Effects of Long-Term High-Sucrose and Dexamethasone on Fat Depots, Liver Fat, and Lipid Fuel Fluxes Through the Retroperitoneal Adipose Tissue and Splanchnic Area in Rats

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In affluent societies high caloric intake and chronic stress are currently associated with upper body fat. We investigated the effects of a high-sucrose (S) diet and dexamethasone (DEX) on fat depots (experiment 1) and lipid fuel fluxes (experiment 2) in male Wistar rats. In experiment 1, a liquid diet of commercial powdered milk containing 31% calories as carbohydrate or an isocaloric S diet (80% calories as carbohydrate) was offered to male rats. One half of the rats on each diet received a daily dose of 3 μg DEX in their diet. Intake was measured daily and body weight 3 times a week. Rats were killed after 7 weeks, and fat depot weights and carcass lipid were determined. In a second experiment, other rats received only the S diet with or without DEX. After 7 weeks, under pentobarbital anesthesia, arterial, portal, and iliolumbar vein blood was drawn, and the liver was extracted. Plasma concentration of triacylglycerides (TAG), nonesterified fatty acids (NEFA), glycerol (GOL), and lactate (L) and liver TAG were measured. Rats on the S diet ingested less and gained less weight. DEX treatment significantly reduced body weight gain. All fat depots as percentage of body weight were increased only in the S-DEX group. The S-DEX group had more liver TAG and less arterial NEFA and GOL than the S group. TAG determinations showed unexpected results: portal levels in the S-DEX group and iliolumbar levels in both groups were significantly higher than in the arterial plasma. This fact, together with high NEFA/GOL ratios in these veins, may signify incomplete TAG hydrolysis by lipoprotein lipase. L levels were higher in the S-DEX group and higher in arterial versus venous blood in both groups, indicating L uptake both in the splanchnic area and the retroperitoneal fat. These results show that, in rats, a long-term high-sucrose diet has peculiar effects on L turnover, and when associated with DEX, it also increases fat depots, induces liver steatosis, and, presumably, inhibits complete hydrolysis of TAG by lipoprotein lipase (LPL).

OBESITY IS A health problem in affluent societies and is commonly attributed to excessive fat intake combined with low levels of physical activity.[1] In humans it was shown that calories from high-fat (HF) foods are consumed in excess compared with high-carbohydrate (HC) foods, at least in part, because fat is both higher in energy content and less satiating than carbohydrate.[2,3] The general recommendation for obese people is to reduce fat intake.[4,5]

Some investigators sustain that ad libitum HC intake in humans reduces body weight,[5] while others propose that glycogen levels are inversely related with body fat oxidation; in this way, a HC diet, by replenishing glycogen depots, will favor glycogen and consequently reduce fat utilization.[6]

In rats, when a HF diet was offered in only such amounts as to equal the average caloric consumption of a HC group, absolute weight gain change was not significantly different.[7,8] This suggests that what is most probably involved in gaining excess weight is the overconsumption of calories in the HF diet.

Corticosteroid (Cs) hormones were repeatedly shown to be involved in the control of energy metabolism and body weight. The synthetic Cs, dexamethasone (DEX), binds with very high-affinity to type II Cs-receptors[9] and has catabolic effects, involving weight loss, accelerated lipolysis, decreased food intake, and increased thermogenesis.[10] High levels of Cs induce insulin resistance,[11,12] most likely by a type II effect, as it is repeatedly obtained by DEX administration in humans and rats.[13-15]

Visceral obesity is also associated with insulin resistance in humans[16] and in rats.[17] When corticosterone was administered to rats, most of the fat accumulated in the mesenteric region.[18] DEX is more efficiently bound to mesenteric adipose cells than in adipocytes of other fat depots.[19]

In a previous study,[17] it was shown that long-term DEX treatment in rats reduced body weight gain and food efficiency and increased both cholesterol and triacylglycerides (TAG) in plasma, as well as TAG in liver. When rats were treated with DEX after lipectomy of the retroperitoneal and inguinal fat depots, mesenteric fat and liver TAG were significantly increased, and glucose tolerance was reduced.

