solution added to it, thoroughly mixed with a clean glass rod and allowed to stand for a minute. Boehmite and kaolinite absorb the dyes and are similarly coloured, but boehmite shows a positive relief and kaolinite a negative relief in the immersion medium used, which can be readily ascertained by the movement of the Becke line. Boehmitic portions in boehmite-gibbsite or boehmite-diaspore intergrowths may easily be distinguished after staining [Figs. 2(*a*) and 2(*b*)]. The transparent minerals commonly found in bauxites may be identified by using the characteristics shown in Table I.

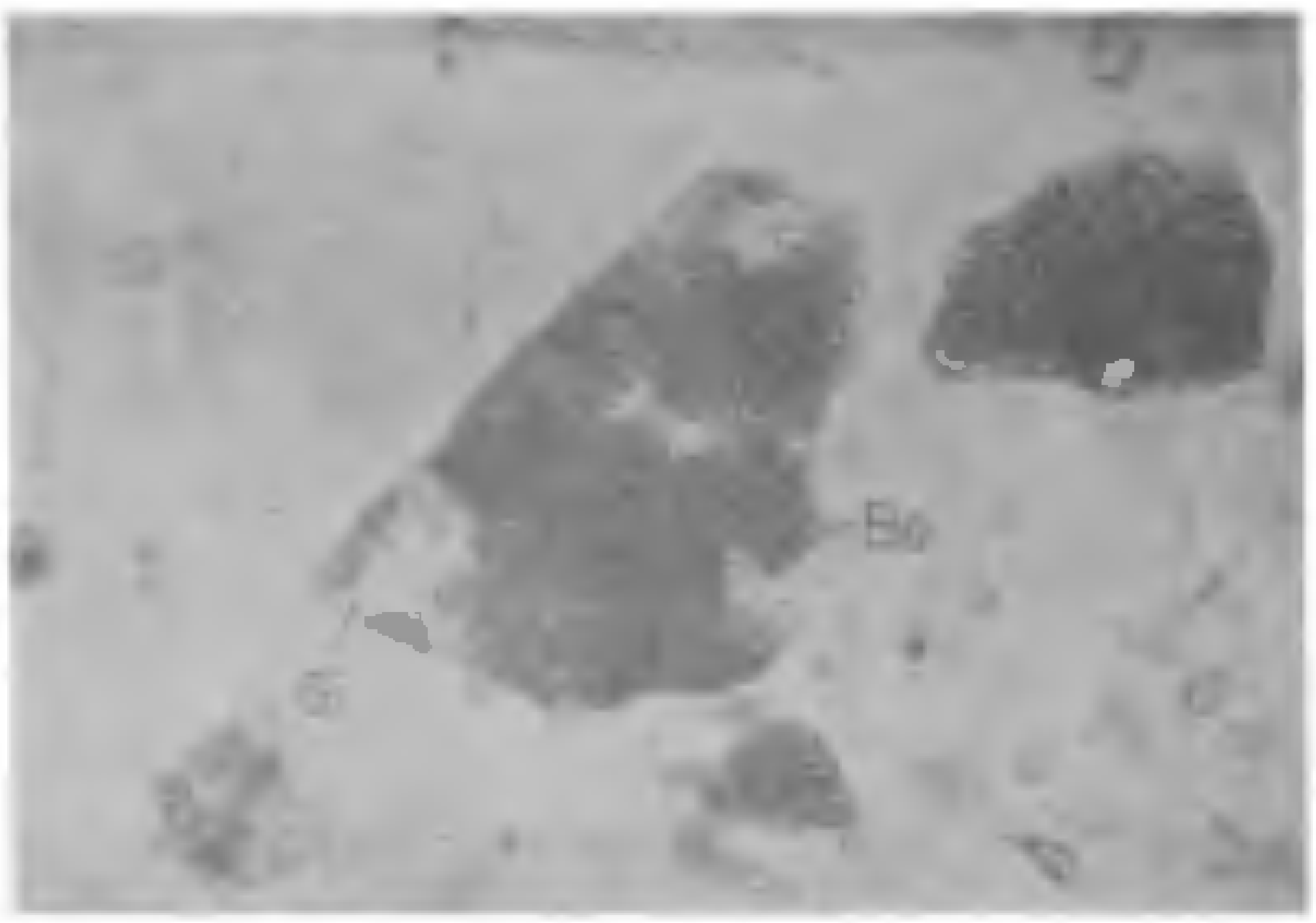

FIG. 2(*a*). Boehmite-gibbsite intergrowth stained with Malachite green, plain light (×40).

