Increase of Adipose Tissue Lipoprotein Lipase Activity with Weight Loss

ROBERT S. SCHWARTZ and JOHN D. BRUNZELL, *Division of Metabolism and Endocrinology, Department of Medicine, University of Washington, Seattle, Washington 98195*

ABSTRACT Obese subjects have elevated adipose tissue lipoprotein lipase activity per fat cell when compared with lean control subjects. This enzyme, which is rate limiting for the uptake and storage of lipoprotein triglyceride in adipose tissue, has been shown to be further elevated in a group of previously obese subjects who had been weight stable at a reduced weight for 4–28 mo. In the present prospective study of eight obese subjects, adipose tissue lipoprotein lipase activity was demonstrated to increase after weight stabilization at a reduced weight (9.33 mU/10^6 cells) when compared with basal obese levels (2.17 mU/10^6 cells). In three subjects who lost weight and subsequently regained their lost weight, the enzyme activity increased after weight loss and then returned toward the original basal level with weight gain. One subject who maintained his weight loss for 10 mo continued to have an elevated level of enzyme activity. Because adipose tissue lipoprotein lipase activity does not "normalize" after weight loss, we hypothesize that this enzyme may play a counterregulatory role in resisting deviation from a "set point" for fat mass or fat cell size and thereby predispose to reattainment of the original obese state.

INTRODUCTION

Obesity is a major public health problem affecting a great number of adults in the United States (1). Treatment of this disorder is considered by most physicians and patients to be discouraging; <5% of subjects losing a significant amount of weight by any technique (2, 3) other than surgery are able to keep it off for 3–5 yr. Because of this impressive tendency for obese patients to regain lost weight and for thin individuals to lose weight they have gained by enforced overeating (4), a specific "set point" for fat mass or fat cell size has been postulated (4, 5). Deviation either above or below the set point might stimulate counterregulatory mechanisms to come into play, driving the patient back toward this set point.

Obesity is known to be accompanied by many metabolic abnormalities: insulin resistance with elevations in basal and glucose-stimulated insulin concentrations (6, 7); increased fat cell volume (8); increased glucose incorporation into triglyceride by adipose tissue (9, 10); enhanced basal and stimulated lipolysis (11); and elevated fasting triglyceride concentrations (7). All of these abnormalities are reversible, however, and normalize with weight loss (6–11). For this reason, it is likely that they are a consequence of the obesity, not a cause.

Adipose tissue lipoprotein lipase (AT-LPL)[1] is the rate-limiting enzyme responsible for the uptake and storage of lipoprotein triglyceride by the adipocyte (12). This enzyme is made in the cytoplasm of the adipocyte and transported to the capillary endothelium, where it hydrolyzes fatty acids from the triglyceride circulating in the core of very low density lipoproteins and chylomicrons (13, 14). The released fatty acids are then taken up by the adipocyte and reesterified to triglyceride, which is stored in the lipid droplet (12). The unique position of the enzyme AT-LPL with regard to fat storage suggests that it is a candidate for a role in the development of human obesity and/or the maintenance of the obese state.

AT-LPL activity per cell has been demonstrated to be elevated in obesity and is positively correlated with fat cell size as well as relative weight by actuarial table (percent of ideal body weight [%IBW]) (15, 16). If this elevation in AT-LPL, like most other metabolic abnormalities associated with obesity, is a consequence of the obese state, its activity would decline with weight

Dr. Schwartz is the recipient of National Institutes of Health fellowship IF 32 HL 18687. His present address is University of Vermont, Department of Medicine, Metabolic Unit, Burlington, Vermont 05405.

Received for publication 21 June 1980 and in revised form 30 December 1980.

[1] *Abbreviations used in this paper*: AT-LPL, adipose tissue lipoprotein lipase; HDL, high density lipoprotein; %IBW, percent of ideal body weight; VLDL-TG, very low density lipoprotein triglyceride.

loss. If, however, the AT-LPL activity remains elevated or increases further with weight loss, this enzyme might be playing a causal role in the maintenance of obesity.