Dallman et al[20] proposed that glucocorticoids and insulin represent a bihormonal system that regulates overall energy balance. However, under glucocorticoid treatment, the system may turn maladaptive due to increases of both hormones, inducing fat accumulation. Because insulin secretion in rats is increased both by HC diets and DEX treatment,[21-23] we were interested to find out what effect these 2 treatments would have on different fat depots and carcass lipids. In the present study, rats receiving or not receiving DEX treatment were isocalorically-fed a diet containing either 47% calories as fat and 31% as carbohydrate or a high-sucrose (80%) low-fat (13%) diet. All fat depots were increased by the combined treatment: DEX + high-sucrose. In a second experiment, we measured the effects of the same high-sucrose diet with or without DEX administration on plasma concentration of lipid fuels in aorta, porta, and the right iliolumbar vein that drains abdominal cutaneous muscle and the retroperitoneal fat pad.[24]

On the other hand, it is known that adipose tissue produces

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Table 1. Percent Caloric Composition in Macronutrients of the Two Diets

Macronutrient/Group	Control	High-Sucrose
Protein	22	7
Carbohydrate		
Lactose	31	9
Sucrose	—	71
Lipid	47	13
Total	100	100

lactate,[25] but also that lactate may be a precursor for adipocyte lipogenesis.[26,27] To find out whether the portal and iliolumbar net arteriovenous flux of lactate would be positive or negative in these particular conditions, lactate levels were also measured in the 3 vessels.

MATERIALS AND METHODS

Experiment 1

Twenty-four male Wistar rats weighing initially 261 ± 3 g were kept in individual metalic cages in a temperature-controlled room (22 ± 1°C) and light from 8 AM to 8 PM. Rats were randomly distributed in 4 groups of 6 rats each. Two control groups (C) were offered a diet of commercial powdered milk (Nido; Nestlé, Mexico City, Mexico) containing per 100 g: 26.4 protein, 26.2 fat, and 38.6 lactose, summing up 5 cal (20.9 kJ)/g. All rats received daily 440 kJ in the following proportion: 2 C groups, 21 g milk; and 2 HC (S) groups, 6 g milk plus 19 g sugar. All diets were prepared in 55 mL tap water. The rats of 1 C and 1 S group received in their diet a daily dose of 3 µg DEX (Decadrón; Merck Sharp & Dohme, Mexico City, Mexico). These 2 subgroups were labeled C-DEX and S-DEX, respectively. The macronutrient caloric composition of the 2 diets is shown in Table 1.

All diets were freshly prepared each day. Daily food intake was measured by weight difference of the tubes containing the liquid food on an electronic balance (precision, 0.1 g); rats were weighed 3 times a week. After 7 weeks and a 24-hour fast, rats were killed under 0.8 g/kg urethane (Sigma Chemical, St Louis, MO) anesthesia. Retroperitoneal and epididymal fat pads were dissected, weighed, and discarded; the gastrointestinal tract was frozen. After shearing the head, the 4 paws, and the tail, the remaining carcass was frozen. Mesenteric fat was later easily dissected from the gastrointestinal tract, weighed, and discarded. At a later time, carcasses were thawed, weighed, and autoclaved for 30 minutes. Bones were separated and discarded, and the remaining carcass was ground in a blender. The resulting homogenates were dried at 70°C and ground to a powder. From approximately 2 g of each sample, weighed in an electronic analytical balance with a precision of 0.1 mg, total fat was extracted in a Soxhlet apparatus (Kimax, Mexico City, Mexico) with petroleum ether.

Experiment 2

Sixteen male Wistar rats (body weight, 343 ± 8 g) received the S diet of experiment 1 and were maintained in similar conditions. Eight rats had DEX included in their daily food at the same dose as experiment 1. One rat in the S-DEX group died during the experiment.

After 7 weeks, the rats were anesthetized with 35 mg/kg sodium pentobarbital (Anestesal; SmithKline Beecham, Farmacéutica, México) after a 24-hour fast. Their abdomens were opened, and 0.5 mL of blood was drawn from each of the following vessels: right iliolumbar (IL) vein, porta, and abdominal aorta, in this order. Plasma and the excised livers were rapidly frozen.

Concentrations of the following circulating metabolites were measured in plasma: triacylglycerides (TAG) and glycerol (GOL), using a Sigma Chemical kit; nonesterified fatty acids (NEFA), using a kit from Wako Chemicals, Richmond VA, and; lactate (L), using an apparatus and kit of Analox Instruments, Lunenburg MA. Livers were thawed and homogenized. Approximately 25 mg were weighed, mixed in a 0.9% NaCl solution, and centrifuged for 35 minutes. TAG present in 10 µL of supernatants was quantified using the Sigma kit mentioned above.

