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.

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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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A group of 6 infected guinea-pigs was given isoniazid solution (0.005%) in lieu of drinking water from the 8th to the 38th day postinfection. Tuberculous animals were decapitated between the 3rd and the 4th week postinfection, whereas isoniazid-treated animals were sacrificed after 30 days of drug therapy. The kidneys were promptly removed and homogenates were prepared as described earlier[4]. The tissue concentration was adjusted to 200 mg/ml of homogenate with the aid of biuret reaction[9]. The glucose-6-phosphatase activity was determined by the phosphate release method of SWANSON[10] by adding 0.1 ml of kidney homogenates in the incubation mixture. Phosphorus was determined by the method of TAUSSKY and SHORR[11].

In the present studies, the assay of glucose-6-phosphatase was performed in the kidney homogenate since this organ did not show any gross tuberculous lesions. The results obtained on glucose-6-phosphatase activity are reported in the Table. A small but significant rise ($p < 0.025$) in the enzyme activity of kidney homogenates of tuberculous animals as compared to those of control animals was observed. Administration of isoniazid for 30 days did not significantly lower the elevated glucose-6-phosphatase activity. In the previous findings we have reported that infection with *M. tuberculosis* caused drastic effects on the metabolism of the host tissues[1-5]. Some enzymes, particularly those dependent on NAD and NADP for their activities, are more easily deranged during the progressive infection[3,4,12]. The increased glucose-6-phosphatase activity in the kidney homogenate of tuberculous animals, as reported here, may be due to enzyme adaptation since several studies have shown that a decrease in liver glycogen causes a marked adaptive increase in glucose-6-phosphatase activity[7,13,14]. Unlike its influence on various enzymes reported previously[2-4], the isoniazid therapy on tuberculous animals for 30 days was ineffective in lowering the glucose-6-phosphatase activity indicating that the elevated activity was secondary to tuberculous infection. This supports the view of adaptive increase in enzyme level as a result of decreased glycogen content.

	No. of animals	μM of phosphate liberated/100 mg of tissue in 15 min (mean ± S.E.)	% increase
Control	10	6.08 ± 0.15	–
Tuberculous	6	8.30 ± 0.18	36.5
Tuberculous animals given isoniazid	6	7.66 ± 0.12	26.0

Zusammenfassung. Bei den tuberkulösen Meerschweinchen wurde in der Niere eine erhöhte Aktivität der Glukose-6-Phosphatase beobachtet. Isoniazid hat die erhöhte Aktivität des Enzyms nicht erniedrigt, wenn es tuberkulösen Tieren vom 8. bis zum 38. Tage nach der Infektion oral verabreicht wurde. Es wird angenommen, dass die erhöhte Aktivität Ausdruck der Adaptionsfähigkeit von Glukose-6-Phosphatase ist.

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Strukturspezifischer Abbau von Polypeptid-Metall-Komplexen III[1]. Abbau des Ni^{2+}-Angiotensin II-Komplexes durch H_2O_2[2]

Der Abbau des Cu^{2+}-Komplexes des cyclischen Dekapeptides Polymyxin B durch H_2O_2 wird, wie wir gezeigt haben[1], vom Metallion gesteuert (vgl. auch[3]), wobei diese Reaktionen – dasselbe gilt für den entsprechenden Ni^{2+}-Komplex[4] – über ternäre Komplexe verlaufen[5].

Um zu prüfen, ob eine derartige Degradation nicht nur bei einem cyclischen Peptid – dessen Komplex strukturmässig eine Sonderstellung einnimmt –, sondern auch bei linearen Peptiden auftritt, untersuchten wir den Abbau von Val^5-Angiotensin II-Asp^1-β-amid[6]. Dieses lineare Oktapeptid besitzt folgende Aminosäuresequenz[7] (vgl. auch [8]):

H · Asp(NH_2)-Arg-Val-Tyr-Val-His-Pro-Phe · OH

Wir untersuchten – ähnlich wie beim Polymyxin B[5,9] – auch beim Angiotensin II zuerst die katalytische Aktivi-

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