Dietary fructose *v.* glucose lowers ferrous-iron absorption in rats

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The effect of dietary fructose *v.* glucose on Fe solubility in the small intestine and apparent Fe absorption was studied in rats. Female rats were fed for 4 weeks on low-Fe (10 mg Fe/kg) or normal-Fe (40 mg Fe/kg) diets containing either fructose or glucose (709·4 g monosaccharide/kg). Fe was added to the diets in the form of $FeSO_4$. The low-Fe diets did not lower levels of haemoglobin and packed cell volume, but significantly lowered Fe concentration and Fe mass in the liver, kidney and spleen. Fructose *v.* glucose also lowered Fe concentrations in these organs, but did not alter absolute Fe contents. Low Fe intake reduced the amount of Fe in the intestinal lumen. The total amount of Fe and Fe concentration in the liquid phase of the proximal intestinal lumen were depressed by fructose irrespective of Fe intake. Fructose also lowered the amount of Fe in the liquid phase of the distal intestine. In keeping with these observations, dietary fructose significantly lowered apparent absorption of Fe at the two levels of Fe intake. Decreasing the intake of Fe raised the percentage of apparent Fe absorption.

Fructose: Iron: Glucose: Rat

Under *in vitro* conditions at neutral to alkaline pH, fructose can form with Fe^{3+} a stable, soluble chelate (Charley *et al.* 1963). In guinea-pigs, Fe absorption from the orally administered Fe^{3+}–fructose complex was more efficient than that from $FeSO_4$ (Bates *et al.* 1972). In a feeding trial with rats, sucrose *v.* maize starch raised Fe absorption (Johnson & Hove, 1986). When giving Fe-depleted rats a single meal containing $^{59}FeCl_3$ and either fructose or glucose, fructose was found to stimulate Fe absorption (Pabón de Rozo *et al.* 1986). Thus, fructose in the diet may enhance Fe absorption. In contrast, the influence of fructose on Fe stores in the liver is not clear. Fructose *v* glucose either lowered (Landes, 1975; Fields *et al.* 1984) or raised (Landes, 1975) the amount of Fe in the liver of rats. Fructose *v.* starch either lowered (Fields *et al.* 1984) or did not influence (Holbrook *et al.* 1986) the absolute amount of Fe in the liver.

In the present experiment with rats the effect of dietary fructose on Fe solubility in the small intestine was investigated. It was hypothesized that in rats fed on fructose instead of glucose there is more soluble Fe in the ileal lumen due to formation of the complex. This would explain the earlier observed (Bates *et al.* 1972; Pabón de Rozo *et al.* 1986) stimulatory effect of fructose on Fe absorption because soluble rather than insoluble Fe in the ileal lumen is absorbed (Hallberg, 1981). We also determined the effect of fructose on apparent Fe absorption and on Fe stores in the liver.

MATERIALS AND METHODS

Animals, housing and diets

Female Wistar (Cpb:WU) rats, aged about 7 weeks, were used. The rats were derived from the colony of the Laboratory Animals Centre of the Wageningen Agricultural University.

Table 1. *Composition of the diets*

Diet...	Normal-Fe		Low-Fe	
	Glucose†	Fructose	Glucose	Fructose
Ingredients				
Glucose (g)	709·4	—	709·4	—
Fructose (g)	—	709·4	—	709·4
$FeSO_4.7H_2O$ (mg)	174	174	17·4	17·4
Constant components* (g)	290·6	290·6	290·6	290·6
Chemical analysis				
Fe (mg/kg)	42·0	38·8	9·5	9·0

