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Xylitol Metabolism in the Isolated Perfused Rat Liver

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1. Loading the isolated perfused liver from well-fed rats with xylitol (20mM) caused a depletion of adenine nucleotides and P_i and an accumulation of α-glycerophosphate. The ATP content fell to 66% of the control value after 10min and to 32% after 80min. The ADP and AMP contents also fell. After 80min 63% of the total adenine nucleotides and 59% of the P_i had been lost. 2. The α-glycerophosphate content rose from 0.13 to 4.74μmol/g at 10min and reached 8.02μmol/g at 40min. 3. Xylitol was rapidly metabolized, the main products being glucose, lactate and pyruvate. 4. The [lactate]/[pyruvate] ratio in the presence of xylitol rose to 30–40. 5. On perfusion of livers from starved animals the main product of xylitol metabolism was glucose and the mean ratio xylitol removed/glucose formed was 1.29 (corrected for endogenous glucose and lactate production). This is close to the predicted value of 1.2. 6. Evidence is presented indicating that the loss of adenine nucleotides caused by xylitol is not due to the increased ATP consumption but to the accumulation of α-glycerophosphate and depletion of P_i. 7. The loss of adenine nucleotides accounts for the hyperuricaemia which can occur after xylitol infusion in man. 8. The relevance of the findings to the clinical use of xylitol as an energy source is discussed.

Substances that are rapidly phosphorylated in the liver, such as fructose and glycerol, cause a depletion of hepatic ATP and also of total adenine nucleotides and of P_i (Mäenpää *et al.*, 1968; Raivio *et al.*, 1969; Burch *et al.*, 1970; Woods *et al.*, 1970). This is due to the fact that ATP and P_i inhibit the enzymes responsible for the irreversible breakdown of AMP (AMP deaminase and 5-nucleotidase). The present experiments were undertaken to test whether xylitol can affect hepatic nucleotides in the same way as fructose and glycerol. Xylitol is known to be rapidly converted into glucose in the liver (Ross *et al.*, 1967; Jakob *et al.*, 1971). This process involves phosphorylations (see Krebs & Lund, 1966), and the rate of additional ATP consumption on addition of xylitol can be of the same order as that in the presence of fructose. The expected depletion of adenine nucleotide was in fact observed. These results are of special interest in connexion with the clinical use of xylitol as a source of energy in special situations.

Materials and Methods

Animals and diet

Female Wistar rats weighing about 200g were obtained from Carworth (Europe) Ltd., Alconbury, Hunts., U.K. and were fed on a standard small-animal diet (Spillers Mills Ltd., Gainsborough, Lincs., U.K.), water being provided *ad libitum*. This strain of rats differs in respect to several metabolic properties from that used as a routine in the Metabolic Research Laboratory. In the present strain the hepatic contents of glutamate and α-oxoglutarate are lower, the rate of gluconeogenesis from xylitol is higher and that from alanine is lower.

Reagents

Xylitol was obtained from Sigma (London) Chemical Co. Ltd., London S.W.6, U.K., and nucleotides, coenzymes and crystalline enzymes were obtained from Boehringer Corp. (London) Ltd., London W.5, U.K.

Liver perfusion

The method of liver perfusion was that described by Hems *et al.* (1966). The composition of the perfusion medium has been described previously (Woods *et al.*, 1970).

Analytical methods

Citrate was determined with citrate lyase (Gruber & Möllering, 1966), ammonia by the method of Kirsten *et al.* (1963) and glutamate by the method of Bernt & Bergmeyer (1963), as modified by Lund

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Table 1. *Metabolite concentrations in the*

Livers from well-fed rats were perfused with a medium containing initially 5, 10 or 20mM-xylitol. The xylitol xylitol was present and four in the control experiments. The initial volume of the medium was 150ml and sampling and Methods section.

