noticed that in addition to SG some other larval tissues such as midgut (MG) and malpighian tubules (MT) also possess adequately large and well-reproducible PC.

The mitotic karyotype of this species comprises five pairs of rod-shaped chromosomes (Acrocentric) and one pair of dot-like chromosomes. The *X* and *Y* chromosomes represent one of the five pairs of rods, the *Y* being heterochromatic and deeply stained[6].

The polytene nuclei consist of five long euchromatic arms and a very short strand. Similar to Chironomid and other Agromyzid species, *M. obtusa* also do not possess a clear common chromocentre. In the absence of a common chromocentre all chromosome elements lie freely within the nucleus[6].

The most striking feature noticed in this species is the occurrence of considerably large PC not only in SG but also in MG and MT. The MG and MT chromosomes are as good as the SG chromosomes of *Drosophila* (figures 1–4). In addition, chromosomes are compact and moderate-sized and possess distinct morphology for quick identification. The tissues (SG, MG, and MT) possess a large number of polytene nuclei and are comparatively bigger in size facilitating easy dissection and handling.

With these unique properties, *M. obtusa* is found to be a suitable material for a detailed comparative study of band-interband and puffing patterns in different tissues as well as at different stages of development. Further, it would be good material for the study of gene response against different experimental treatment in tissues having different physiological requirements.

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1. Makino, S., *Cytologia*, 1930, **9**, 272.
2. Beermann, W., Chromosoma, 1952, **5**, 139.
3. Ribbert, D., *Chromosoma*, 1979, **74**, 269.
4. Redfern, C. P. F., *Chromosoma*, 1981, **83**, 221.
5. Spencer, K. A., *Agromyzidae (Diptera) of economic importance*, W. Junk B. V., The Hague, 1973.
6. Singh, O. P. and Gupta, J. P., *Caryologia*, 1981, **34**, 275.
7. Gupta, J. P. and Singh, O. P., *J. Heredity*, 1983, **74**, 365.
8. Singh, O. P. and Gupta, J. P., *Chromosoma*, 1985, **91**, 359.

OCCURRENCE OF A BUD BLIGHT DISEASE OF MULBERRY CAUSED BY *FUSARIUM LATERITIUM* F. SP. *MORI* TYPE-A IN INDIA—A NEW REPORT

J. SUKUMAR and B. R. DAYAKAR YADAV
Moriculture Division, Karnataka State Sericulture Development Institute, Thalaghattapura, Bangalore 560 062, India.

THE bud blight caused by *Fusarium lateritium* Nees f. sp. *mori* (Desmazieres) Matuo et Sato Type-A is a serious disease of mulberry in Japan and other temperate regions[1]. The pathogen affects the buds, causing them to wither and die, resulting in heavy crop and capital loss. During our survey for new diseases and pests of mulberry (*Morus alba* L.) from different geographical regions, we noticed a severe bud blight disease at Ootacamund in the Nilgiris District of Tamil Nadu. Literature survey revealed no report from India of this disease. Considering the severity of the problem a preliminary study on the nature and cause of the disease was undertaken and the results are reported in the present communication.

The disease was noticed during February and March 1987 in varying degrees of intensity on both exotic and indigenous varieties of mulberry viz. Selection-54, Mildew resistant-I, Mysore-5, Dehradun, Mildew resistant-II, Japan-I, Japan-II, Sukasakava and Kosen (table 1), killing more than 50% of their buds. A close examination of the infected plants revealed that the nodal regions surrounding the buds were discoloured and necrotized (figure 1). The necrotic region extended both horizontally and

Table 1 *Intensity of bud blight disease caused by F. lateritium f. sp. mori Type-A on different mulberry varieties*

Variety	Per cent plants infected
Indigenous	
Selection-54 (S54)	100
Mildew resistant-I (MR I)	87
Mysore-5 (M5)	76
Dehradun (DD)	29
Mildew resistant-II (MR II)	6
Exotic	
Japan-I	93
Japan-II	77
Sukasakava	40
Kosen	30

Figure 1a, b. a. A bud blight infected mulberry shoot; **b.** A portion of the stem showing killed bud and sporodochia in the necrotic lesion.

vertically across the stem killing the tissues on the way and blocking the translocation of food, thus starving the buds to death. Severely infected stem portions became dry as the pathogen continued to colonize the entire surface of the stem. In the nectrotic regions numerous reddish brown conidial stroma (sporodochia) were formed producing conidia. The matured conidial stroma appear like reddish brown pustules erupting through the bark. Sexual bodies were not observed in the present study.

The pathogen was isolated on potato dextrose agar medium frequently from the infected stem portions. Following standard descriptions[2,3] it was identified as *F. lateritium* Nees f. sp. *mori* (Desmazieres) Matuo et Sato. In culture the colonies were pink, producing plenty of macroconidia which were falcate with beaked apex, and usually with pedicillate foot cell. A majority of the conidia. were three-septate measuring 21–44 μm in length and 3–4.5 μm in breadth. Chlamydospores were rarely formed but intercalary in position. Based on the conidial length, the number of septa, and the disease symptomatology, four types, designated as A, B, C and D, in the pathogen have been identified[1]. The pathogen presently isolated belonged to the A-type.

Pathogenecity tests were conducted on healthy potted plants. The conidia harvested from a 7-day-old culture was spray-inoculated on to the stem portions and the plants were incubated under high humidity at 20–22°C overnight and later removed to an isolated place for disease expression. Plants receiving sterilized distilled water in place of conidial suspension served as control. Almost all field symptoms of the disease were reproduced consistently on the inoculated plants while the plants kept as control did not develop the disease symptoms. Reisolations from infected tissues yielded a high percentage of *F. lateritium* f. sp. *mori* Type-A.

Mulberry leaves form the chief source of food for silk worms (*Bombyx mori* L.). The bud blight disease now reported may assume epidemic proportions if left uncontrolled and can become a serious constraint in leaf production. Pathogen is reported to enter the host through the wounds caused by insects, wind, frost, leaf harvesting and pruning[1]. The disease appears to be favoured by cool and humid climate prevalent in hilly areas. Detailed investigations on the epidemiology and control of the disease are under progress.

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1. Kimura, K., *Mulberry diseases in Japan*, Kenpakusha Publishing Co., Tokyo, Japan, 1979, p. 163.
2. Booth, C., *The genus Fusarium*, Eastern Press, London, 1977, p. 237.
3. Joffe, A. Z., *Mycopathol. Mycol:, Appl.*, 1974, **53**, 201.