GLUCOSE AS A PRECURSOR OF MILK CONSTITUENTS IN THE INTACT DAIRY COW*

by

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Earlier research on the role of glucose in the synthesis of milk has been reviewed in 1949 by FOLLEY[1]. The uptake of glucose by the mammary gland has been demonstrated repeatedly by the measurement of arterio-venous difference in glucose concentration[2]. This technique naturally does not produce information on the question how much of the absorbed glucose enters organic milk constituents and how much is catabolized to CO_2. FOLLEY AND FRENCH[3] studied the effect of glucose on the respiratory quotient of mammary gland slices and observed a rise of this quotient above unity suggesting milk fat synthesis from carbohydrate in mammary slices from non-ruminants but no comparable effect in slices from ruminants.

BALMAIN, FOLLEY AND GLASCOCK[4] used tritium, ^{13}C, and ^{14}C in *in vitro* studies of lipogenesis by mammary gland slices. They noted that rat slices incorporated about 8 times more glucose carbon and 6 times less acetate carbon into fatty acids of milk fat than did mammary gland slices from sheep.

DIMANT, SMITH AND LARDY[5] perfused an amputated cow's udder with blood containing 1-^{14}C glucose and observed nearly equal amounts of radioactivity in the glucose and galactose moieties of the lactose. This result was consistent with that of BARRY[6] who injected 1-^{14}C labeled glucose into an intact goat and concluded that glucose as well as galactose moiety of the lactose were derived, at least in part, from blood glucose. REISS AND BARRY[7] later injected glucose uniformly labeled with ^{14}C into an intact goat. They noted that the specific activity of ^{14}C in the lactose followed closely the corresponding specific activity in the glucose of the blood with about 1 hour's delay. This observation led to the conclusion that blood glucose is the principal source of glucose as well as galactose moieties in lactose.

The present paper deals with the carbon transfer from glucose to respiratory CO_2 and to lactose, casein, milk fat, and citric acid in the intact dairy cow measured by intravenous injection of glucose uniformly labeled with ^{14}C, and subsequent determination of the ^{14}C levels in these substances and in blood glucose and blood acetate.

* This investigation was supported by a contract with the U.S. Atomic Energy Commission.

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Glucose is present in cows' blood at lower concentration than in blood of non-ruminants and is derived from fatty acids produced by rumen micro-organisms fermenting organic constituents of the feed. Yet glucose is a more effective general source of carbon in the components of milk than the fatty acids are. It contributed 80% of the C in lactose and in addition about 6% of the C in casein and in butterfat. Over 2% of the ^{14}C injected in glucose appeared in the organic constituents per kg of milk, which is twice as much as the ^{14}C transfer from 2-^{14}C acetate, and amounted to a recovery in the milk of over 50% of the ^{14}C injected.

EXPERIMENTAL

Cows. Two 6 year-old Jersey cows were used. The cow for Trial I was in her fourth month of lactation. She weighed 457 kg and produced 10 kg of milk per day. Her plasma volume, measured with Dye-T-1824 (Evan's Blue)[8] amounted to 20 liters. The initial glucose level in the plasma was 60 mg%. The cow for Trial II was used three months after calving. She weighed 526 kg and produced daily 15 kg of milk. Her plasma volume was also 20 liters. Her plasma contained 61 mg% glucose at the start and 45 mg% at the end of the trial.

Tracer. The uniformly labeled glucose was prepared by exposing a Canna leaf to $^{14}CO_2$ and then isolating the glucose according to the method of PUTMAN AND HASSID[9]. Degradation of glucose prepared by this method has shown uniform labeling[10].

Injection and dose. A total of 2.9 mc of ^{14}C in 1 gram of glucose was injected into cow I and 1.45 mc in 0.85 grams of glucose into cow II. The relative injected dose thus amounted to 6.27 μc/kg body weight in Trial I and 2.76 μc/kg in Trial II.

The injection of the labeled material, dissolved in about 30 ml of sterile saline solution, was performed through plastic tubes that had been inserted into the jugular veins of the cows on the day before the trial by the method previously described[11]. The injected glucose amounted to less than 10% of the total plasma glucose.

