The metabolism of tartrate in man and the rat

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Summary

1. Sodium tartrate labelled with 14C was given orally and parenterally to man and rats, and by direct injection into the caecum in rats. From the differences in urinary excretion after oral and parenteral administration intestinal absorption of tartrate was calculated as 18% of the dose in man and 81% in rats. Urinary tartrate was equivalent to 14% of the dose in man and 70% in rats, the difference between absorption and urinary excretion representing metabolism in body tissues.

2. Both man and the rat excreted part of the UC as respiratory carbon dioxide. This occurred to a small extent after parenteral injection, suggesting metabolism of tartrate by body tissues, but was greater after oral or intracaecal administration, indicating that the main site of tartrate metabolism is the intestine.

3. Several genera of intestinal bacteria were shown to liberate 14C carbon dioxide from labelled tartrate, and in a faecal incubation system L-tartrate, the natural isomer, was metabolized five times as rapidly as D-tartrate.

4. Oral sodium L-tartrate, 1.5 mmol day−1 kg−1, was given to two subjects and was shown to alkalinate the urine like sodium salts of other organic acids which are metabolized in the body. The reduction in renal hydrogen ion excretion showed that an average of 84% of the dose was metabolized.

5. Only 5% of labelled tartrate given by mouth appeared in faeces, and pharmacological doses of unlabelled L-tartrate had little or no aperient effect.

6. No evidence of toxicity of L-tartrate was encountered.

Key words: colonic bacteria, renal hydrogen-ion excretion, tartrate.

Introduction

L-Tartaric acid (2,3-dihydroxysuccinic acid) is present in many fruits, and is widely used as an aperient, in baking powder, and as a constituent of effervescent drinks. Extensive use over many years suggests that it is not toxic, but Robertson & Lönnell (1968) reported a death in man, associated with renal tubular changes, after ingestion of approximately 30 g of tartaric acid, and Krop & Gold (1945) described renal tubular damage in a dog ingesting tartrate (1.0 g/kg) daily for 3 months.

Previous studies of tartrate metabolism in animals and man used non-radioisotopic techniques with variable and sometimes conflicting results, though some of these differences may have been due to species variation (Underhill, Leonard, Gross & Jaleski, 1931a). Most workers agree that in man after oral ingestion tartrate is not found in the stool and urinary recovery is incomplete (Underhill, Peterman, Jaleski & Leonard, 1931b; Finkle, 1933), but whether it is absorbed or metabolized within the intestinal lumen by bacteria, or disappears by a combination of these two mechanisms, is not yet established. In most species, including man, parenteral tartrate appears largely unchanged in the urine (Finkle, 1933), which suggests that any metabolism occurring after oral ingestion takes place within the intestinal lumen.
The end result of complete oxidation of tartrate, as with other organic anions, must be the production of bicarbonate, which would have an alkalinizing effect on body fluids (Post, 1914; Pratt & Swartout, 1933).

Any toxicity of tartrate is probably dependent on the amount of anion absorbed from the intestine, whereas the purgative effect is likely to depend on the osmotic activity of the fraction unabsorbed. The alkalinizing effect will be determined by the proportion metabolized, whether by intestinal bacteria or by body tissues after absorption. A clearer understanding of the fate of ingested tartrate is needed, therefore, from a medical and toxicological viewpoint.

The purpose of this study was first to determine the extent of absorption and metabolism of sodium $^{14}$C-tartrate in the rat and man, and second to study quantitatively the effect of tartrate ingestion upon acid–base status in man.

Materials and methods

**Isomeric forms of tartaric acid**

Tartaric acid has two asymmetrically substituted carbon atoms, and so exists in three isomeric forms: L-tartaric acid, D-tartaric acid and meso-tartaric acid. L-Tartaric acid is the naturally occurring form which concerns us here, and was used in human experiments which did not involve $^{14}$C labelling. No commercial preparation of L-$^{14}$C-tartaric acid is available, and our tracer experiments were therefore performed with the $^{14}$C-labelled racemic DL mixture.

