : —

The first visible symptom appears as stripes of deep green tissue over the surface of the leaf, these running from the mid rib to the leaf margin. Diseased plants appear pale from a distance. When the disease is fully developed, the stripes become evenly distributed. Under natural conditions, the plants are susceptible at all stages of growth, though the disease is seldom seen in nursery beds and appears only after transplanting. The affected plants quickly deteriorate; newly formed clumps from infected shoots are small, and show gradual reduction in longevity, ultimately followed by death. Many clumps wither before beginning to yield (Uppal *et al.,* 1945; Varma and Capoor, 1953).

Transmission : —

That the disease is not soil-borne has been proved (Uppal *et al.,* 1945). Sap inoculation gave negative results (Kulkarni, 1924; Uppal *et al.,* 1945; Varma, 1956). The disease is transmitted by the banana aphid, *Pentalonia nigronervosa* Coq. (Varma and Capoor, 1953). The symptoms of the disease appear on healthy plants 21 to 46 days after feeding by the vector (Uppal *et al.,* 1945).

Although banana is the most important host for the aphid, it has been found to breed on cardamom during the winter months, and sometimes on members of Aroideae (Renga Ayyar, 1954; Varma and Capoor, 1953). In areas, where cardamom is propagated vegetatively, infected rhizomes transmit the disease. The virus is found not to affect banana (Varma and Capoor, 1953).

It has been shown that a single feed by the vector makes it capable of transmitting the disease, and that the percentage of infection depends upon the number of insects transmitting the disease (Varma, 1956). Aphids pick up the virus within a feeding time of 15 minutes, and viruliferous aphids transmit the disease within 5 minutes. The maximum time for the insect to acquire the virus and transmit it, has been found to be one hour. The minimum incubation period for the virus in the aphid is 30 minutes, though the ability of the vector to transmit the disease is increased after one hour. The vector is found to loose all the virus it has acquired immediately after feeding either on a healthy banana or cardamom plant.

No congenital transmission has been reported. But, it is shown that nymphs could also act like the adult, as vectors and transmit the disease (Varma, 1956).

Control Measures : —

Total eradication of all old and diseased plants, constant and ruthless roguing at regular intervals, and transplanting healthy seedlings raised under disease-free conditions, are some of the control measures recommended (Galloway, 1935; Varma and Capoor, 1953).

Other plants affected by similar disease : —

(a) *Amomum subulatum* (Greater cardamom), is reported to suffer from, "Foorkey" disease, stated to be very destructive in Darjeeling area (Anonymous, 1955e). Diseased plants produce many stunted shoots, which fail to flower, and the leaves are slightly chlorotic. The disease is transmitted by the same vector as that of cardamom mosaic, namely *Pentalonia nigronervosa*. The disease is said to be quite distinct from the mosaic of cardamom, as evidenced by the distinct symptoms the two viruses produce on wild *Amomum* sp. (Anonymous, 1955b).

(b) A wild species of *Amomum* is reported to suffer from a disease identical with the mosaic disease of cardamom, transmitted by *Pentalonia nigronervosa*, and controllable by roguing (Anonymous, 1955b).

14. *Mosaic of Papaya* (*Carica papaya* L.).

A mosaic disease affecting papaya, (*Carica papaya* L.) is stated to have been prevalent in Bombay during 1940-'50, and in Poona during 1947 (Capoor and Varma, 1948d; Kamat, 1955).

Symptoms : —

Dot-like, solid necrotic spots all over the blades of the newly opened leaves are the first symptoms visible 20 days after inoculation. The foliage of a fully developed plant presents marked signs of mosaic, with blister-like patches of green tissue scattered at random over the surface of the pale green lamina, which is occasionally dwarfed and malformed. The petiole becomes very short. Elongated water soaked areas are seen on the stem and petiole. The fruits are few, abnormally small and are elongated. Numerous circular water soaked lesions, with a solid central spot covering their surface are seen on these fruits (Capoor and Varma, 1948d).

Transmission and Host Range : —

The disease is sap-transmissible. *Aphis gossypii*, collected from cotton and brinjal, *A. malvae*, from *Lagenaria vulgaris*, *Aphis* sp. from *Euphorbia prolifera* and *Myzus persicae* from *Brassica* sp., are also capable of transmitting the disease and it is supposed that one or more of these insects is responsible for the disease in the field. *Myzus persicae* is shown to be a more efficient vector than *Aphis gossypii* (Anonymous, 1954c). Seed transmission has not been reported.

Papaya mosaic virus is shown to infect a large number of papaya varieties like, Washington Special, Honey Dew, Hawaiian, Ranchi, Ceylon, Bombay, Poona Long and Poona Round (Capoor and Varma, 1948d). Among other plants susceptible to the virus are Cucumber, Vegetable marrow, Musk melon, Watermelon, *Luffa acutangula, Lagenaria vulgaris, Trichosanthes anguina, Impatiens balsamina,* sunflower and *Tropaeolum majus* (Anonymous 1954c; d).

Physical properties of the Virus :—

The virus is quite unstable and apparently of the non-persistent type. The infectivity of the virus is lost after 28 hours' storage *in vitro* but it withstands 10 minutes' of 53°C, not 55°C. It is found to be infectious at a dilution of 1: 1,000 (Capoor and Varma, 1948d).

Affinities of the Virus :—

Though capable of infecting a large number of Cucurbitaceous hosts, the virus is shown to be distinct from the mosaic viruses on

Lagenaria and *Cucurbita moschata* by cross-inoculation experiments (Anonymous, 1954c.).

15. *Mosaic of Egg plant (Brinjal) (Solanum melongena* L.)

Egg plant (*Solanum melongena* L.) is reported to suffer from a mosaic disease in Bombay (Kulkarni, 1924) and Delhi (Raychaudhuri, 1947b).

Symptoms : —

Diseased plants are characterised by bright green mottling of the leaves, followed by puckering and crinkling of the lamina. Occasionally, fine, pale, straw-coloured rings, which are either concentric or irregular, develop on the leaf. In some cases, the midrib or its branches extend beyond the lamina, while in others the entire leaf is changed into a filamentous structure. Flowers and fruits are greatly reduced (Raychaudhuri, 1947b).

Transmission and Host Range : —

The disease is transmissible by sap inoculation, by grafting, and by *Myzus persicae* (Anonymous, 1954c; Raychaudhuri, 1947b). *Empoasca devastans* is suspected to act like vector (Raychaudhuri, 1947b). The disease is transmissible to tobacco plants.

Physical properties of the Virus :—

The thermal death point of the virus is found to be between 60° and 70°C. It is found to be infective even after 15 days' storage at 35°F, but is inactivated at a dilution of 1: 1,000 (Anonymous, 1954c).

Affinities of the Virus :—

The virus is shown to be the same as that causing 'Little leaf' of *Vinca* and *Cucurbita moschata* mosaic (Anonymous, 1954b).

16. *Mosaic of Tobacco (Nicotiana tabacum* L.)

In spite of the fact that, tobacco mosaic is widely prevalent in all tobacco growing centres in India, information regarding the disease is meagre. McRae (1926b) reported the disease from Bombay, and Kulkarni (1924) found that it is one of the most destructive diseases of tobacco in Deccan, Karnatak and Gujerat.

Experiments to differentiate *Dolichos* enation mosaic virus and Tobacco mosaic virus, which agree in physical properties, by means of cross-immunization tests, reveal that the two are distinct viruses, the presence of one not inhibiting the entry of the other (Capoor, 1954). Chromatographic studies of the infected plant show the absence of a ninhydrin reacting substance, and the presence of 2 new bands corresponding to asparagine and histidine-lysine (Laloraya *et al.*, 1955).

17. *Mosaic of Tomato (Lycorpersicon esculentum* Mill.)

Incidence of tomato mosaic has been recorded from Bombay (McRae, 1923; Kulkarni, 1924), Pusa (Desai, 1933a) and Delhi (Vasudeva and Lal, 1947). According to Vasudeva and Lal (1947), no variety of tomato in India is resistant to the disease.

Symptoms : —

The diseased plants appear stunted, the young leaves are small, crinkled, deformed, pale yellow and brittle. Sometimes small necrotic areas are seen on the leaves, while in a few cases the leaf margin becomes brown. The diseased plants produce very few, small fruits (Desai, 1933a; Kulkarni, 1924). In Delhi, during 1941-'42, the disease incidence was 24·5 % (Vasudeva and Lal, 1947).

Desai (1933a) believed that the virus represented a filterable cyclostage in the life-history of a bacterium, which he found to be closely associated with the disease.

Control Measures : —

Spraying the plants with insecticides does not cause any reduction in the incidence, although it reduced the insects. Pruning and mixed cropping similarly does not reduce the infection. Roguing is recommended only when the incidence is low. Timely sowing is thought to favour less infection (Vasudeva and Lal, 1947).

A suspected strain of Tomato Aucuba mosaic virus : —

During 1949-'50, a mosaic disease affected tomato plants at the Indian Agricultural Research Institute, New Delhi, causing 2·7 to 4·5 % infection in experimental plots. It showed to be a strain of tobacco mosaic virus, differing from the typical virus, in that it withstood heating for 10 minutes' at 98°C, and produced local necro-

tic lesions, instead of stripes on leaves of eggplant, mottling and vein-banding on tobacco and interveinal mottling on *Solanum nigrum* (Das and Raychaudhuri, 1954).

18. *Mosaic of Chilli* (*Capsicum* sp.)

The occurrence of mosaic disease of chilli, is reported from Bombay (McRae, 1923; Kulkarni, 1924) and Delhi (Raychaudhuri and Jha, 1954).

Symptoms : —

Mosaic mottling and occasional malformation of the leaves are the main symptoms described for *Capsicum fruitescens* L. In some cases, the leaves get reduced to small tendrilar structures. Diseased plants often have diminutive leaves, and few flowers and fruits. The yield of affected plants is very low (Kulkarni, 1924; Raychaudhuri and Jha, 1954).

Transmission and Host Range : —

The disease is transmissible by sap inoculation, by *Aphis gossypii* and thrips. *Aphis gossypii* is thought to be responsible for the natural spread of infection. Transmission through seed does not take place.