Other studies (17) have revealed that previously obese subjects, who by careful chronic dieting were able to maintain a stable reduced weight for 4–28 mo, had AT-LPL activity threefold greater than predicted for their degree of obesity (%IBW) or their fat cell size. This retrospective analysis supports the hypothesis that AT-LPL activity was increased in these stable, reduced subjects as a counterregulatory attempt to increase triglyceride storage and return fat cell size, and thus fat mass, toward the original obese set point. Alternatively, those unusual individuals who were able to maintain their weight loss might have been a unique subgroup that always had greatly elevated AT-LPL activity. To evaluate these possibilities, prospective weight loss studies were performed, and the AT-LPL response was determined.

METHODS

Eight obese (175±68 %IBW) but otherwise healthy male volunteers were admitted to the University of Washington Clinical Research Center for weight loss (Table I). Seven volunteers were moderately obese, with onset of obesity after adolescence. The eighth had life-long massive obesity. None of them had known endocrinological causes of obesity and all had normal fasting plasma glucose levels. They were placed on a weight maintaining "basal" liquid formula diet comprised of 45% carbohydrate, 40% fat and 15% protein. The calorie intake was determined by the relationship between calories per kilogram and relative weight as previously reported (18). The last 4 d of the 1-wk weight stabilization period showed a rate of weight change of <50 g/d in each subject. After this weight stabilization period, a baseline adipose tissue biopsy for lipoprotein lipase activity was obtained at 8 A.M. after an overnight fast. The subjects then began a 600-kcal (50% protein, 50% carbohydrate) liquid formula weight reduction diet, which was continued until the subject felt he could no longer lose weight and wished to discontinue the diet. Each was then restabilized for 1 wk at the reduced weight on the original "basal" formula diet, and the adipose tissue biopsy was repeated. AT-LPL also was measured in four of the subjects at the end of a 1- to 2-wk period of weight stabilization at the midpoint of the weight loss protocol.

All subjects were given multiple vitamins and a stool softener throughout the study but took no other drugs. Two of the eight subjects (Nos. 2 and 4) were cigarette smokers, and except for the mornings before studies, when tobacco was withheld, they continued their usual smoking habit throughout the study.

AT-LPL was measured as the heparin-releasable enzyme as previously described (16). Adipose tissue specimens were

TABLE I
Effect of Weight Reduction

Patient	Age	Weight	IBW	Fat cell size	AT-LPL	VLDL-TG	HDL cholesterol	LDL cholesterol
		kg	*%*	*μg TG/cell*	*mU/10⁶ cells*	*mg/dl*	*mg/dl*	*mg/dl*
1 Obese	44	149	207	0.794	4.13	351	37	87
Reduced		138	192	0.673	11.81	204	33	93
2 Obese	34	103	148	0.576	1.43	204	28	115
Reduced		80	115	0.573	4.33	115	61	103
3 Obese	27	92	149	0.606	1.62	91	32	137
Reduced		83	134	0.576	12.16	173	33	145
4 Obese	34	80	131	0.705	2.72	136	24	124
Reduced		74	120	0.613	3.13	69	46	114
5 Obese	31	97	144	0.619	0.88	188	28	85
Reduced		81	119	0.541	0.81	126	35	85
6 Obese	24	99	143	0.770	2.01	171	31	153
Reduced		83	120	0.678	8.42	126	31	99
7 Obese	25	95	148	0.484	1.09	135	33	71
Reduced		84	130	0.571	29.03	75	37	108
8 Obese	39	252	333	0.743	3.49	50	28	123
Reduced		213	285	0.560	4.97	31	41	99
Mean±SD								
Obese	32±7	121±56	175±68	0.662±0.108	2.17±1.17	166±90	30±4	112±28
Reduced		105±48	152±60	0.598±0.052	9.33±8.93	115±56	40±10	106±18
P value		0.01	0.01	0.05	0.03	0.09	0.05	>0.3

obtained by suction needle biopsy of subcutaneous tissue in the area of the buttock. The tissue pieces were rinsed with 100 ml of cold Krebs-Ringer phosphate buffer (pH 7.4) dried on lipid-free sharkskin filter paper and cut into pieces of uniform size. Approximately 45 mg of tissue was incubated in duplicate flasks for 45 min at 37°C in 2.5 ml of Krebs-Ringer buffer with heparin (2 U/ml). At the end of the incubation period, two 1-ml samples of heparin-eluted enzyme were taken from each flask for assay.