As a general rule naturally occurring L-compounds are more rapidly and completely metabolized than their un-natural D-isomers. This was so in our own studies of tartrate metabolism in a faecal incubation system, and our isotope studies with DL-tartrate must therefore have over-estimated metabolism of D-tartrate and under-estimated metabolism of the L-isomer. However, in some experiments more than 50% of the tracer dose was metabolized, providing firm evidence that some of the D-form was metabolized, as was also shown by our faecal incubation studies.

**Experimental**

**Metabolism of sodium DL-$^{14}$C-tartrate in rats.**

Three groups of rats weighing 200–250 g were given 20 μCi of sodium DL-$^{14}$C-tartrate with 18·8 mg/kg (0·081 mmol/kg) of L-tartrate as carrier, by three different routes: intraperitoneal, by gastric tube, and by injection directly into the caecum at open operation. Specific radioactivity of CO$_2$ in breath was measured at 30 min intervals for 6 h, and urinary excretion of labelled tartrate was measured over 24 h. Urine was first acidified to pH 2 to eliminate any $^{14}$C present as bicarbonate, causing loss of less than 1% of total urinary radioactivity, and thin-layer chromatography (see below) showed that the remaining radioactivity was in tartrate. Mean total recovery of the isotope was 72%; the remainder could have been excreted in the breath after 6 h, or in the faeces, which were not collected.

Metabolism of sodium DL-$^{14}$C-tartrate in man.

Five normal subjects aged 28–45 years took 5 μCi of sodium DL-$^{14}$C-tartrate, with sodium L-tartrate as carrier at one of three different doses (2·5, 5·0 or 10·0 g), together with 12·5 g of D-xylose, in 80 ml of water. The specific radioactivity of labelled CO$_2$ in breath was measured at hourly intervals, and urinary $^{14}$C-tartrate excretion in hourly collections for the first 7 h. Stools were weighed immediately they were passed, and analysed as described below. The excretion of $^{14}$C-tartrate in urine and stool and as respiratory CO$_2$ were expressed as percentages of the radioactivity administered. Hourly excretion profiles were plotted for urinary $^{14}$C-tartrate and respiratory $^{14}$CO$_2$ and compared with the excretion profile of D-xylose in urine, a substance of similar molecular size to tartrate which is known to be passively absorbed from the small intestine and partly excreted unchanged in the urine.

One subject received 10 μCi of DL-$^{14}$C-tartrate intravenously together with 125 mg (0·55 mmol) of sodium L-tartrate as carrier, and urine and breath were analysed for 8 h in the same way.

All experiments had the approval of the University College Hospital Ethical Committee.

Metabolism of sodium DL-$^{14}$C-tartrate by intestinal bacteria. The technique used estimated the proportion of the carboxyl groups in tartrate which were converted by bacteria into CO$_2$. Pure cultures of representative organisms from each of the major groups of intestinal bacteria were studied. Each organism was cultured for 48 h in Robertson's cooked meat broth, and 0·1 ml was then transferred to 10 ml of tartrate broth at pH 7·2 (Difco brain–heart infusion broth with 1% yeast extract and L-tartrate, 43·5 mmol/l, labelled with 0·10 μCi of DL-$^{14}$C-tartrate/ml) and incubated anaerobically for 48 h at 37°C. This period approximates to...
The colonic transit time of most normal subjects and was chosen to give anaerobic organisms ample time for growth and metabolism. After incubation 1 ml of perchloric acid solution (2·0 mol/1) was added and the broth was shaken with a few glass beads for 1 h at 37°C to release any 14CO2. Samples were then analysed for residual [14C]tartrate. Uninoculated broths containing labelled tartrate were similarly treated, and showed 98·5·101·6% recovery of isotope.

**Metabolism of unlabelled sodium D- and L-tartrate in a faecal incubation system.** In the previous experiments representatives of each group of faecal bacteria were found to be capable of converting labelled DL-tartrate (the racemic mixture) into CO2. Although on first principles it was likely that L-tartrate was metabolized more rapidly than the D-isomer, it was necessary to compare the metabolism by faecal bacteria of the two isomers by use of a chemical method, and the procedure of Underhill, Peterman & Krause (1931c) was followed.

