Seeds treated with a solution of Agrimycin and Ceresan wet for 12 hr followed by hot water treatment at 52° to 54° C for 30 min served as control[8].

In a different experiment, seedlings of cultivars Karuna, IR 8 and TN 1 were raised from 9-month-old seeds outside in the the open in 30 cm diameter earthen shallow pans filled with mango orchard soil supplemented with 1 g of ammonium sulphate per kg of soil. Similarly, seedlings were also grown from seeds of the cultivar IR 8 collected from Amritsar. Seedlings were regularly observed for symptom appearance.

Wilting started to appear 15 to 20 days after sowing and the number of wilted seedlings increased gradually until 30 days after sowing. Similarly, Srivastava and Rao[3] reported that seedlings raised from infected seeds were killed in 3 to 5 weeks indicating that the disease was systemic and transmitted internally. The wilted seedlings were carefully uprooted and their roots washed thoroughly in running tap water. Sections of roots, coleoptile, leaf sheath and leaves were mounted on a slide in a drop of water and observed under the microscope. Profuse bacterial streaming was observed in sections from coleoptiles, leaf sheaths and first leaves and the bacterial oozing decreased in the top portions of the tubular leaf sheaths. The topmost wilted leaf showed only feeble oozing of the bacterium. However, vascular bundles of roots did not possess the bacterium indicating that the bacterial cells present in the seed are activated by moisture. These multiply and move upwards along with the transpiration stream finally resulting in the wilting of seedlings. The bacterium was isolated from such wilted seedlings in potato-sucrose-agar medium and its pathogenicity was confirmed by inoculating on healthy plants of IR 8.

However, the seedlings raised in the open showed typical leaf blight symptoms 60 days after sowing to the extent of 10, 3 and 7% in Karuna, IR 8 and TN 1, respectively. Seedlings grown from seeds (cv. IR 8) collected from Amritsar also showed leaf blight infection in 60-day-old plants.

Interestingly, the susceptible rice cultivars differed markedly in the extent of disease transmission. The highly susceptible TN 1 did not develop the symptoms of wilting, though it expresses severe disease incidence at the maximum tillering or heading stages of the crop. This may be because TN 1 possesses larger quantities of phenolic prohibitions toxic to the pathogen[9] than IR 8 or to the presence of high number of bacteriophages in the seed[10] The later development of the disease may be due to escape of few remaining cells of the bacterium into the vascular system, which invariably took longer time in symptom expression. On the other hand, IR 8, Karuna and Parwanipur 1 showed an efficient transmission of the disease to an extent of 5, 23 and 85% of wilted seedlings, respectively.

The movement of the bacterium in the diseased seedlings was monitored for the first time in this study. Upward movement of the bacterium has been observed in seedlings artificially inoculated by dipping the roots in bacterial suspension to induce wilting[11]. My results show that the bacterial cells carried by the seeds, multiply under favourable conditions, move upwards with the growth of the seedlings and infect the aerial parts.

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ON THE DISTRIBUTION OF PLASMODESMATA IN THE EUPHORBIALES

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THE presence or absence of plasmodesmata in the mature foliar epidermis of the family Euphorbiaceae

or, in fact, any other family of the order Euphorbiales of Cronquist[1] is an aspect not reported earlier. The incidence of cytoplasmic connections between the guard cells of two adjacent stomata or between the guard cell of a stoma and the subsidiary/epidermal cell in four species of the Euphorbiaceae was first reported by the present authors[2]. Reports about the presence of these structures in the foliar epidermis of angiosperms, in general, are very rare; it is perhaps not because they are truly absent but because they have not been thoroughly observed.

As regards the presence of plasmodesmata between mature guard cells and the nearby cells (subsidiaries and/or epidermal cells), there is a difference of opinion. For example, according to Carr[3], no plasmodesmata can occur in the walls of the guard cells of a fully formed stoma. He was not convinced with the evidence provided by Pallas and Mollenhauer[4] who noted the incidence of plasmodesmata between the mature guard cells and the epidermal cells. Carr[3] is of the opinion that plasmodesmata will get lost during maturation even if present in the developing stomata. But, on the contrary, Litz and Kimmins[5] observed plasmodesmata between mature guard cells and the subsidiaries of stomata in Fabaceae and Solanaceae. The present authors also observed clear plasmodesmata which are associated with mature stomata in the foliar epidermis of Euphorbiales in general and the Euphorbiaceae in particular (table 1).

