(species of *Mus* and *Rattus*). In the following months during summer (February–May), there is an increase in seed-shed. By the end of summer (last week of May to first week of June) there are enough seeds on the forest floor (Figure 2 *a*, *b*) and a large number of rats relishing on the abundant food supply. We have noticed many rats, which are normally nocturnal, feeding on bamboo seeds even during mid-day. Then starts the rainy season. The transition from summer to the rainy season is very rapid and crucial. With the onset of the rains, bamboo seeds germinate in a few days. Now, instead of a layer of bamboo seeds there is a lush green carpet of bamboo seedlings (Figure 2 *c*). All of a sudden there is no food for the seed predators. But there is sufficient food for herbivores.

In places where there are large bamboo forests, the sudden transition from plenty of food to the near-total absence of food forces millions of rats out of the bamboo forests. They land in farms in the vicinity and play havoc with the standing crops, and devour the grains stored in granaries, thus bringing about famine as an aftermath of bamboo flowering. This dynamic interaction between bamboo flowering, bamboo-seed predators (mainly rats), climatic change (the sudden transition from summer to the rainy season) and the rapid transformation of seeds into seedlings (not a gradual depletion), was not realized earlier. Interestingly, Janzen[7] has stated that 'As the seed is exhausted by predation and germination, the rats emigrate.' Nag[11] has quoted the following from a letter to the administration by the Reverend Lorrain, 'It appears that the rats began to get more than extraordinarily troublesome years before the simultaneous seeding of the mau-thing bamboos, but as soon as the seeding was over they increased to such an extent that no human power could save the crops from their depredation.' This also indicates that the exodus of rats occurs at the end and not during seeding.

In Mizoram, people have been experiencing famines at regular intervals, that they can predict the impending famine. The famine of 1862 is well documented. About 30 years later there was another famine (as was expected)[11]. In more recent history, in the early 1960s there was a coincidence of large-scale bamboo flowering and a severe famine in northeast India. In Mizoram, when there was large-scale flowering of bamboos in the latter half of 1970s, there was a phenomenal increase in the rat population. About 2.5 million rats were reportedly killed in just one year, i.e. 1978 (ref. 9).

The northeastern region of India is very rich in bamboo forests. Slash and burn shifting cultivation (clearing a part of the forest, burning the resultant biomass and adding it as a manure, and cultivating the land for few years, before shifting to a new site to do the same) is very common in this part of the country[15]. This practice leaves many small farms in the midst of (bamboo) forests. Given the peculiar geography and cultivation practice in northeast India, we strongly feel that this belief, of a connection between incidences of bamboo flowering and famines, may not be a myth but a real happening.

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C. K. JOHN
R. S. NADGAUDA*

Tissue Culture Pilot Plant,
National Chemical Laboratory,
Pune 411 008, India
**For correspondence*
e-mail: rsn@ems.ncl.res.in

Antibacterial properties of cyanobacteria: A cost-effective and eco-friendly approach to control bacterial leaf spot disease of chilli

Excessive use of toxic chemicals for controlling crop diseases has caused unprecedented ill-effects on the environment. The growing concern about the harmful effects of such chemicals has necessitated the need for a change in our approach to manage crop diseases in an eco-friendly manner. Search for alternatives with less harmful effects on the environment attracted scientists to look for biocides from plants, including those of microbial origin[1,2]. This has particular concern for countries like ours where agriculture is the prime occupation. Cyanobacteria, which constitute a versatile group of microorganisms, occur in diverse habitats ranging from alkaline hot springs to permanent snow fields in the poles. In addition to their ability to fix atmospheric carbon, many cyanobacteria are capable of fixing molecular nitrogen. Certain cyanobacterial species are known for their toxin-production properties[3,4]. Three such strains namely *Lyngbya majuscula*, *Microcystis aeruginosa* and *Plectonema boryanum* were tested for their antibacterial properties

towards the control of leaf spot disease of chilli caused by *Xanthomonas vesicatoria*. First reported in 1953 at Pune[5], the bacterial leaf spot disease has become a problem in most of the chilli-growing areas in the country, including Rajasthan. With distinctly raised spots on chilli leaves, this bacterial disease causes great economic loss to the farmers.

Cyanobacterial samples were collected from natural habitats (Fateh Sagar Lake of Udaipur). Standard microbial techniques were employed for selection, isolation and clonal propagation of cyanobacteria[6]. In brief, cyanobacterial mats collected from natural habitats were crushed with sterilized glass beads, diluted in sterilized double distilled water and spread on agar plates containing BG-II medium[7] and incubated until visible colonies appeared. Identification was made following Desikachary[8]. Colonies were picked up using sterilized micro-capillaries and transferred to culture tubes containing 10 ml liquid BG-II medium. Growth was visible in tubes after 10 days of incubation. Such clonal cultures were washed thrice with sterilized double distilled water through repeated centrifugation and subsequently transferred to fresh BG-II medium to obtain desirable population size. Cultures were incubated at 24 ± 1°C and illuminated for 10 days with cool daylight fluorescent tubes (intensity 14.4 W m^{-2}) on surface of the culture vessel with a 18/6 h light/dark cycle.

