of TAN was prepared from 1-(2-thiazolylazo)-2-naphthol (Fluka) and also aqueous solution of Triton X-100 (Fluka). Buffer solution (pH 6.6) was prepared from 0.5 M solutions of hexamine and hydrochloric acid (A.R.).

The optimum pH for the reaction between beryllium(II) and TAN is 6.3 to 6.9 and the reaction was studied at pH 6.6, maintained by hexamine-HCl buffer. The sparingly soluble complex dissolves in polar organic solvents requiring at least 80% (v/v) of the solvent, and maximum absorbance is noticed in acetone. The complex is however found to be water soluble in the presence of non-ionic surfactant, Triton X-100. Although the complex has equal sensitivity in both media, surfactant medium has been preferred for the determination to avoid the use of high concentration of organic solvent. At least 2.5 ml of 10% Triton X-100 solution is required for solubilization and constant maximum absorbance is obtained in the presence of 2.5 to 4.5 ml of surfactant and 4 to 7 ml of 0.1% methanolic solution of TAN. The complex exhibits maximum absorbance at 547 nm in 80% (v/v) acetone and 555 nm in surfactant medium. The instantaneously formed complex is stable for 24 hr in acetone but starts precipitating after 2 hr in surfactant medium.

The 1 : 2 complex obeys Beer's law over the concentration range 0.02 to 0.34 ppm of beryllium, has molar absorptivity 2.25×10^4 l mol^{-1} cm^{-1}, Sandell sensitivity 0.4 ng cm^{-2} and coefficient of variation ± 0.76%. Tolerance limits of various ions in determination of 4 μg of beryllium(II) in 25 ml are (in μg): Cl^-, Br^-, I^-, urea, thiourea 20000; $S_2O_3^{2-}$ 10000; ascorbate 7500; SO_4^{2-} 5000; $NH_2OH.HCl$ 4000; Mo(VI), W(VI), Pt(IV) 2000; phthalate, Au(III) 1000; succinate, Al(III) 500; $N_2H_6SO_4$ 400; CN^- 300; V(V), Th(IV), Cr(III), Ag(I) 200; oxalate 150; Ga(III) 100; In(III), Hg(II) 40; Sb(III), Bi(III), Sn(II) 20; PO_4^{3-}, malonate, Pb(II) 10; F^-, U(VI), Tl(III), Pd(II) 5. Alkali and alkaline earth metal ions, CO_3^{2-}, SCN^- did not interfere even in large amounts. EDTA and citrate gave negative interference, whereas Fe(II, III), Mn(II), Co(II), Ni(II), Cu(II) and Zn(II) gave positive interference.

An aliquot of the solution containing ≤8.5 μg of beryllium(II) is taken, added 5 ml of 0.5 M hexamine-HCl buffer, 4 ml of 10% Triton X-100, 6 ml of 0.1% methanolic TAN, the contents diluted to 25 ml with distilled water and absorbance measured at 555 nm against a reagent blank prepared under identical conditions.

Beryl (0.5 g) was fused with sodium carbonate and brought into solution[4]. An aliquot of this solution was taken, beryllium separated by reversed phase extraction chromatography[5] and determined as given earlier. Duplicate analysis gave the beryllium content as 4.68 and 4.69% against the theoretical amount of 4.71%.

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1. Anderson, R. G. and Nickless, G., *Analyst*, 1967, **92,** 207.
2. Havard R. Hovind, *Analyst*, 1975, **100,** 769.
3. Vogel, A. I., *A textbook of quantitative inorganic analysis*, Longman, London, 1975, p. 518.
4. Shankar Das, M. and Athavale, V. T., *Anal. Chim. Acta*, 1955, **12,** 414.
5. Chhaya Sharma and Khopkar, S. M., *Anal. Chim. Acta*, 1985, **167,** 403.

EFFECTS OF CODEINE ON RAT INTESTINE

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CODEINE, first reported by Robiquet 1832 (see ref. 1), is an alkaloid of opium, obtained from the milky exudate of the incised unripe seed capsules of the poppy plant, *Papaver somniferum*. This belongs to the opiate group such as morphine and codeine which are used principally as analgesics, to induce sleep in the presence of pain, to check diarrhoea and to suppress cough. It has adverse effects like euphoria, drowsiness, decreased gastrointestinal motility, reduced propulsive movement and increased constipation[2].

The effects and mechanism of action of opiates on the gastrointestinal nonpropulsion have been reported earlier[3–6]. The constipating effects of opiates have been investigated in several mammalian species[7–9] In rats a delay in transit time in small intestine induced by opiate has been associated with decreased contractile activity[10, 11].