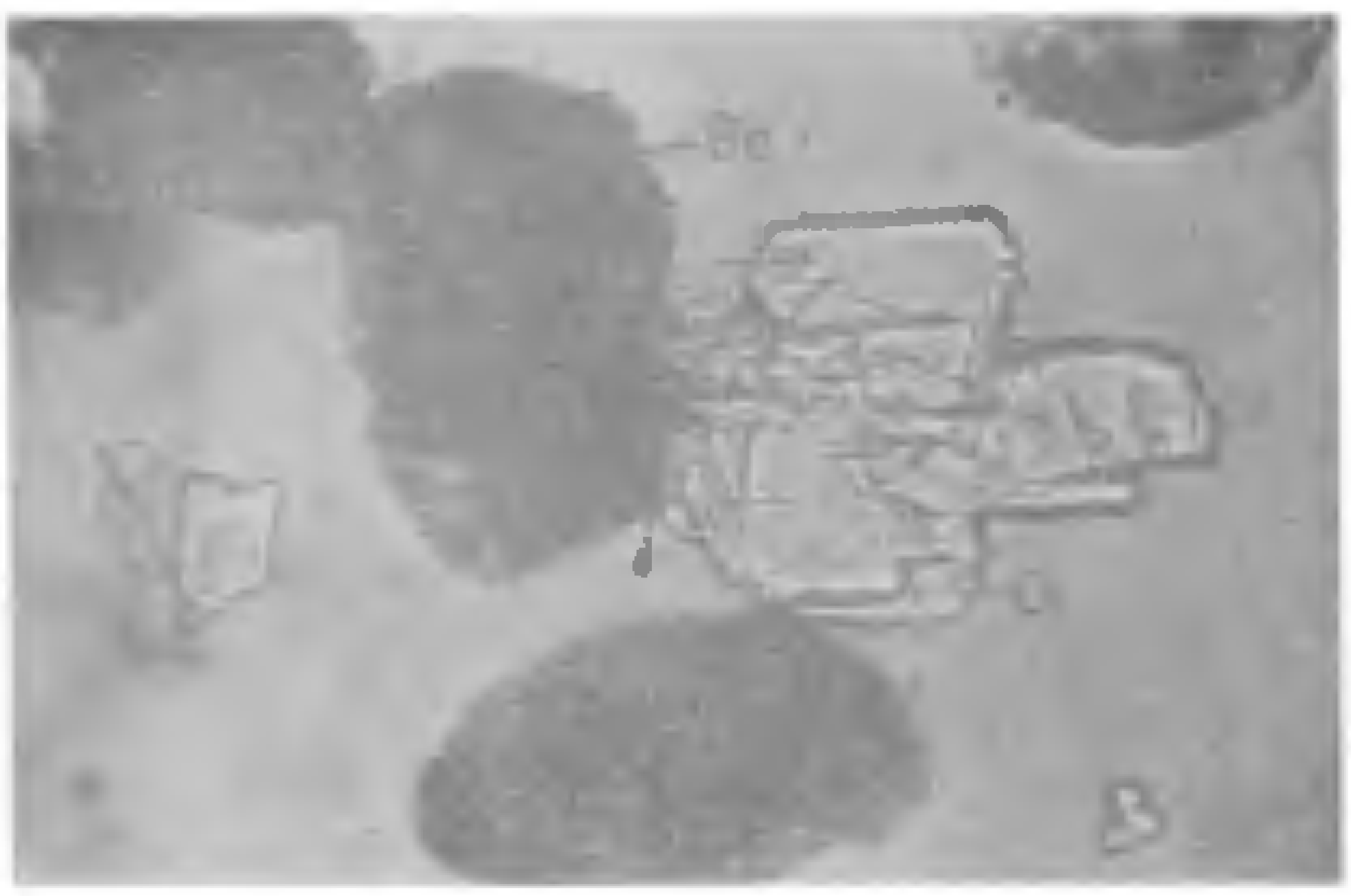

FIG. 2(*b*). Boehmite and diaspore (Di) grains stained with Malachite green, plain light (×40).