Cyanobacterial cell extracts were prepared following Shibib *et al.*[9]. For this purpose, 500 g fresh weight of cyanobacterial mass was blended in 1000 ml of 95% ethanol and left at room temperature with occasional shaking for 48 h. The suspension was filtered through a cheesecloth and the filtrate was evaporated at 40–50°C to a residue. On the day of experimentation, extracts were diluted with distilled water to make 100 mg l^{-1}, 200 mg l^{-1} and 500 mg l^{-1} concentrations for foliar spray.

The pathogen, *X. vesicatoria*, was isolated by the standard pour-plate technique and the culture was maintained at 30°C in a bacteriological incubator, according to Dye *et al.*[10]. The bacterium was allowed to grow in the culture medium containing different concentrations of cyanobacterial cell extract and growth was recorded at 0, 24, 48 and 72 h.

For disease spread on plants, the pathogen was inoculated through soil mix. For this purpose, severely infected dried leaves of chilli were powdered and sieved; 500 g of the fine powder was properly mixed with 30 kg of sterilized garden soil (pale brown-coloured sandy loam, pH 7.4; organic carbon, 0.89%; total nitrogen; 0.09%; available P, 0.006%; exchangeable K, 0.12%; cation exchange capacity, 16.3 meq 100 g^{-1}). The soil mix was filled in 15 cm diameter pots. Seeds of chilli (*Capsicum annuum* L.) obtained from Shri Udaipur Krishi Kendra, Udaipur, were sown directly into thirty such pots. Approximately two weeks after emergence, the germinants were thinned to one per pot. To maintain constant soil moisture, all pots were uniformly watered thrice a week with distilled water. The disease appeared on the oldest leaves first and after 4–5 days it spread over on the whole plant. Cell extracts were applied by means of a hand-operated agro sprayer (Turbhe Polycans (P) Ltd, India) at the rate of 0.04 ml cm^{-2}. Extracts were applied separately on each set and each

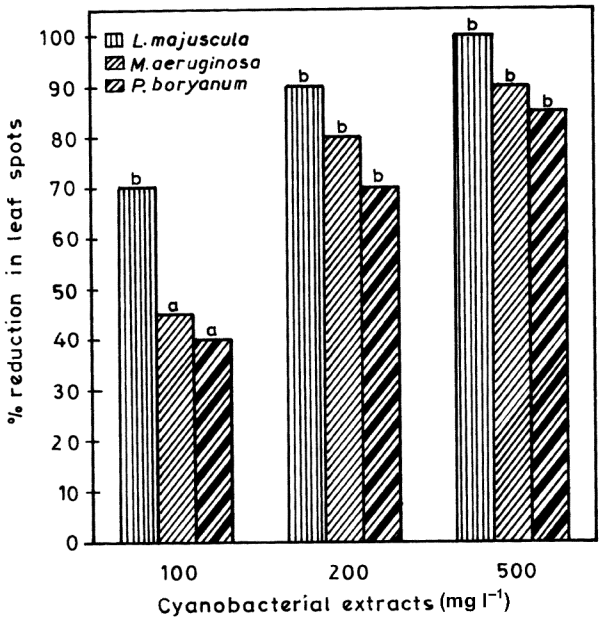

Figure 1. Effect of cyanobacterial extracts on leaf spot disease of chilli. Explanation for a, b as in Table 1.

Table 1. Effect of cyanobacterial cell extracts (100, 200 and 500 mg l^{-1}) on growth of *X. vesicatoria*

Time (h)	Control	Growth of *X. vesicatoria* (NTU)*								
		L. majuscula cell extract (mg l^{-1})			*M. aeruginosa* cell extract (mg l^{-1})			*P. boryanum* cell extract (mg l^{-1})		
		100	200	500	100	200	500	100	200	500
0	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04
24	3.2	1.0^b	0.30^b	0.04^b	1.4^b	0.62^b	0.32^b	1.5^a	1.0^b	0.46^b
48	6.5	2.1^b	0.56^b	0.04^b	2.9^b	1.3^b	0.58^b	3.0^a	1.6^b	0.82^b
72	6.5	2.1^b	0.56^b	0.04^b	2.9^b	1.3^b	0.58^b	3.0^a	1.6^b	0.82^b

*NTU, Nephllo Turbidity Unit.
^{a,b}Differences from respective controls are significant at $P > 0.02$ and $P > 0.01$, respectively (analysis performed using paired *t*-tests).

treatment was replicated thrice. Untreated plants sprayed with distilled water were used as control. Disease rating was made by counting the number of leaf spots per plant.