In the opiate group, the effects of morphine have been extensively worked out. Since codeine has also been found to be present in the natural product of opium, the effect of this substance on rat intestine has been taken up for the present investigation.

Young albino rats weighing about 100–120 g were obtained from King Institute, Madras. Codeine phosphate was obtained from Government Opium and Alkaloids Works, Ghazipur, India. The ^{14}C glucose and ^{14}C glycine were obtained from Bhabha Atomic Research Centre, Bombay. The chemicals were obtained from Sigma Chemicals Co., New York.

The test animals received 50 mg/kg body weight of codeine phosphate daily by oral administration through stomach tube. The rats from two groups were employed to estimate the *in vivo* obsorption of ^{14}C glucose and ^{14}C glycine at 3rd and 5th day and for enzyme assays.

Adenosine triphosphatase was assayed by the method of Hokin *et al*[12], and alkaline phosphatase by the method of Moog[13], as modified by King[14]. *In vivo* absorption of ^{14}C glucose and ^{14}C glycine was measured by perfusion technique according to the method of Yonuosza *et al*[15].

Our results (table 1) show that codeine phosphate reduces the intestinal adenosine triphosphatase (ATPase) and alkaline phosphatase activities as compared with the control. The *in vivo* intestinal absorption (figures 1 and 2) of ^{14}C glucose and ^{14}C glycine had decreased when compared with the control.

The intestinal absorption of nutrients are carrier-mediated and energy-dependent[16]. Involvement of alkaline phosphatase in the intestinal transport has been reported by Moog[17], while ATPase is known to take part in the "sodium and potassium pump" and amino acid transport[18]. Codeine-treated rats exhibited reduction in the activities of ATPase and alkaline phosphatase, which is reflected in the decreased absorption of glucose and glycine. The reduction of glucose and glycine absorption is dose-dependent as the 5th day animal exhibited greater reduction than the 3rd day animal. Concomitant reduction in the activities of alkaline phosphatase and ATPase has also been noticed.

It has been reported that administration of opiates consistently delays the passage of intraluminal contents through the gastrointestinal tract[3,8]. Morphine

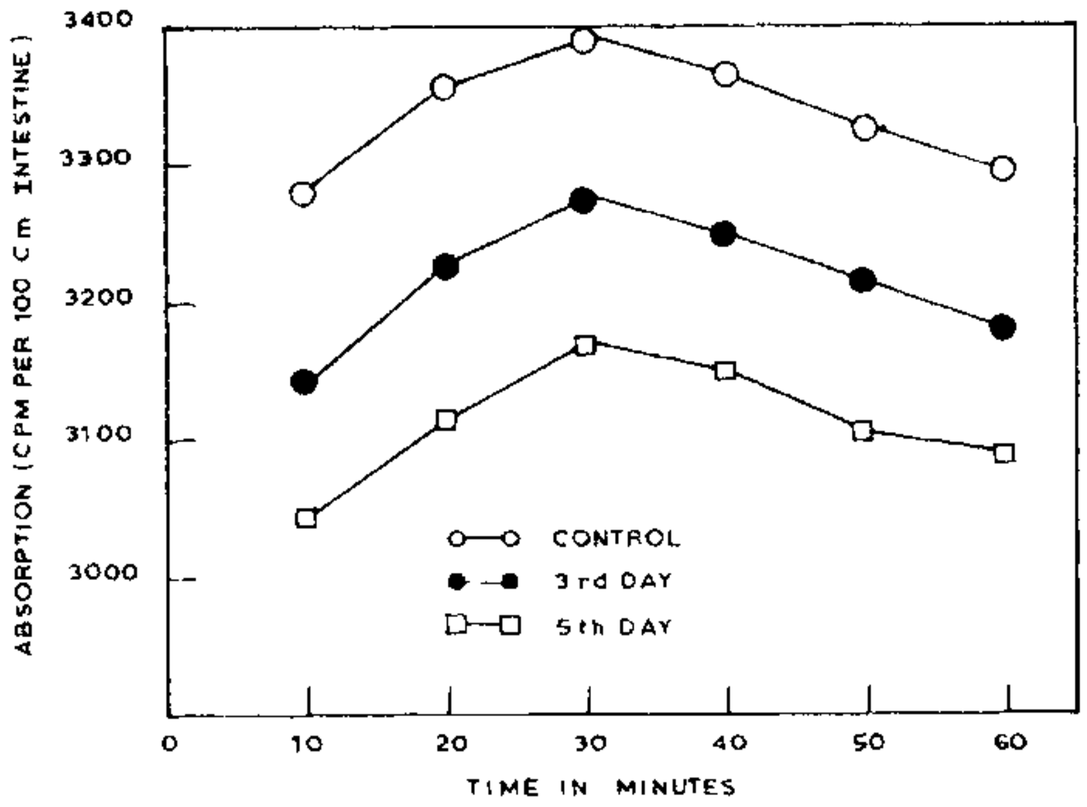

Figure 1. *In vivo* absorption of ^{14}C glucose by the intestine of control and codeine-treated rats expressed as counts per minute 100 cm intestine at various time intervals.

