reported in figure 1. For this experiment, the Sephadex was swelled and eluted with a solution of 5 mM tris-HCl buffer (pH 8.0) containing 0.2 mM UTP and 2 mM $MgCl_2$, because Mg^{++} and particularly UTP stabilize the unstable form of nucleoside phosphotransferase[6]. The figure shows 2 peaks of activity: the first corresponds, as previously demonstrated[6], to the unstable nucleoside phosphotransferase and it prefers UMP as phosphate donor, while the second is represented by a stable form which employes preferentially AMP as substrate.

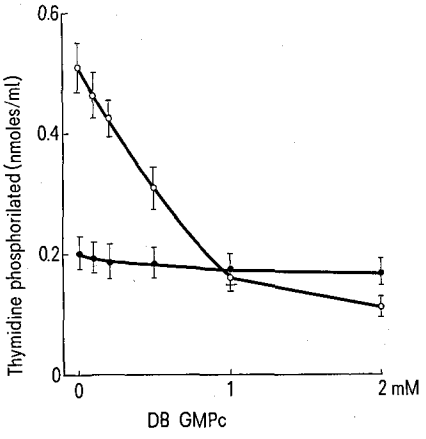

Fig. 2. Effects of DBcGMP on the 2 forms of nucleoside phosphotransferase of chick embryo retina. Peak I (tube numbers 11–15) and peak II (tube numbers 19–23) were collected separately and 200 µl of each peak were utilized for the incubation sample. The activity was measured by using as phosphate donor UMP (○—○) for peak I and AMP (●—●) for peak II. Data are the means ± SE of 6 separate experiments.

As shown in figure 2, DBcGMP markedly inhibits the nucleoside phosphotransferase of peak I, while any significative effect was not observed for the activity of peak II. Previously[6] we have hypothesized that the nucleoside phosphotransferase is present in the chick embryo retina at least in 2 different forms, which could be an expression of the same enzyme at different aggregation states.

It is possible that DBcGMP facilitates the conversion of the form with higher m.wt into a disaggregated state. This state could be represented by the stable nucleoside phosphotransferase, an enzymatic activity which is able to utilize as phosphate donors also the adenine nucleotides. These considerations could explain why the DBcGMP causes an increment of the thymidine phosphorylating rate when the reaction is measured, by using AMP as phosphate donor, in the 105,000 g supernatant. Furthermore, because it seems that the nucleoside phosphotransferase takes part in the control of the endogenous pools of nucleosides and nucleotides, the effects of DBcGMP on this activity could indicate the participation of this compound in the regulation of nucleotide metabolism.

Absorption and biotransformation of L(+)-tartaric acid in rats

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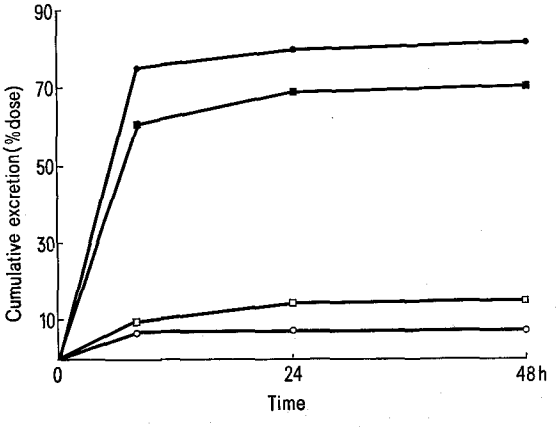
Summary. Oral or parenteral doses of monosodium ^{14}C-L(+)-tartrate (400 mg/kg) are rapidly excreted by rats and a proportion completely metabolized to CO_2. The oral dose was well-absorbed.

Tartaric acid and its salts are used in medicine and in the food industry. In humans, the acid is thought to be poorly absorbed[1] and when given orally, to be metabolized by the gut flora[2,3], since it is readily metabolized by microorganisms such as Pseudomonas putida[4] and Penicillium charlesii[5], which convert it to glycerate and CO_2. Studies in dogs and rabbits have shown that oral doses of tartaric acid were excreted in the urine as unchanged compound, the proportion of which decreased with increasing doses[6]. Much of the tartaric acid used is obtained as a byproduct of wine manufacture and is therefore the naturally-occurring L(+) form[7]. Thus the absorption and biotransformation of tartaric acid has been evaluated using the ^{14}C-L(+) form.



Cumulative excretion of radioactivity in the urine (■, ●) and expired air (□, ○) of rats dosed orally or i.v. respectively with monosodium ^{14}C-L(+)-tartrate (400 mg/kg).