Analyses. A description of the respiration apparatus used and the technique followed for measuring the CO_2 production of the cows, and the ^{14}C activity in the expired CO_2, is found in an earlier publication[12].

The ^{14}C in the non-fat organic compounds lactose, albumin, casein, citric acid, and glucose was measured in carbonate resulting from dry combustion of these compounds in a semi micro combustion oven. The method of PREGL AND GRANT[13] was modified by GABOUREL[14] to avoid ^{14}C cross contamination between samples.

Milk fat combustion was carried out by wet combustion with periodate[15].

The acetate prepared out of blood plasma by steam distillation[16] was transferred to a small volume of 5% butanol in chloroform by the method of ELSDEN[17] and then chromatogrammed on silica gel according to MARVEL AND RANDS[18]. The fraction containing the acetate was extracted with 0.1 *N* NaOH. The organic solvents were boiled off from the extract and then the acetate was oxidized with potassium persulfate according to CALVIN *et al.*[15]. The isolation of plasma glucose is described by BAXTER *et al.*[19].

Citric acid was prepared out of the milk by LUCAS, HIRAHARA AND KANEKO[20] who adapted to milk a chromatographic method described by BUSH, HURBERT AND POTTER for preparing citric acid out of liver homogenates[21].

Unit for expressing specific radioactivity. As in our earlier publications we express the tracer concentration in standard specific activity given in microcuries per gram atom C in the various substances, per unit of the relative injected dose, which latter is expressed as microcuries injected per kg body weight.

To simplify the discussion we shall call the quotient $\frac{\mu c \text{ per gramatom C in substance}}{\mu c \text{ injected per kg body weight}} = \text{s.u.}$, the number of "standard units".

RESULTS AND DISCUSSION

The standard specific activity of the respiratory CO_2 shows good agreement between the two trials. It reached a maximum of about 9 s.u., between 30 and 40 minutes after injection of the uniformly labeled glucose. The results for the first 3 hours after injection are shown on Fig. 1.

This maximum activity in respiratory CO_2 after glucose injection is lower, and

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occurs later, than the corresponding maxima observed after injection of ^{14}C labeled acetate, propionate, or butyrate[22,23,21].

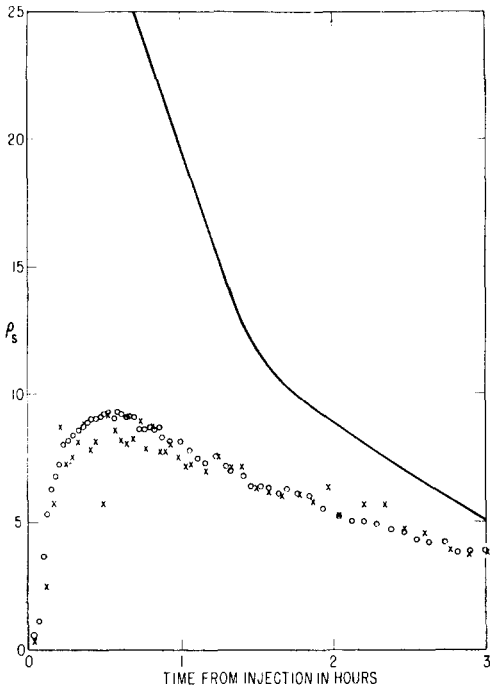

Fig. 1. ^{14}C in respiratory CO_2 after intravenous injection of uniformly labeled glucose and carbonate. ϱ_s in $\frac{\mu c/\text{mole } CO_2}{\mu c \text{ inject./kg body wt}}$ — carbonate; O glucose trial I; × glucose trial II.

TABLE I

PERCENT OF INJECTED DOSE EXCRETED AS $^{14}CO_2$

Time from injection hours (1)	*^{14}C-labeled material injected*		
	*Carbonate** (2)	*Acetate*** (3)	*Glucose I* (4)
1	58	17	9
2	72	30	17
3	78	38	23

* Carbonate Trial IV described before[22] but without the percent excretion.