* The constant components consisted of (g): casein 151, maize oil 25, coconut fat 25, cellulose 30, $CaCO_3$ 12·4, $NaH_2PO_4.2H_2O$ 15·1, $MgCO_3$ 1·4, KCl 1·0, $KHCO_3$ 7·7, Fe-free mineral premix 10, vitamin premix 12. The mineral premix consisted of (mg): MnO_2 79, $ZnSO_4.H_2O$ 33, $NiSO_4.6H_2O$ 13, NaF 2, KI 0·2, $CuSO_4.5H_2O$ 15·7, $Na_2SeO_3.5H_2O$ 0·3, $CrCl_3.6H_2O$ 1·5, $SnCl_2.2H_2O$ 1·9, NH_4VO_3 0·2, maize meal 9853·2. The vitamin premix consisted of (mg): thiamin 4, riboflavin 3, niacinamide 20, calcium DL-pantothenate 17·8, pyridoxine 6, cyanocobalamin 50, choline chloride 2000, folic acid 1, biotin 2, menadione 0·05, DL-α tocopheryl acetate 60, retinyl acetate and retinyl palmitate 8 (4000 IU), cholecalciferol 2 (1000 IU), maize meal 9826·15.

† This diet also served as the pre-experimental diet.

The rats had been fed *ad lib.* on a commercial, pelleted diet (RMH-B; Hope Farms, Woerden, The Netherlands) and tap-water.

All rats went through a pre-experimental period of 2 weeks during which they received a purified diet containing 709·4 g glucose and 40 mg Fe/kg (Table 1) and demineralized water. The rats had free access to feed and water. During the pre-experimental period the rats were housed in groups of four animals in stainless-steel cages (600 × 210 × 190 mm) with wire-mesh bases.

At the end of the pre-experimental period (day 0 of the experiment) the rats were divided into four groups of twelve animals each, so that body-weight distributions within the groups were similar. Each group was randomly assigned to one of the purified diets given in Table 1. From day 0 the rats were housed individually in stainless-steel cages (240 × 170 × 170 mm) with wire-mesh bases. The cages were placed in a randomized position in a room with a controlled temperature (20–22°), lighting (light on 07.00–19.00 hours) and relative humidity (40–65%).

During the experimental period one group remained on the diet containing 709·4 g glucose and 40 mg Fe/kg. The other groups were fed on diets with either 709·4 g fructose and 40 mg Fe/kg, 709·4 g glucose and 10 mg Fe/kg or 709·4 g fructose and 10 mg Fe/kg. The diet containing 40 mg Fe/kg can be considered a normal-Fe diet (National Research Council, 1978), whereas that containing 10 mg Fe/kg is a low-Fe diet. The purified diets, which were in powdered form, were stored at 4° until needed. Feed and demineralized water were provided *ad lib.* Feed intake and body weights were recorded. The experiment lasted 28–29 d.

Collection of samples

From day 21 to day 26 the faeces of each rat were collected quantitatively. On day 28, between 09.00 and 12.00 hours, six rats from each dietary group were anaesthetized in random order by exposure to diethyl ether. Blood was taken by orbital puncture and the anaesthetized rats were immediately killed by decapitation. The entire small intestine between the stomach and the caecum was removed. It was divided into a proximal and a distal half. The digesta of both halves of the intestine were collected separately in

Table 2. *Effect of glucose or fructose on growth performance and organ weights of rats fed on low- and normal-Fe diets*‡

(Mean values with their pooled standard errors for twelve rats per dietary group)

Diet...	Normal-Fe		Low-Fe			Statistical significance (ANOVA) of effect of
Variable	Glucose	Fructose	Glucose	Fructose	Pooled SE	dietary monosaccharide†
Feed intake (g/d)	16·0	14·2[a]	15·6	13·9[a]	0·92	*
Body wt (g)						
Initial (day 0)	177·1	176·7	175·4	176·1	9·4	
Final (day 28–29)	217·2	214·9	217·8	206·7	13·8	
Organ wts (g/100 g body wt)						
Liver	3·49	4·57[a]	3·52	4·46[a]	0·26	*
Left kidney	0·32	0·38[a]	0·32	0·39[a]	0·03	*
Right kidney	0·33	0·39[a]	0·33	0·40[a]	0·03	*
Heart	0·36	0·38	0·36	0·37	0·02	*
Spleen	0·20	0·22[a]	0·20	0·22	0·02	*