Experiments were performed on faeces from five healthy subjects aged 22–61 years. In each study a fresh stool from a single subject was mixed by kneading through a polythene bag in 1·5 times its weight of sodium chloride solution (15·4 mmol/1). Three 50 ml aliquots were taken and mixed (1) with 30 ml of sodium chloride solution (154 mmol/1), (2) with 30 ml of sodium L-tartrate (250 mmol/1), and (3) with 30 ml of sodium D-tartrate (250 mmol/1). Samples were then incubated anaerobically at 37°C for 24 h with the system described by Vince, Down, Murison, Twigg & Wrong (1976). At zero time and at 3, 6, 7·5 and 24 h, 5 ml samples were removed for measurement of residual tartrate.

**Changes in systemic acid–base state after oral sodium L-tartrate.** Urinary hydrogen ion excretion and stool weights were measured daily in two normal subjects before, during and after ingestion of unlabelled sodium L-tartrate, 1·5 mmol/kg daily in three divided doses. In one subject changes in plasma total CO2 and urinary tartrate were measured and in both all urine samples were analysed for protein and serial measurements of creatinine clearance were performed. Since the complete metabolism of 1 mmol of sodium tartrate would produce 2 mmol of bicarbonate, and a consequent reduction in urinary hydrogen ion excretion by 2 mmol, the fraction of ingested tartrate metabolized to bicarbonate could be calculated from the change in urinary hydrogen ion excretion.

**Analytical**

DL-[1,4-14C]Tartaric acid from The Radiochemical Centre (Amersham, Bucks, U.K.) was shown to be at least 98% radiochemically pure by thin-layer chromatography on cellulose acetate, with p-gpant-1-ol/eucalyptol/formic acid (14·5:41·5:15, by vol.). The stable sodium L- and D-tartrate preparations used both had a purity of over 99%.

Urine samples were collected in plastic bottles containing a few crystals of thymol. In the radioisotopic experiments samples of urine were counted for 14C content directly in water-soluble scintillation fluid (PCS) with internal standard quench correction. Further samples were applied to a Bio-Rad AG 2-X8 anion-exchange resin column (acetate phase), and the urinary organic acids eluted with formic acid (19·5 mol/l), evaporated to dryness under nitrogen, dissolved in isopropyl alcohol and chromatographed as described above. Duplicate plates were developed with Bromocresol Green to identify organic acids and ammonium metavanadate to stain tartric acid. After identification the 14C content of individual spots was measured by liquid scintillation counting. In non-isotopic experiments sodium tartrate was measured in urine by a micro-modification of the metavanadate method of Underhill, Peterman & Krause (1931c).

Stools passed after ingestion of labelled sodium tartrate were homogenized and a sample centrifuged at 40 000 g. The supernatant was counted for 14C content, and the radioactivity shown to be in tartrate by the method described for urine. In the faecal incubation experiments 5 ml samples of incubate were taken into 30 ml of boiling 2% trichloroacetic acid, cooled, made up to 50 ml and decolorized with activated charcoal as described by Underhill, Peterman & Krause (1931c). The pH was then adjusted to 7·0 with sodium hydroxide solution (1·0 mol/l), 1 ml of sample was mixed with 1 ml of sodium metavanadate solution (164 mmol/l) and 1 ml of acetic acid and left for 55 min at room temperature and absorption then read at 550 nm.

Excretion of labelled CO2 was measured in man by collecting 2 mmol samples of expired CO2 in plastic scintillation pots containing 2 ml of Hyamine hydroxide (1·0 mol/l) and 2 ml of absolute ethanol. Thymolphthalein was used as indicator and the 14C content measured by liquid scintillation counting. The total 14C output in breath was calculated by multiplying the mean specific radioactivity of
expired CO₂ over the measured interval by the amount of endogenous CO₂ produced in the same time, obtained from tables relating CO₂ output at rest to body size (Winchell, Stahelin, Kusubov, Slanger, Fish, Pollycove & Lawrence, 1970). To measure CO₂ excretion by rats, each animal was housed in a glass metabolic chamber through which air was pumped into a series of glass columns filled with small glass rings and containing Hyamine hydroxide, ethanol and thymolphthalein. Columns were arranged in series so that samples (2-5 mmol) of CO₂ were collected at 30 min intervals and the specific radioactivity of CO₂ was measured as described above. Endogenous CO₂ production in the rat was measured directly in this system.

Urinary D-xylose was estimated by the method of Roe & Roce (1948) and urinary hydrogen ion excretion during the acid-base studies was measured as ammonium plus titratable acid minus bicarbonate, as described by Wrong & Davies (1959).