The virus has been shown to produce mosaic symptoms on White Burley tobacco, *Nicotiana glutinosa, Nicotiana* selections 78 and 78 A, *Solanum nigrum, Petunia hybrida, Carthamus tinctorius, Cucumis melo* var. *utilissimus* and *C. sativus*. President and Craig's Defiance potato varieties, and *Datura stramonium* carry the virus symptomlessly.

Physical properties of the Virus :—

The virus is inactivated when exposed to 60°C for 10 minutes, and diluted to 1: 30,000; it loses its infectivity when stored for 22 days at 15° to 33°C and 23 days at 7° to 10°C. 50 % nicotine sulphate, 1% silver nitrate, 50% alcohol for 4 hours, 0·25% carbolic acid for 24 hours, and ultra violet radiation for 6 hours also inactivate the virus.

Effect of the virus on Meiosis and Seed fertility : —

Mosaic infection in chilli, causes meiotic disturbances, delayed embryo-sac development and few seeds per fruit. Only the

gametes free from virus-infected tissue are functional. The virus is shown not to penetrate the seed embryo, and the presence of some virus inhibiting substances is suspected to be present in the seed. These two reasons probably explain the non-seed transmission of the chilli mosaic virus (Swaminathan, 1956).

Affinities of the Virus :—

The virus causing mosaic in chilli, is considered to be a distinct one from other mosaic viruses (Raychaudhuri and Jha, 1954).

19. *Mosaic of Radish* (*Raphanus sativus* L.).

A mosaic disease of raidsh (*Raphanus sativus* var. *caudatus*) has been recorded from Bombay (McRae, 1923; Kulkarni, 1924), Poona (Kulkarni, 1924) and Delhi (Raychaudhuri and Pathanian, 1956).

Symptoms :—

The disease is characterized by mosaic mottling of the leaves, accompanied by necrosis and stunting of plants (Raychaudhuri and Pathanian, 1956).

Transmission and Host Range :—

The disease has been shown to be transmissible by sap inoculation and seed transmission does not take place. The host range of the virus is confined to members of Cruciferae, including oil producing species of *Brassica*, in which it produces severe necrosis and death.

Physical properties of the Virus :—

The virus withstands 85°C, for 10 minutes, dilution of 1:10,000,000 and 17 days' storage at 17° to 22°C, and 101 days' storage at 6° to 8°C (Raychaudhuri and Pathanian, 1956).

Affinities of the Virus :—

The virus is considered to be a distinct one from Radish mosaic virus and other viruses affecting cruciferae in its host range and physical properties (Raychaudhuri and Pathanian, 1956).

20. *Yellow Mosaic of Lettuce* (*Lactuca sativa* L.)

During 1946-47, lettuce plants at the Indian Agricultural Research Institute, New Delhi, showed 50 to 80% of Yellow mosaic infection (Vasudeva *et al.*, 1948).

Symptoms :—

The diseased plants show vein-clearing of young leaves, followed by yellow mosaic mottling. Later, pronounced laminar distortion and malformation of inflorescence axis are seen. The flowers are reduced and the market value of the infected leaves is appreciably low.

Transmission and Host Range :—

The virus is shown to be sap-transmissible and 30% seed transmission has been recorded. The virus infects, besides lettuce, Harrison's Special and White Burley tobaccos, and Sutton's Early Market tomato.

Physical properties of the Virus :—

The virus withstands 86°C for 10 minutes, a dilution of 1 : 60,000, and 60 days' storage at 15° to 25°C.

Affinities of the Virus :—

The virus is considered to be a distinct one from other viruses affecting lettuce (Vasudeva *et al.*, 1948).

21. *Distortion Mosaic of* Datura.

The virus :

Datura virus 3, (Capoor and Varma, 1948, n. sp.)
Marmor daturae,
Distortion mosaic virus.

A disease of *Datura alba* Nees. was first observed in Poona in 1939, and later, the disease has been recognised from several places in Bombay Province.

Symptoms :—

The disease is characterized by light and dark green mosaic with blisters of dark green patches on the leaf, and laminar distortion. Diseased plants are pale in appearance. The flowers are severely malformed but dwarfing of plants is rarely seen (Capoor and Varma, 1948b).

Transmission and Host Range :—

The disease is shown to be transmissible by sap, grafting and by insects. *Myzus persicae* Sulz. transmitted the disease from

Datura fastuosa to *D. fastuosa* and *D. alba*, under controlled conditions. The virus is shown to infect tobacco, *Petunia*, and potato producing mosaic symptoms. Datura *stramonium* and *Nicotiana glutinosa* show only local lesions, while it does not affect *Phaseolus vulgaris, Vigna sinensis* and *Solanum melongena* (Capoor and *Varma, 1948b*).

Physical properties of the Virus :—

The virus withstands heating for 10 minutes at 60°C, a dilution of 1 : 10,000 and 13 days' storage in the laboratory. 95% alcohol does not affect its infectivity when treated for 30 hours at 45°C.

The virus is shown to be present in small quantities inside the host plants. The purified virus solution is opalescent and highly birefringent, and shows anisotropy of flow, visible to the naked eye. The virus, iso-electric at pH 4·5, forms long, rod-shaped paracrystals at pH 5·5 and aggregates into small spindles on further concentration. True crystals are not formed, remaining in a liquid crystaline state. The purified preparation, free from pigmentation, gives protein reactions (Capoor, 1950b).

Other species of Datura *affected by Distortion Mosaic virus* :—

(a) *Datura fastuosa* L. is reported to be affected by "Distortion mosaic virus" of *Datura alba*, but the symptom is only a mild mosaic mottle (Capoor and Varma, 1948b).

(b) *Datura inoxia* Mill. shows severe puckering and distortion of leaves and flowers (Capoor and Varma, 1952). The disease is sap-transmissible, and the aphids *Myzus persicae* and *Aphis gossypii* are also capable of transmitting the disease. The host range appears to be confined to solanaceae, except for *Tetragonia expansa* in which the virus causes concentric rings. The virus is inactivated by 6 days' exposure to air at room temperature, and by dessication at room temperature. It withstands a temperature of 45°C for more than 100 days and also complete dessication at this temperature (Capoor and Varma, 1952).

22. *Mosaic of other plants* :—

(a) *Mosaic of Banana* (*Musa* sp.).

Banana is known to suffer from a mosaic disease, which is transmitted by sap as well as by the banana aphid, *Pentalonia*

nigronervosa Coq. (Kamat, 1955). The disease has been successfully transmitted to cucumber (Anonymous, 1954d).

(b) *Mosaic of Maize (Zea mays.).*

The mosaic of maize has been reported to be sap-transmissible and by *Aphis miadis, A. gossypii* and *Myzus persicae.* The disease has been transmitted to Sudan grass, *Euchlaena mexicana, Dactyloclenium aegyptiacum* and *Coix-lacryma-jobi;* to maize by *Aphis maidis* and *A. gossypii* and to maize and sorghum by *Myzus persicae* (Anonymous, 1954d, 1955b).

(c) *Mosaic of* Malvastrum coromandelianum *Garcke.*

Vein-clearing of young foliage, and the upward curling of leaves, are the symptoms seen in a mosaic disease of *Malvastrum coromandelianum* Garcke. The condition resembles the vein-clearing mosaic of *Hibiscus esculentus* and tobacco leaf-curl virus. It is suggested that *Malvastrum coromandelianum* might represent a secondary host for the 'Yellow vein mosaic' virus of *Hibiscus esculentus* (Venkatarayan, 1947).

(d) *Enation mosaic of* Hibiscus rosa-sinensis :—

Enation mosaic of *Hibiscus rosa- sinensis* is transmitted by the white-fly, *Bemisia tabaci* to *Hibiscus esculentus,* cotton and *Zinnia elegans* (Anonymous, 1952a).

(e) *Mosaic of* Nicotiana glauca *R. Grah.*

During 1951, a mosaic disease of *Nicotiana glauca* R. Grah. was observed in New Delhi causing 20% infection. The disease increased to 40% during 1952. Dark green mottling, reduction of leaf-size, wavy margins and slight malformation of the leaves are the main symptoms of the disease (Nariani and Singh, 1952).

Transmission and Host Range :—

The virus is readily transmitted by sap-inoculation and better results are obtained by using carborundum. First symptoms appear 15 days after inoculation. The virus has a wide host range of solanaceae and cucurbitaceae. In tomato, fern leaf symptoms are produced. In tobacco varieties Harrison's Special and White Burley, the lamina is reduced to tendrilar structures.

Physical properties of the Virus :—

The virus in standard extract remains infective after an exposure to 57°C for 10 minutes, but loses infectivity at 60°C. Crude juice withstands dilution up to 1 : 100, and at 1 : 200, it is rendered innocuous. In standard extract it remains infective for 7 days at 6° to 8°C and after 8 days at the same temperature, the virus becomes innocuous.

Affinities of the Virus :—

The virus is considered to be closely related to *Cucumis* virus 1. (Nariani and Singh, 1952).

(f) *Mosaic of Mustard* (Sarson) (*Brassica* sp.).

A severe mosaic of Indian mustard (Sarson) is reported from Simla (Anonymous, 1955b).

(g) *Momardica charantia* L. and

(h) *Helianthus annuus* L. have been reported to show mosaic symptoms in Bombay (Uppal, 1933).

STREAK DISEASES

The symptoms of 'streak' being variable, it is often used to indicate necrotic or light green areas or stripes, usually arranged in a definite manner, often parallel to the veins. The incidence of streak diseases in India is very low.

1. *Streak of Sugar cane (Saccharum officinarum* L.)

The virus : *Saccharum* virus 4, Smith.

Very few records of streak disease of sugar cane are available in India. Dastur (1926) reported the disease from Central Provinces, and McRae (1928) recorded the same from Pusa. In both places the disease was found affecting the Uba cane variety.

Symptoms :—

The leaves of the affected plant show numerous stripes of light green areas, running parallel to the veins. In the bleached areas, thin, dark green lines are visible. The affected young leaves assume a stiff, erect position and sometimes show slight crinkling. The streaks do not become confluent; sometimes irregularly outlined designs are seen (Dastur, 1926).

No secondary effects are seen due to the streak disease and no reduction in the yield of molasses is recorded (Dastur, 1926).

2. *Streak of tomato* (*Lycopersicon esculentum* Mill.)

