THE ORIGIN OF *l*-XYLOKETOSE (URINE PENTOSE)

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In a previous paper (1) we suggested a possible relationship between glucuronic acid and urine pentose.[1] In this communication it will be shown that *d*-glucuronic acid is the precursor of *l*-xyloketose. It was observed that certain of the drugs which are excreted as compound glucuronates produce an increased elimination of urine pentose. We attributed this increase to glucuronic acid formed in response to the administration of the drug rather than to the drug itself and accordingly fed glucuronic acid. A similar increase in pentose excretion resulted. These experiments establish *d*-glucuronic acid as the source of *l*-xyloketose.

Early in our investigation we were impressed by the remarkable uniformity of the daily output of pentose. This is illustrated in Table I which is typical of many other experiments with the same patients. The constant daily excretion was affected neither by altering the carbohydrate or protein content of the diet nor by periods of violent exercise followed by prolonged rest in bed (1). No attempt was made, therefore, to regulate the diet or restrict the activities of the patients in our further studies.

The effect of the feeding of various drugs on the excretion of urine pentose is shown in Table II. Following the ingestion of pyramidone the excretion of pentose is increased enormously and continues at a high level for several days after medication is stopped. Antipyrine is chemically allied to pyramidone and has a similar influence on pentose excretion; borneol and menthol, on the other hand, are members of a different class of drugs yet they also produce an increase in pentose.[2] There is little doubt

[1] We have identified *l*-xyloketose in thirty-eight different cases of pentosuria.

[2] Experiments with melubrin in the former group and thymol in the latter were without appreciable effect on pentose excretion.

that these drugs are excreted, both by the normal and by the pentosuric subject, in the form of glucuronates. As corroborative evidence that these four drugs are so excreted, we note that both normal and pentosuric urine give a strongly positive naphthoresorcinol test (3) after medication, indicating the presence of glucuronic acid or glucuronates.[3]

Glucuronic acid exists in the body, preformed, in mucin. According to Miller and Conner (7), glucuronic acid is liberated in

TABLE I

Constancy of Reducing Substances in Pentosuria

Subject	No. of consecutive days	Reducing substances (as xyloketose*)		
		Average per day	Average deviation	Maximum deviation
		gm.	*gm.*	*gm.*
H.B.	28	1.1	0.2	0.6
	7	1.2	0.2	0.4
	7	1.1	0.2	0.3
	7	1.2	0.1	0.2
A.G.	8	2.0	0.2	0.3
	5	2.0	0.2	0.2
	6	2.0	0.1	0.2
J. L.	26	2.9	0.2	0.7
	16	3.1	0.2	0.8
B.B.	9	3.1	0.3	0.5
	30	2.8	0.3	0.7
L.B.	6	3.5	0.2	0.2
	12	3.7	0.3	0.9

* In estimating xyloketose we have made use of Greenwald's (2) equivalent: 1 mg. of xyloketose = 1.22 mg. of glucose, by the Benedict method.

the digestion of mucin and is available for conjugation with toxic substances. We were particularly interested in feeding mucin,

[3] Enklewitz (unpublished work) has obtained a glucuronic acid compound from the urine after feeding pyramidone to human subjects and rabbits. Lawrow (4) showed that antipyrine administered to dogs is excreted in combination with glucuronic acid. Neuberg and Lachmann (5) and others fed menthol to rabbits as a means of procuring glucuronic acid, while Quick (6) prepared it from the urine of dogs after borneol feeding. We ourselves isolated glucuronic acid from normal and pentosuric urine following the ingestion of borneol.