[a] Significantly different from glucose for the same dietary Fe content, $P < 0·025$.
* $P < 0·05$.
† There were no significant effects of dietary Fe level.
‡ For details of diets and procedures, see Table 1 and pp. 171–172.

preweighed centrifuge tubes by gently squeezing the intestine between finger and thumb, and total weight was determined. The digesta were immediately centrifuged (10 min, 10000 ***g***) at room temperature, and the supernatant fraction and pellet were separated. The weights of the pellet and supernatant fraction were determined; pH of the supernatant fraction was measured promptly with an electrode (Russell combination pH electrode, Type RS-53, Auchtermuchty, Fife). The heart, kidneys, liver, spleen and tibia were excised, weighed and frozen at −20°. On day 29 the entire procedure was repeated with the remaining rats.

Chemical analysis

In whole heparinized blood, haemoglobin and packed cell volume were measured using a Sysmex K1000 (Automated Hematology Analyzer; Toa Medical Electronics Co. Ltd, Kobe, Japan). Trichloroacetic acid (TCA) was added to the supernatant fraction to a final concentration of 50 g/l and the TCA-soluble fraction isolated by centrifugation (2 min, 10000 ***g***). The pellet of whole digesta was freeze-dried overnight, weighed, ashed (500° for 17 h) and dissolved in 0·1 ml 6 M-HCl and 0·9 ml demineralized water. Fe in intestinal fractions, plasma Fe, total Fe-binding capacity and transferrin saturation were determined using a commercial kit (Iron FZ Test; Roche, Roche Diagnostics, Basel, Switzerland) and a COBAS-BIO auto-analyzer (Hoffmann–La Roche BV, Mijdrecht, The Netherlands). Cholesterol and triacylglycerols in plasma were determined enzymically using kits purchased from Roche Diagnostics and the COBAS-BIO auto-analyzer.

The heart, kidneys, spleen and tibia were dried overnight (105°), weighed and ashed (500°, 17 h). The ash was dissolved in 1 ml 6 M-HCl and 4 ml demineralized water. Fe in the liver was measured after wet-ashing with 14 M-nitric acid. Fe in the feed was determined after wet-ashing: 5 g feed was dissolved in 5 ml 18 M-H_2SO_4 and 14 M-HNO_3 and heated at 120° until a clear solution was obtained. Appropriate dilutions were made for the determination of Fe by atomic absorption spectrometry (Varian AA-475; Varian Techtron, Springvale, Australia).

Calculations

The distribution of Fe between the solid and liquid phases of digesta was calculated. The pellet obtained after centrifugation of whole digesta contains the solid phase contaminated with the liquid phase. The weight of the solid phase was obtained after freeze-drying the pellet. The weight of the liquid phase was calculated as the sum of weights of the liquid phase in the pellet (total pellet weight minus solid phase) and supernatant fraction. The concentration of Fe in the supernatant fraction was taken for that in the liquid phase. The amount of Fe in the solid phase was calculated as that in the total pellet minus that in the liquid phase of the pellet. Multiplying Fe concentration (μg/g) in the supernatant fraction by the weight of the liquid phase gave the amount of Fe in the liquid phase.

The proportion of Fe in the liquid phase was computed as the percentage of total Fe in intestinal contents. Apparent digestibility of Fe was calculated as intake minus faecal excretion and expressed as a percentage of intake.

Statistical analysis

Treatment effects were statistically evaluated by two-way analysis of variance. The probability of a type 1 error $P < 0{\cdot}05$ was taken as the criterion of significance. The effects of fructose *v.* glucose with Fe constant and the effects of dietary Fe concentration with sugar constant were statistically analysed using Student's t test and Bonferroni's adaptation; the level of significance was pre-set at $P < 0{\cdot}025$. Data were analysed by computer using the SPSS/PC+ statistical package.