During 1941-'42 streak, associated with leaf-roll of tomatoes has been recorded in Delhi as causing 9·2% infection. The highest incidence of streak as well as other virus diseases of tomato was observed in October, and plants showing streak symptoms were more than the others. Metereological conditions affected the disease incidence probably because, they affected the insect population. Of the virus diseases of tomato, recorded, streak causes the greatest reduction in yield (Vasudeva and Lal, 1947).

CHLOROTIC DISEASES

Pronounced yellowing of the leaf, distinguishes this type of disease from mosaic. As in mosaic, in chlorosis also, other subsidiary symptoms are often seen. A few plants are known to suffer from chlorotic diseases of viral nature in India.

1. *Chlorosis of Jute* (*Chorchorus* sp.)

Jute (*Corchorus capsularis* and *C. olitorius*) is known to suffer from a disease, marked by extreme chlorosis of the leaves, and the cause of the disease was not established for a long time. *Corchorus capsularis* is more susceptible to the disease (Gosh and Kundu, 1954).

The disease sometimes affects only one branch, and the tendency for recovery from the disease is often pronounced. Nitrogenous cattle manure in combination with potash and phosphorous is known to increase the symptoms, while a mixture of calcium, nitrogen, phosphorous and potash, or ammonium sulphate and super-phosphate reduce the disease (Anonymous, 1940).

Transmission :—

That the disease is due to a virus is gathered from the transmission studies. The disease has been found to be graft-transmissible, and to some extent through seed (Gosh and Kundu, 1954).

Pollen from chlorotic plants can transmit the disease during fertilisation. If one of the fusing gametes is from chlorotic tissue, the seed becomes virus-borne (Gosh and Kundu, 1954).

2. *Yellow Chlorosis of* Salvia :—

A mosaic disease of *Salvia*, soon developing into an acute chlorosis, has been recorded from Lucknow (Verma and Bose, 1954).

Symptoms :—

The symptoms start on the young leaves as small yellow specks, later growing bigger, coalescing and giving a chlorotic appearance. Vein-clearing, distortion and curling of leaves, and stunting of the entire plant, are often seen accompanying the chlorotic symptom (Verma and Bose, 1954).

The disease has been transmitted to healthy plants by cleft and bud-grafting.

NECROTIC DISEASES

Pronounced death of tissues either localized or complete, is the main symptom of necrotic diseases. Only potato and tomato are known to suffer from such a disease in India.

1. *Necrosis of Potato* (*Solanum tuberosum* L.)

Phulwa potato plants show necrotic spots on young and old leaflets, though more of them are seen on the lower leaflets. At its worst, the margins and even the entire leaflets die out. The disease has been shown to be sap- and graft-transmissible (Vasudeva and Gupta, 1946).

The disease has been transmitted to White Burley tobacco, *Capsicum annum, Datura stramonium* and potato variety President and Epicure. The virus is identified as *Solanum* virus 6, a closely related strain of *Solanum* virus 1 (Vasudeva and Gupta, 1946).

Darjeeling Red Round potatoes, raised in insect proof cages, and from virus-free tubers, showed acropetal necrosis, leading to a wilted condition; the leaves were slightly malformed, and gradually they dried, the whole plant later dying. Virus-free potato plants of the varieties President and Craig's Defiance, also showed malformation, browning of leaves, drying but not death of plants. Sap inoculation and other methods of mechanical transmission were unsuccessful. But the disease was successfully grafted to potato and a few other hosts (Vasudeva and Azad, 1948).

Bemisia tabaci was able to transmit the disease from potato to tobacco variety Harrison's Special and tomato. The virus causing necrosis of Darjeeling Red Round potatoes is identified as a mild strain of Tobacco leaf-curl virus (*Nicotiana* virus 10) (Vasudeva and Azad, 1948).

2. *Necrosis of Tomato* (*Lycopersicon esculentum* Mill.)

Sutton's Early Market tomato plants, produced veinal necrosis and necrotic spots when inoculated with the leaf extracts of potato plants showing severe crinkling and mottling of leaves. The first symptom on tomato, 20 to 22 days after inoculation is the slight inward rolling of leaf margins, followed by both veinal and foliar necrosis. Necrotic streaks are also produced on the petioles. The infected plants are dwarfed and tend to shed their leaves (Vasudeva *et al.*, 1949).

The sap from the infected tomato plants has been inoculated to tobacco, *Datura stramonium, Solanum nodiflorum, Petunia hybrida,* and *Nicotiana glutinosa* (Vasudeva *et al.*, 1949; Anonymous, 1955).

From the study of symptoms on these hosts, it is concluded that the virus causing necrosis in tomato is a combination of *Solanum* viruses 1 and 2 (Vasudeva *et al.*, 1949).

LEAF CURL AND LEAF ROLL DISEASES

The main symptom of these diseases is an excessive curling of leaf margins or apices, leading to a gross distortion of the shape of the lamina, often followed by other symptoms like mottling. There are a few reports regarding these diseases, most of them pertaining to crop and ornamental plants.

1. *Leaf-curl of Tobacco* (*Nicotiana tabacum* L.)

The virus: *Nicotiana* virus 10, Smith.

Tobacco suffers from a highly destructive virus disease, chiefly in North India, caused by the tobacco-leaf-curl virus. In some years the incidence is low ranging from 5 to 10%, while in others, as occurred during 1934-'35 in North India, the disease virtually destroys the entire crop (Pal and Tandon, 1937). The disease has been studied mostly on Pusa H 142 variety (Pal and Tandon, 1937; Pruthi and Samuel, 1937). In the Gangetic Delta, comprising

Bihar in east and Baroda in west, the cigarette variety of tobacco is known to be seriously affected (Pruthi, 1945).

Symptoms : –

The general symptoms of the disease are dwarfing of plants, reduction of leaf surface and size, curling of a portion or the entire lamina, vein-banding or thickening, greening of veins of calyx and ovary wall (Pal and Tandon, 1937). The disease does not appear in the seed bed, but is known to occur only after transplanting. The incidence of the disease increases in late October to early November, and declines in mid-January, showing a second rise in March. Monsoon favours infection (Pal and Tandon, 1937).

The symptoms of tobacco leaf-curl are so variable on tobacco, that it has been possible to identify five types of diseases, classified mainly on symptamatology, and supposed to be caused by different strains of the virus (Pal and Tandon, 1936; Pruthi and Samuel, 1937, 1939; 1941; Pruthi, 1945). Types A, B, C, D, and X are recognised as follows : —

Extreme stunting of plants, profuse greening and enations on leaves ——Type A. and B.

Leaves small, much curled and thickened, rugose brittle and dark green ——Type A.

Leaves large, slightly curled, wrinkled, pale or yellowish green with no thickening or brittleness ——Type B.

Stunting less pronounced, vein-clearing seen and enations absent ——Type C. and D.

Vein-clearing pronounced, with "stitches" in old leaves ——Type C.

Vein-clearing less pronounced with no "stitches" in old leaves ——Type D.

Symptoms variable, often a combination of two or more types ——Type X.

That the type X, is a combination of one or more of the other types is also proved by synthetically inducing the symptoms by a mixture of sap of other types (Pal and Tandon, 1937). The various types can be separated by passage through different hosts. Thus, types A and B are separable from C and D, by symptoms

on *Nicotiana glutinosa, Solanum nigrum* and *Petunia* sp., while A is distinguished from B, and C from D, by reactions on *Nicotiana rustica* (Pal and Tandon, 1937).

Transmission and Host Range : —

The disease has been experimentally transmitted by injection of the infective sap into healthy plants, and by grafting. There is now overwhelming evidence to show that in nature the transmission takes place through the agency of the white-fly *Bemisia tabaci* Gen. (*B. gossypiperda*) (Pal and Tandon, 1937; Pruthi and Samuel, 1937, 1939, 1941, 1942; Pruthi, 1945). Attempts to transmit the disease by the capsid-bug *Cyrtopeltis* (*Gallobelicus*) *crassicornis* Dist. gave negative results (Pruthi and Samuel, 1937). Seed transmission is shown not to take place (Pal and Tandon, 1937). The disease incidence depends on the white-fly population, which increases in autumn and falls down in winter, with a secondary rise in March (Pruthi and Samuel, 1942).

A number of alternate hosts have been described for the tobacco leaf-curl virus, and their presence as weeds, makes it difficult to eradicate the disease. *Ageratum conyzoides, Althaea rosea, Crotalaria juncea, Euphorbia hirta, Hibiscus rosa-sinensis, Launaea asplenifolia, Petunia* sp., *Schizanthus* sp., *Scoparia dulcis, Sida rhombifolia, Solanum nigrum, Vernonia cinerea* and *Zinnia elegans* have been reported to show leaf-curl symptoms (Pal and Tandon, 1937; Garga, 1939; Pruthi and Samuel, 1939, 1941; 1942). The following types of the tobacco leaf-curl virus have been suspected on the various hosts : —

Euphorbia hirta	——B, rarely C, often mixed with X.
Launaea asplenifolia	——X, sometimes D and X.
Lycopersicon esculentum	——A.
Scorparia dulcis	——X.
Sida rhombifolia	——C, sometimes A.
Solanum nigrum	——B, C and D, and all the 3 combined.
Vernonia cinerea	——A and C, both mixed with X.
Zinnia elegans	——C, D and X. (Pruthi and Samuel, 1941).

Nicotiana plumbaginifolia is suspected to be a symptomless carrier (Pal and Tandon, 1937).

Control Measures :

Constant roguing brought down the infection to 19 %. Selection of resistant varieties, spraying and dusting the leaves at 10-day intervals for two months from seed-bed, and abolition of alternate hosts, are some of the control measures recommended (Pal and Tandon, 1937; Pruthi, 1945).

Experiments on the control of the disease by the use of fertilisers reveal, that plants treated with nitrogen, carried the maximum infection; those treated with phosphorous, none; and those with P N, intermediate type of infection. The P/N ratio of healthy plants was higher than that of the diseased. Addition of nitrogenous fertilisers increased the disease, phosphates reduced it while boron, copper, manganese and zinc slightly reduced the infection (Ramamoorthy *et al.*, 1948). Diseased plants show the absence of a ninhydrin reacting substance, which is present in the healthy, and the presence of 2 new bands corresponding to asparagine and histidine-lysine (Laloraya *et al.*, 1955).