TABLE II

Effect of Administration of Various Drugs on Excretion of Reducing Substances in Pentosuria

Period	Date	Total reducing substance (as xyloketose)		Xyloketose		Dosage
		Average per day	Increase	Average per day	Increase	
		gm.	gm.	gm.	gm.	
Subject L.B.						
Control	Dec. 26–30	3.8				
Pyramidone	" 31–Jan. 1	5.5	1.7			2 gm. daily
	Jan. 2–8	12.5	8.7			2 " "
	" 9–16	13.4	9.6			2 " "
Control	" 21–Feb. 2	3.7				
Pyramidone	Feb. 3–4	4.9	1.2			2 gm. daily
	" 5–10	12.5	8.8			2 " "
After	" 11	10.0	6.3			
	" 12–14	4.4	0.7			
Control	" 15–20	3.5				
Borneol	" 21–23	4.3	0.8			2.4 gm. daily
	" 25–26	5.2	1.7			4 gm. daily
Subject A.G.						
Control	Mar. 9–14	2.0				
Borneol	" 15	2.3	0.3			2.9 gm.
	" 16	2.7	0.7			3 gm.
	" 17	3.3	1.3			4 "
After	" 18–21	1.9				
Subject B.B.						
Gastric mucin	May 18–22	3.1	0.3	2.3	0.3	50 gm. daily, 3 days 25 gm. daily, 2 days
Control	May 23–30	2.8		2.0		
"	June 23–27	2.8		2.0		
Pyramidone	" 28–30	6.6	3.8	5.1	3.1	2 gm. daily
Control	July 1–2	3.0		2.0		
Borneol	July 8	3.8	0.8	2.6	0.6	2.5 gm.
	" 9	4.2	1.2	3.1	1.1	4 gm.
Control	Mar. 17–20	3.0				

TABLE II—*Concluded*

Period	Date	Total reducing substance (as xyloketose)		Xyloketose		Dosage
		Average per day	Increase	Average per day	Increase	
		gm.	*gm.*	*gm.*	*gm.*	
Subject B.B.—*Concluded*						
Antipyrine	Mar. 21	3.5	0.5	2.6	0.6	0.6 gm.
	" 22	4.0	1.0	3.2	1.2	1.3 "
	" 23–26	5.2	2.2	4.2	2.2	1.3 " daily
After	" 27	4.2	1.2	3.1	1.1	
	" 28–30	2.9		1.9		
Control	Apr. 3–7	3.1		2.0		
Borneol	" 8–10	4.6	1.5	3.4	1.4	4 gm. daily
	" 11	5.4	2.3	4.8	2.8	6.5 gm.
After	" 12	3.6	0.5	2.8	0.8	
	" 13–17	2.6		2.0		
Control	" 28–May 1	3.0		2.0		
Menthol	May 2	3.3	0.3			1 gm.
	" 3–5	4.7	1.7	3.3	1.3	4 " daily
After	" 6	3.4	0.4	2.2	0.2	
	" 7	3.0		2.0		
	" 8–11	3.0				

because it seemed possible that glucuronic acid contained in it might be the source from which xyloketose is derived, but in view of the large amount of mucin administered and the slight response in increased pentose excretion, this does not seem at all likely. Margolis (8) observed an increased excretion of reducing substances following the taking of pyramidone by the pentosuric but he could offer no satisfactory explanation for this. Greenwald proved the increase to be pentose by estimating it as the osazone and determining its optical activity. Our experiments with pyramidone (9) confirmed this finding. We also noted that the increased reducing substances reacted with Benedict's reagents at

room temperature,[4] a property exhibited only by *l*-xyloketose, of all the hexoses and pentoses we examined (9).[5]

The influence of pyramidone can be demonstrated in striking fashion by comparing the hourly rate of pentose excretion during

TABLE III

Effect of Administration of Pyramidone to Subject B.B. on Hourly Rate of Excretion of Reducing Substances in Pentosuria

	Time	Volume of urine	Total reducing substances per hr. (as xyloketose)	Xyloketose per hr.
		cc.	*gm.*	*gm.*
Control day	7.00 a.m.			
	11.30 "	135	0.08	0.05
	3.30 p.m.	237	0.14	0.10
	6.45 "	175	0.15	0.11
	7.00 a.m.	393	0.13	0.09
Pyramidone medication* 1st day	7.00 "			
	11.00 "	58	0.09	0.08
	3.00 p.m.	492	0.30	0.22
	7.00 "	112	0.29	0.22
	9.00 "	57	0.30	0.23
	7.00 a.m.	469	0.23	0.17
2nd day	7.00 "			
	9.35 "	210	0.21	0.15
	11.00 "	121	0.23	0.19
	3.00 p.m.	158	0.32	0.25
	7.00 "	105	0.34	0.27
	9.10 "	128	0.50	0.36
	7.00 a.m.	488	0.27	0.20
3rd day	7.00 "			
	11.00 "	255	0.22	0.16
	3.00 p.m.	70	0.33	0.26
	7.00 "	70	0.36	0.28
	7.00 a.m.	407	0.29	0.22

This table comprises 4 consecutive days.

