→ Cl → Br; and the transformation S → SO_2 resulted in a decreased inhibition. *p*-Bromophenylthio-acetohydroxamic acid is the most potent inhibitor of the tested group ($pI_{50} = 4.0$); its inhibiting power is of similar order to that of other catalase inhibitors (cf. Table).

When the inhibitor (hydroxamic acid) was added to the enzyme solution, before the addition of substrate, there resulted an initial considerable inhibition which progressed only slowly by increasing the incubation time (2–10 min). In this respect, the arylthio-acetohydroxamic acids behave similarly to sulphide (BEERS and SIZER[8]). The presence of substrate (added simultaneously with the inhibitor to the enzyme solution) did not protect catalase against inhibition. This suggests that the inhibition of catalase does not involve the attachment of the hydroxamic acid to the substrate-binding group.

Zusammenfassung. Leberkatalase wird durch (Arylthio)-acetohydroxamsäure gehemmt. I_{50} Werte: $4.5 \cdot 10^{-3}$ to $1.0 \cdot 10^{-4} M$. *p*-Bromoderivat ($pI_{50} = 4.0$) zeigt die stärkste Hemmung. Substitution in der Para-Stellung des Benzol Ringes verursacht eine zunehmende Hemmung in der Reihenfolge: CH_3 → OCH_3 → Cl → Br. Es wird vermutet, dass sich der Hemmstoff nicht mit dem aktiven Zentrum des Ferments verbindet.

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Chemical Investigations of *Alangium lamarckii* III. Isolation of Steroids and Terpenoids from the Leaves

Alangium lamarckii Thw. (N.O. Alangiaceae) is a widely-grown small tree, and is known in different parts of India as *ankola, ankota, ankora, dhera, akarkanta* etc. The bark of the plant has been used in Indian indigenous systems of medicine for the treatment of leprosy, syphilis, various skin diseases and dysentery. It has also been used as a diaphoretic, antipyretic, emetic, laxative and anthelmintic. The leaves are used as a poultice to relieve rheumatic pains[1]. In a recent communication[2], the total tertiary alkaloidal fraction, a mixture of at least 5 alkaloids as revealed by paper chromatography, isolated from the leaves was found to be pharmacologically quite active and showed antispasmodic, hypotensive, anticholinesterase and adrenolytic activities. One of the 5 alkaloids was isolated as a new crystalline phenolic alkaloid, Ankorine[3], $C_{19}H_{29}O_4N$, m.p. 174–176°, which was found to possess hypotensive action of a prolonged duration[4]. Besides, choline chloride[5] was also isolated from the water-soluble quaternary basic fraction, which was pharmacologically found to be cholinergic in nature, after separation of the chloroform-soluble tertiary bases. Preliminary pharmacological studies[6] indicated that *Alangium lamarckii* leaves significantly increased the inflammatory reaction during the first 5 days and then significantly reduced the foot volume from the eleventh day onwards in formalin-induced arthritis in albino rats. Ascorbic acid content of the adrenal gland was found to be significantly raised. These findings suggested that some steroidal principles might be responsible for the anti-inflammatory property of the leaves. This is further corroborated by the fact that the leaves are used in the indigenous systems of medicine in relieving rheumatic pains when applied in the form of a poultice. Attempts were therefore made to isolate the steroids, terpenoids and other principles present in the leaves. For this purpose the petroleum ether extract of the leaves was saponified under different conditions either with strong alkali or acid, and then distilled in steam to remove volatile oils. The non-volatile, non-saponifiable fraction was obtained by extraction with ether. The product was then chromatographed over Brockmann aluminium oxide for chromatography, eluting with petroleum ether, benzene and chloroform in different proportions. Under different conditions of hydrolysis, the following 4 compounds were isolated after repeated chromatography and crystallization.

(1) *Compound A*, needles, m.p. 152–154°, $[\alpha]_D^{20°}$ – 33.8°, – 24.72° ($CHCl_3$). Anal.[7] – Found: C, 84.78, 84.83, 83.96, 83.92; H, 11.42, 11.55, 11.34, 11.44; *M* weight, 310, 302, 365 (Rast); CCH_3, 7.05, 9.40. Episterol[8], $C_{28}H_{46}O$, m.p. 151°, $[\alpha]_D$ – 5°, requires C, 84.35; H, 11.63; *M* weight, 398.65. Campesterol[8], $C_{28}H_{48}O$, m.p. 158°, $[\alpha]_D$ – 33° requires C, 83.93; H, 12.08; *M* weight, 400.66. Compound A has IR-absorption peaks at 2.72, 2.85, 3.35, 6.1, 6.85, 7.28, 7.9, 8.82, 9.2, 9.6, 9.78, 10.25, 10.42, 11.2, 11.88 μ, and UV λ_{max} at 204 nm. With acetic anhydride and pyridine compound A forms an *acetate*, flakes, m.p. 137–138°, $[\alpha]_D^{20°}$ – 38.68°, – 40.45° ($CHCl_3$). Anal. – Found: C, 82.44, 82.27, 82.49, 82.48; H, 10.97, 11.12, 10.86, 11.18; *M* weight, 396, 392, 364 (Rast); CH_3CO, 8.80, 3.84. Episterol acetate, $C_{30}H_{48}O_2$, requires C, 81.76; H, 10.98. Campesterol acetate, $C_{30}H_{50}O_2$, requires C, 81.39; H, 11.38. With benzoyl chloride and pyridine compound A forms a *benzoate*, m.p. 138–140°, $[\alpha]_D^{20°}$ – 14.8°, – 14.24° ($CHCl_3$). Anal. – Found: C, 83.98, 83.81, 83.93, 84.23; H, 9.61, 9.86, 10.28, 10.45; *M* weight, 258, 250, 405 (Rast); C_6H_5CO, 7.15. Episterol benzoate, $C_{35}H_{50}O_2$, requires C, 83.61; H, 10.02. Campesterol benzoate, $C_{35}H_{52}O_2$, requires C, 83.28; H, 10.38. Iodometric titration showed the consumption of 2 moles of perbenzoic acid by compound A, indicating the presence of 2 double bonds.