2. *Leaf-curl of Tomato* (*Lycopersicon esculentum* Mill.).

Tomato is known to suffer from a leaf-curl disease for several years in Delhi (Vasudeva and Samraj, 1948). Depending upon the age of infection, the plants show dwarfing, puckering of leaves, vein-clearing, excessive branching mild to severe mosaic, and partial to complete sterility. During 1947, 90 % of plants were affected, and from 1943 to '45, 60 varieties have been found to be susceptible to the disease (Vasudeva and Samraj, 1948).

Transmission is achieved by white-fly *Bemisia tabaci* (*B. gossypiperda*) and 30 to 100% of the plants, allowed to feed by the vector developed symptoms within 15 to 25 days. The disease can be grafted to tobacco varieties Harrison's Special, White Burley, German Samson, Potato variety Craig's Defiance, *Datura stramonium, Nicotiana sylvestris* and *N. glutinosa*. On *Datura stramonium* the reaction is violent (Vasudeva and Samraj, 1948).

The symptoms on various hosts reveal that the disease in tomato is due to both mild and severe strains of the leaf-curl virus, both types of strains producing similar symptoms on tomato. The virulence is shown to be reduced, when the virus is allowed to pass through tomato plants (Vasudeva and Samraj, 1948).

3. *Leaf curl of Papaya* (*Carica papaya* L.)

Leaf-curl disease of papaya has been reported from several parts of India. Thomas (1939) reported the disease from Madras, Thomas and Krishnaswami (1939b) an analogous disease from Coimbatore, Sen *et al.* (1946) from Bihar, and Nariani (1956) from Delhi.

Symptoms : —

The disease is characterized by severe curling, crinkling and distortion of leaves accompanied by vein-clearing and reduction in leaf size. Leaf margins are rolled downwards and inwards in the form of inverted cups, and the veins get thickened and dark green in colour. The petioles are highly twisted. The leaves become leathery and brittle, and the inter-veinal areas become rugose, due to hypertrophy. The affected plants fail to flower, or bear very few fruits, and in the advanced stages, defoliation takes place and the growth of the plant is arrested (Nariani, 1956).

Transmission and Host Range : —

The disease has been transmitted by juice inoculation (Sen *et al.*, 1946), and by grafting (Nariani, 1956). White-fly *Bemisia tabaci* has been found capable of transmitting the disease (Nariani, 1956).

The disease is transmissible to White Burley tobacco and tomato by means of the white-fly. Ranchi, Bombay, Honey Dew and Washington varieties of papaya were tested and shown to be susceptible (Nariani, 1956). The disease is not seed-borne Sen *et al.*, 1946).

Effect of the disease on host physiology : —

Paper chromatographic analysis of the sap of healthy papaya leaves and those infected by the leaf-curl virus reveal a marked increase of asparagine in the latter (Laloraya *et al.*, 1956).

Etiology of the disease :—

There appears to be some confusion regarding the etiology of the disease. Sen *et al.*, (1946) found that the disease could be caused by water-logging in the soil, independent of infection. Khan and Mehta (1956) report the presence of galls in the roots of affected plants induced by *Meloidogyne marioni*, a nematode. The nematode is reported to attack besides Papaya, *Phaseolus mungo*, *Solanum melongena*, tomato, *Ficus carica*, and *Impatiens balsamina* (Khan and Mehta, 1956). Nariani (1956) identified the causal agent of the leaf-curl of papaya at Delhi as tobacco leaf-curl virus. Thus, it appears that the leaf-curl condition can be brought about by several causes—soil, nematode and virus.

4. *Leaf Curl Mosaic of Sandal* (*Santalum album* L.)

Venkata Rao (1933) observed leaf-curl symptoms accompanied by mosaic on sandal (*Santalum album* L.) in Bangalore and Mysore.

Symptoms :—

There appears to be two stages for the disease. In the first stage, conspicuous spots develop between the veins, followed by inter-veinal mottling and slight rolling of mature leaves; In the pigmented varieties of sandal, a reddish-brown marginal discolouration is seen. The leaves retain their normal size and the flowering and fruiting is normal. The small branches and the leaves generally show a drooping habit.

During the second advanced stage, the leaves show ruffling at the edges even when quite young, followed by wrinkling, mottling, progressive dwarfing, curling and discolouration, ranging from greenish yellow to reddish brown. In contrast to the 'spike disease', the surface reduction in mosaic leaves is usually greater in length than in breadth. The thickness of the petiole is considerably diminished, the leaves become brittle and fall off early. The twigs and branches die back, and adventitious branches arise, showing highly attenuated leaves. The fruits of the affected trees are small and are shed early (Venkata Rao, 1933).

Transmission :—

The virus is transmissible by ring-bark grafts, but not by sap-inoculation. The cause of natural infection is yet unknown.

5. *Leaf Curl of* Zinnia elegans :—

A leaf-curl disease affecting *Zinnia elegans* is reported from Dehra Dun (Mathur, 1933).

Symptoms :—

Thickening of lower surface of vein-lets, preceded by curling of leaf blades, dwarfing of plants (sometimes less than 1 foot), and few, dwarfed flowers, which are pale and partially sterile. During the warm damp season, the axillary buds give rise to short stunted, rosette-like branches with malformed leaves. Leaf-curl is most prominent from July to September, when the white-fly population is also high. Infection begins at the young leaves near the growing points (Mathur, 1933).

Transmission :—

The disease has been found transmissible to healthy plants by the white-fly *Bemisia tabaci.*

Affinities of the virus :—

The virus causing leaf-curl of *Zinnia elegans* is identified as that causing leaf-curl of cotton at Sudan (Mathur, 1932).

6. *Leaf-curl of other plants* :—

(a) Potato (*Solanum tuberosum* L.)

Bismarck variety potato has been known to suffer from a leaf-curl disease, characterized by stunting of the apical shoots, downward curling of leaf-tips and outward or inward curling of leaf-margins. The cause of the disease has been attributed to the tobacco leaf-curl virus (Anonymous, 1951b).

(b) *Hibiscus rosa-sinensis* L.

The plant when affected with the leaf-curl virus shows faint mottling, thickening of veins and reduction of leaves; subsequently enations develop on the veins on the lower side. Puckering is common, and the leaf margins usually curl upwards (Anonymous, 1951b). The disease has been found transmissible by grafting and by white-fly *Bemisia tabaci. Hibiscus esculentus,* H. *tuberculatus, H. manihot,* hollyhock and cotton are the alternate hosts for the virus (Anonymous, 1954d).

(c) *Saponaria vaccaria* L.

The disease was first observed on *Saponaria vaccaria* L. in Delhi during 1952.

The main symptoms of the disease are stunting of plants, curled and reduced leaves, profuse irregular granular outgrowths on the veins of lower side of leaves, scanty flowering, and in some cases of late infection, production of purple leaves (Azad, 1953). The disease has been transmitted to healthy plants by grafting. The disease is shown to be due to a virus different from the tobacco leaf-curl virus (Azad, 1953; Anonymous, 1954-d).

(d) *Physalis peruviana* L.

The disease was first observed in 1953, in New Delhi. The main symptoms of the disease are stunting, dwarfing and curling of leaves, and enations on the lower veins of the leaves. The virus has been identified as tobacco leaf-curl virus (Nariani and Pathanian, 1954).

(e) *Sann hemp* (*Crotalaria juncea* L.)

Production of enations, curling of leaves and dwarfing of plants are the main symptoms of the leaf-curl disease of sann hemp (*Crotalaria juncea* L.). The disease is graft-transmissible (Raychaudhuri, 1948).

7. *Leaf roll of potato.* (*Solanum tuberosum* L.)

Leaf-roll of potatoes is one of the highly destructive diseases of potatoes, and the disease appears to be fairly common in North India. During 1937-'38, 1,000 maunds of seed potatoes of Dunbar, Cavalier and Majestic varieties were obtained from abroad, to replace the mosaic and leaf-roll affected seed potatoes of Agra (Pushkarnath, 1943). Imperia and Sattoo variety are reported to be resistant to the leaf-roll virus (Pal and Pushkarnath, 1951). The disease causes the highest yield reduction in potatoes compared to other potato virus diseases, and in India, the loss is estimated as 72 to 90% (Vasudeva and Azad, 1952). High temperature (summer temperature) reduces the disease incidence in seed potatoes (Thirumalachar, 1954).

Venkata Rao (1930) inoculated the infective juice of potato leaves suffering from leaf-roll, by means of hypodermic needles and fine pin punctures into healthy tomato plants. Symptoms of leaf-roll were observed 50 days after inoculation (Venkata Rao, 1930).

8. *Leaf roll or 'Bobri' of Chilli* (*Capsicum fruitescens* L.)

Leaf-roll or 'bobri', a virus disease of chilli, has been reported from Rajasthan, causing serious havoc on chilli plants (Anonymous, 1951-c). The affected plants are small, and stunted; the leaves exhibit intense rolling, fruiting is retarded and the fruits are small. During 1950-'51, the disease caused 100% damage in Kapasin district (Rajasthan).

9. *Leaf-roll of Tomato* (*Lycopersicon esculentum* Mill.)

Tomato plants are known to suffer from a virus disease, characterized by leaf-roll, associated with streak and mosaic. Typical leaf-roll has however, not been observed in India (Vasudeva and Lal, 1947).

SPIKE DISEASES

In the foregoing diseases, the main symptoms are foliar, while in "spike", the disease is manifested by the entire plant, and particularly by the branches, which appear like a spike, with attenuated leaves and branchlets, often similating a condition described as "witches broom" or "rosette".

1 *Spike of Sandal* (*Santalum album* L.).

The virus:

Sandal spike virus, Coleman.
Santalum virus 1, Smith.
Sandal spike rosette virus, Rao and Iyengar, 1934.
Chlorogenus santali Holmes.

Spike disease of sandal (*Santalum album* L.) is one of the earliest known virus diseases in India. The disease was first observed in Coorg in 1899, by McCarthy. By 1903, the disease extended to other parts, chiefly Mysore, Madras and North Coimbatore. The disease soon spread towards south and south-east India. Salem experienced heavy losses due to the disease and during 1917-'25, 98,734 sapwood trees were removed in North Salem, due to the disease, which increased from 30 to 20,000 acres (Hart and Rengaswamy, 1926) and the ultimate loss in controlling the disease amounted to Rs. 47 lakhs. The annual loss due to spike disease of sandal, in South India, is estimated as Rs. 6 to 7 lakhs (£ 45,000 to £ 52,000) (Narasimhan, 1933).