* 2 gm. of pyramidone were administered daily at 9.00 a.m.

the medication period with that of a control day. Within 6 hours after the taking of pyramidone, pentose elimination is increased

[4] It is improbable that the increase in reduction is due to free glucuronic acid, since glucuronic acid does not reduce Benedict's reagents at room temperature.

[5] Schmidt and Treiber (10) have recently prepared the enantiomorph, *d*-xyloketose, from *d*-xylose and shown that it also reduces in the cold.

to 0.22 gm. per hour; on the 2nd day it reaches 4 times the average normal output and continues at a very high level throughout the medication period (Table III).

It would appear that the increased pentose excretion obtained from the feeding of the drugs, pyramidone, antipyrine, borneol, and menthol, could be attributed to glucuronic acid which is formed in response to these drugs.[6] On this assumption one would expect

TABLE IV

Effect of Administration of Glucuronic Acid on Excretion of Reducing Substances in Pentosuria

	Date	No. of days	Total reducing substances		Xyloketose	
			Average per day	Increase	Average per day	Increase
			gm.	*gm.*	*gm.*	*gm.*
Subject B.B.						
Fore period	July 29–Aug. 5	8	2.9		1.9	
Glucuronic acid (5-gm.)	Aug. 6	1	4.1	1.2	2.7	0.8
After period	" 7	1	3.6		2.2	
	" 8	1	2.7		1.9	
Fore period	Oct. 18–23	6	3.3		1.9	
Glucuronic acid (10 gm.)	" 24	1	5.9	2.6	4.1	2.2
After period	" 25–27	3	3.4		2.2	
Subject M.C.						
Glucuronic acid (5 gm.)	Nov. 5	1	4.3	1.3	3.0	1.0
Control day	" 10	1	3.0		2.0	
Subject J.L.*						
Glucuronic acid (5 gm.)	Nov. 6	1	3.6	2.1	2.3	1.4
Control day	Dec. 26	1	1.5		0.9	

* Urine was collected for 12 hours only, from 7.00 a.m. to 7.00 p.m.

the administration of glucuronic acid itself to produce an increased elimination of urine pentose. We therefore fed glucuronic acid[7] (Table IV).

[6] In order to learn whether glucuronic acid, in conjugated form, may be the source of xyloketose, we fed 5 gm. of borneol glucuronic acid to subject B.B. and found that it had no influence on the output of pentose. In 16 hours, 2.5 gm. of the borneol glucuronic acid were recovered from the urine as the zinc salt.

[7] Glucuronic acid was prepared from the urine of human subjects after

The marked increase in xyloketose establishes *d*-glucuronic acid as the precursor of urine pentose. In the three cases the increase of 0.8, 1.0, and 1.4 gm. follows the administration of 5 gm. of glucuronic acid. In subject B.B. the ingestion of 10 gm. in two equal doses produces more than twice the increase of xyloketose obtained from a single dose of 5 gm.

The effect of glucuronic acid feeding[8] is brought out very clearly in Table V, which shows again the hourly rate of pentose excretion during control and medication periods. During the control periods pentose excretion proceeds at a fairly uniform rate. Immediately following the ingestion of glucuronic acid the output of pentose increases about 4-fold and remains at a very high level for the greater part of the day.

Xyloketose was estimated by the method already reported by us (9); that is, by determining the amount of reduction of Benedict's quantitative sugar reagent at 55°. This method has received recent support from Exton, Rose, and Roehl (11) who demonstrate its value in the identification of reducing sugars, using their modification of Sumner's reagent in place of Benedict's.