** Mean values of individual results obtained with 1-^{14}C and 2-^{14}C acetate[19].

Table I compares the amount of ^{14}C expired as $^{14}CO_2$ during the first three hours after injection of uniformly labeled glucose, acetate[22] and carbonate[25]. Carbon from glucose goes to CO_2 at a slower rate than carbon from any other compound tested in our laboratory; that includes, besides acetate and carbonate mentioned in Table I, the other fatty acids from formate to caproate. It appears as if the cow were very generous in providing glucose for milk formation but very reluctant in using glucose as a fuel, or, looking at the situation from the point of view of competition between organs, the mammary gland seems to be very efficient in "catching" glucose from the blood stream and incorporating it in milk constituents, a view expressed before by FRENCH (POPJAK[26]).

During the first 3 hours after injection 1.6 times as much ^{14}C was recovered in the organic components of milk as was expired in CO_2. These results with glucose differ markedly from those with the fatty acids from which in each case more ^{14}C was given off as respiratory CO_2 during the first 3 hours after injection than was transferred to the milk constituents.

In glucose Trial II, unfortunately, we lost the information on total ^{14}C excretion. An impossible result (150% recovery of ^{14}C) led to the discovery of an abnormal measurement of the ventilation rate, presumably the result of a leak in the air line after the absorber for CO_2.

Table II shows the appearance of the tracer in milk. Of the ^{14}C injected as glucose in Trial I, 56% was transferred to organic compounds in the milk in two days, (2.7% per kg of milk) and 40% appeared in respiratory CO_2; that left only 4% that might have been incorporated in body constituents with a low turnover rate. Of the ^{14}C transferred to milk components in two days 83% went to lactose, 11% to milk fat, 4% to casein and 2% to other compounds, mainly albumin.

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TABLE II

^{14}C IN MILK IN % OF ^{14}C INJECTED IN GLUCOSE

Trial	Time from injection h	Milk produced Total kg	^{14}C in milk in % of injected ^{14}C					
			Lactose %	Milk fat %	Casein %	Albumin %	Citric acid %	Total %
I	3.5	1.6	34.2	2.0	1.4	0.4	—	38.0
	10.0	4.4	44.0	4.9	2.1	0.7	—	51.7
	22.2	9.9	46.1	6.0	2.3	0.8	—	55.2
	35.0	15.7	46.5	6.3	2.4	0.8	—	56.0
	46.0	20.6	46.6	6.4	2.5	0.8	—	56.3
II	3.3	2.0	27.5	0.6	1.6	0.4	0.2	30.3
	10.8	6.6	48.2	5.6	3.0	1.0	0.4	58.2
	23.1	13.8	53.7	7.7	3.5	1.3	0.5	66.7
	38.4	20.9	55.0	9.1	3.6	1.3	0.5	69.5
	47.2	27.9	55.4	9.3	3.8	1.4	0.5	70.4

In Trial II 70% of the injected ^{14}C appeared in the organic milk constituents during two days. This transfer amounted to 2.5% of the injected dose per kg of milk. Of this ^{14}C in milk, 79% was found in lactose, 13% in milk fat, 5% in casein, 2% in albumin, and 0.7% in citric acid. The amino acids of the labeled casein were isolated for a study of the role of glucose in the synthesis of amino acids[27].

TABLE III

SPECIFIC ^{14}C ACTIVITY IN MILK CONSTITUENTS

Period	Time from injection end of period hours		λs, in s.u. = $\frac{\mu c \text{ per gram atom C in constituent}}{\mu c \text{ injected per kg body weight}}$						
			Lactose		Casein		Milk fat		Citric acid
	Trial I	Trial II	I	II	I	II	I	II	II
1	3.5	3.4	59.76	43.23	2.80	2.84	1.52	0.39	9.41
2	10	11	9.62	13.95	0.70	1.10	1.24	1.42	3.02
3	22	23	1.01	2.38	0.13	0.25	0.24	0.39	0.85
4	35	35	0.18	0.58	0.05	0.07	0.07	0.27	0.20
5	46	47	0.08	0.18	0.03	0.05	0.03	0.04	0.26
6	58	59	0.05	0.15	0.03	0.03	0.02	—	0.12
7	70	72	0.07	0.07	0.04	0.01	0.02	—	0.24

Table III shows the specific ^{14}C activity in the major constituent of the milk as a function of the time from injection. The standardized specific activity in the 3 hour lactose sample, 60 and 43 s.u., from Trial I and II is 10 and 8 times respectively as high as that of the "hottest" milk constituent resulting from our previous trials with fatty acids, namely lactose after injection of 2-^{14}C labeled propionate[23]. The specific activity in all milk components falls off rapidly after the first or, for fat, after the second sample. This fast decrease indicates rapid utilization of glucose.