The substrate for the AT-LPL assay was prepared using 200 μl of unlabeled triolein (25 mg/ml in benzene); 100 μl of [1-^{14}C]-triolein (2 μCi/ml in benzene); and 20 μl of purified egg lecithin (12 mg/ml in 1:1 chloroform:methanol). These were evaporated with nitrogen and emulsified in 2 ml of a mixture of 10% fatty acid-free bovine serum albumin (pH 8.0), pooled human serum as a source of enzyme activator, 2 M Tris–HCl buffer (pH 8.2 at 37°C), and distilled water (vol/vol 4:1.5:5:9.5) for a total of 3 min with a Branson 125 Sonifier (Branson Sonic Power Co., Danbury, Conn.). The substrate was kept cool on ice during and after sonification for a minimum of 30 min before use.

The 1-ml aliquot of the medium containing the heparin-eluted enzyme activity was added to 0.2 ml of substrate and incubated at 37°C in a metabolic shaker. The reaction was stopped at 45 min by adding Dole's reagent, and free fatty acids were extracted and specific activity was determined. AT-LPL activity is expressed as milliunits per million fat cells with one milliunit equal to a nanoequivalent of free fatty acids released per minute.

Fat cell diameter was determined from formalin-fixed frozen sections of fat by the method of Sjöstrom et al. (19). Fat cell volume (micrograms of triglyceride per cell) was calculated by the equation of Goldrick (20), and fat cell number was determined by dividing the tissue weight by the mean fat cell volume. To reduce assay variability, plasma activator pool, triolein substrate, and albumin lots were not changed during this study. The precision of replicate analysis was 5.9% within an assay and 8.9% between assays in subjects at their usual weight. The coefficient of variation between assays in a single, weight-stable, reduced individual biopsied seven times over a period of 25 mo was 13.6% (n = 7, 8.44±1.15 mU/10^6 cells).

Plasma lipoprotein was analysed in each subject in the fasting weight-stable state before and after weight reduction by the plasma ultracentrifugation technique used in the Lipid Research Clinics Program (21). %IBW was calculated from the Metropolitan Life Insurance Co. (New York) tables, using the midpoint of the range for medium build. The Wilcoxon signed rank test for paired values was used to evaluate differences in subjects before and after weight loss. Results are reported as mean±SD.

RESULTS

The mean %IBW for the eight patients in the obese state was 175 (Table I). After weight loss, it decreased to 152. Both of these means are somewhat inflated by the inclusion of one massively obese subject. The mean %IBW for the other seven subjects was 153 before and 133 after weight loss. The mean weight loss for the eight subjects was 16 kg.

The level of AT-LPL activity of the eight subjects before and after weight loss was compared to the relationship between AT-LPL activity and %IBW in a group of normal control subjects (Fig. 1). The subjects making up this control line were nonreduced, nonsmoking males who had been stable at their usual weight for a minimum of several months.

Seven of eight patients showed a rise in their AT-LPL activity after weight loss. The AT-LPL activity in the obese state was 2.17±1.17 compared with 9.33±8.93 mU/10^6 cells after weight loss (difference: 7.16±9.20, P = 0.03). The data points of the patients studied when obese fell on or about the control regression line, developed from data of other individuals, and deviated upward off the line after restabilization at a reduced weight (Fig. 2). Four subjects were restudied 8–14 mo after their weight reduction (Table II). Three subjects had regained their lost weight with a return of AT-LPL activity to near their obese levels. One subject had maintained most of his lost weight and continued to have elevated AT-LPL activity.

The fractional rate of weight loss was greater during the initial period of the hypocaloric diet (0.30±0.06%) than during the second period of the hypocaloric diet (0.11±0.07%, $P < 0.01$) in the four subjects weight stabilized halfway through the weight loss protocol (Fig. 3). In these subjects the initial period of weight loss was 41±5 d; the second period was 44±11 d. There was an inverse correlation between the change in AT-LPL activity and the fractional rate of weight loss (Fig. 3).