Our chief concern, of course, is to establish the identity of the increased reducing substances in the possible presence of glucuronic acid, which might easily have been excreted unchanged.[9] Although glucuronic acid gives many of the same reactions as the pentoses, we found that it did not interfere with the estimation of xyloketose when added to the urine even in such large amounts as 0.6 per cent of glucuronic acid in 0.2 per cent xyloketose.

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borneol administration (5 gm. daily over periods of 3 and 4 days). We applied the method used by Quick (6) for dog urine. It was isolated and used in the form of the lactone.

[8] As a control experiment 5 gm. of glucuronic acid were fed to a normal person. Tests made on the urine showed no pentose, but bear evidence that some glucuronic acid was excreted unchanged. The urine, within 8 hours, contained reducing substances equivalent to 0.8 gm. of glucose but none thereafter. There was no reduction of Benedict's reagent at 55°; a naphthoresorcinol test on the urine was strongly positive. Unlike the pentosuric urine it formed, with phenylhydrazine, a dark brown sticky substance, somewhat resembling glucosazone microscopically, insoluble in alcohol, and giving a heavy naphthoresorcinol test.

[9] A naphthoresorcinol test indicated that glucuronic acid, or glucuronate, was present in some, but not in all, of the urine specimens of the experimental periods.

TABLE V

Effect of Administration of Glucuronic Acid on Hourly Rate of Excretion of Reducing Substances in Pentosuria

	Time	Volume of urine	Total reducing substances per hr. (as xyloketose)	Xyloketose per hr.	Remarks
		cc.	*gm.*	*gm.*	
Subject B.B.					
Control day	6.45 a.m.				
	10.00 "	118	0.13	0.08	June 16
	12.40 p.m.	83	0.09	0.06	
	2.10 "	272	*	*	
	4.50 "	197	0.13	0.09	
	6.50 "	200	0.14	0.09	
	8.40 "	192	0.14	0.08	
	6.45 a.m.	455	0.12	0.09	
	7.00 a.m.				
	10.45 "	87	0.10	0.06	Oct. 23
Glucuronic	1.30 p.m.	485	0.44	0.29	5 gm. at 10.50 a.m.
acid	3.00 "	280	0.35	0.23	5 " " 3.00 p.m.
	4.45 "	108	0.51	0.34	
	6.15 "	70	0.45	0.36	
	9.15 "	160	0.30	0.22	
	7.00 a.m.	237	0.14	0.10	
Subject M.C.					
Control day	7.00 a.m.				
	10.30 "	260	0.12	0.07	Dec. 9
	2.30 p.m.	300	0.12	0.07	
	7.10 "	324	0.10	0.06	
	12.40 a.m.	235	0.13	0.08	
	7.00 a.m.	330	0.16	0.10	
	6.30 a.m.				
	10.20 "	204	0.07	0.05	Nov. 4
Glucuronic	12.40 p.m.	285	0.37	0.28	5 gm. at 11.00 a.m.
acid	2.15 "	65	0.35	0.27	
	3.20 "	39	0.30	0.22	
	4.55 "	70	0.26	0.19	
	5.50 "	310	0.16	0.10	
	7.30 "	121	0.14	0.09	
	1.10 a.m.	257	0.15	0.10	
	9.30 "	245	0.11	0.07	

* The method used was not sufficiently delicate to determine the amount of reducing substances in this very dilute urine.

TABLE V—*Concluded*

	Time	Volume of urine	Total reducing substances per hr. (as xyloketose)	Xyloketose per hr.	Remarks
		cc.	*gm.*	*gm.*	
Subject J.L.†					
Control day	7.00 a.m.				
	12.00 noon	455	0.10		May 29
	2.30 p.m.	385	0.10		
	5.00 "	255	0.19		
	7.00 "	80	0.14		
	7.00 a.m.				
	9.00 "	90	0.12	0.07	Nov. 5
	10.30 "	85	0.12	0.07	
Glucuronic acid	12.00 noon	150	0.35	0.26	5 gm. at 10.55 a.m.
	4.30 p.m.	218	0.41	0.27	
	7.00 "	145	0.32	0.20	

† Urine was collected over a 12 hour period.