RESULTS

Feed intakes were significantly lower in rats fed on the fructose diets, but final body weights did not differ between the experimental groups (Table 2). Fructose increased the weights of liver, kidney, heart and spleen. The livers of rats fed on the fructose diets had a grey-white appearance. Fructose *v.* glucose in the diet did not affect plasma cholesterol, but produced hypertriacylglycerolaemia. Plasma cholesterol and triacylglycerol concentrations for rats fed on the glucose and fructose diets were: 2·16 (SE 0·22) and 2·36 (SE 0·22), and 0·78 (SE 0·24) and 1·14 (SE 0·43) mM, (n 24). The amount of Fe in the diet did not influence plasma lipids.

Haemoglobin and packed cell volume values were not influenced by the type of monosaccharide or the amount of Fe in the diet (Table 3). Decreasing the amount of Fe in the diet lowered the plasma Fe concentration and transferrin saturation. Fructose *v.* glucose in the diet slightly, but significantly, elevated total Fe-binding capacity and reduced transferrin saturation.

In all organs except the heart, Fe concentrations were significantly lowered in rats fed on the low-Fe diets (Table 3). Fructose *v.* glucose had a similar effect and also reduced heart Fe concentrations. For tibia Fe, the lowering effect of fructose failed to reach statistical significance. Looking at the Fe mass in organs it is clear that the low-Fe diets reduced Fe in the liver, kidney and spleen. However, fructose did not lower Fe mass in the liver, kidney and spleen. For kidney there was even a slight increasing effect.

Fructose significantly reduced the weight of the liquid and solid phase in the proximal half of the small intestine (Table 4). In the distal half such fructose effects were not seen. Fructose significantly raised the pH of the liquid phase of the proximal intestine, but not that of the distal intestine. The total amount of Fe and Fe concentration in the liquid phase of the proximal intestine were depressed by fructose. Fructose instead of glucose in the diet also lowered the amount of Fe in the liquid phase of the distal intestine, but did not affect Fe concentration. Low Fe intake reduced the amount and concentration of Fe in the liquid

Table 3. *Effect of glucose or fructose on blood Fe variables and tissue Fe of rats fed on low- and normal-Fe diets*‡

(Mean values with their pooled standard errors for twelve rats per dietary group)

Diet...	Normal-Fe		Low-Fe			Statistical significance (ANOVA) of effect of:	
Variable	Glucose	Fructose	Glucose	Fructose	Pooled SE	Dietary monosaccharide	Dietary Fe level
Blood							
Haemoglobin (mmol/l)	9·31	9·17	9·19	9·07	0·02		
Packed cell volume	0·47	0·46	0·45	0·46	0·003		
Plasma Fe (μmol/l)	56·31	59·66	54·13	49·15[b]	8·57		*
Total Fe-binding capacity (μmol/l)	92·32	105·3[a]	96·33	105·7	11·24	*	
Transferrin saturation (%)	61·99	57·49	56·41	47·23[ab]	10·97	*	*
Tissue Fe (μg/g dry wt)							
Liver	772	535[a]	464[b]	380[b]	112·6	*	*
Kidney	309	269[a]	244[b]	245	35·0	*	*
Spleen	2848	2210	1675[b]	1561[b]	632·9	*†	*†
Heart	422	363[a]	369	365	48·4	*	
Tibia	74·6	66·1	61·1[b]	56·5	13·6		*
Tissue Fe (μg/organ)							
Liver	1875	1805	1146[b]	1162[b]	274·0		*
Kidney	108	111	83[b]	87[b]	11·9	*†	*†
Spleen	281	231	164[b]	158[b]	69·0		*†
Heart	74·5	65·4	64·4	63·0	11·2		

[a] Significantly different from glucose for the same dietary Fe content, $P < 0·025$.
[b] Significantly different from normal Fe for the same dietary monosaccharide, $P < 0·025$.
* $P < 0·05$.
† After log-transformation of the data (unadjusted pooled standard errors are given).
‡ For details of diets and procedures, see Table 1 and pp. 171–173.

and solid phases of both the proximal and distal halves of the intestine, but increased the proportion of Fe in the liquid phase. There were no interactions between the effects of fructose and Fe.