Materials. (1,4-^{14}C)-DL-Tartaric acid of specific activity 2–10 mCi/mmoles was obtained from The Radiochemical Centre, Amersham, England, and was resolved into the L(+)-isomer[8]. The resulting monosodium ^{14}C-L(+)-

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tartrate was shown by paper chromatography in a solvent system of butan-1-ol:acetic acid:water (12:3:5, v/v) to be at least 99% radiochemically pure. Non-radioactive monosodium L(+)-tartrate was provided by Fides, Union Fiduciaire, Switzerland.

Methods. Adult CFY rats (b.wt 200–250 g), a strain of Sprague-Dawley origin were obtained from Anglia Laboratory Animals, Huntingdon, England, and were allowed a pellet diet and water ad libitum. The rats were dosed by oral intubation or by injection into a tail vein with monosodium ^{14}C-L(+)-tartrate at a dose level of 400 mg/kg in aqueous solution. The rats were kept singly in glass metabolism cages, which enabled urine, faeces and expired air to be separately collected, the urine into receivers cooled in solid CO_2, and the expired air ($^{14}CO_2$) into traps containing ethanolamine: 2-ethoxyethanol (1:4, v/v). The rats were sacrificed after 2 days. Radioactivity was measured using procedures previously described[9].

Results. An oral dose of monosodium ^{14}C-L(+)-tartrate was rapidly absorbed and excreted by rats. Excretion of radioactivity in the urine was almost completed within 12 h and in the expired air within 24 h (figure). At 48 h after the oral dose to rats (3♂ + 3♀), 70.1 ± 4.1%, 13.6 ± 7.3% and 15.6 ± 2.7% had been excreted in the urine, faeces and expired air respectively (± SD). After the i.v. dose, 81.8 ± 4.9%, 0.9 ± 1.1% and 7.5 ± 0.5% had been excreted by these routes respectively (± SD). Rates of excretion of radioactivity by male and female animals were similar.

Discussion. The urinary excretion data obtained in these studies are in agreement with an earlier investigation which reported that rats excreted in the urine a mean of 68% of an oral dose of 400 mg/kg of tartrate given as Rochelle salt[6]. However, the earlier work[2] suggested that tartaric acid was only metabolized by the gut flora and not by the tissues. The results obtained in these studies contradict this view since significant amounts of $^{14}CO_2$ were excreted after parenteral administration of ^{14}C-L(+)-tartrate showing that systemic metabolism of tartaric acid occurred. Comparison of results obtained after oral or i.v. doses indicates that an oral dose of L(+)-tartrate was extensively absorbed and that a part was completely metabolized to $^{14}CO_2$ after oral or parenteral administration.

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On a relation of low phosphoglucomutase activity to starch accumulation in spiked sandal

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Summary. A considerable decline in the activity of phosphoglucomutase appeared to be a cause for the starch accumulation in the leaves of sandal affected by spike disease.

In the sandal (Santalum album L.) affected by spike disease, mycoplasmal in nature, the leaves show stunted growth, chlorosis and accumulation[1] of large amounts of starch and sugars, and there is necrosis of phloem elements in the diseased state. The sugar accumulation in the chlorotic spiked leaves, presumably occurring as a result of impaired translocation due to the necrosis of phloem elements, naturally leads to increased starch formation to prevent abnormal rise in the osmotic pressure of the tissue. However, the enzymes related to starch breakdown also determine starch-balance in the tissue. Examination of the diastatic activity of the diseased sandal leaves, showed it, contrary to expectation, to be at a high level[2], thus apparently showing no correlation to the high starch content therein. It is now known that in the plant the breakdown of starch in tissues other than storage tissues and germinating seeds[3–5] is largely brought about by phosphorylase[6–8]. Glucose-1-phosphate (G-1-P) formed during the phosphorolysis, is converted to G-6-P by phosphoglucomutase for entry into glycolysis. Therefore, these 2 enzymes, of relevance to starch balance in the tissue, were studied in the healthy and spiked sandal leaves to examine their relation to the starch accumulation in the diseased state.

Material and methods. Samples of young and mature leaves from healthy and spiked sandal trees were taken during July and September respectively, selecting 6 trees in each case. As the spiked trees remain vegetative throughout, the healthy leaf samples also were taken from trees in vegetative stage for proper comparison.

Phosphorylase and phosphoglucomutase activities and starch content in healthy and spiked sandal leaves

	Healthy Leaves		Spiked Leaves	
	Young	Mature	Young	Mature
Phosphorylase activity* (µg Pi liberated/100 mg tissue/30 min)	4.15 (0.36)	5.20 (0.49)	18.30 (0.56)	18.70 (0.61)
Phosphoglucomutase activity* (µg Pi converted/150 mg tissue/30 min)	51.7 (2.7)	78.0 (4.4)	45.4 (3.5)	10.3 (1.7)
Starch content* (mg/100 g dry leaf)	105.7 (11.8)	73.3 (12.1)	179.2 (16.5)	461.0 (48.5)

*Average of 6 replications. Figures in parenthesis represent SD.

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