The specific activity in milk fat, 1.5 s.u. in the first and 1.4 s.u. in the second sample for Trials I and II respectively, is lower than the corresponding specific activity in milk fat after injection of labeled acetate[22] which in the second (10 hour) sample averaged 2.4 s.u. That acetate would be more lipogenic than glucose could be expected

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but we were surprised to note that glucose labeled milk fat more highly than did 1 or 2 labeled butyrate[21], which led to a specific activity of butterfat in the 2nd (10 hour) milk sample of only 0.3 to 0.6 s.u. The rather effective labeling of butterfat by ^{14}C labeled glucose in our cow differs from the observations of PETERSEN[28] and those of DIMANT, SMITH AND LARDY[29] who injected uniformly labeled and 1-^{14}C labeled glucose respectively into amputated perfused udders of cows and detected only insignificant amounts of the tracer in the milk fat. We have at present not enough data for an explanation of this difference in the behavior of the intact cow and the perfused isolated udder. Possibly the conversion of carbohydrate carbon to fat carbon in the intact cow occurs outside the udder, but it is also possible that the isolated udder has lost a metabolic ability which normally operates in the udder of the intact cow.

As in previous trials with other metabolites[22,23,24] we may estimate the relative importance of carbonate fixation in the transfer of carbon glucose to the constituents of the milk.

Table IV shows this calculation and its result for lactose formation. Less than 1% of the carbon transferred from glucose to lactose goes *via* the plasma carbonate pool. The analogous calculation for the transfer to milk fat indicates also a minor role of carbonate in the C transfer from glucose to milk fat. This result is, however, unreliable because the carbonate transfer quotient for milk fat formation is inaccurate[25]. In the transfer of carbon from glucose to casein, the plasma carbonate pool is more important, about 10% (Trial I) and 4% (Trial II) of the carbon appearing in casein from glucose came *via* carbonate.

TABLE IV

^{14}C TRANSFER TO LACTOSE VIA CO_2 IN PERCENT OF TOTAL ^{14}C IN LACTOSE

(Calculated without delay)

		Total ^{14}C transf. to lactose				*via CO_2 to lactose*			$\frac{\sum_0^t \lambda_S \Delta t}{\int_0^t q\, \varrho_S dt}$
Trial and period	*Time at end of period hours*	λ_S	Δt *in period minutes*	$\lambda_S \Delta t$ *in period*	$\sum_0^t \lambda_S \Delta t$	$\int_0^t q\, \varrho_S dt$ *minutes*	q *(from carbonate trials)*	$\int_0^t \varrho_S \Delta t$	%
I. 1	3.5	59.76	210	12,550	12,550	1180	0.04	47	0.38
2	10	9.62	390	3,752	16,302	1380	0.07	100	0.61
3	22	1.01	720	727	17,029	1444	0.09	130	0.76
4	35	0.18	780	140	17,169	1485	0.10	148	0.86
II. 1	3.4	43.24	204	8,821	8,821	732	0.03	22	0.24
2	10.8	13.95	444	6,195	15,016	1130	0.08	90	0.60
3	23.1	2.38	738	1,760	16,777	1286	0.09	116	0.69
4	34.8	0.58	702	404	17,180	1360	0.10	136	0.79

λ_s = standardized spec. ^{14}C activity in lactose in s.u. = $\frac{\mu c \text{ per gram atom C}}{\mu c \text{ inject. per kg wt.}}$

ϱ_S = standardized spec. ^{14}C activity in respiratory CO_2 in s.u.

$\bar{q}$ = carbonate fixation quotient for lactose formation determined in earlier trials with labeled carbonate. (KLEIBER, SMITH, AND BLACK, 1952).