Intake of Fe was significantly lower for groups fed on fructose instead of glucose and for groups fed on a low-Fe diet *v.* a high-Fe diet (Table 5). Faecal output of Fe was depressed in rats given the low-Fe diets, but not in rats fed on the fructose diets. Fructose *v.* glucose caused a significantly decreased absorption of Fe. The low-Fe diets raised the percentage of apparent Fe absorption.

DISCUSSION

Lowering Fe intake in rats generally produces reduced values for haemoglobin and packed cell volume (Sørensen, 1965; Wien & Van Campen, 1991). This was not seen in the present experiment and may have been related to the somewhat advanced age of the animals (7 weeks at the beginning of the experiment). Possibly the Fe stores were sufficiently large to prevent changes in haemoglobin and packed cell volume after feeding the low-Fe diets for 4 weeks. The rats fed on the low-Fe diets did show other signs of Fe deficiency, such as lowering of Fe in the liver, kidney, spleen, tibia and a slight decrease of plasma Fe and transferrin saturation.

Table 4. *Effect of glucose or fructose on the distribution of Fe between liquid and solid phases of digesta in small intestine of rats fed on low- or normal-Fe diets*‡

(Mean values with their pooled standard errors for twelve rats per dietary group)

Diet...	Normal-Fe		Low-Fe			Statistical significance (ANOVA) of effect of:	
Variable	Glucose	Fructose	Glucose	Fructose	Pooled SE	Dietary monosaccharide	Dietary Fe level
Proximal intestine							
Liquid phase							
Wt (g)	0·23	0·14[a]	0·27	0·18[a]	0·10	*	
pH	6·04	6·52	6·12	6·43	0·52	*	
Solid phase wt (g)	0·04	0·02[a]	0·06	0·03	0·02	*	
Fe:							
Amount in liquid phase (μg)	1·58	0·69	0·55[b]	0·31[ab]	0·68	*†	*†
Amount in solid phase (μg)	11·0	7·20	2·07[b]	1·59[b]	4·25		*†
Percentage in liquid phase (%)	13·0	13·3	22·2[b]	18·6	8·30		*
Concentration in liquid phase (μg/g)	6·56	5·00	1·98[b]	1·75[b]	1·42	*†	*†
Distal intestine							
Liquid phase							
Wt (g)	0·44	0·37	0·44	0·40	0·11		
pH	7·72	7·79	7·52	7·62	0·31		*
Solid phase wt (g)	0·12	0·10	0·11	0·11	0·04		
Fe:							
Amount in liquid phase (μg)	1·27	1·04	0·69[b]	0·55[b]	0·30	*	*
Amount in solid phase (μg)	50·2	41·4	7·8[b]	8·2[b]	12·55		*†
Percentage in liquid phase (%)	2·60	3·01	8·59[b]	6·99[b]	2·66		*†
Concentration in liquid phase (μg/g)	2·93	2·85	1·51[b]	1·38[b]	0·48		*

[a] Significantly different from glucose for the same dietary Fe content, $P < 0·025$.
[b] Significantly different from normal Fe for the same dietary monosaccharide, $P < 0·025$.
* $P < 0·05$.
† After log-transformation of the data (unadjusted pooled standard errors are given).
‡ For details of diets and procedures, see Table 1 and pp. 171–174.