	Concn. of metabolites in perfusion medium (mM)							
	5mM-Xylitol				10mM-Xylitol			
Time (min)	Xylitol	Glucose	Lactate	Pyruvate	Xylitol	Glucose	Lactate	Pyruvate
0	4.73±0.01	0.25±0.04	0.29±0.07	0.01±0.006	8.28±0.21	0.44±0.06	0.57±0.18	0.04±0.01
15	—	3.13±0.33	1.17±0.21	0.05±0.04	—	3.12±0.22	1.68±0.11	0.04±0.003
30	2.53±0.36	4.43±0.12	1.58±0.20	0.02±0.003	5.27±0.23	4.50±0.39	2.23±0.12	0.06±0.02
45	—	5.31±0.07	2.01±0.39	0.03±0.01	—	5.63±0.34	2.52±0.27	0.04±0.01
60	1.01±0.08	5.82±0.19	2.13±0.29	0.12±0.04	3.14±0.28	6.80±0.43	2.77±0.33	0.05±0.01
90	0.72±0.20	6.55±0.33	2.46±0.46	0.19±0.05	1.59±0.21	8.95±0.42	3.28±0.34	0.05±0.01
120	0.73±0.25	6.75±0.36	2.92±0.56	0.28±0.10	0.91±0.29	9.64±0.50	3.32±0.32	0.29±0.07

(1971). Xylitol was determined by the method of West & Rapoport (1949). Oxoglutarate was determined by the method of Bergmeyer & Bernt (1963), malate by that of Hohorst (1963) and xylulose 5-phosphate by that of Racker (1963).

Results

Effect of xylitol concentration of the medium on the production of glucose, lactate and pyruvate by livers from well-fed rats

Xylitol was added to the perfusion medium at initial concentrations of 5, 10 or 20mM and the time-course of metabolic changes was followed for 120min. Table 1 shows the metabolite concentrations in the perfusate and Table 2 the rates of changes in μmol/min per g of liver. Xylitol removal was rapid and the initial rates rose somewhat with the xylitol concentration from 1.55 at 5mM to 1.93μmol/min per g of liver at 20mM.

The initial concentration of xylitol giving half-maximal rates of removal in perfused livers was 3.2mM. This value is higher than the K_m for the NAD-linked purified cytoplasmic polyol dehydrogenase of sheep liver (0.18mM at pH9.6; Smith, 1962) and that of guinea-pig liver (0.6mM at pH8.1; Hollmann, 1969).

Glucose production was increased by xylitol over that from endogenous precursors and the final glucose concentration in the medium was greater in the xylitol perfusion than in the control perfusions. The increase was smaller than that expected on the assumption that the xylitol removed was quantitatively converted into glucose. This discrepancy was partly due to the formation of products other than glucose, i.e. lactate, and partly to an inhibition of glucose formation via glycogenolysis.

In all experiments lactate was produced throughout the perfusions, and the final lactate concentration in the medium was increased by xylitol above the control values, in which 2mM was seldom exceeded. The [lactate]/[pyruvate] ratio rose in the presence of xylitol above physiological limits to 30–40, i.e. as expected since xylitol introduces reducing equivalents (see Jakob *et al.*, 1971).

Xylitol metabolism in livers from rats starved for 48h

The main product formed from xylitol in livers from starved rats was glucose; small and variable amounts of lactate were produced (Table 3) and pyruvate production was negligible.

The rate of xylitol removal was of the same order as in the livers of fed rats. The initial rates of glucose formation rose with increasing xylitol concentration from 1.10μmol/min per g of liver at 5mM- to 1.71μmol/min per g of liver with 20mM-xylitol. These rates are somewhat higher than those previously measured under similar conditions [0.67μmol/min per g of liver (Ross *et al.*, 1967) and 0.65μmol/min per g of liver (Jakob *et al.*, 1971)].

If glucose is the only product of xylitol metabolism the ratio (xylitol removed)/(glucose formed) should be 1.2. The values for the ratio obtained were between 1.08 and 1.25. It is uncertain whether this should be corrected for the endogenous production of glucose and lactate, because it is not known whether addition of xylitol affects the endogenous metabolism. If the correction is made the ratio has a mean value of 1.29.

Contents of intermediary metabolites in the freeze-clamped perfused liver after xylitol loading

The changes in the contents of liver metabolites at different time-intervals after xylitol loading (addition

perfusion medium after xylitol loading

concentrations in the medium are expressed as mM (mean±S.E.M.). The number of observations was three when decreased this by 0.5ml every 15min. The liver weights were about 7g. For experimental details see the Materials