Using a light microscope, it is fairly easy to spot the plasmodesmata associated with mature stomata although they are less frequent (say, one among a thousand or more stomata). However, their detection is difficult with an electron microscope. Light and electron microscopic studies reveal that plasmodesmata occur in two different sizes[6-8]. This view is also supported by Clowes and Juniper[6] who contemplated and suggested the name *microplasmodesmata* for plasmodesmata which are seen under electron microscope and whose diameter is c. 25 nm. Similarly the name *macroplasmodesmata* was suggested for plasmodesmats which are described by light microscopists and whose size (across) is 0.2 μm.

The present study, which included nearly 300 taxa of Euphorbiales, revealed plasmodesmata even in paradermal sections (epidermal peels) prepared from herbarium specimens. The differences in the formation of plasmodesmata within stomatal complexes, and between epidermal cells and stomatal complexes can be conveniently categorised into *four* types. Plasmodesmata occur between the guard cells of nearby stomata (Type I—figure 1A); between the guard cell of a stoma and the subsidiary of another stoma (Type II—figure 1B); between the guard cell of a stoma and its own subsidiary (Type III—figure 1C) and between the guard cell of a stoma and the nearby epidermal cell (Type IV—figure 1D). However, where the distinction between the subsidiary and the epidermal cell is difficult, differentiation between Types III and IV is also difficult.

TABLE 1

Distribution of macroplasmodesmata[a] in the Euphorbiales

Taxon	Plasmodesmatal Type[b]			
	I	II	III	IV
1. Aextoxicaceae	−	−	−	−
2.. Buxaceae (*s.l.*)	−	−	−	−
3. *Daphniphyllaceae*	−	−	−	+
4. Euphorbiaceae				
i) *Phyllanthoideae*	+	−	+	+
ii) *Oldfieldioideae*	+	−	−	−
iii) *Acalyphoideae*	+	−	−	−
iv) *Crotonoideae*	+	+	−	−
v) *Euphorbioideae*	+	−	+	+
5. Pandaceae	−	−	−	−
6. Dichapetalaceae[c]	+	−	−	+

[a] This term is independent of the ultrastructural nomenclatural types erected by Robards[10] for describing the plasmodesmata.
[b] For definitions of these types, see text.
[c] This family was not actually included in Euphorbiales by Cronquist[1].
+ Present; − Absent.

While plasmodesmata are not observed in Aextoxicaceae, Buxaceae and Pandaceae (these are the families which are often removed from Euphoribiales and placed in Celastrales)[9], they are common in Euphorbiaceae, Daphniphyllaceae and Dichapetalaceae. Interestingly the family Dichapetalaceae is kept under Euphorbiales by some systematists[9] though not by Cronquist.

According to Robards[10] the lack of plasmodesmata between adjacent cells may be as much significant as their presence with a high frequency, even if it is more