It is known that fructose in the diet enlarges the liver of rats (Landes, 1975; Fields *et al.* 1984; Holbrook *et al.* 1986; Lewis *et al.* 1990). In the present experiment this was also found. Fructose reduced the concentration of Fe in the liver, but did not affect the total hepatic Fe content. In previous studies with rats fructose *v.* glucose either lowered (Fields *et al.* 1984; Landes, 1975) or raised (Landes, 1975) the absolute amount of Fe in the liver. Thus, fructose has no systematic influence on Fe stores in the liver.

Fructose *v.* glucose reduced the total amount of Fe in the small intestine. This fructose effect could result from an altered feed intake pattern of the rats given fructose or from an altered flow of digesta through the small intestine. The reduced food intake by rats given fructose may have contributed to the observed reduction in intestinal Fe content. Fructose also lowered the concentration of Fe in the liquid phase of small intestinal contents. As it has been suggested that only soluble Fe may cross the intestinal epithelium (Hallberg,

Table 5. *Effect of glucose or fructose on apparent absorption of Fe in rats fed on low- and normal-Fe diets*†

(Mean values with their standard errors for twelve rats per dietary group*)

Diet...	Normal-Fe				Low-Fe			
	Glucose		Fructose		Glucose		Fructose	
Fe balance	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Intake (μg/d)	660	51	557[a]	38	142[b]	11	125[ab]	11
Faecal output (μg/d)	383	28	389	55	64·5[b]	10	71·0[b]	15
Absorption (% of intake)	41·8	4·5	29·9[a]	9·7	54·4[b]	7·2	42·5[ab]	14·0

[a] Significantly different from glucose for the same dietary Fe content, $P < 0{\cdot}025$.
[b] Significantly different from normal Fe for the same dietary monosaccharide, $P < 0{\cdot}025$.
* Because of different variances between the groups, even after log-transformation, ANOVA was not possible.
† For details of diets and procedures, see Table 1 and pp. 171–174.

1981), this should reduce Fe absorption. Indeed, apparent absorption of Fe was significantly lower in the fructose groups. The relationship, at a fixed Fe status, between soluble Fe concentration in the intestinal lumen and apparent Fe absorption is not known. Thus, it cannot be assessed to what extent the fructose-induced reduction in Fe absorption may be explained by the lowering of Fe concentration in the liquid phase of intestinal contents.

The observed fructose-induced impairment of Fe absorption is at variance with earlier reports. Fructose and Fe^{3+} can form soluble, stable complexes *in vitro* (Charley *et al.* 1963) and oral administration of such a complex has been shown to facilitate Fe absorption (Bates *et al.* 1972). Fe^{3+}–fructose complexes can also be prepared *in vitro*, but only at high carbohydrate:Fe values (Charley *et al.* 1963). Possibly, Fe–fructose complexes were not formed in the gastrointestinal tract of our rats because the pH of the digesta and/or the Fe:fructose molar ratio were not suitable (Charley *et al.* 1963). The use of Fe^{3+} salts as the dietary Fe source and/or a different experimental design may explain the earlier observed sucrose-induced (Johnson & Hove, 1986) and fructose-induced (Pabón de Rozo *et al.* 1986) stimulation of Fe absorption in rats. Johnson & Hove (1986) compared sucrose with maize starch and used $Fe_2(SO_4)_3$ as the Fe source. Pabón de Rozo *et al.* (1986) added $^{59}FeCl_3$ to meals containing either glucose or fructose and determined the absorption of the label in rats.

The previously stated reasoning implies that the effect of fructose on Fe absorption depends on the Fe source in the diet. With Fe^{3+} salts, fructose may improve Fe absorption, which may relate to the formation of Fe^{3+}–fructose complexes in the gastrointestinal tract. The present study using $FeSO_4$ as the Fe source demonstrates that fructose *v.* glucose lowers Fe absorption in rats, which may relate to the decreased Fe concentration in the liquid phase of intestinal digesta. Why dietary fructose lowers this concentration is not known.

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