Concn. of metabolites in perfusion medium (mM)						
20mM-Xylitol				No substrate added		
Xylitol	Glucose	Lactate	Pyruvate	Glucose	Lactate	Pyruvate
18.1 ±0.33	0.28±0.05	0.85±0.37	0.01±0.01	0.27±0.26	0.26±0.02	0.06±0.01
—	3.03±0.27	2.12±0.09	0.02±0.006	2.44±0.26	0.83±0.08	0.13±0.05
15.1 ±0.25	4.07±0.29	2.99±0.23	0.03±0.01	3.44±0.27	1.30±0.04	0.15±0.06
—	5.13±0.47	3.41±0.57	0.04±0.003	4.08±0.34	1.59±0.06	0.15±0.08
11.8 ±0.11	6.09±0.55	3.87±0.64	0.04±0.01	4.85±0.36	1.87±0.05	0.19±0.08
9.54±0.44	8.61±0.74	4.76±0.91	0.06±0.01	5.18±0.30	1.91±0.16	0.22±0.12
6.79±0.72	11.56±0.83	5.63±0.99	0.06±0.02	5.91±0.60	2.00±0.24	0.25±0.13

of 20mM-xylitol to the perfusion medium) are shown in Table 4. The main changes were, as expected, a depletion of adenine nucleotides and P_i together with an accumulation of α-glycerophosphate. The content of ATP fell to 66% of the control value after 10min and was 32% of the control value after 80min. The loss of ATP was accompanied by a fall in the contents of ADP and AMP, the sum of the adenine nucleotides being 70% of the control value after 10min and 37% after 80min. In contrast to the findings after fructose loading (Woods *et al.*, 1970), the lost adenine nucleotides were not partly accounted for by the accumulation of IMP. There was a substantial fall in the P_i content, from 4.25 to 1.64μmol/g after 10min, and it then rose a little to 1.73μmol/g at 80min.

The content of α-glycerophosphate rose 37-fold at 10min to 4.74μmol/g and further to 8.02μmol/g at 40min. The contents of the other phosphorylated products fell but the changes were small in absolute terms. The content of xylulose 5-phosphate reached 0.18±0.01μmol/g at 80min.

The content of some non-phosphorylated intermediates (glucose, malate, citrate and glutamate) decreased at 10min, probably because they were washed out from the tissue. The contents of glucose and ammonia rose to near-normal values after 80min.

The [fructose 6-phosphate]/[glucose 6-phosphate] ratio was of the order expected for equilibrium, and the mass-action ratio of the adenylate kinase system ([ATP][AMP]/[ADP]^2) remained within the expected range at 10 and 40min in spite of the large changes in the contents of the individual adenine nucleotides. The [2-phosphoglycerate]/[3-phosphoglycerate] ratio was outside the order expected for equilibrium.

A depletion of adenine nucleotides and P_i together with α-glycerophosphate accumulation also occurred after loading with 10mM-xylitol but not with 5mM-xylitol (Table 5).

Effect of xylitol on the redox state of the hepatic [NAD$^+$]/[NADH] couple

Xylitol shifted the redox state of the couple in the cytoplasm in the direction of reduction without affecting that of the mitochondria (Table 6). The cytoplasmic [NAD$^+$]/[NADH] fell to about one-fifth of the normal value. Such an effect was reported by Jakob *et al.* (1971), but these authors also record a change in the mitochondria under similar conditions in rats starved for 24h. However, their initial mitochondrial redox state was abnormally low. The transient fall of the mitochondrial ratio shown in Table 6 is relatively small and of doubtful significance.

Discussion

The experiments demonstrate that xylitol, like fructose (Mäenpää *et al.*, 1968; Woods *et al.*, 1970) and glycerol (Burch *et al.*, 1970; Woods & Krebs, 1973), can cause a substantial loss of hepatic ATP, total adenine nucleotides and P_i, and a large accumulation of a phosphorylated intermediate. The concentration of ATP fell to 32%, that of total adenine nucleotide to 37%, that of P_i to 40%, and that of α-glycerophosphate rose up to 60-fold, compared with initial values. The formation of α-glycerophosphate was greater than the loss of the phosphate in the form of P_i, ATP and ADP, so that an uptake of phosphate by the liver from the medium must be postulated. Like fructose and glycerol, xylitol is rapidly metabolized by the liver (as already known), with glucose as the main end product. Unlike fructose, xylitol also causes a shift of the redox state of

Table 2. *Rates of xylitol uptake and glucose and lactate output by perfused livers from well-fed rats*

The rates were calculated from the results in Table 1 for consecutive 30min periods. Negative rates denote uptake by the liver. The rates are expressed as μmol/min per g wet wt. (means±S.E.M.).