The following experiment corroborates our finding that the xyloketose content of urine is much greater after glucuronic acid feeding. Urine of any given pentose content, at a fixed temperature, requires the same length of time for the onset of reduction whether glucuronic acid is present or not; the greater the content of xyloketose the shorter the time required. At 40° a urine with a xyloketose content of 0.3 per cent takes 20 minutes for the onset of reduction; after the addition of 0.6 per cent glucuronic acid it still takes 18 minutes. Pentosuric urine, to which xyloketose has been added to a total of 0.55 per cent, takes 5 minutes. Specimens after glucuronic acid medication (B.B., M.C., and J.L.), calculated by reduction to contain 0.55 per cent, require only 6 minutes.

Formation and Yield of Phenylosazone—Since xyloketose cannot be easily crystallized, we did not attempt to isolate it from the post-glucuronic acid urine; instead we relied on the formation of the pentosazone for identification.[10,11] From ten of the post-glu-

[10] It is known that ketoses are unaffected by bromine. Some authors (2, 12) have applied this fact in identifying xyloketose. A specimen of urine from the experimental period was shaken with an excess of bromine

curonic acid specimens of urine of the three patients a phenylhydrazine compound was formed, which had the same properties as the osazone obtained from control pentosuric urine, but was produced in greater quantities and gave better yields and melting points. In all ten of these the yield of osazone was greater than

TABLE VI

Influence of Glucuronic Acid Feeding on Yield of Osazone

Subject		Volume of urine	Xyloketose by reduction	Phenylosazone	Yield of osazone	Melting point
		Control				
		cc.	*gm.*	*gm.*	*per cent*	*°C.*
B.B.	4 hr. period	126	0.24	0.28	52	149
	Recrystallized from alcohol			0.18	33	157–158
	Extracted with chloroform			0.14*	26	157–159
		Experimental				
J.L.	4 hr. period	201	1.10	2.30	95	149–150
	Recrystallized from alcohol			1.81	75	156–159
	Extracted with chloroform			1.39†	58	161–163
M.C.	1 hr. period	40	0.22	0.49	85	152–154
	Extracted with chloroform			0.36	62	160–164
	Twice recrystallized and re-extracted with chloroform			0.17‡	30	163

* See osazones in control Period I of protocols given in the section "Optical activity" below.

† See osazones in experimental Period I of protocols given in the section "Optical activity."

‡ See osazones in experimental Period II of protocols given in the section "Optical activity."

that of any control urine pentosazone we have ever prepared. The yield of recrystallized osazone in every case was also greater after

and left in contact with it for 30 days. Before bromination it contained 0.18 per cent xyloketose; after bromination there still remained the same amount as measured by the reduction at 55°.

[11] Polariscopic readings of the post-glucuronic acid urine cannot be used in identifying the additional reducing substances, for the glucuronic acid has a specific rotation similar to that of *l*-xyloketose.

glucuronic acid feeding than in control periods. Typical experiments are shown in Table VI.

Method Used in Preparing Osazone—Recrystallized phenylhydrazine and sodium acetate were added directly to the urine in the proportion 4 gm.:6 gm.:1 gm. of total reducing bodies. It was heated for $1\frac{1}{4}$ hours in a boiling water bath, then cooled in air. In urine which contained a large amount of xyloketose a precipitate began to form in the hot solution. We filtered off the precipitated osazone as soon as the solution was cold, in this way producing a much purer product than if allowed to stand until the next day as in Greenwald's method of preparation (8). The osazone was washed with water, dried with suction, and later over phosphorus pentoxide in the desiccator. It was recrystallized from 35 per cent alcohol and, when dry, extracted with chloroform. Sometimes repeated recrystallizations were needed before it attained a melting point of 162°.

The phenylhydrazine compound was identified as *l*-xylosazone in the following ways: (1) by its gross and microscopic appearance and by its solubility, (2) by its melting point, (3) the formation of *dl*-xylosazone, (4) the optical activity, (5) by the nitrogen content.