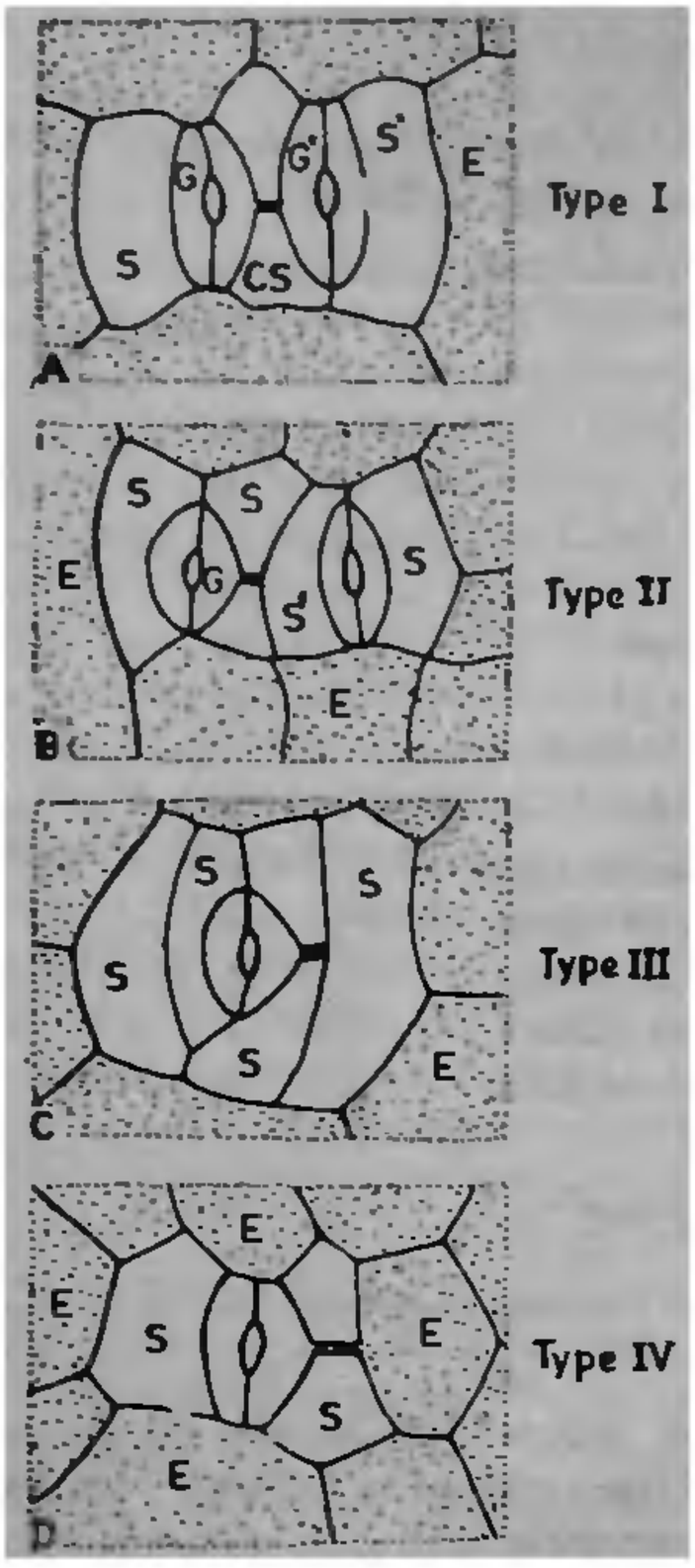

Figure 1. Diagrammatic representation of macro-plasmodesmata in the Euphorbiales. (E–Epidermal cell; G,G′–Guard cells; S,S′–Subsidiary cells; CS–Common subsidiary cell.)

difficult to prove. The present authors also agree with this view point after the present study.

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CHOLINESTERASE SYSTEM DURING 5TH INSTAR OF THE SILK-WORM, *BOMBYX MORI*, L

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INDIVIDUAL tissues are of vital physiological significance, particularly in relation to cocoon formation in silk-worms[1,2]. Investigations were made on the biochemical alterations of proteins[3], fibroin[4], glucose-trehalose[5], total free amino acids, RNA and DNA[6] in the silk-worm to understand the metabolic processes associated with the preparation for spinning of the cocoon. The present investigation deals with changes in the acetylcholine (ACh) content and acetylcholinesterase (AChE) activity in the silk-gland and central nervous system (CNS) of 5th instar larvae of the silk-worm, *Bombyx mori*.

The eggs of silk-worm, *Bombyx mori* were incubated (28 ± 2° C) for about 9 days after which the first instar larvae hatched out. The larvae were reared in the laboratory and fed with fresh mulberry leaves. The lifespan for 5th instar larvae was 6 days after which the spinning of the cocoon started. The ACh content and AChE activity were estimated in the silk-gland and the CNS of these 5th instar larvae daily from the first to the last day of the instar. ACh content was estimated by the method of Augustinsson[7] and AChE activity by the method of Metcalf as given by Glick[8].

The levels of ACh content and AChE activity of the silk-gland (figure 1A) and nervous system (figure 1B) of the silk-worm presented interesting correlation during the development of 5th instar larvae. A sharp rise in the level of ACh content was observed in silk-gland (from 12.6 to 29.7 μmol) and CNS (from 13.1 to 34.7 μmol) towards the end of 5th instar. In contrast to ACh content, AChE activity presented a sudden