Time period (min)	Rates of metabolic change (μmol/min per g wet wt.)											
	No added substrate		5mM-Xylitol			10mM-Xylitol			20mM-Xylitol			
	Glucose	Lactate	Xylitol	Glucose	Lactate	Xylitol	Glucose	Lactate	Xylitol	Glucose	Lactate	
0–30	2.16±0.22	0.73±0.07	−1.55±0.042	2.78±0.25	0.84±0.10	−1.84±0.038	2.12±0.20	1.00±0.25	−1.93±0.27	2.30±0.11	1.35±0.44	
30–60	0.88±0.15	0.37±0.04	−0.97±0.22	0.95±0.20	0.39±0.26	−1.24±0.22	1.63±0.39	0.32±0.17	−2.13±0.18	1.20±0.11	0.55±0.29	
60–90	0.26±0.13	0.02±0.08	−0.26±0.17	0.38±0.16	0.24±0.17	−0.95±0.36	1.22±0.09	0.29±0.03	−1.42±0.40	1.50±0.15	0.54±0.20	
90–120	0.58±0.28	0.18±0.09	−0.08±0.04	0.10±0.14	0.31±0.12	−0.40±0.11	0.37±0.28	0.01±0.05	−1.71±0.25	1.73±0.02	0.51±0.11	

the NAD couple in the direction of reduction (see also Jakob *et al.*, 1971).

The formation of glucose from xylitol, fructose or glycerol involves a consumption of ATP. The rate of the additional ATP consumption after loading the liver with the various precursors can be calculated on the somewhat simplifying assumptions that glucose, lactate and pyruvate are the only metabolic products, that the formation of glucose from fructose or glycerol or dihydroxyacetone requires 2 molecules of ATP/molecule of glucose, that the formation of glucose from xylitol requires 1.2 molecules of ATP/molecule of glucose, and that the formation of lactate or pyruvate from fructose, glycerol, dihydroxyacetone or xylitol yields 1 molecule of ATP/molecule of lactate or pyruvate. The formation of the phosphorylated intermediates and their accumulation in the liver may be neglected because these are small in relation to the other metabolic products. For the purpose of the calculations it is further assumed that all the glucose formed was derived from the added substrate. This was very nearly correct in the liver of starved rats. The error in the liver of fed rats was relatively small, as at least 80% of the glucose was formed from the added precursors. The results of the calculations, based on the rates in Tables 2 and 3, and those given by Woods (1970), Woods *et al.* (1970) and Woods & Krebs (1973), are shown in Table 7. The rates of additional ATP consumption are all of the same order of magnitude. There is no simple correlation between the extra ATP consumption and the loss of adenine nucleotides. Thus fructose, glycerol and xylitol cause a loss of adenine nucleotides whereas dihydroxyacetone does not. The loss is greatest with fructose, yet the rate of extra ATP consumption is higher at high xylitol concentrations and with dihydroxyacetone.

This lack of correlation becomes understandable when the rate of extra ATP consumption is compared with the total ATP turnover in the liver. The latter may be assessed from the O_2 consumption (about 2.5μmol/min per g; Woods *et al.*, 1970) and an assumed P/O ratio of 3. This gives a value of 15μmol of ATP synthesized (and utilized)/min per g. Thus the additional ATP consumption (although it removes the whole ATP content of the tissue in about 1min) is rather less than 20% of the basal turnover.

However, there is a correlation between the accumulation of phosphorylated intermediates and the depletion of P_i in the liver on the one hand, and the loss of adenine nucleotides on the other. Dihydroxyacetone or 5mM-xylitol (which in contrast to fructose, glycerol and 10 or 20mM-xylitol do not cause a depletion of adenine nucleotides) do not cause an accumulation of phosphorylated intermediates, though they cause an increased rate of ATP consumption comparable with that caused by fructose and glycerol. The fall of the concentration of P_i therefore must be taken as the main factor responsible for the depletion of the

Table 3. *Effect of xylitol concentration on the rate of glucose formation in perfused livers from starved rats*

Rats were starved for 48h. Xylitol was added after a preliminary perfusion for 38min and zero time was 2min later. By that time the concentration of xylitol was somewhat below the initial concentration. The rates were calculated from a plot of the metabolite concentrations in the medium against time and represent the initial rates. They are expressed as μmol/min per g (means±S.E.M.). For other experimental details see the Materials and Methods section. The number of experiments is shown in parentheses.