Physical Properties—The osazones of the experimental period precipitated out suddenly from the urine in a jelly-like mass which was composed of long, yellow, needle-shaped crystals scattered irregularly, typical of a pentosazone. A similar precipitation occurred with the osazone from the control urine. The crystals were soluble in hot water and in alcohol and acetone.

Melting Point—The melting point was about 149–153° for the crude material and ranged from 160–164° on purification. This agrees with the melting point of urine pentosazone found by other investigators.[12] The melting point remained the same when the osazone was mixed with *l*-xylosazone formed from the urine of a control unmedicated period.

dl-Xylosazone—According to Levene and La Forge (12), "a dextrorotatory pentosazone, which shows an elevation of its melting

[12] Elliott and Raper (13) state 148–150° after recrystallization from 20 per cent alcohol and 163–164° after further purification. Levene and La Forge (12) report 160° for urine pentosazone (164° for *d*-xylosazone). Hiller (14) gives 159°; Greenwald (2) 157–160°, 156–157°, 161–163°, and 156–158°; Hari (15) 161–164°.

point after being mixed with a levorotatory xylosazone from 163° to 205°C., has to be regarded as xylosazone."

The melting point of the osazone from the experimental period was elevated from 163° to 204° when mixed with an equal quantity of *d*-xylosazone (from *d*-xylose). 0.1 gm. of material (see † in Table VI), after being mixed with 0.100 gm. of *d*-xylosazone and recrystallized from alcohol, produced 0.175 gm. of a substance which differed in solubility and general appearance as well as in crystalline structure and melting point from both the dextro and levo variety. Short boat-shaped crystals were formed, which melted at 200–204° and were identical with *dl*-xylosazone prepared from a normal period.

Optical Activity—Levene and La Forge (12) state that the xylosazone nature of the urine pentosazone is further substantiated by the character of its mutarotation. They write, "the initial optical rotation of xylosazone is lower than the equilibrium rotation. . . The optical rotation of the urine osazone increases in magnitude on standing."

We determined the optical activity of the purified osazones of the experimental period and of the control period. All specimens showed an initial dextrorotation and an increased dextro activity on standing. A comparison with the rotation observed by other investigators shows that the character of the mutarotation is the same in each case.

Osazones, Experimental Period I—Read in a 1 dm. tube[13] after 25 minutes $[\alpha]_D = +0.09°$; after 24 hours, +0.23°; after 48 hours, +0.23°.

Osazones, Experimental Period II—Read in a 0.5 dm. tube after 10 minutes $[\alpha]_D = +0.06°$; after 24 hours, +0.15°; after 48 hours, +0.17°.

Osazones, Control Period I—Read in a 1 dm. tube after 10 minutes $[\alpha]_D = +0.12°$; after 24 hours, +0.30°; after 46 hours, +0.31°.

Osazones, Control Period II—Subject B.B. Read in a 1 dm. tube after 10 minutes $[\alpha]_D = +0.10°$; after 24 hours, +0.35°; after 48 hours, +0.35°.

Osazones, Levene and La Forge (12)—Read in a 0.5 dm. tube.

Period I, l-Xylosazone—Soon after preparation of the solution $[\alpha]_D = +0.15°$; after 8 hours, +0.47°.

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[13] Our osazones were dissolved in the usual mixture of 4 cc. of pyridine and 6 cc. of methyl alcohol at half the concentration used by all the other investigators.

Period II, l-Xylosazone—Soon after preparation of the solution $[\alpha]_D$ = +0.27°;[14] after 18 hours, +0.57°.[14]

Period III, d-Xylosazone—Soon after preparation of the solution $[\alpha]_D$ = −0.10°; after 18 hours, −0.36°.

Period IV, d-Xylosazone—Soon after preparation of the solution $[\alpha]_D$ = −0.09°; after 24 hours, −0.43°.

l-Xylosazone, Greenwald (2).