Statistical Analysis

The data of the first experiment were analyzed by bifactorial analysis of variance (ANOVA) (diet × treatment) with Neuman-Keuls posthoc analysis between individual means when presenting a significant interaction. Data of experiment 2 were analyzed by 2-tail unpaired or paired Student *t* test. Pearson product moment correlation analysis was applied between plasma metabolite concentrations. Results are expressed as mean ± SEM. The level of significance was set at $P < .05$.

RESULTS

Experiment 1

Rats did not always consume their daily portion, and there was a significant difference by bifactorial ANOVA in mean daily intake between diets, but not between treatments (Fig 1A). This difference between diet groups is more evident for the mean daily weight gain, S groups having gained less weight (Fig 1B). The difference between treatments was also significant due to less weight gain induced by DEX in both groups. Bifactorial ANOVA shows that diet induced a significant difference only in mesenteric fat relative weight, while DEX treatment increased significantly both mesenteric and epididymal fat. The effect of DEX on retroperitoneal fat was close to statistical significance (Fig 2). The significant interaction in mesenteric and epididymal depots indicates that DEX effect depended on diet, and Neuman-Keuls test shows that the S-DEX group presented more fat than the other 3 groups.

Carcass total lipids were also elevated in the S-DEX group, but the difference did not reach statistical significance (C, 8.08 ± 0.84; C-DEX, 8.02 ± 0.57; S, 7.78 ± 0.44; S-DEX, 10.22% ± 0.52% of body weight). However, S-DEX and C means are significantly different by 2-tail *t* test ($P = .05$).

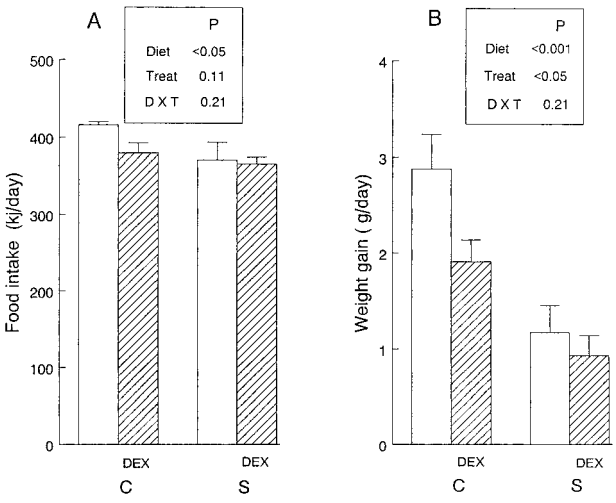

Fig 1. (A) Daily food intake and (B) body weight gain in rats fed control (C) or high-sucrose (S) diet with or without DEX treatment. Inserts: 2-way ANOVA (diet × treatment).