Results

Metabolism of sodium DL-[¹⁴C]tartrate in rats

After intraperitoneal injection, 63.1 ± SEM 5.2% was excreted in urine unchanged in 24 h and 9.4 ± 0.5% was excreted in 6 h as CO₂, indicating significant tissue metabolism of tartrate. After oral administration 51.0 ± 4.5% was excreted unchanged in the urine, but a greater proportion, 21.8 ± 1.4%, appeared in expired CO₂ than after intraperitoneal injection, suggesting that some of the tartrate had been metabolized within the intestine to bicarbonate. That this probably occurred as a result of bacterial metabolism in the caecum was suggested by the results of intracecal injection, which showed that most of the isotope, 66.6 ± 4.6%, was excreted as CO₂ in breath, and only a very small amount, 1.4 ± 0.3%, was absorbed from this site and appeared in the urine.

Metabolism of sodium DL-[¹⁴C]tartrate in man

Oral tartrate. From the raw data the percentage of administered ¹⁴C recovered over 7 h in breath and urine appeared to be independent of the dose given (three dosages) and results from all five experiments have therefore been pooled. In contrast to the findings in the rat, the major route of excretion was as labelled CO₂ in breath (46.2 ± 8.1%) and only 12.0 ± 1.2% of the dose was excreted unchanged in the urine. Four of the five subjects produced faeces in the 7 h after administration, and in some cases several fluid stools were passed, but mean faecal recovery of the label was equivalent to only 4.9 ± 1.7% of the dose, and later stools contained negligible amounts of the label.

Intravenous tartrate. After intravenous sodium tartrate in one subject, 63.8% of the dose was excreted unchanged in the urine over 22 h and 18.0% was excreted in breath as ¹⁴CO₂ over 8 h.

Comparison between [¹⁴C]carbon dioxide profiles in expired air after oral and intravenous DL-[¹⁴C]tartrate. After intravenous tartrate the peak of ¹⁴CO₂ in breath occurred during the first hour (Fig. 1), suggesting rapid metabolism by tissue enzymes. Peak excretion after oral administration was at 4 h, consistent with an increase in ¹⁴CO₂ production at the time of exposure of the tartrate to colonic bacteria.

Fig. 1. Oral and intravenous administration of [¹⁴C]tartrate in man: recovery of radioactivity in expired CO₂, expressed as percentages (±SEM, vertical bars) of the administered dose.
Metabolism of tartrate

Comparison between urinary excretion profiles for D-xylose and $[^{14}C]$tartrate after intravenous administration. The difference between the excretion profile after intravenous and oral administration must reflect the time required for intestinal absorption. After oral ingestion the peak excretion of $[^{14}C]$tartrate in urine was 1 h later than after intravenous injection and coincided with peak urinary D-xylose excretion.

Metabolism of labelled DL-tartrate by intestinal bacteria

All groups of intestinal organisms metabolized tartrate when grown for the first time in broth containing tartrate (Table 1). On several occasions more than 50% of the radioisotopic label was converted into CO$_2$ showing that both L- and D-forms of tartrate were metabolized. When tested a second time several aerobic organisms metabolized more tartrate in 48 h than they had metabolized on primary exposure.

Metabolism of unlabelled D- and L-tartrate in a faecal incubation system

The rate of disappearance of tartrate varied widely with different samples of faeces, but in every case L-tartrate disappeared more rapidly than D-tartrate (Table 2). Because of insensitivity of the method it was not possible to measure accurately tartrate concentrations below 25% of the initial concentration, and so the time of complete disappearance of tartrate could not be determined. When tartrate concentrations were plotted logarithmically against time, 50% loss occurred at approximately 4 h with L-tartrate and 20 h with D-tartrate.