The colour test employing *p*-aminophenol may be used to confirm the presence of montmorillonoids[3]. *p*-aminophenol did not give a distinctive colour test in the case of boehmite samples used in this study.

In the presence of kaolinite on staining with safranine-Y boehmite takes on a brownish pink to yellowish brown colour compared to deep pink colour of kaolinite. However, staining with Malachite green produces distinctly different colours in the two minerals as shown in Table I, and hence appears to be best suited for differentiating between boehmite and kaolinite. The identification of kaolinite, gibbsite, boehmite and diaspore used in these tests was confirmed by X-ray diffraction analysis; montmorillonite by the *p*-aminophenol colour test and quartz by optical methods.

The staining technique reported here has the following advantages—(1) It is a rapid and cheap method of identification of the transparent bauxitic minerals, (2) It helps in distinguishing between boehmite and crypto-crystalline gibbsite/diaspore during modal analysis and (3) It is possible to detect the presence of boehmite even when it is present in amounts less than the detection limits[4] of Derivatography (0·3%) and X-ray diffraction (0·5%) by microscopic observation of the stained powdered sample.

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VARIATIONS IN TISSUE GLYCOGEN CONTENT SERUM LACTATE AND GLUCOSE LEVELS DUE TO COPPER INTOXICATION IN THREE FRESHWATER TELEOSTS

COPPER is widely used as an algacide and in the treatment of disease and parasitism in fishes[1]. The latter application sometimes poses the problem of toxicity to the fish treated. In the present investigation the author has studied the effect of copper sulphate (5, 10 and 15 ppm) on glycogen content in liver, muscle, brain and kidney tissues and the lactate and glucose levels in serum of *Labeo rohita* (Ham.), *Ophicephalus punctatus* (Bloch.) and *Clarias batrachus* (Linn.).

Twenty-four live *L. rohita, O. punctatus* and *C. batrachus* (18–20 cm) were acclimatized in the laboratory for 3–4 days. A group of six fishes (*L. rohita, O. punctatus* and *C. bagrachus* each) were placed in a glass aquaria containing 5, 10 and 15 *ppm* of copper

TABLE I

Changes in tissue glycogen content of three teleosts copper sulphate intoxication

	Control	Copper intoxication		
		5 ppm	10 ppm	15 ppm
L. rohita				
Liver	4216·30±196·80	3458·70±250·56	2156·44±156·38	1456·10±32·50
Muscle	3106·45±126·38	2230·56± 95·80	1750·68±128·75	1050·26±65·12
Brain	1075·24± 84·26	820·16± 24·36	596·52± 56·42	324·54±12·38
Kidney	580·32± 25·42	710·40± 15·10	948·92± 36·40	406·12±18·56
O. Punctatus				
Liver	3096·12±140·58	2646·18±190·36	2024·12±27·12	1626·58±58·42
Muscle	2185·09±147·56	1448·36± 89·54	1250·24±18·36	826·28±61·30
Brain	905·64± 49·49	690·12± 28·42	530·70±12·50	415·14±28·19
Kidney	415·16± 18·94	584·28± 38·42	736·62±21·72	253·78±19·56
C. batrachus				
Liver	2645·38±115·55	2158·40±165·38	1720·80±41·50	1428·16±72·24
Muscle	1726·40± 90·58	1238·24± 58·56	1025·58±32·46	710·32±54·61
Brain	720·16± 24·58	560·64± 36·27	452·84±22·12	380·15±38·12
Kidney	306·18± 12·99	458·52± 23·84	622·18±64·10	180·47± 9·50

Values (μgm/gm wet wt. of tissue) are mean ± SE of 5 replicates.

sulphate for a period of 3 hours. Their liver, muscle, brain and kidney tissues were collected quickly after decapitation and blotted on a filter paper. The preparation of tissue samples, processing and expression of glycogen values were described elsewhere[2]. The method of collection of serum and estimation of lactate was described earlier[2-3]. Glucose in serum was estimated by method of Hawk *et al.*[4]. The tissues and serum of untreated fishes for glycogen, lactate and glucose were taken as controls. The experiment was repeated 5 times to subject the data to statistical analysis.