Initial xylitol concentration (mM)	Rates of			Ratio $\frac{\text{xylitol removed}}{\text{glucose formed}}$	
	Glucose formation	Lactate formation (as glucose equivalents)	Xylitol removal	Uncorrected	Corrected for lactate production
0	0.13±0.01 (3)	0.06±0.04 (3)	—	—	—
5	1.10±0.10 (3)	0.04±0.03 (3)	1.30±0.18 (3)	1.18	1.37
10	1.38±0.07 (4)	0.12±0.06 (4)	1.50±0.14 (4)	1.08	1.15
20	1.71±0.07 (3)	0.05±0.02 (3)	2.14±0.16 (3)	1.25	1.36

Table 4. *Contents of intermediary metabolites in the perfused rat liver*

Livers of fed rats were perfused with a medium containing xylitol (initial concentration 20mM) and were freeze-clamped at various times after the start of perfusion. The initial values refer to livers of fed rats freeze-clamped *in vivo* after cervical dislocation. The results are μmol/g (mean±S.E.M.) for three observations except for the adenine nucleotides, P_i, α-glycerophosphate, lactate and pyruvate, where there were four observations.

	Metabolite content (μmol/g)			
	Livers not perfused	Livers perfused with xylitol		
Time of perfusions (min) ...	0	10	40	80
ATP	2.22±0.07	1.47±0.12	0.92±0.12	0.70±0.09
ADP	0.79±0.08	0.66±0.05	0.47±0.03	0.48±0.01
AMP	0.26±0.05	0.18±0.02	0.21±0.013	0.12±0.01
Total adenine nucleotides	3.27±0.12	2.30±0.16	1.61±0.12	1.20±0.08
P_i	4.25±0.25	1.64±0.42	1.86±0.35	1.73±0.24
IMP	0.12±0.03	0.11±0.22	0.008±0.01	0.12±0.01
Xylulose 5-phosphate	<0.01	0.04±0.003	0.16±0.07	0.18±0.01
α-Glycerophosphate	0.13±0.10	4.74±1.24	8.02±1.76	6.99±1.19
Glucose	7.22±0.77	2.42±0.23	4.27±0.21	7.00±0.74
Lactate	0.79±0.09	0.81±0.14	1.29±0.19	1.57±0.24
Pyruvate	0.08±0.01	0.02±0.0004	0.017±0.0004	0.02±0.01
Fructose 1,6-diphosphate	0.02±0.002	0.01±0.002	0.01±0.002	0.01±0.002
Glucose 6-phosphate	0.25±0.03	0.10±0.02	0.14±0.01	0.16±0.02
Fructose 6-phosphate	0.06±0.01	0.02±0.01	0.04±0.01	0.04±0.004
Glucose 1-phosphate	<0.01	<0.01	<0.01	<0.01
Phosphoenolpyruvate	0.16±0.02	0.02±0.01	0.02±0.01	<0.01
2-Phosphoglycerate	0.04±0.01	0.02±0.001	0.02±0.002	0.03±0.004
3-Phosphoglycerate	0.26±0.06	0.05±0.01	0.06±0.002	0.04±0.002
Glyceraldehyde 3-phosphate	0.006±0.0002	0.003±0.0003	0.004±0.0006	0.004±0.0006
Dihydroxyacetone phosphate	0.04±0.003	0.02±0.0003	0.03±0.01	0.03±0.002
Ammonia	0.95±0.07	0.44±0.23	0.50±0.06	0.81±0.10
Glutamate	1.87±0.26	1.33±0.11	1.23±0.11	0.86±0.13
α-Oxoglutarate	0.11±0.003	0.11±0.01	0.08±0.03	0.06±0.01
Malate	0.73±0.12	0.01±0.02	0.07±0.02	<0.01

Table 4. (*Continued*)