Table V shows the specific ^{14}C activity in glucose and acetate of the blood plasma. The level of activity in plasma glucose is consistently higher in Trial II than in Trial I. Presumably the difference is, at least in part, the result of a relatively smaller plasma

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volume in the cow of Trial II. Both cows contained 20 liters of blood plasma. In cow of Trial I, who weighed 457 kg, this meant 4.4%, in cow of Trial II, however, with a body weight of 526 kg, the plasma amounted to only 3.8%, of her body weight. The specific activity in plasma glucose decreased rapidly. Three hours after the injection it amounted to only 1% of the theoretical activity at the start. The calculation of this initial activity is explained in Table V. The ratio of the specific activities in blood glucose in Trial II to those in Trial I increased with time from injection. At the same time the glucose concentration in the plasma of cow II decreased. Both observations suggest a relative decrease in the rate of glucose synthesis in cow II during the trial. The plasma acetate contains very little ^{14}C (see Table V). This result indicates that in the cow only small amounts of acetate are formed from plasma glucose. Transfer through the plasma acetate pool cannot, therefore, account for the fact that after injection of labeled glucose the specific activity in milk fat rose as high as 60% of the maximum specific activity of milk fat observed in our trials after injection of labeled acetate. The high labeling of fat from glucose is explained by the recent observation of T. ROGERS in our laboratory[30] that almost all ^{14}C in milk fat after injection of labeled glucose is located in the glycerol moiety of the fat.

TABLE V

SPECIFIC ^{14}C ACTIVITY IN GLUCOSE AND ACETATE OF BLOOD

Trial I				*Trial II*		
Plasma glucose		*Plasma acetate*		*Plasma glucose*		*Plasma acetate*
t *min*	π_S	t	π_S	t *min*	π_S	π_S
0	(1143)	—	—	0	(1308)	—
22	183	29	2.26	9	282	0.27
39	124	—	—	16	218	0.31
60	79	66	0.72	25	166	0.26
89	47	—	—	40	106	0.25
121	32	126	1.15	60	72	—
153	21	—	—	90	52	—
181	13	187	1.48	120	27	—
402	1.9	—	—	150	19	—
655	0.6	—	—	180	18	—
1309	0.2	—	—	309	4.7	—
				614	0.7	
				1450	0.2	

t = time from intravenous injection of all-labeled glucose.

π_S = standard spec. ^{14}C activity in s.u. = $\frac{\mu c \text{ per gramatom C in glucose}}{\mu c \text{ injected per kg body weight.}}$

* $\pi_{S,0,I}$ calculated as follows: Cow contained 20 liters of plasma (measured) with 60 mg% glucose (measured), that is 12 g glucose which contains 0.4 gramatom carbon. Injected 2.87 mc ^{14}C. This amounts to 7180 μc ^{14}C per gram atom C in plasma glucose, or 7180/6.28 = 1143 s.u.

** $\pi_{S,0,II}$ calculated as follows: Cow contained 20 liters of plasma (measured) with 61 mg% glucose (measured), that is 12.2 g glucose which contains 0.4 gram atom carbon injected 1.45 mc ^{14}C. This amounts to 3630 μc per gram atom C in plasma glucose or 3630/2.76 = 1308 s.u.

Table VI shows the calculation of the glucose–lactose transfer quotient for carbon. This quotient indicates the ratio between the rate of transfer of glucose carbon to lactose and the rate of lactose formation expressed in terms of all carbon atoms incorporated into lactose during a given period. This quotient (q) increases with time from injection

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TABLE VI

CARBON TRANSFER FROM PLASMA GLUCOSE TO MILK CONSTITUENTS

Period	Hour end of per.	Δt in per.	$\int_0^t \pi_S\, dt$* In plasma	Milk constituents: Lactose λ_S	$\lambda_S \cdot \Delta t$	$\sum \lambda_S \Delta t$	Lactose q**	Casein q** %	Fat q** %	Citric acid q** %
Trial I										
1	3.5	210	20750	59.76	12000	12000	58	2.8	1.5	—
2	10	390	21890	9.62	3752	15752	72	3.9	3.7	—
3	22	720	22200	1.01	727	16479	74	4.3	4.4	—
4	35	780	22290	0.18	140	16619	75	4.5	4.6	—
Trial II										
1	3.4	204	17890	43.24	8821	8821	49	3.2	0.4	11
2	10.8	444	19560	13.95	6195	15016	77	5.5	3.6	17
3	23.1	738	19980	2.38	1760	16776	84	6.3	5.0	19
4	34.8	702	20160	0.58	404	17180	85	6.5	5.9	20

* The time integral of the specific activity, π_S, in plasma glucose was obtained by plotting the results of Table V, using interpolated means for 10 minute periods for integration, and then plotting the integrals against time. From the resulting curve the integral at any particular time (3.5, 10 h) could be read.