Copper sulphate intoxication has brought about a number of significant variations in the glycogen content of liver, muscle, brain and kidney tissues and lactate and glucose levels in serum of *L. rohita*, *O. punctatus* and *C. batrachus*. Coagulated mucus, accumulated in the gill cavity, hung on the surface of the fishes and clouded the water of glass aquaria. It is evident from Table I, that the effect of copper intoxication on tissue glycogen content varies with the tissue. In liver, muscle and brain of three fishes, the author observed an inverse relationship between the concentration of copper and the fall in glycogen content, whereas in kidney the glycogen level increased upto 10 ppm of copper exposure and at 15 ppm the process of glycogenolysis was maximum. There was a direct relationship between the cncentration of copper and accumulation of lactate and glucose in the serum of *L. rohia*, *O. punctatus* and *C. batrachus* [Table II (*a*) and (*b*)]. The elevation in the levels of serum lactate was 14, 10 and 8 fold to the initial concentration in *L. rohita*, *O. punctatus* and *C. batrachus* respectively. More than two fold rise in serum glucose level was recorded to the initial values in *L. rohita*, *O. punctatus* and *C. batrachus* [Table II (*b*)].

The deposition of mucus on the gills of fishes observed in the present study might have reduced the capacity of blood to carry oxygen to various internal organs[5], which in turn might have influenced the process of tissue acidosis. The accumulation of metabolic products in serum (lactic acid and glucose) might be related with the above process. Whenever the metabolic products start accumulating, it will lead to a change in pH of the blood, thus disturbing the buffering system of organs and this might have favoured the process of glycogenolysis in tissues[3].

It is also known that heavy metal intoxication deaccelerates the cardiac function, lowering the blood capacity to carry oxygen change in pH of blood

TABLE II

Copper sulphate intoxication and accumulation of (a) *lactate and* (b) *glucose in serum of three teleosts*

Name of the fish	Control	Copper intoxication		
		5 ppm	10 ppm	10 ppm
		(a)		
L. rohita	112·26±24·12	350·17±16·90	680·86±36·22	1568·90±41·54
O. punctatus	68·44±10·58	156·56± 8·70	390·74±15·11	680·12±29·14
C. batrachus	45·12± 5·38	118·36±12·42	240·92± 8·34	364·26±16·38
		(b)		
L. rohita	975·24±68·36	1096·44±105·09	1450·12±84·40	2096·44±180·12
O. punctatus	504·62±32·48	590·48± 28·36	820·11±24·18	1024·18± 76·34
C. batrachus	636·42±53·12	724·80± 34·48	915·04±59·91	1290·48± 84·88

Values (μgm/ml of serum) are mean ± SE of 5 replicates.

and the fall in osmolality of blood serum[1,6] may be true in present study and the fall in tissue glycogen content and the rise in serum lactate and glucose levels of *L. rohita*, *O. punctatus* and *C. batrachus* may be related to the above phenomena. The rise in renal glycogen content up to 10 ppm copper exposure may be associated with the increased levels of glucose.

It appears that the copper sulphate intoxication causes severe anaerobic stress resulting in the breakdown of tissue glycogen possibly to meet the energy demands in the muscle. The initial accumulation of glycogen in the kidney may be an attempt by the kidney to conserve glycogen and therefore to restore normalcy. At a later stage however the toxicity is so high creating an imbalance in the glycogen content of kidney also. Further studies on these lines are in progress.

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PIPER NIGRUM L. A NEW HOST OF *XANTHOMONAS BETLICOLA* PATEL *ET AL.*

DURING June–July 1977, a bacterial disease was observed on pepper plants (*Piper nigrum* L.), growing adjacent to betelvine plants severely infected with bacterial leaf spot, at the College of Agriculture, Vellayani, Kerala. Subsequently the disease was also observed to be prevalent in farmer's field at Mannanthala area of Trivandrum District. This is the first report of a bacterial leaf spot on pepper plants.

The disease appeared as minute water-soaked lesions on the leaf-lamina. As the lesions grew older, the centre of the spot became black, surrounded by a yellowish halo. Sometimes infection began from the leaf margins also. Often a dark pigmentation developing at the infection spot, diffused into surrounding parts of the leaf-lamina, eventually causing defoliation (Figs. 1 and 2).

On microscopic examination of the infected peppervine leaves, plenty of bacterial ooze could be detected in the lesions. Isolations from diseased host tissue yielded yellow coloured bacterial colonies. Pathogenicity trials on healthy hosts gave positive results. Morphological and physiological characters of the pathogen such as yellow colony colour, rod shape, negative gram reaction, negative oxidase test, nega-