Time of perfusions (min)	Metabolite content (μmol/g)			
	Livers not perfused	Livers perfused with xylitol		
	0	10	40	80
Citrate	0.45±0.06	0.27±0.01	0.22±0.03	0.11±0.01
[Lactate]/[pyruvate]	9.6±0.96	44.8±7.5	84.6±13.5	105±27.2
[Fructose 6-phosphate]/[glucose 6-phosphate]	0.25±0.02	0.24±0.09	0.30±0.08	0.30±0.05
[2-Phosphoglycerate]/[3-phosphoglycerate]	0.20±0.06	0.53±0.13	0.43±0.02	0.70±0.08
[α-Glycerophosphate]/[dihydroxyacetone phosphate]	3.55±0.40	305±82.1	349±64.5	276±59.4
[ATP][AMP]/[ADP]^2	0.97±0.17	0.53±0.11	0.99±0.27	0.36±0.06

Table 5. *Effect of xylitol on the metabolite contents of rat liver*

The experimental conditions were as described for Table 4 except for the xylitol concentrations. The tissue was freeze-clamped 40 min after the start of the perfusion. Control values for livers freeze-clamped immediately after cervical dislocation and values obtained with 20mM-xylitol are given in Table 4.

Initial xylitol concn.	Metabolite content (μmol/g wet wt.)	
Metabolite	5mM	10mM
ATP	2.16	1.48
ADP	0.47	0.66
AMP	0.16	0.21
Total adenine nucleotides	2.79	2.35
P_i	4.64	2.34
α-Glycerophosphate	0.86	3.59
Xylulose 5-phosphate	0.06	0.21

adenine nucleotides. It is known that a fall in the concentration of P_i (and ATP) de-inhibits the ATP-degrading enzymes and causes a formation of IMP and adenosine (see Woods *et al.*, 1970).

The high rates of xylitol utilization by the liver (already noted by previous investigators, see Ross *et al.*, 1967; Jacob *et al.*, 1971) are remarkable in view of the fact that xylitol is present in natural diets in mere traces, if at all, and that the rate of glucose formation in the course of the degradation of *myo*inositol and D-glucuronate is exceedingly low in rat liver (less than 0.2μmol/min per g; R. Hems, unpublished work). The high rates of xylitol utilization indicate that the enzymes converting xylitol into xylulose 5-phosphate, i.e. polyol dehydrogenase and D-xylulose kinase, as well as the enzymes of the pentose phosphate cycle, possess a high capacity in rat liver.

Relevance of the findings to therapeutic uses of xylitol

Intravenous infusion of xylitol has in recent years been recommended as a source of energy, especially during post-operative starvation periods (Baessler *et al.*, 1962; Spitz *et al.*, 1970; Horecker *et al.*, 1969). According to Schumer (1971) large intravenous doses of xylitol (4.8g/h per kg) can cause abdominal pain, nausea, vomiting and rise in the serum concentrations of lactate, uric acid, bilirubin, P_i, glutamate–oxaloacetate aminotransferase and alkaline phosphatase. The hyperuricaemia (which also occurs after fructose loading; Perheentupa & Raivio, 1967) is in all probability a direct result of the increased degradation of the hepatic adenine nucleotides. Since the intactness of the adenine nucleotide system is a key factor in cell function, it is likely that the other abnormalities are also consequences of the loss of the adenine nucleotides. Thus intravenous infusion of relatively large amounts of xylitol is obviously not without risk.

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Table 6. *Effect of xylitol on the redox state of the NAD couple in perfused rat livers*

The [NAD^+]/[NADH] ratios were calculated as described by Williamson *et al.* (1967) from the values in Table 4. The tissue pH was taken to be 7.0.

Material	[Free NAD^+]/[free NADH] ratio, calculated from: Lactate dehydrogenase (cytoplasm)	Glutamate dehydrogenase (mitochondrial matrix)
Freeze-clamped fed liver	910	13.7
Liver perfused with xylitol (initially 20mM)		
10min	225	9.6
40min	140	8.5
80min	114	14.7

Table 7. *Hepatic ATP consumption after substrate loading*

The rates of additional ATP consumption have been calculated as described in the text by using rates of metabolism derived from the results in Tables 2 and 4 and the following references: Woods (1970), Woods *et al.* (1970) and Woods & Krebs (1973).

Addition to perfusion medium	(mM)	Additional ATP consumption (μmol/min per g wet wt.): Well-fed animal	48h-starved animal
Fructose	10	1.66	2.03
Glycerol	10	2.36	1.24
Dihydroxyacetone	10	3.04	2.77
Xylitol	5	1.82	1.52
	10	2.08	1.68
	20	1.95	2.52

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