Daily foliar spray showed significant effects ($P > 0.01$) of cyanobacterial cell extracts in controlling bacterial leaf spots of chilli. Both foliar application tests and laboratory studies proved the superiority of *Lyngbya* cell extract in controlling disease development and growth of the pathogen. Foliar application of 100 mg l^{-1} *Lyngbya* cell extract reduced chilli leaf spots by 70%, whereas for other strains the effectiveness was below 50% (Figure 1). High concentrations were found to be more effective in disease control. At 500 mg l^{-1} of *Lyngbya* cell extract, no foliar spots were observed. At this concentration, for *Microcystis* and *Plectonema*, the effectiveness remained 90%. Laboratory trials also indicated concentration-dependent effects of different cell extracts on growth of *X. vesicatoria*. All the concentrations of the three cyanobacterial extracts significantly reduced the growth of the pathogen (Table 1). Parallel to the foliar spray, no growth of the pathogen was observed at 500 mg l^{-1} of *Lyngbya* cell extract. The study indicated that the cell extracts of all the three cyanobacterial strains have anti-bacterial properties against *X. vesicatoria*; however *Lyngbya* cell extract is most effective. These observations form the first report of antibacterial properties of three cyanobacteria in controlling leaf spot disease of chilli. This may be a cost-effective and eco-friendly approach to control bacterial leaf spot disease of chilli.

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USHA PANDEY*
J. PANDEY†,#

**Department of Botany, G.N.P.G. Girls College, Udaipur 313 002, India*
†Department of Environmental Sciences, M.L. Sukhadia University, Udaipur 313 001, India

Heparinase in purple fluid of the sea hare, *Bursatella leachii*

The physiological role of heparin has been a subject of considerable research in recent years because of its strong anti-coagulant property and its ability to induce the appearance of lipoprotein lipase in the circulatory system of vertebrates, including man. Heparin may be hydrolysed with acid only under rigorous conditions, which results in considerable caramelization, especially of the glucuronic acid, and extensive splitting of heparin. Thus, the products are mono- or di-saccharides from which all the sulphate groups are removed[1]. Studies on enzymatic hydrolysis and isolation and characterization of partial hydrolysis products, would reveal the fine structure of heparin.

Heparin can induce some bacteria to produce a series of enzymes, including glycuronidase, sulphoesterase and sulphamidase as well as heparinase and heparatinase[2]. Heparinase is an α1-4 eliminase which acts specifically on the glycosidic linkage between N-sulphated D-glucosamine and sulphated D-glucuronic acid (or L-iduronic acid) present in heparin[3]. The elimination reaction at this site, catalysed by heparinase, results in the formation of smaller polysaccharides with both a reducing end and an α,β-unsaturated acid end-group. This enzyme, despite a high demand for it by researchers, has not yet been made commercially available. In this present study, we tried the degradation of heparin by the purple fluid of *Bursatella leachii*.

The purple fluid of *B. leachii* was obtained by irritating the animal. The fluid was centrifuged (4000 rpm in 15 min) and the supernatant was lyophilized and stored at 4°C until it was used for determining the heparinase activity. Two methods were followed and are described below.

In azure-A assay, heparinase activity was determined in terms of decrease in heparin content when mixed with the purple fluid extract. The content of heparin was quantified based on decrease in metachromasia with a reagent, azure-A, with some modifications[4]. Heparin was incubated with the purple fluid of *B. leachii* (1 mg of heparin per ml of fluid) for 1 h at room temperature (28 ± 2°C). Placing the mixture in a water bath at 60°C for 10 min stopped the reaction. Various concentrations of the reaction mixture were drawn and added to 10 ml of 0.02 mg/ml azure-A dye solution. The dye showed a metachromatic shift from blue to red in the presence of heparin. The change in optical density was measured within 1 h at 620 nm and compared with a standard curve and controls. Two controls were maintained – heparin + azure-A as a positive control, and heparin + inactivated sample (by heating at 60°C for 1 h) + azure A as negative control.

The heparinase activity was measured with some modifications[5]. Heparin was incubated with the purple fluid of *B. leachii* (1 mg of heparin per ml of fluid) for 1 h at room temperature (28 ± 2°C), and placing the mixture in a water bath at 60°C for 10 min stopped the reaction. The turbidity was removed by centrifugation and the supernatant solution was