Period I—Read in a 1 dm. tube, initial rotation $[\alpha]_D$ = +0.25°; after 48 hours, +0.60°.

Period II—After pyramidone (8), initial rotation $[\alpha]_D$ = +0.20°; after 24 hours, +0.40°.

l-Xylosazone, Hari (15)—Read in a 1 dm. tube.

Period I-Initial rotation $[\alpha]_D$ = +0.15°; read the next morning, +0.72°.

Period II—Initial rotation $[\alpha]_D$ = +0.30°; read the next morning, +0.63°.

l-Xylosazone, Bálint (16)—Read in a 0.5 dm. tube. Initial rotation $[\alpha]_D$ = +0.16°;[15] after 24 hours, +0.35°.[15]

Analysis for Nitrogen—A specimen of purified osazone from the experimental period (see † in Table VI) was analyzed by Dr. H. K. Alber, of New York University. Using the Dumas method in the micromodification by Pregl, he reported as follows: weight of sample = 2.661 mg.; net volume of nitrogen = 0.394 cc.; temperature, 27°; pressure, 755 mm.; percentage of nitrogen, 16.73 (calculated, 17.07).[16]

SUMMARY

It has been shown that the administration of glucuronic acid causes a greatly increased elimination of pentose in the urine; that this increase is *l*-xyloketose is established by the reduction at 55°, and by the formation of the calculated amount of osazone. The identity of the osazone has been determined by its melting point, by the elevation of its melting point when mixed with *d*-xylosazone to form *dl*-xylosazone, by optical activity and nitrogen content, and the yield shown to be greatly in excess of the amount obtained from a control period.

[14] It is noted that the rotations reported by Levene and La Forge (12) for a 0.5 dm. tube are those found by others in a 1 dm. tube.

[15] Average rotation of osazones from four patients. The maximum deviation was 0.01°.

[16] Mayer (17) prepared a compound from glucuronic acid and phenylhydrazine which melted at 159–164° and had a nitrogen content of 11 per cent; also another compound melting at 199–205° contained 15.5 per cent nitrogen.

In spite of the fact that it is difficult, on theoretical grounds, to see how *d*-glucuronic acid can be converted into *l*-xyloketose, the conclusion to be drawn from this work is that *l*-xyloketose arises from *d*-glucuronic acid.

BIBLIOGRAPHY

1. Enklewitz, M., and Lasker, M., *Am. J. Med. Sc.*, **186**, 539 (1933).
2. Greenwald, I., *J. Biol. Chem.*, **88**, 1 (1930).
3. Tollens, B., *Ber. chem. Ges.*, **41**, 1788 (1908).
4. Lawrow, D., *Ber. chem. Ges.*, **33**, 2344 (1900).
5. Neuberg, C., and Lachmann, S., *Biochem. Z.*, **24**, 416 (1910).
6. Quick, A. J., *J. Biol. Chem.*, **69**, 549 (1926).
7. Miller, C. O., and Conner, J. A., *Proc. Soc. Exp. Biol. and Med.*, **30**, 630 (1932–33).
8. Margolis, J. I., *Am. J. Med. Sc.*, **177**, 348 (1929).
9. Lasker, M., and Enklewitz, M., *J. Biol. Chem.*, **101**, 289 (1933).
10. Schmidt, O. T., and Treiber, R., *Ber. chem. Ges.*, **66**, 1765 (1933).
11. Exton, W. G., Rose, A. R., and Roehl, E. J., *Proc. Soc. Exp. Biol. and Med.*, **32**, 417 (1934).
12. Levene, P. A., and La Forge, F. B., *J. Biol. Chem.*, **18**, 319 (1914).
13. Elliott, J. H., and Raper, H. S., *J. Biol. Chem.*, **11**, 211 (1912).
14. Hiller, A., *J. Biol. Chem.*, **30**, 129 (1917).
15. Hari, P., *Biochem. Z.*, **224**, 474 (1930).
16. Bálint, P., *Biochem. Z.*, **274**, 305 (1934).
17. Mayer, P., *Z. physiol. Chem.*, **29**, 59 (1899–1900).

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