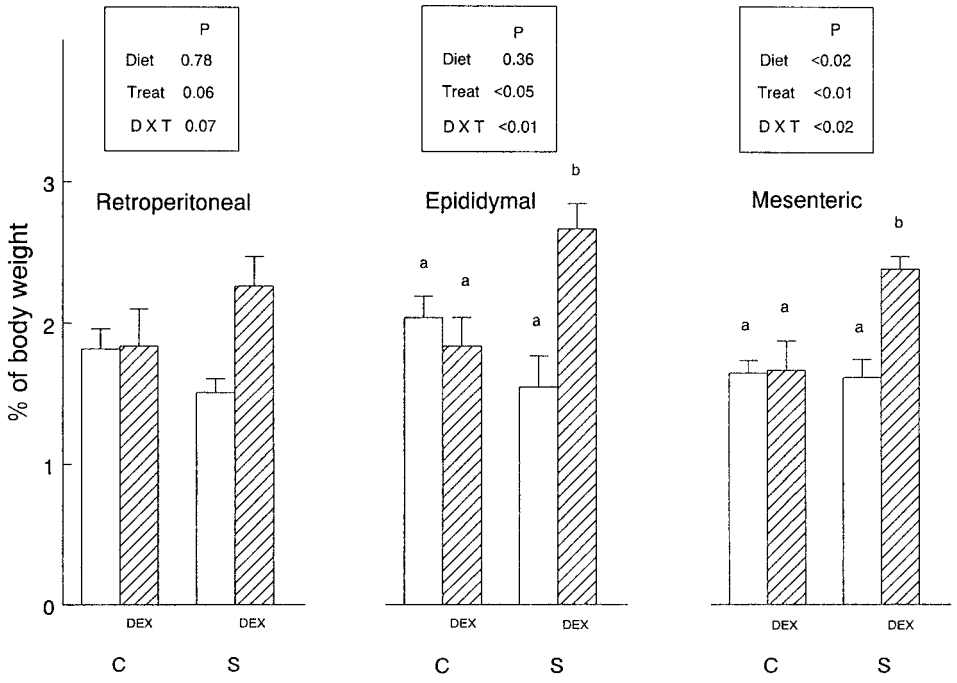

Fig 2. Relative weight of fat depots in rats fed control (C) or high-sucrose (S) diet, with or without DEX treatment. Inserts: 2-way ANOVA (diet × treatment). Bars not sharing the same superscript are significantly different at $P < .05$ by Neuman-Keuls post hoc analysis.

Experiment 2

The 2 groups did not present significant differences in body weight gain (S, 0.83 ± 0.12; S-DEX, 0.81 ± .12 g/d). However, the energy intake of S-DEX was lower than that of the S group (391 ± 2.2 *v* 414 ± 2.6 kJ/day, respectively; $P < .001$). Liver weighed more in the S-DEX group (3.07% ± 0.14% *v* 2.67% ± 0.05% of body weight; $P < .05$) and contained twice more TAG (33.5% ± 4.0% *v* 17.6% ± 0.9% mg/g liver; $P < .02$) than the S group.

Table 2 presents the data for serum metabolites in the 3 vessels. GOL and NEFA arterial concentrations were significantly decreased by DEX treatment. Lipolysis is commonly considered to be reflected by GOL venoarterial difference (v-a); however, our results show that, apparently, this process took place only in the tissue drained by the iliolumbar vein in both groups. NEFA, the other component of lipolysis, showed similar concentrations in this vein in both groups, but due to lower arterial levels, v-a was significantly higher for both veins in S-DEX. These differences between the 2 groups of rats are also reflected in NEFA/GOL ratios, which were significantly higher in the S-DEX group in both veins, while not different in the arterial serum.

TAG determinations gave very unexpected results: in both groups v-a was positive for the ileolumbar vein. Because TAG are hydrolyzed by lipoprotein lipase (LPL) in the capillaries of adipose tissue, venous levels should be lower than in the artery. These results will be analyzed in the Discussion.

Significant or nearly significant correlations were found in the S group: NEFA versus TAG in aorta and ileolumbar vein, GOL versus NEFA in aorta, GOL versus TAG in the 3 vessels (Table 3). In the S-DEX group, only GOL versus NEFA levels were positively correlated in the iliolumbar vein, while GOL versus TAG concentrations in aorta and porta vessels showed negative trends. In general then, DEX treatment either canceled some positive relationships between these circulating metabolites or even reversed the normal trends.

L arterial concentration was significantly elevated by DEX treatment. Venoarterial differences show that L was consumed in the tissues drained by both veins, significantly more in S-DEX group (Table 2). L levels were not correlated with any of

Table 2. Arterial, Portal, and Iliolumbar Venous Plasma Levels of TAG, NEFA, GOL, and L and NEFA/GOL Ratios

Metabolite/ Group	S (n = 8)	v-a	S-DEX (n = 7)	v-a
TAG (mmol/L)				
A	.56 ± .03		.66 ± .08	
P	.52 ± .04	−.04 ± −.003	.76 ± .13	.10 ± .13
IL	.87 ± .07*	.31 ± .07	1.00 ± .10*	.34 ± .08
GOL (mmol/L)				
A	.45 ± .03		.24 ± .05†	
P	.39 ± .03	−.06 ± .03	.23 ± .03†	−.01 ± .45
IL	.80 ± .13*	.35 ± .11	.53 ± .06*	.29 ± .09
NEFA (mmol/L)				
A	.84 ± .09		.44 ± .03†	
P	.76 ± .05	−.08 ± .06	.72 ± .07*	.28 ± .05*
IL	1.57 ± .13*	.73 ± .10	1.58 ± .17*	1.14 ± .06†
NEFA/GOL				
A	1.87 ± .15		2.21 ± .45	
P	1.99 ± .14		3.34 ± .44*†	
IL	2.08 ± .17		3.02 ± .19†	
L (mmol/L)				
A	3.39 ± .42		6.80 ± .68†	
P	2.90 ± .41*	−.49 ± .18	4.94 ± .37*†	−1.86 ± .63†
IL	2.55 ± .32*	−.84 ± .18	4.25 ± .38*†	−2.55 ± .57†