Symptoms:—

The characteristic symptom of the disease, is the extreme reduction of leaf size and the attenuation of branches into stiff pointed structures resembling a spike. The internodes are shortened, phyllody of flowers is common and the root ends and haustoria die, which gradually leads to the death of the tree (Narasimhan, 1928). Sreenivasaya (1934) has recorded two types of disease, causing almost similar symptoms; one, a non-infectious stunting in South India, due to adverse environmental conditions, not transmissible by grafting and curable by the supply of good soil and a favourable host, the other, the infectious and highly destructive spike disease, transmissible by grafting. The girth of the stem, affected by the spike disease is adversely reduced, and the minimum girth observed for a fairly old tree is ½″ (Hart and Rengaswamy, 1926). The ratio of length and breadth of the affected leaves (L/B) is significantly higher, and can be used as a test to identify the disease (Varadaraja Iyengar, 1938a).

In some cases, the leaves become pale in colour, and in later stages, the spike leaves attain a reddish tinge. The average period from the appearance of the symptoms to the death of the tree is about 15 months, the time varying with the girth of the tree (Rangaswami Iyengar and Griffith, 1940).

Masking of symptoms is sometimes observed in sandal, and hence external appearance sometimes proves useless in detecting the disease (Sreenivasaya, 1930b). Symptomless sandal however, develops symptoms when mechanically injured as when pruning or making holes in stem. Apparently healthy trees can be tested for the virus by the leaf or bark grafting. The masking is influenced by sunshine and temperature. During the masked period the virus appears to be localized in the phloem, where it multiplies and exists in a highly virulent form (Sreenivasaya, 1930b).

Transmission:—

Earlier observations on the cause of sandal spike revealed that the disease is caused by a difficulty in obtaining water to the plant, due to unfavourable selection of the host plant (Jivanna Rao, 1921a, 1921b and 1925). Coleman (1923) showed that the disease could be transmitted by grafting, and through haustoria. Bud-grafting, patch-grafting and leaf-insertion methods gave very good results of transmission (Sreenivasaya, 1930c; Sreenivasaya and Naidu,

1928). Transmission did not take place by patch-grafting of root bark on root, leaf mutilation, wood grafting or injection of tissue fluids. According to Sreenivasaya (1933c), best results of transmission are obtained by the leaf-insertion method. According to Venkata Rao and Iyengar, (1934b) twig grafting gave 100% infection, budding 63%, patch-grafting 18% and leaf insertion 17%. The average incubation period for both bud and twig grafting is three to four months. Callous formation has been shown to occur 10 to 15 days after the insertion of infected leaf into the healthy plant. Transmission of the disease by pollen and seed does not take place (Venkata Rao, 1935).

As early as 1923, Coleman suspected the role of an insect vector in the natural spread of the disease. Dover and Appanna (1934) obtained indirect evidence that a species of *Macrosiphum* from *Lantana camara* can cause infection. Similar results were obtained for studies on transmission by *Moonia albimaculata*. Rangaswami and Sreenivasaya (1935) reported that the vector of the disease is to be found among three types of pentatomidae, two of jassidae, and three of fulgoridae, and that the vectors are active during night. Rangaswami and Griffith (1941) showed that *Jassus indicus* Walk. can effectively transmit the disease, the maximum and minimum incubation period for the virus inside the vector, being 122 and 102 days respectively. The vector of the spike disease has been shown to be active between the months of April and June, with a secondary climax in October to December.

Histopathology :—

In the spiked leaf, six to seven lines of mesophyll cells are packed very closely without any intercellular space, and the cells show a high accumulation of starch (Jivanna Rao, 1921a). The starch thus accumulated disintegrates in the later stages of infection (Narasimhan, 1933). Necrosis of phloem due to collapse of sieve tubes is often seen (Narasimhan, 1934).

A characteristic symptom of the infected leaf is the presence of round or oval vacuolate intra-cellular bodies, in close association with the nucleus. Generally, only a single such body is present in a cell, though rarely, two or three have been observed. These bodies, stain readily with iron-alum haematoxylin or Fleming's triple stain and reacts with Good pastur's Carbol anilin-fuchsin stain, similar to those found in animal virus diseases like

rabies (Narasimhan, 1933). The inclusion bodies have a diameter of 4·3 to 8·7 μ. They have a reticulate matrix containing usually 1 to 11 vacuoles of different sizes. As many as seven proliferations have been observed arising from these bodies.

Effect of environment on the incidence of the disease :—

Many workers have shown that environment exerts a considerable influence on the incidence of spike disease. Hart and Rengaswamy (1926) showed that the spread of the disease is from southwest to northeast, i.e., in the direction of the prevailing monsoon winds at the flowering time. This is in keeping with the view of Sreenivasaya and Rangaswami (1934) that the viruliferous insects, acting as vectors spread along the direction of the wind. The highest infection is in June, while the lowest is in October (Venkata Rao and Iyengar, 1934b). Norris (1930) showed that the nature of host parasitized by the sandal has a considerable bearing on the incidence of infection. The disease is reported to be very high in areas where *Lantana camara* dominates the vegetation, which not only depletes the soil moisture and nutrients but also might harbour insect vectors (Anonymous, 1931). That the role of undergrowth has a pronounced effect on the disease is stressed by many workers. *Zizyphus oenoplia, Dodonaea viscosa, Stachytarpheta indica* and *Vinca rosea* are known to suffer from spike-like diseases and these are considered to act like alternate hosts for sandal spike. Sandal trees parasitic on *Cajanus indicus, Cassia montana, Acacia suma* and *Pongamia glabra* become susceptible to the disease, while those parasitic on *Azadirachta indica, Murraya koenigii, Eugenia jambolana, Toddalia aculeata* and *Dodonaea viscosa* seem to resist infection (Mathew, 1955). Shade, rainfall followed by excessive heat, dense stands of sandal trees, and an annual rainfall of 15 to 20 inches, contribute to the disease (Anonymous, 1931; Mathew, 1955).

Physiology of the spiked sandal :—

Considerable work has been done on the various physiological aspects of the spiked sandal and only the salient points are mentioned here.

The infected tissues show increased diastatic activity, the increase being sometimes up to four times that in the healthy plant (Sreenivasaya and Sastri, 1928; Sastri and Sreenivasaya 1929). Higher enzyme concentration, osomotic concentration, depression

in the freezing point, (due to an increased soluble matter in the sap) more reducing sugars, lower specific conductivity of sap, and lower ratio of specific conductivity to depression of freezing point of the sap characterize the diseased sandal (Sreenivasaya and Sastri, 1928, 1929; Varadaraja Iyengar, 1928; Rao and Sreenivasaya, 1928). Infected leaves show higher total nitrogen, increased water-soluble nitrogen and basic nitrogen, and decreased nitrate nitrogen (with reference to dry weight and total nitrogen content) (Varadaraja Iyengar, 1928; Rao and Sreenivasaya, 1928; Narasimhamurthy and Sreenivasaya, 1929). The accumulation of total organic nitrogen takes place in the diseased foliage, and this is increased by an increase in the proteoclastic activity. The higher peptonase activity of the diseased tissues and tissue fluids accounts for the presence of a higher concentration of amino nitrogen (Subrahmanyam and Sreenivasaya, 1941). Histidine is found in large quantities in the infected leaves, which is found in negligible amounts in the healthy, and its conversion into histamine is believed to explain the necrosis of root ends of spiked plants (Rao, 1933). Mannitol has been found in the spiked plants but not in the healthy (Sreenivasaya 1930a). Increased production of ammonia due to an increased oxidative deamination and increased carbon-di-oxide are observed in the spiked plants. While the healthy plants show pyrocatechin group of tannins, the infected ones show the pyrogallol group. The buffering capacity of tissue fluids is greatly disturbed consequent on infection. (Varadaraja Iyengar, 1933a 1935a and 1937). Oxalic, malic and succinic acids are seen to accumulate in the spiked leaves, and is explained as due to respiratory changes, the oxygen uptake being half and the oxidase and peroxidase activities about the double of the healthy leaves. Lower concentration of calcium either hinders their neutralization or removal by precipitation (Sastri, 1946).

The spiked plants contain lower amounts of calcium and potassium, while phosphorous and manganese are found in greater quantities than in healthy plants (Sreenivasaya and Sastri, 1928; Varadaraja Iyengar, 1928, Rao and Sreenivasaya, 1928; Sreenivasaya and Sastri, 1929b Badami and Iyengar, 1932). The ratio of calcium to nitrogen is low in the infected plant (Varadaraja Iyengar, 1929). The inability of plants to assimilate calcium is the immediate reaction of sandal stems and leaves to spike infection (Varadaraja Iyengar, 1937). There is a high gradient of calcium concentration from stem towards leaves, which tends to flatten out

as the disease progresses. Due to the decreased concentration of calcium, irregularities in the translocation of starch are seen. The root of the infected plant often contains a high concentration of calcium, and the translocation of calcium from the roots to the aerial parts is checked. Nitrogen on the other hand, rapidly reaches the aerial parts, causing proliferation of the vegetative parts at the expense of the reproductive. The reduced size of the affected leaves is probably due to calcium deficiency. In the affected areas, the soil beneath the spiked plants contain twice as much calcium as in healthy plants. It is observed that the immediate cause of spike is poor intake of calcium by roots and growth, due to some toxin secreted after infection. The importance of this view is that it serves to connect together the virus and the physiological theories of the nature of the disease (Varadaraja Iyengar, 1938b).

The higher manganese in the spiked plants has little effect on protein synthesis (Badami and Iyengar, 1932).

Movement of the Virus in the Host :—

That the sandal spike virus moves through the phloem and parenchyma of the cortex has been shown by ringing experiments. The fact that the virus does not pass through the xylem is indirectly shown by the failure of disease transmission by grafting the wood of the infected plant. The most rapid movement recorded is twelve inches in 89 days after inoculation (Venkata Rao and Iyengar, 1934a).