Changes in systemic acid–base state in man after oral unlabelled sodium L-tartrate

Fig. 3 shows the effect of oral sodium L-tartrate (130.5 mmol/day) on urine pH and hydrogen ion concentration.
excretion in subject A. During the 3 control days mean urine pH was 5.8. During the 2 days of tartrate administration urine pH rose to 7.69 with a marked bicarbonate diuresis, urinary hydrogen ion excretion becoming negative on the second and third days. The deficit in hydrogen ion excretion from control values during the two experimental and subsequent 3 recovery days was 390 mmol, equivalent to 76-6% of the ingested dose of tartrate and indicating metabolism of approximately this proportion of the dose to bicarbonate. There was no proteinuria, and creatinine clearance was 115 ml/min before and 120 ml/min after ingestion of tartrate. Sodium tartrate had only a slight purgative effect (see faecal weight), which disappeared by the second day of administration.

Fig. 4 shows the results from subject B, who took a constant diet in order to prevent fluctuations in hydrogen ion production caused by variations in diet. Urine pH on the second to fourth control days was 6.2–6.6, and urinary hydrogen ion excretion averaged 63 mmol/day. During tartrate administration and over the next 3 days the reduction in hydrogen ion excretion totalled 771 mmol, equivalent to 92.3% of the ingested dose. The total CO₂ content of venous plasma was 31.4 mmol/l on the fourth control day, 34.0 on the fourth tartrate day, and 30.3 mmol/l on the fourth day of the recovery period, showing that the alkaline urine caused by tartrate was not a consequence of renal damage impairing the mechanism of urinary acidification, but a normal renal response to the mild systemic alkalosis caused by the conversion of ingested sodium tartrate into bicarbonate. In keeping with the greater metabolism of tartrate in this study, the purgative effect was negligible, but urine output rose to 3500 ml/day, an effect attributable to the osmotic diuresis caused by excretion of the sodium bicarbonate load. There was no proteinuria, and creatinine clearance was 108 ml/min before and 104 ml/min after tartrate administration. Urinary tartrate excretion was 9.8, 9.4, 8.9 and 10.3 mmol on the 4 tartrate days respectively; total renal excretion was thus 38.4 mmol or 7.4% of the total administered dose.

**Fate of carbon skeleton of tartrate anion**

In the radioactive studies only atoms C-1 and C-4 of the tartrate molecule were labelled with ¹⁴C; it was possible therefore that atoms C-2 and C-3 if not completely metabolized to CO₂ could have resulted in the production of an unlabelled, non-metabolizable C₂ fragment such as oxalate. However, in the acid–base experiments conversion of tartrate into oxalate or other unmetabolized anion would have caused a proportional reduction in the change in urinary hydrogen ion excretion. In subject B 92.3% of ingested tartrate was metabolized to bicarbonate on the basis of the change in urinary hydrogen ion excretion and 7.4% was accounted for as unchanged tartrate in
urine. Clearly, therefore, no significant formation of oxalate or other unmetabolized anion could have occurred. Urinary oxalate excretion was measured in this subject and was 0.24 ± SEM 0.04 and 0.22 ± 0.05 mmol/day on the control and tartrate days respectively, confirming no conversion of ingested tartrate into oxalate.

Discussion

Intestinal absorption of tartrate

Up to 60% of labelled tartrate was absorbed in rats, in agreement with previous estimates (Underhill et al., 1931a). In man only 12% of oral tartrate appeared in the urine; this was unrelated to dose and similar in time-course to D-xylose, suggesting that absorption is passive. The marked difference in urinary excretion after an oral dose (12%) and an intravenous dose (66%) suggests that only a small proportion of the oral dose was absorbed in man.

Fate of tartrate after absorption

In rats up to 10% of labelled parenteral tartrate was expired as CO₂ within 6 h, whereas 63% was excreted in the urine over 24 h, which indicates significant metabolism by tissues. Underhill et al. (1931a) found that after parenteral injection of tartrate recovery ranged from 70% in rabbits, dogs and rats to almost 100% in guinea pigs, so that tissue metabolism could not be excluded in any of these species, though clearly it played a minor role in disposal of tartrate.

In man Finkle (1933) found that 91% of an intramuscular dose of unlabelled tartrate was recovered in urine, and concluded that the human body cannot metabolize tartrate. With intravenous [¹⁴C]tartrate we found that 66% was excreted in the urine in 22 h, but that 18% was excreted as CO₂ over 8 h, indicating slow tissue metabolism.