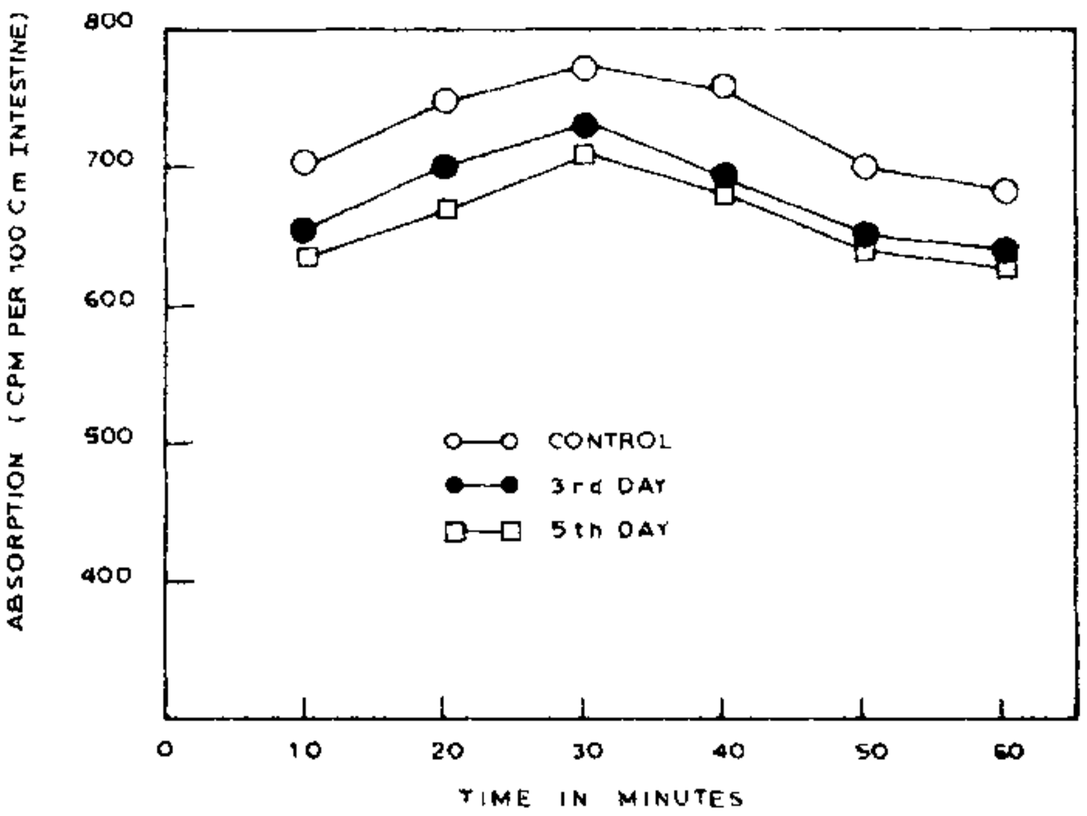

Figure 2. *In vivo* absorption of ^{14}C glycine by the intestine of control and codeine-treated rats expressed as counts per minute 100 cm at various time intervals.

Table 1 *Activities of ATPase and alkaline phosphatase of control and codeine treated rat intestine*

Enzymes	Control	Experimental 3rd day	Experimental 5th day
ATPase	4.03 ± 0.19	3.75 ± 0.18	3.41 ± 0.21
Alkaline phosphatase	1.92 ± 0.13	1.62 ± 0.15	1.47 ± 0.17

Enzyme activities are expressed as μmol of product liberated per mg protein under incubation conditions (mean ± SD).

induces an increase in contraction of small and large intestines[19], and inhibits the transit of material through gastrointestinal tract[9,10,20]. Our observations on the enzyme activities and absorption of ^{14}C glucose and ^{14}C glycine in codine-treated rat intestine also add further evidence for such adverse effects of codeine. The observed reduction in absorption could be due to the cumulative effects of codeine on the enzyme systems and on the intestinal motility.