Abbreviations: TAG, triacylglycerides; GOL, glycerol; NEFA, nonesterified fatty acids; L, lactate; A, aorta; P, porta; IL, iliolumbar; v-a, venoarterial differences.

*$P < .05$, *P* or IL *v* A by paired *t* test.

†$P < .05$ *v* S by unpaired *t* test.

Table 3. Correlations Between Lipid Fuels in the Same Blood Vessel

		Aorta		Porta		Iliolumbar	
Group	Metabolites	*r*	*P*	*r*	*P*	*r*	*P*
S	AG-NEFA	0.85	<.01	NS		0.86	<.01
	AG-GOL	0.66	.07	0.68	.07	0.89	<.01
	NEFA-GOL	0.73	.04	NS		NS	
S-DEX	AG-NEFA	NS		NS		NS	
	AG-GOL	−0.62	.14	−0.87	.01	NS	
	NEFA-GOL	NS		NS		0.88	.01

Abbreviations: NS, not significant; AG, acylglycerides; GOL, glycerol; NEFA, nonesterified fatty acids.

the other measured metabolites, but significant correlations were found between aorta-porta and aorta-iliolumbar L concentrations (not shown).

DISCUSSION

The 2 isocaloric diets offered to rats in experiment 1 induced significantly different effects on both caloric intake and body weight gain. Mean daily weight gain was 1 g with the S diet and at least 2 g for the C diet. This may have been caused by the relatively low protein content in the S diet, decreasing food conversion efficiency as shown by other investigators,[28,29] possibly due to an increase in triiodothyronine plasma levels.[30] DEX administration significantly reduced body weight gain without significantly affecting food intake.

High-sucrose diets induce increased glucose uptake in adipocytes.[31] In the present experimental conditions, only when the S diet was combined with DEX administration did fat accumulation occur. Although blood glucose and insulin levels were not determined, the S diet should have induced chronic increases in their concentration[32] and, as a result, in liver lipogenesis. Glucocorticoids (GC) also stimulate liver lipogenesis in rats.[33] The activity of glucose-6-phosphate dehydrogenase, the key regulator enzyme of the pentose phosphate cycle, producing reducing equivalents in the form of nicotinamide adenine dinucleotide phosphate (NADPH), increases in the presence of insulin, GC, and dietary carbohydrates.[34] However, in the present experimental conditions, mesenteric fat was not preferentially increased in the S-DEX group as reported by other investigators to occur in stress exposed rats.[18] Nevertheless, it is interesting to note that the 2 fat stores that showed a significant increase in this group, that is, the mesenteric and epididymal depots, were reported to grow in Wistar rats mostly due to hypertrophy, while retroperitoneal fat growth is predominantly due to hyperplasia.[35]

The effect of DEX on lipid accumulation when the diet was very rich in sugar could also be interpreted according to Flatt's model.[6] In the presence of high glucose and GC plasma levels, liver glycogen is significantly increased mainly from circulating glucose.[36] The model proposes that in the presence of a high glycogen reserve, glucose is preferentially used as metabolic fuel, thus sparing lipid utilization and maintaining or even increasing adipose tissue weight.

Of the variables measured in both experiments, food intake showed significant differences in experiment 2: S-DEX group mean ingestion was lower than that of the S group, but this might be due to body weight (age) differences between the rats of the 2 experiments.

Arterial levels of TAG were similar in both groups in experiment 2, confirming that the effects of sucrose and DEX on TAG plasma levels are not additive.[32] High sucrose diet (S group) induced high arterial NEFA levels, confirming the results of other investigators.[37]

A surprising result of this experiment was the significant positive TAG v-a in the iliolumbar vein of both groups. LPL-induced hydrolysis of TAG might not be complete; the enzyme hydrolyzes a TAG molecule to 2 FA and 1 2-MAG at the luminal surface of endothelial cells.[38] In order that LPL may completely split TAG, an isomerization of 2-MAG to the 1 or 3-isomer is necessary, a reaction that is considered to be rate-limiting. According to Belfrage et al,[39] 2-MAG has to be interiorized into adipose cells and presumably split by a specific 2-MAG hydrolase.