Resistance to the disease :—

Sandal possesses (i) an autogenic resistance, which is largely independent of the nature of the host it parasitises, many sandal varieties remaining healthy in severely infected areas and (ii) an acquired resistance, exhibited when specific hosts are provided under certain natural environmental conditions, the disease incidence and spread being dependent upon the floristic composition of the area. Based on this, two areas have been established in North Salem using resistant strains of sandal, reinforced by hosts imparting immunity like *Melia indica, Dodonaea viscosa* and *Ruta graveolens* (Sreenivasaya, 1948).

In pot culture studies, sandals growing in large pots were more resistant than those in small ones. Deep-rooted hosts are bene-

ficial to sandal. Infected sandals succumb to disease soon, in the absence of a host (Sreenivasaya, 1948; Sreenivasaya and Rangaswami, 1938).

The identification of sandal plants resistant to spike is difficult. Profuse root development, and a strong tendency to haustorial formation are taken to be characters of such varieties. Some resistant varieties are said to have ovate leaf, stem with rich lenticels, and a root with high haustorial capacity (Sreenivasaya, 1930c; Anonymous, 1932). Tissue fluids of sandals, grown in combination with *Ruta graveolens, Murraya koenigii, Azadirachta indica,* and *Toddalia aculeata,* are more buffered than those grown with *Acacia farnesiana.* Thus, a significant correlation would appear to exist between disease resistance and the buffering capacity of the tissue fluids of sandal grown with different hosts (Srinivasan and Sreenivasaya, 1935).

Control measures :—

In certain areas, prompt removal of infected plants appears to have controlled the disease (Hart and Rengaswamy, 1926; Rangaswamy and Griffith, 1939a). Lopping or ringing the affected part is found to be useful in some cases, but proves unsatisfactory when the disease is in the latent form (Anonymous, 1932). Abolition of *Lantana camara,* and other unfavourable hosts of sandal, has given excellent results.

Several methods have been adopted to destroy the affected plants. The older method was to burn down the plant after removing the heartwood, but recently certain chemicals are shown to do the job better. Thus the patent arsenical chemical "Atlas", has become popular (Varadaraja Iyengar, 1935c). Potassium and sodium chlorates, potassium arsenate, (2½ lbs. per gallon) arsenic oxide (4 lb. in 1 lb. of NaoH per gallon) and sodium arsenate also give good results (Varadaraja Iyengar and Rangaswamy, 1935).

2. *Pendulous Spike of Sandal (Santalum album* L.)

Venkata Rao and Iyengar (1934a) described a disease, distinct from the spike disease, and which they designate "Pendulous spike".

Symptoms :—

The individual infected shoots show continuous apical growth usually attaining a height of one or three feet. The leaves are

confined to the upper portions of such branches. The characteristic feature of the disease is the drooping habit of the branchlets, these bearing leaf-blades, which are usually large and broad. Extensive branching is rare, due to the non-development of the axillary buds.

The flowers of the diseased plants are invariably abnormal, the pedicels often elongating to three or four times the length of those in healthy plants. Anthers show a reddish tinge, the pistil is usually seen as a thick cylindrical body, the apex of which is bent on itself in the bud condition. Sterility being very pronounced, fruits are seldom produced. Death of root ends and haustoria does not occur in this disease.

This disease is transmissible by the same grafting methods as applicable to spike disease, and inclusion bodies similar to those found in spike disease are reported in sandals affected with pendulous spike.

3. *Other plants affected with the spike disease* :—

(a) *Dodonaea viscosa* L.
(b) *Vinca rosea* L.
(c) *Zizyphus oenoplia* Mill.

These plants are often seen to suffer from spike-like diseases, producing symptoms similar to those of spiked sandal, like stunted growth, excessive branching, diminutive leaves and internodes and phyllodic flowers. In the leaves of *Vinca rosea* affected with spike, inclusion bodies similar to those found in the leaves of spiked sandal, have been described (Narasimhan, 1928).

In *Vinca rosea,* the diseased parts usually contain 1½ times as much starch as in healthy parts. The ash content and nitrogen show an increase during infection (Varadaraja Iyengar, 1933b). Calcium is seen in greater concentration in roots, less in the leaves of the infected plants, while nitrogen is seen in greater concentration in leaves and less in roots, suggesting a rapid mobilisation of nitrogen to the aerial parts. The protein content of the different parts of the infected plant are generally lower than similar parts in healthy plant, but the reverse condition is seen in the case of ammonia. The infected leaves are further characterized by starch accumulation and increased diastatic activity (Varadaraja Iyengar, 1935b).

In *Dodonaea viscosa* L., non-protein nitrogen constituents of spiked trees show a higher value for nitrate, nitrite, ammonia and

amide nitrogen. Sugars and starch accumulate in the spiked tissues and the diastatic activity of such tissues is always higher than in healthy ones. Low quantities of calcium is yet another feature of infected parts (Sastri and Narayana, 1931).

In *Zizyphus oenoplia* Mill., the affected leaves show a higher accumulation of starch and sugars, with an increased diastatic activity and low calcium content (Sastri and Narayana, 1931, Varadaraja Iyengar, 1935b).

It is obvious from the above observations, that spike disease causes similar disturbances in the physiology of the host in addition to the display of similar symptoms. Muniyappa *et al.* (1941) have shown that the diseased leaf tissue from spiked *Vinca rosea* when grafted to healthy sandal, causes the production of spike symptoms in the latter. These facts definitely show that the sandal spike virus has allernate hosts in the above-said plants.

4. *Rosette of Groundnut* (*Arachis hypogea* L.)

A disease, similar to spike, called the "rosette" disease, has been known to affect groundnut (*Arachis hypogea* L.). The main symptoms of the disease are reduction of leaf and internodes, phyllodic flowers and a bushy appearance of the plant (Anonymous, 1945).

PHYLLODY AND BUNCHY-TOP DISEASES

Phyllody is characterized by the conversion of the floral parts into foliaceous structures, resulting in the sterility of the flower.

1. *Phyllody of Sann hemp* (*Crotalaria juncea* L.)

Bose and Misra (1938) reported twelve different types of phyllody observed from 0·05 to 6·61% in seven varieties of *Crotalaria juncea* at Pusa. The floral parts in the affected plants are transformed into greenish leaf-like structures. Proliferation of floral parts, adherence of vegetative shoots formed by the converted stamen and carpel and fasciation are other symptoms of the disease. Sterility is pronounced in the affected plants. Occurrence of similar disturbance in *Crotalaria striata*, and *C. saltiana* is recorded from Bombay.

Transmission :—

The disease has been successfully transmitted to healthy plants by grafting (Bose and Misra, 1938). Sap-inoculation, the white fly *Bemisia tabaci* and the jassid *Deltocephauls* sp. also caused transmission. (Anonymous, 1952a; 1955d).

Physical properties of the Virus :—

Sap from *Crotalaria juncea* infected with the phyllody virus is shown to remain infective for 370 days at 6° to 8°C. (Anonymous, 1953b).

2. *Phyllody of Sesame* (*Til*) (*Sesamum* sp.)

Dey (1948) recorded the phyllody disease of sesame for the first time in India. Mehta (1952) reported the occurrence of the disease from Uttar Pradesh, and it is now known that the disease is fairly widespread throughout North India.

Symptoms :—

The disease in some areas is called the 'green flowering' disease. The infected plants of *Sesamum indicum* show flowers in which all floral parts except the stamen, are modified into leaf-like structures. The anthers seldom contain pollen, and the plants are often sterile. Fasciation is a common feature. The leaves are small, and near the floral axis they become pale. Phylloid flowers are radially symmetrical, with polysepalous calyx, and the veins of the sepals heavily thickened. The fifth anterior stamen is usually developed unlike in normal flowers, the style is reduced and the carpellary wall is reduced to a leaf-like structure (Pal and Nath, 1935).

Twenty varieties of *Sesamum orientale* as well as *S. occidentale, S. indicum* and *S. radiatum* proved susceptible to the disease.

Transmission :—

Sap transmission is not successful; but the disease has been successfully transmitted by grafting (Pal and Nath, 1935). The jassid *Deltocephalus* sp. has been shown to effectively transmit the disease, the symptoms of which appeared 33 to 59 days after feeding by the vector (Vasudeva and Shambi, 1956).

3. *Bunchy-top of Banana* (*Musa* sp.).

Bunchy-top of banana is a very destructive virus disease, recorded from various parts of India. The disease is fairly widespread in Assam, Bihar, Bombay, Hyderabad, Khandesh, Orissa, Poona, Surat, and Travancore (Kottayam) (Nandhi, 1941; Verghese, 1945; Kamat and Patel, 1951; Varasi, 1954). The high incidence of the disease prevented the export or transport of

banana plants or any articles used in packing them, from the states of Orissa and Travancore-Cochin (Anonymous, 1953a). The disease appears to have been first observed in India during 1943, and is presumed to have come from Ceylon (Varasi, 1954).

Symptoms :—

The disease is characterized by stunted growth of the shoot, dwarfing of leaves (lamina and leaf-stalk), failure of the leaf to emerge from the overlapping leaf-sheaths, chlorotic leaf-margin and lamina, and failure to flower. The disease gets the name, due to the bunchy appearance of the entire plant after infection, and is known to affect all varieties of banana and at all stages of growth (Verghese, 1945).

Transmission :—

The disease is transmitted by the aphid *Pentalonia nigronervosa* Coq.

Control measures :—

Avoiding the use of infected suckers, pouring boiling water on diseased stumps, addition of lime to the underground portions of the diseased plants, destroying the affected plants by cutting them into bits and deeply burying them in the ground and the eradication of insects are some measures of control recommended (Verghese, 1945; Varasi, 1954).

STENOSIS AND LITTLE LEAF DISEASES

1. *Stenosis of Cotton* (*Gossypium* sp.).

The virus :

Cotton small-leaf virus.

Kottur and Patel (1920) described a disease of cotton plants, in all parts of West India, characterized by a malformation of leaves leading to sterility, and of unknown cause, Afzal (1933a, b) reported a similar disease affecting American varieties of cotton grown in the canal waters of Punjab and ascribed symptoms of "stenosis" of cotton. Gokhale (1936) reported a small leaf disease of cotton, the cause of which was not known, and hence he considered it to be a teratological case. It is now clear, that the small leaf disease or stenosis of cotton, is a virus disease, (Uppal *et al.*, 1944). These authors report the occurrence of the disease in Bombay, Madras and Punjab.