Fate of unabsorbed tartrate

In rats after intracaecal injection of [¹⁴C]tartrate 67% was expired as CO₂ within 6 h, whereas less than 2% was recovered in the urine. This suggests only very limited absorption of caecal tartrate, but that active bacterial metabolism to bicarbonate or some precursor of bicarbonate occurred with rapid equilibration with the bicarbonate and CO₂ in blood to allow for the rapid appearance of the carbon label as ¹⁴CO₂.

In man 46% of oral [¹⁴C]tartrate was metabolized to CO₂ within 7 h and only 5% appeared in stool, even when purgative doses were taken, suggesting that very little unabsorbed tartrate escapes metabolism by intestinal bacteria. The change in urinary hydrogen ion excretion after ingestion of non-radioisotopic sodium L-tartrate in subject B suggests that up to 92.3% of ingested tartrate was metabolized to bicarbonate, and since our tracer experiments showed poor absorption of oral tartrate most of this metabolism must have been by bacteria.

Bacterial metabolism of tartrate

We confirm that a wide variety of human intestinal bacteria metabolize tartrate (Maasen, 1895). Some organisms (e.g. micrococci, streptococci and Proteus mirabilis) metabolized more tartrate on repeated growth in tartrate broth, suggesting an adaptive process, which might explain the rapid decrease in purgation when tartrate was ingested for several days (Fig. 3 and Fig. 4).

Our faecal incubates metabolized both L- and D-tartrate, but L-tartrate, which exists naturally in foods, was destroyed faster, suggesting that intestinal bacteria have adapted their metabolism to utilize it as a substrate. Many strains of coliform bacteria destroy the L-isomer more rapidly than D-tartrate (Vaughn, Marsh, Stadtman & Cantino, 1946).

The colon is likely to be the main site of tartrate metabolism, as it has the most abundant bacterial flora. Despite our finding that intestinal bacteria formed bicarbonate from tartrate when grown in vitro in pure culture, it is possible that bicarbonate is not formed directly in the colon, where the low oxygen tension (Bornside, Cherry & Bert, 1973) and negative Eh (Wostmann & Bruckner-Kardoss, 1966; Vince et al., 1976) favour reductive rather than oxidative reactions. Splitting of the four-carbon-atom tartrate molecule to glycollate, glyoxyxlate or oxalate also appears unlikely as these substances would be expected to increase urinary oxalate excretion, which was not found. Intestinal bacteria may split the tartrate molecule to two molecules of acetate, which is produced in large amounts by colonic bacteria (Rubinstein, Howard & Wrong, 1969). Acetate has been shown to be a product of tartrate metabolism by lactobacilli (Radler & Yanissis, 1972), and is readily absorbed from the colon (Dawson, Holdsworth & Webb,
### Fate of ingested tartrate in man

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FIG. 5. Fate of ingested tartrate in man. Calculations have been made on the assumption that complete absorption of oral tartrate would yield urinary recoveries of labelled tartrate identical with those seen after parenteral tartrate; as a result some figures are slightly different from the direct results of the isotope recoveries. The figures in parentheses show the relevant values, where available, for the rat.

1964; Bond & Levitt, 1976); its complete metabolism after absorption would produce the observed effects on acid–base metabolism.

### Effect of tartrate on acid–base state

Complete metabolism of sodium tartrate, as with sodium salts of other organic anions, would produce an equivalent amount of sodium bicarbonate and hence would in turn produce an equivalent reduction in urinary hydrogen ion excretion (Reiman, Lennon & Lemann, 1961), irrespective of whether tartrate was metabolized in the tissues (Pratt & Swartout, 1933) or in the bowel, provided that the products of intestinal metabolism were absorbed. Incomplete oxidation of tartrate would result in proportionally reduced changes in urinary hydrogen ion excretion. In our two human studies with pharmacological doses of sodium L-tartrate, the reduction in urinary hydrogen ion excretion was equivalent to 76-6% and 92-3% of the dose, indicating that most of the ingested tartrate was metabolized, and our evidence that tartrate was poorly absorbed in man leads us to conclude that this metabolism was mainly intestinal.

### Toxicity of L-tartrate

In our two human studies using large pharmacological doses of L-tartrate there was no evidence of renal toxicity as creatinine clearance was normal and there was no proteinuria. In man the fact that so little tartrate is absorbed may help to explain its use over many years without evidence of harmful effects.

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### References

Metabolism of tartrate