** The transfer quotient, q indicates the percentage of the carbon in lactose, casein, fat, or citric acid that came to these compounds *via* the plasma-glucose pool.

reaching a plateau at 35 hours. This apparent dependence of the transfer quotient on time from injection may be explained as a result of several different metabolic paths from glucose to lactose as discussed earlier for carbonate fixation[25]. An intermediary pool between plasma glucose and lactose would also explain the observed correlation between transfer quotient and time from injection.

About 4/5 of the lactose carbon apparently was derived from the plasma glucose pool*, about 1/5 of the carbon in citric acid of the milk was contributed by this pool but only 4 to 6% of the carbon in casein and in butterfat had this origin. This result does not necessarily mean that only 20% or less of the lactose originates from precursors synthesized in the mammary gland. It is possible that some glucose (or related metabolite) is synthesized in the mammary gland and joins the glucose pool. If the exchange between plasma glucose and mammary glucose is rapid enough, then the specific activity measured in one (in our case plasma) is also representative for that of the other.

ACKNOWLEDGEMENT

The authors gratefully acknowledge the help of C. HIRAHARA, J. KANEKO AND J. LUCAS who, aside from their routine tasks in the laboratory, succeeded in preparing citric acid out of our cow's milk. We are also indebted to T. ROGERS for help in preparation of the sample of labeled glucose used in the second trial.

* "Plasma glucose pool" stands for the sum of all carbon compounds which for a given measurement are in rapid enough carbon exchange with the glucose of the plasma to behave for that measurement as a kinetic unit. The term "plasma" indicates that the measurements of the specific activities from which the characteristics of this pool are derived, are carried out with glucose prepared from plasma.

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We also wish to express our gratitude to our colleagues of the Dairy Husbandry group, especially Dr. ROBERT LABEN, for their help in providing the cows for our experiments.

SUMMARY

1. Glucose uniformly labeled with ^{14}C was injected into the jugular veins of two normal lactating dairy cows as single doses of 3 and 6 microcuries per kg body weight respectively. The injected glucose, about 1 gram, amounted to less than 10% of the plasma glucose.

2. Between 30 and 40 minutes after injection the radioactivity of the respiratory CO_2 reached a maximum of 9 microcuries per unit of the relative injected dose (μc injected per kg body weight).

3. During the first 3 hours after injection of uniformly labeled glucose less ^{14}C appeared in the respiratory CO_2 than was expired during the corresponding period in earlier trials after injection of ^{14}C labeled acetate, propionate, or butyrate.

4. More than 50% of the ^{14}C injected as uniformly labeled glucose appeared in the organic constituents of the milk within 10 hours after injection. Over 80% of this ^{14}C in milk components was found in lactose.

5. In the first milk sample, 3 hours after injection, the specific ^{14}C activity in the components of the milk decreased in the following order: Lactose $\longrightarrow$ Citrate $\longrightarrow$ Casein $\longrightarrow$ Milk fat.

6. Only about 1% of the carbon transferred from plasma glucose to lactose passed the carbonate pool, 4 to 10% of the carbon transfer to casein followed this path.

7. Three hours after injection the specific ^{14}C activity in blood glucose had decreased to about 1% of its theoretical level at the time of injection, the latter calculated from injected dose, plasma volume and glucose level in plasma.

8. About 4/5 of the lactose carbon came from carbon in plasma glucose or a pool in rapid exchange with plasma glucose, 1/5 of the carbon in citrate originated from this pool and about 5% of the carbon in casein and milk fat.

RÉSUMÉ

1. Du glucose, marqué par ^{14}C à toutes les positions 6 du carbone, fut injecté intraveineusement à deux vaches en lactation en doses uniques de 3 et 6 microcuries par kg du poids vif.

2. 30 à 40 minutes après l'injection, la radioactivité de l'acide carbonique respiratoire atteignit un maximum de 9 microcuries par unité de la dose relative injectée (microcuries injectés par kg de poids vif).