Symptoms :—

The diseased plants show extreme stunting of the aerial parts, crinkled deformed and highly reduced leaves, sometimes with yellow patches, reduction of the number of leaf-lobes, normal sized petioles light in colour, discoloured epicalyx, and reduction of root system. A large proportion of developing bolls and young buds on the diseased plants are shed, and the few bolls that are allowed to ripen, contain a few small seeds. The fibre obtained from the diseased plants is weak in quality. (Afzal, 1933a, b).

Starch accumulation takes place in the tissues of leaves, vascular bundles of petioles, stems, and roots of stenosised plants. (Likhite and Desai, 1934).

The disease affects plants at all stages of growth, especially after the plants become six weeks old, and is very virulent during August-September.

Transmission :—

The disease is not seed-borne, nor is it sap-transmissible. While the possibility of a vector relationship is strongly suspected (Afzal, 1933b) the disease at present is shown to be graft-transmissible (Uppal *et al.*, 1944).

Susceptible varieties :—

The most susceptible variety of cotton, to stenosis is reported to be the American variety, while indigenous cottons are shown to be fairly immune to the disease. The common susceptible varieties are Rozi, Jarilla, American, Gaorani and Mungari (Uppal *et al.*, 1944).

2. *Little leaf diseases* :—

Synonyms : 'Small leaf' disease; 'Rosette' disease.

In this group, the characteristic symptom of the disease is manifested mainly on the leaf, which undergoes a striking reduction in size, simulating a "little" or "small" leaf. It should be noted however, that this symptom occurs in other diseases like spike and phyllody. Since no better name for the disease is available and since many have used this foliar symptom to identify the disease, the convention is followed here.

(a) *Little leaf of egg plant (Brinjal) (Solanum melongena* L.).

The virus :

Cranberry False-blossom virus, Dobroscky.
Vaccinium virus 1, Smith.
Chlorogenus vaccinii Holmes (Smith, 1957).

Symptoms :—

The main symptom of the disease is the reduction in leaf size, the new leaves becoming smaller and smaller and sessile. The lamina becomes thin, soft, glabrous and pale green. In thorny varieties the thorns are attenuated or absent. The stimulation of the axillary buds to grow, results in a bushy appearance of the affected plants. Phyllody is very common, and the plants are sterile. The disease affects the plants at all stages of growth (Thomas and Krishnaswami, 1939a) and is widespread in South India.

Transmission and Host range :—

The virus is not sap-transmissible, but is transmitted by grafting. Two species of jassids *Empoasca devastans* and *Eutettix phycitis* are shown to be vectors of the disease. The virus occurs naturally on *Datura fastuosa,* and it has been experimentally transmitted both by grafting and by leaf-hoppers, to tomato, tobacco, potato, *Solanum trilobatum, S. pubescens, S. xanthocarpum, S. seaforthianum* and *Withania somnifera* (Thomas and Krishnaswami, 1939a).

(b) *Little leaf of Vinca rosea* L.

The little leaf of *Vinca rosea* L. has already been described under the spike diseases. The virus has been shown to be transmissible by grafting, and by *Eutettix phycitis* (Anonymous, 1954b).

Histopathological investigations of "little leaves" of Brinjal, *Datura fastuosa,* and *Vinca rosea* reveal that a "little leaf" can be identified by the peculiar anatomical features, from a similar-sized young healthy leaf and a mature healthy leaf. Furthermore, the stomata per unit area is shown to be highest in a small young leaf and lowest in a "little leaf". The nuclei in the "little" leaf are often irregular and sometimes crescent-shaped (John and Malini, *unpublished*). Intra-cellular inclusion bodies have been described in the spiked leaves of *Vinca rosea* and are shown to be similar to those described in the spiked leaves of sandal (Narasimhan, 1928).

(c) *Small leaf of Sann hemp (Crotalaria juncea* L.).

Sterility is shown to be the most important symptom of the small leaf disease of sann hemp which is shown to be due to a sap-transmissible virus. The physical properties of the virus are thermal death point of 85°-90°C, longevity *in vitro* 41-50 days and dilution end point of 1:25:000 to 1:30,000 (Raychaudhuri, 1948).

OTHER VIRUS DISEASES

Under this group a few virus diseases, for which no distinct names are available, are included.

(a) *Big bud of tomato* :—(*Lycopersicon esculentum* Mill.).

The virus :

Cranberry False Blossom virus, Dobroscky.
Vaccinium virus 1, Smith
Chlorogenus vaccinii Holmes (Smith, 1957).

During 1943, Sutton's Early Market tomato plants at the Imperial Agricultural Research Institute, New Delhi, showed symptoms of tomato big bud virus, not previously known in India. Erect growth habit, floral anomalies such as adhesion, certain types of overgrowth, virescence and thickening of the pedicels due to the formation of an abnormal tissue in the phloem, were the chief symptoms of the disease.

Transmission :—

The disease has been found to be transmissible by bud grafting and inarching. The symptoms appear 45 days after bud grafting and 105 days after inarching in healthy plants (Vasudeva and Lal, 1946).

(b) *A latent virus disease of tomato* (*Lycopersicon esculentum* Mill).

The cause of a faint mottling of tomato plants suffering from "smalling", has been ascribed to a latent virus, which is known to produce a very faint mottling even in the healthy tomato plants, variety Sutton's Early Market.

Transmission :—

The disease is transmissible by sap, using carborundum. In addition to tomato, the virus affects White Burley tobacco.

Inclusion bodies :—

X-bodies have been described from the epidermal cells of the diseased plants. Usually only one X-body is seen in each cell, and the nuclei in such cells are enlarged and deformed. Prior to the formation of the X-body, small granules appear in the cells, which gradually aggregate, and finally become the X-body. They are usually round, with an average diameter of 25 μ. The crystalline inclusions are cubical, and their number and size are variable. The maximum diameter of one such block is 6 μ.

Physical properties of the Virus :—

The virus has a thermal death point of 59°C. for 10 minutes, a dilution end point of 1:10,000 and longevity *in vitro* of 24 hours at room temperature (Vasudeva and Samraj, 1947).

(c) *Grassy shoot of sugarcane* (*Saccharum officinarum* L.).

A new virus disease has been described as, "grassy shoot" affecting sugarcane, from Lucknow (Anonymous, 1956), and Bombay (Vasudeva, 1956). In Lucknow, the affected variety is Co 453, while in Bombay Co 419 showed the disease.

Symptoms :—

On Co 453, the symptoms are stunted grassy shoots, with narrow chlorotic yellowish white leaves, thin stalks, with the leaves of the spindle chlorotic, and premature sprouting of side shoots, with yellowish white leaves (Anonymous, 1956). In general, the affected plants show a bushy appearance, and the growth of these plants is adversely arrested (Vasudeva, 1956).

Transmission :—

The disease appears through infected cane setts, and *Aphis sacchari* is suspected to be the vector for the disease (Vasudeva, 1956).

Control measures :—

Treatment of cuttings in hot water (50°C.) for two hours appreciably reduced the disease (Vasudeva, 1956).

(d) *Line Pattern of Plums* (*Prunus* sp.).

A new virus disease of plums is reported by Bhargava and Bist (1957), which they call "Line pattern". The disease was first noticed in Ranikhet and Ramgarh orchards in Kumaon during 1955.

Symptoms :—

The leaves of the infected plant show yellow or creamy white chlorotic lines often leading to a net-work. Vein-banding is a common feature. Sometimes "oak-leaf" like symptoms are also seen.

Transmission :—

The disease has been shown to be bud-graft transmissible.

(e) *Virus disease of* Solanum jasminoides *Paxt.*

A virus disease of *Solanum jasminoides* has been described from Simla (Pushkarnath, 1952).

Symptoms :—

The disease produces usually a fleeting type of mottle. On White Burley tobacco, complete collapse of the lower leaves occur within 4 to 5 days after inoculation. Vein-banding and necrotic banding along the veins are also observed. On *Nicotiana glutinosa* mottling is the only symptom. On *Solanum nigrum,* floral anomalies are seen. The disease is also transmissible to *Hyocyamus niger, Petunia* and tomato.

Transmission :—

The virus is sap-transmissible.

Physical properties of the Virus :—

The dilution end point of the virus lies between 1:1,900 and 1:2,000. It can withstand 24 to 48 hours' storage at room temperature, and the thermal death point is 60°C. Fifty per cent alcohol inactivates the virus.

Affinities of the Virus :—

From studies on cross-immunity and host range, the virus is found to behave like Potato virus Y in many respects, yet differing from it appreciably. It is believed that the virus occurring on *Solanum jasminoides* plants at Simla has come from South America, the original home of the host plant.

(f) *Virus disease of* Thevetia neriifolia *Juss* :—

A virus disease affecting *Thevetia neriifolia* Juss. is reported from Delhi (Garga, 1953).

Symptoms :—

The leaves of the affected plant show reduction in leaf-size and extreme curling of margins. Fine chlorotic streaks are seen

along the veinlets, giving a unique pattern. During 1948, there was 100% incidence of the disease.

Transmission and Host Range :—

The disease has been grafted by wedge-grafting. In addition to *Thevetia neriifolia*, the disease has been transmitted to *Datura stramonium*, tobacco varieties Harrison's Special, and White Burley, and Sutton's Early Market tomato. In all these hosts, leaf rolling and smalling are seen.

(g) *Yellowing of* Sorghum (*Sorghum* sp.).

Thomas (1937) has reported a virus disease affecting *Sorghum*, characterized by yellowing of leaves. The disease is not sap-transmissible, but is shown to have a vector in *Pundaluoya simplicia* Distant.