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Colour reaction	Compound A	Compound B	Compound C	Compound D
1. Liebermann-Burchard	Red → violet → blue → green	Brown → violet → blue → green	Interface pink, $CHCl_3$ layer light brown on standing	Brown → violet → blue (very slowly)
2. Salkowski	Acid layer orange to brown, $CHCl_3$ layer light violet → pink → brown	Interface brown, $CHCl_3$ layer brown → violet on standing	No colour, on standing interface light brown	No colour
3. Rosenheim	No colour	No colour	No colour	No colour
4. Tschugaeff	Red	Blue → red	No colour	No colour
5. Tortelli-Jaffé	No colour	Green colour at interface	No colour	No colour
6. Zimmermann	Negative	Negative	Positive (magenta colour)	Negative

(2) *Compound B*, needles, m.p. 191–193°, $[\alpha]_D$ nil. Anal. – Found: C, 83.37, 83.67; H, 11.78, 11.72; *M* weight, 500, 487 (Rast). It has IR-absorption peaks at 2.7, 2.85, 3.32, 5.65, 5.85 (w), 6.09, 6.82, 7.25, 9.35, 9.6, 9.9, 10.1, 10.25, 11.2 μ, and UV λ_{max} at 201 nm.

(3) *Compound C*, needles, m.p. 252–255°, $[\alpha]_D$ – 21.18° ($CHCl_3$). Anal. – Found: C, 85.34, 85.12; H, 11.99, 11.98; M weight, no depression (Rast); CCH_3, 7.60. It has IR-absorption peaks at 2.8, 3.32, *5.82 (s)*, 6.88, 7.2, 7.35, 7.6, 7.7, 7.8, 8.5, 8.8, 9.0, 9.3, 9.95, 10.15, 10.4, 10.9 μ, and UV λ_{max} at 199 nm.

(4) *Compound D*, m.p. 262–265°. It has no IR-carbonyl absorption.

The 4 compounds give the following colour reactions. Both compound A and B appear to be sterols. A positive Tortelli-Jaffe test indicates that the compound B might contain a double bond between 2 ditertiary carbon atoms (C_8–C_9) at a bridgehead, as in Fecosterol and Ascosterol[8]. From elemental analysis, positive Zimmermann colour reaction and strong carbonyl absorption at 5.82 μ in the IR-spectrum, compound C appears to be a triterpenoid containing a carbonyl function. Compound D also might belong to the triterpenoid series. Further chemical characterization of the 4 compounds and their pharmacological studies are in progress. It may be mentioned that a sterol, Alangol, $C_{42}H_{84}O_7$, m.p. 296°, was isolated by BHARGAVA and DUTT[9] from the seeds, and β-sitosterol, stigmasterol, and a compound, m.p. 128–129°, were isolated by ROY and PAKRASHI[10] from the stem-bark.

Zusammenfassung. Es wird über die Isolierung und Charakterisierung von Inhaltsstoffen, vermutlich Steroide, aus einer indischen Pflanzenart, *Alangium lamarckii*, berichtet. Medizinische Zubereitungen aus dieser Pflanze werden für verschiedene Indikationen, z. B. auch antiinflammatorische, verwendet.

B. DASGUPTA and (Mrs.) SHEELA SHARMA

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Glucose-6-Phosphatase Activity in Experimental Tuberculosis and Following Isoniazid Treatment

Previous studies from this laboratory have shown that experimental infection in guinea-pigs with *Mycobacterium tuberculosis*, strain H 37 Rv, lowers the succinic dehydrogenase[1,2], isocitric dehydrogenase[3] and NADH dehydrogenase[4] and elevates the nicotinamide adenine dinucleotidase (NADase)[5] activities in the kidney. Earlier it was reported that the glycogen content was significantly depleted in the tissues of tuberculous animals[6]. WEBER and CANTERO[7] have shown that the depletion of liver glycogen causes an adaptive increase in hepatic glucose-6-phosphatase activity. This prompted us to investigate the levels of glucose-6-phosphatase in guinea-pigs during the experimental infection with tuberculosis and following isoniazid therapy.

Male albino guinea-pigs, 250–300 g, were infected by injecting intraperitoneally 0.1 ml of a 7-day-old culture of *M. tuberculosis*, var. H 37 Rv, grown in DUBOS media[8].

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