3. Pendant 3 heures après l'injection du glucose marqué nous trouvâmes moins de ^{14}C dans l'acide carbonique respiratoire que nous n'en avions trouvé pour la période correspondante dans des expériences avec des acides acétique, propionique, et butyrique marqués par ^{14}C.

4. Dix heures après l'injection, plus de 50% du ^{14}C injecté était dans les substances organiques du lait, 80% du ^{14}C du lait se trouvent dans le lactose.

5. Parmi les constituants du lait tiré 3 heures après l'injection du glucose marqué l'activité spécifique était la plus grande dans le lactose, moindre dans l'acide citrique, encore moindre dans la caséine et la plus petite dans la graisse.

6. Une fraction très petite, 1%, du carbone transféré du glucose du plasma au lactose passa par le stade ("pool") carbonate, mais 4 à 10% du carbone du glucose transféré dans la caséine passa par CO_2.

7. Pendant les 3 heures qui suivirent l'injection du glucose marqué, l'activité spécifique du ^{14}C dans le glucose du plasma diminua rapidement. Trois heures après l'injection, l'activité ne fut plus que 1% de l'activité théorique au moment de l'injection. Cette activité théorique originale fut calculée d'après le rapport entre la quantité de ^{14}C injecté et la quantité de glucose mesuré dans le sang.

8. Environ 4/5 du carbone dans le lactose a son origine dans le glucose du sang ou dans un "pool" métabolique qui est en échange rapide avec le glucose du sang. Un cinquième du carbone de l'acide citrique du lait et 5% du carbone de la caséine et de la graisse du lait proviennent de ce "pool" de glucose du sang.

ZUSAMMENFASSUNG

1. Gleichmässig mit ^{14}C signierte Glucose wurde in die Jugularvenen zweier laktierender Kühe injiziert und zwar in Eizelinjektionen von 3, resp. 6, Mikrocuries pro kg Körpergewicht. Die Menge der injizierten Glukose, ungefähr ein Gramm, machte weniger als 10% der Plasmaglukose der Kühe aus.

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2. In 30 bis 40 Minuten nach der Injektion erreichte die Radioaktivität der ausgeatmeten Kohlensäure ein Maximum von 9 Mikrocurie pro Einheit der relativen injizierten Dosis (Mikrocurie pro kg Körpergewicht).

3. Während der ersten 3 Stunden nach der Injektion von signierter Glukose erschien weniger ^{14}C in der Atmungskohlensäure als in der vergleichbaren Zeitdauer in früheren Versuchen nach Injektion von signiertem Azetat, Propionat und Butyrat.

4. Innerhalb 10 Stunden nach der Injektion von signierter Glukose erschien mehr als 50% der injizierten Radioaktivität in organischen Bestandteilen der Milch, über 80% hiervon im Milchzucker.

5. Die spezifische ^{14}C-Aktivität in Bestandteilen der ersten Milchprobe (3 Stunden nach der Injektion) nahm nach folgender Reihenfolge ab: Milchzucker → Zitronensäure → Kasein → Milchfett.

6. Nur ungefähr 1% des Kohlenstoffs der von Plasmaglukose zu Laktose ging, passierte den Karbonat"pool", aber 4 bis 10% des Kohlenstofftransportes von Laktose zu Kasein ging über Kohlensäure.

7. Nach der Injektion der signierten Glukose nahm die spezifische ^{14}C-Aktivität der Plasmaglukose rasch ab. In 3 Stunden war sie auf ungefähr 1% des theoretischen Niveaus zur Zeit unmittelbar nach der Injektion abgesunken, welch theoretisches Niveau man aus der gemessenen Menge von Plasmaglukose und der Menge injizierten Radiokohlenstoffs schätzen konnte.

8. Ungefähr 4/5 des Kohlenstoffs im Milchzucker kam vom Kohlenstoff der Plasmaglukose oder einem Glukose"pool" in raschem Austausch mit Plasmaglukose; aus diesem "Pool", oder Plasmaglukose, stammten 1/5 des Kohlenstoffs in der Zitronensäure der Milch und 5% des Kohlenstoffs im Kasein und im Milchfett.

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Received December 20th, 1954