The kit that was used to measure TAG contains a lipase, which completely hydrolyzes acylglycerides (AG) and actually determines free GOL plus the GOL contained in any AG molecule. A second treatment of the samples, without lipase reagent, measures free GOL. In this way, AG concentration is obtained by substracting free GOL from the total GOL value of the same sample. If any MAG (or diacylglycerol) is present in the sample, it will be equal to TAG, as both MAG and TAG molecules contain 1 GOL residue. Nevertheless, this cannot explain an enrichment of AG in venous blood without considering the calculation implied in determining TAG levels by means of the kit's GOL standard: the GOL standard is equated to a triolein concentration of 250 mg/dL (not in mmol/L). Monoolein has a molecular weight 2.5 times less than triolein. If, for example, a sample contains 250 mg/dL triolein, the inferred value versus GOL standard will be 1; if a sample contains only monoolein, with the same GOL content, the calculated value for TAG will be 2.5 mmol/L, that is, 525 mg/dL. Any intermediate relative proportion of the 2 AG will overestimate the reported value of TAG.

Only taking into account the above-mentioned particularities of lipid metabolite fluxes through an adipose tissue can the present data be interpreted. It should also be considered that while iliolumbar v-a may be attributed mainly to blood-muscle and blood-adipose cell interchanges, in the portal v-a all intraperitoneal organs should be involved. That may explain the nonsignificant NEFA v-a in the S group when considering that some splanchnic organs might use them. In the same S group, the portal v-a for AG was not significant, and the NEFA/GOL ratio was approximately 2, as in arterial serum. This suggests that some TAG were possibly hydrolyzed by LPL, but NEFA and GOL uptake into the different splanchnic organs balanced, on the average, NEFA and GOL produced by mesenteric fat. This results, in the present conditions of fasted rats having received a long-term high-sucrose diet, that the lipolytic participation of mesenteric fat was very low. Part of the NEFA reaching the liver from the portal vein will be reesterified to TAG, which will be retained in fat droplets in hepatocytes and/or secreted in very–low-density lipoprotein. Arterial TAG levels were similar, but more NEFA were retained in the liver in the S-DEX group, as shown by the porta-arterial difference (Table 2). This should also contribute to the liver steatosis of the S-DEX rats. On the other hand, the significant GOL and NEFA positive v-a for the iliolumbar vein in both groups strongly

suggests that it represents mainly adipose metabolism because muscles do not release any of these metabolites into plasma.

DEX treatment reduced both NEFA and GOL arterial levels by 40% to 50%, but only NEFA v-a was higher in both veins (Table 2). This might be interpreted as due either to an activation of lipolysis or to an inhibition of complete TAG hydrolisis, resulting in higher levels of NEFA and MAG (and/or diacylglycerides) in the venous blood. It is known that part of the FA released by LPL will not enter into the adipose or muscular cells, but will be released as NEFA into venous drainage.[38] The high NEFA/GOL ratio in both veins and the positive v-a for what was measured as TAG supports the second hypothesis. In the iliolumbar vein of both groups, NEFA and GOL enrichments were significant, while in the portal vein, NEFA v-a was increased only in the S-DEX rats. A possible interpretation is that the sucrose diet caused an incomplete LPL-induced hydrolysis of TAG at the retroperitoneal adipose tissue endothelium, an effect that was significantly increased by DEX, while the same effect in tissues drained by the portal vein, ie, visceral fat, was induced only by DEX treatment.

In arterial plasma NEFA were positively correlated both with AG and with GOL in the S, but not in the S-DEX group. Also, contrary to the S group, GOL and AG levels were negatively correlated in S-DEX both in arterial and portal plasma (Table 3). Although mean NEFA/GOL ratios were not different between the 2 groups, S-DEX rats had a very wide range of values (1.13 to 4.25) compared with the S group (1.42 to 2.70). This could have been caused by very different effects of DEX on each S-DEX rat: a large systemic reesterification rate in some rats, inducing low NEFA/GOL, but an inhibition of complete hydrolyzing LPL activity in other rats, resulting in MAG plus more NEFA and less GOL. The degree of the latter effect in each rat might explain the negative trend between GOL and AG levels in aorta and porta in the DEX treated group (Table 3). Lack of correlation with NEFA in the portal vein might be due to different utilization and release of these compounds by the organs and tissues that are drained by the portal vein.