HOST-INDEX

Abelomoschus esculentus Moench.	Yellow vein mosaic (389)
Amomum sp.	Mosaic (396)
Amomum subulatum	Foorkey disease (Mosaic) (396)
Arachis hypogea L.	Rosette (424)
Brassica sp.	Mosaic (405)
Cajanus cajan L.	Sterility mosaic (388)
Capsicum fruitescens L.	Leaf roll (Bobri) (416)
Capsicum sp.	Mosaic (400)
Carica papaya L.	Mosaic (396) and Leaf-curl (412)
Corchorus capsularis L.	Chlorosis (406)
C. olitorius L.	Chlorosis ((406)
Cucumis melo L.	Mosaic (393)
C. sativus L.	Mosaic (394)
Cucurbita pepo L.	Yellow vein mosaic (394)
Crotalaria juncea L.	Mosaic (381, 383), Leaf-curl (415), Phyllody (424) and Small leaf (429).
C. mucronata	Mosaic (383)
Datura alba Nees.	Distortion mosaic (402)
D. fastuosa L.	Distortion mosaic (403)
D. inoxia Mill.	Distortion mosaic (403)
Dodonaea viscosa L.	Spike (423)
Dolichos lablab L.	Enation mosaic (386) and Yellow mosaic (389)
Elettaria cardamomum Maton.	Mosaic (Marble or Katte) (395)
Eleusine corocana Gacertn.	Mosaic (378)
Euchlaena mexicana	Mosaic (378)
Glycine soja Sieb and Zucc.	Mosaic (390)
Gossypium sp.	Stenosis (426)
Helianthus annuus L.	Mosaic (405)

Hibiscus esculentus L. (*Abelomoschus esculentus* Moench.)	Yellow vein mosaic (389)
H. rosa-sinensis L.	Enation mosaic (404) and Leaf-curl (414)
Lactuca sativa L.	Yellow mosaic (401)
Lagenaria leucantha Rus.	Mosaic (391)
L. siceraria Standl.	Mosaic (393)
L. vulgaris Ser.	Mosaic (390)
Lycopersicon esculentum Mill.	Mosaic (399), Streak (406), Leaf-curl (411), Necrosis (408), Leaf-roll (416), Bigbud (429) and Latent virus disease (429)
Malvastrum coromandelianum Garcke.	Mosaic (404)
Momardica charantia L.	Mosaic (405)
Musa sp.	Bunchy top (425) and Mosaic (403)
Nicotiana glauca R. Grah.	Mosaic (404)
N. tabacum L.	Mosaic (398) and Leaf-curl (408)
Phaseolus lunatus L.	Yellow mosaic (389)
P. mungo L.	Mosaic (388)
Physalis peruviana L.	Leaf-curl (415)
Prunus sp.	Line pattern (430)
Raphanus sativus L.	Mosaic (401)
Saccharum officinarum L.	Mosaic (374), Streak (405) and Grassy shoot (430)
Salvia sp.	Yellow chlorosis (407)
Santalum album L.	Spike (416), Pendulous spike (422) and Leaf-curl mosaic (413)
Saponaria vaccaria L.	Leaf-curl (414)
Sesamum sp.	Phyllody (425)
Soja max (*Glycine soja*)	Mosaic (390)
Solanum jasminoides Paxt.	? Virus disease (431)
Solanum melongena L.	Mosaic (398) and Little leaf (428)
Solanum tuberosum L.	Mosaic (379), Leaf-curl (414), Leaf-roll (415) and Necrosis (407)
Sorghum sp.	Mosaic and Yellow disease (432)
Thevetia neriifolia Juss.	? Virus disease (432)
Vigna catjang Walp.	Mosaic (384)
V. cylindrica Skeels	Mosaic (385)
Vinca rosea L.	Spike (423) and Little leaf (428)
Zea mays	Mosaic (404)
Zinnia elegans	Leaf-curl (413)
Zizyphus oenoplia Mill.	Spike (423)

TABLE I

Comparison of the physical properties of the sap-transmissible viruses

No.	Virus	Thermal Death Point	Dilution End Point	Longevity *in vitro*	Reference
1.	Bottle gourd mosaic	86-88°C for 10′	1 : 10,000	90 days at 33-35° C	Vasudeva *et al.*, 1954.
2.	„ „ „ (*Cucumis* Virus 2B)	95° for 10′	1.: 1,000,000	308 days	Capoor and Varma, 1948e.
3.	„ „ „ (*Cucumis* virus 2C)	86-88°C for 10′	1 : 1,000 (crude) 1 : 10,000 (pure)	90 days	Vasudeva *et al.*, 1950.
4.	„ „ „ (*Cucumis* virus 3)	60°C for 10′	1 : 500	6 hours	Vasudeva and Lal, 1943.
5.	Chilli mosaic	60°C for 10′	1 : 30,000	22 days	Raychaudhuri and Jha, 1954.
6.	Cowpea mosaic	85°C for 10′	1 : 50,000 to 1 : 10,000	9 days at 24°C	Capoor *et al.*, 1947.
7.	„ „	——	1 : 50,000 to 1 : 100,000	19 days at 24°C	Capoor and Varma, 1956.
8.	*Datura* Distortion mosaic	60°C for 10′	1 : 10,000	13 days	Capoor and Varma, 1948b.
9.	*Dolichos* enation mosaic	90°C for 10′	1 : 5,000,000 to 1 : 3,000,000	——	Capoor and Varma, 1948a.
10.	Egg plant mosaic	60-70°C	1 : 1,000	15 days at 35°F	Anonymous, 1954c.
11.	Lettuce Yellow mosaic	86°C for 10′	1 : 60,000 (crude)	60 days at 15-25°C	Vasudeva *et al.*, 1948.
12.	*Nicotiana glauca* virus (*Cucumis* virus 1)	57°C for 10′	1 : 100	7 days at 6-8°C	Nariani and Singh, 1952.
13.	Papaya mosaic	53°C for 10′	1 : 1,000	28 hours	Capoor and Varma, 1948d.
14.	*Phaseolus mungo* mosaic	60°C for 10′	1 : 2,000	26 hours at 15-37°C	Anonymous, 1952a.
15.	Potato X	75°C for 10′	1 : 100,000	234 days	Vasudeva and Lal, 1945a.
16.	Potato Y	50-54°C	1 : 1,000	24 hours	Vasudeva and Lal, 1945b.
17.	Radish mosaic	85°C for 10′	1 : 10,000,000	17 days at 17-22°C 101 days at 6-8°C	Raychaudhuri and Pathanian, 1956.
18.	Sann hemp mosaic (*Crotalaria juncea*)	68-70°C	1 : 1,000 to 1 : 5,000	71-76 days	Raychaudhuri, 1947a.
19.	Sann hemp mosaic (*C. mucronata*)	80-85° C	1 : 20,000 to 1 : 30,000	134-142 days at 12-15°C	Raychaudhuri and Pathanian, 1950.
20.	(Southern) Sann hemp mosaic (*C. juncea*)	90° C for 10′	1 : 100,000	557 days	Capoor, 1950.
21.	Sann hemp small leaf	85-90°C	1 : 25,000 to 1 : 30,000	41-50 days	Raychaudhuri, 1948.
22.	Sugarcane mosaic virus X	65°C	1 : 100	6 hours	Chona, 1944.
23.	„ „ „ Y	55°C	1 : 100	6 hours	
24.	„ „ „ Z	45°C	1 : 50	2 hours	
25.	*Solanum jasminoides* virus	60°C	1 : 1,900 to 1 : 2,000	24 to 48 hours	Pushkarnath, 1952.
26.	Tomato latent virus	59°C for 10′	1 : 10,000	24 hours	Vasudeva and Samraj, 1947.

TABLE II

Insect vectors and the diseases transmitted by them.

Insect vector	Disease transmitted	Reference
Aphis gossypii Glover	Chilli mosaic	Raychaudhuri and Jha, 1954.
	Cowpea mosaic	Capoor and Varma, 1956.
	Datura inoxia Distortion mosaic.	Capoor and Varma, 1952.
	Maize mosaic	Anonymous, 1954d.
	Papaya mosaic	Capoor and Varma, 1948d.
A. maidis Fitch.	Maize mosaic	Anonymous, 1954d.
	? Sugarcane mosaic	Sundararaman, 1928a.
A. malvae Koch.	Papaya mosaic	Capoor and Varma, 1948d
A. medicaginis Koch. (*A. craccivora* Koch)	Cowpea mosaic	Capoor *et al.*, 1947.
		Capoor and Varma, 1956.
A. sacchari	? Sugarcane grassy shoot	Vasudeva, 1956.
Bemisia tabaci Gen. (*B. gossypiperda* Misra & Lamba)	Bhendi Yellow Vein mosaic	Varma, 1952.
	Dolichos Yellow mosaic	Capoor and Varma, 1950a.
	Double bean Yellow mosaic	Capoor and Varma, 1948c.
	Hibiscus rosa-sinensis Enation mosaic and Leaf-curl	Anonymous, 1952a; 1954d.
	Papaya leaf-curl	Nariani, 1956.
	Pumpkin Yellow Vein mosaic	Varma, 1955b.
	Potato necrosis	Vasudeva and Azad, 1948.
	Sann hemp phyllody	Anonymous, 1952a.
	Tobacco leaf-curl	Pruthi and Samuel, 1937.
	Tomato leaf-curl	Vasudeva and Samraj, 1948.
	Zinnia leaf-curl	Mathur, 1932.
Deltocephalus sp.	Sann hemp phyllody	Anonymous, 1955d.
	Sesame phyllody	Vasudeva and Shambi, 1956.
Empoasca devastans Distant.	Egg plant little leaf	Thomas and Krishnaswamy, 1939a.
	Egg plant mosaic	Raychaudhuri, 1947b.

Insect vector	Disease transmitted	Reference
Eutettix phycitis	Egg plant little leaf	Thomas and Krishnaswamy, 1939a.
	Vinca rosea little leaf	Anonymous, 1954b.
Jassus indicus Walk.	Sandal spike	Rangaswami and Griffith, 1941.
Macrosiphum sp.	? Sandal spike	Dover and Appanna, 1934.
Moonia albimaculata	? Sandal spike	Dover and Appanna 1934.
Myzus persicae Sulz.	Cowpea mosaic	Capoor and Varma, 1956.
	Datura inoxia Distortion mosaic	Capoor and Varma, 1952.
	Egg plant mosaic	Anonymous, 1954c.
	Maize mosaic	Anonymous, 1954d.
	Papaya mosaic	Capoor and Varma, 1948d.
Pentalonia nigronervosa Coq.	*Amomum* mosaic	Anonymous, 1955b.
	Banana bunchy top	Varasi, 1954.
	Banana mosaic	Kamat, 1955.
	Cardamom mosaic	Varma and Capoor, 1953.
	Foorkey disease	Anonymous, 1955e.
Pundaluoya simplicia Distant.	*Sorghum* yellow	Thomas, 1937.

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