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1. Ewart A. Swinyard and Stewart C. Harver., In: *Remington's pharmaceutical sciences*, (ed.) Melvin R. Gibson, Mack Publishing Co., Easton, Pennsylvania, 1970, p. 1124.
2. Jerome H. Jaffe and William R. Martin, *The pharmacological basis of therapeutics*, (eds) A. G. Gilman, L. S. Goodman and A. Gilman, Macmillan Publishing Co., New York, 1980, p. 494.
3. Plant and Miller, *J. Pharmacol. Exp. Ther.*, 1926, **27,** 361.
4. Bass, P., Kennedy, J. A., Wiley, J. N., Villareal, J. and Butler, D. E., *J. Pharmacol. Exp. Ther.*, 1973, **186,** 183.
5. Vaughan Williams, *Pharmacol. Rev.*, 1954, **6,** 159.
6. Pruitt, D. B., Grubb, M. N., Jacquette, O. E. and Burks, T. F., *Eur. J. Pharmacol.*, 1974, **26,** 298.
7. Jeffe, J. H. and Martin, W. R., *The pharmacological basis of therapeutics*, (eds) L. S. Goodman and A. Gilman, Macmillan Publishing Co., New York, 1975, p. 245.
8. Weisbrodt, N. W., Sussman, S. N., Stiwari, J. J. and Burks, T. F., *J. Pharmacol. Exp. Ther.*, 1980, **214,** 336.
9. Galligan, J. J. and Burks, J. F., *J. Pharmacol. Exp. Ther.*, 1983, **226,** 356.
10. Fioramonti, J., Fargeas, M. S. and Bueno., *Arch. Int. Pharmacodyn.*, 1984, **270,** 141.
11. Porreca, F., Filla, A. and Burks, T. F., *Eur. J. Pharmacol.*, 1983, **86,** 135.
12. Hokin, L. E., Dhal, J. L., Deukpree, J. D., Dixon, J. F., Hackney, J. F. and Perdue, J. E., *J. Biol. Chem.*, 1973, **248,** 2593.
13. Moog, F., *J. Cell. Comp. Physiol.*, 1946, **28,** 197.
14. King, F., In: *Practical clinical enzymology*, D. Van Nostrand Co., London, 1965, p. 106.
15. Younosza, M. K. and Schedl, H. E., *Proc. Soc. Exp. Biol. Med.*, 1970, **134,** 564.
16. Curren, P. F., Schultz, S. G., Chez, R. and Fuisz, R. F., *J. Gen. Physiol.*, 1967, **50,** 1261.
17. Moog, F., *J. Exp. Zool.*, 1951, **118,** 187.
18. Rosenberg, L. E. and Scriver, C. R., In: *Duncan's diseases of metabolism*, (eds) P. K. Bondy and L. E. Rosenberg, W. B. Saunders, Tokyo, Japan, 1974, p. 645, 7th edn.
19. Bueno, I. and Fioramonti, J., *Eur. J. Pharmacol.*, 1982, **82,** 147.
20. Burks, T. F., Gallingan, J. J., Hirnino, L. D. and Porrela, F., *Gastroenterol. Clin. Biol.*, 1983, **7,** 729.

TITANIUM-BEARING GARNETS FROM ALKALINE ROCKS OF CARBONATITE COMPLEX OF TIRUPPATTUR, TAMIL NADU

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Six types of Ti-bearing garnetiferous alkaline rocks occur as intrusive bodies, plugs, veins and dykes amidst riebeckite syenites within the carbonatite complex of Tiruppattur (N 12°15′–12°30′ and E 78°25′–78°35′)[1,2] along the southern margin of a structural basin[3] marked by pyroxenites and granitic gneisses. The garnets are black in colour and their composition approaches that of melanite which normally[4] possesses 1–5% of TiO_2. The mineral proportion (table 1) within a single intrusive body varies widely. In the outcrop, the syenite containing the fine-grained riebeckite ($2V_\gamma = 85°$, $(\gamma-\alpha) = 0.013$, $\alpha:(001) = 8°$, N_γ 1.678; X = blue; $Y = Z$ = yellowish green) grades to a coarse-grained melanite-orthoclasite which includes high temperature orthoclase[5,6] with $2V_\alpha = 81°$, $\alpha:(001) = 15°$, $(\gamma-\beta) = 0.002$. The latter grades into a very coarse-grained melanite-aegirine-wollastonite orthoclasite and then to a monomineralic wollastonite rock. The fine-grained melanite-microcline containing high temperature iron rich microcline[6] $2V_\alpha = 39°$, $\alpha:(001) = 19°$ is an ultrapotassic syenite ($K_2O - 16.5\%$) which is in sharp contact with coarse-grained melanite-orthoclasite and wollastonite rock. Syenite porphyries and melanite-