In the iliolumbar vein NEFA and GOL were significantly correlated with AG in the S group, while in the S-DEX group, GOL concentrations showed significant correlations with NEFA, but not with AG (Table 3). In this case, lipid metabolism in the venous effluent represents mainly fat depot drainage. The NEFA/GOL ratio was higher in the S-DEX group. All of this suggests either that there was no reesterification in the retroperitoneal fat pad of this group or that, given the enrichment in venous AG, more MAG plus more NEFA and less free GOL occurred in different proportions in all rats. Figure 3 represents a diagram of the possible origin of lipid metabolites in a vein draining an adipose mass.

A last intriguing result of the present work is L fate. Arterial levels were high in the S group and twice as high in S-DEX. A similar effect of glucocorticoids was reported in dogs.[36,40] In both groups, but significantly more in the DEX-treated rats, L was utilized by the tissues drained by both veins, while, in general, it is considered that adipose tissue releases L.[25,41] High levels of blood L are related to insulin resistance in rats[25] and humans[42] and suggest more production than utilization. Because the tissues whose L v-a was measured consumed lactate,

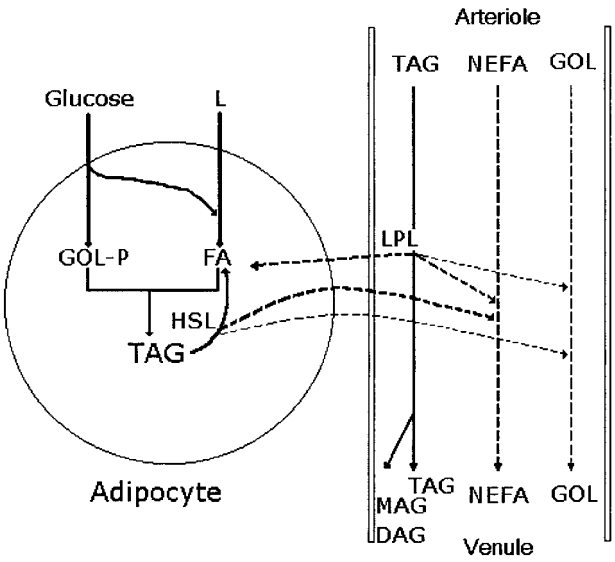

Fig 3. Possible origin of lipid metabolites in a venous drainage of adipose mass. GOL-P, glycerol phosphate.

the increased production ought to be attributed to other body regions, presumably skeletal muscles and/or liver. Adipose tissue lipogenesis which, as already discussed, might be activated in these particular dietary conditions might be responsible for L utilization by organs draining into the portal and IL veins. It is currently considered that both FA synthesized from acetyl-CoA, and glycerol phosphate resulting from dihydroxyacetone phosphate reduction, originate from glucose carbons. However, Botion et al[43] have recently shown that glucose was the substrate for only 10% of the FA synthetized in adipose tissue and 3% in liver. The difference in FA synthesis was obviously covered by other sources of acetyl-CoA, which, in conditions of our low-protein and high-sucrose diet, might be L. Both L and pyruvate have been shown to be very good precursors for adipocyte lipogenesis.[26,27] Therefore, it seems likely that the high-sucrose diet increased muscle glycolytic activity and that part of the produced L was utilized for glyceride-FA synthesis both in mesenteric and retroperitoneal tissues. Higher L levels in the S-DEX group may also explain, at least in part, the lower levels of NEFA and GOL in this group, as shown by other data reporting the same relations between these circulating metabolites in dogs.[44,45]

In conclusion, a very high-sucrose diet increased lipid depots only when combined with DEX treatment. The same combination raised TAG in liver, but not in plasma, decreased arterial nonesterified fatty acids and glycerol, and increased arterial lactate. The results suggest also that, in these conditions, TAG are incompletely hydrolyzed by LPL resulting in a leakage of NEFA and mono- and/or diacylglycerides in the portal and iliolumbar vein. High-sucrose diet induced a net lactate uptake both by splanchnic organs and the retroperitoneal tissue, an effect significantly enhanced by DEX treatment.

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