Measurement of plasma lipoprotein levels (Table I) revealed a reduction in very low density lipoprotein triglyceride (VLDL-TG) from 166±90 mg/dl in the obese state to 115±56 mg/dl after weight stabilization at the reduced weight; however, this was not a statistically significant decline (P = 0.09). Plasma high density lipoprotein (HDL) cholesterol level increased significantly from an obese level of 30±4 mg/dl to 40±10 mg/dl after weight reduction (difference: 9.50±12.55, P = 0.05). The change in HDL cholesterol did not correlate with the change in AT-LPL activity.

DISCUSSION

Most of the metabolic abnormalities associated with obesity have been shown to normalize with weight loss (6–11) and thus are likely a consequence of the obese state, not a cause. In the present study, the high AT-LPL activity usually seen in obesity did not decrease after weight stabilization at a reduced weight but in fact became more elevated. The persistent elevation of enzyme activity for 10 mo in the single individual who was able to maintain most of the weight loss suggests the increase in AT-LPL activity continues indefinitely, as indicated in a previous retrospective study of weight loss (17). In that study, elevation of AT-LPL activity was demonstrated in a weight reduced population studied 4–28 mo after losing weight. Furthermore, AT-

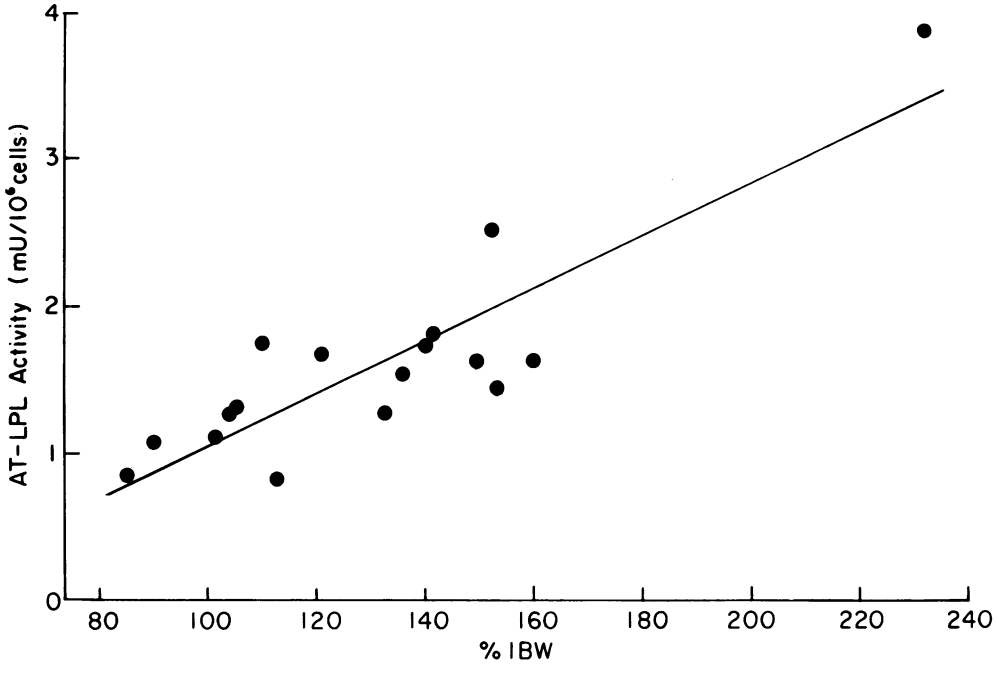

FIGURE 1 Relationship between AT-LPL activity (mU/10^6 cells) and %IBW for 17 weight-stable, nonsmoking controls ($r = 0.87, P < 0.001$). AT-LPL activity also correlated with fat cell size (micrograms of triglyceride per cell) in the control subjects ($r = 0.85, P < 0.001$).

LPL activity returned to the lower level present before weight loss after a gain in weight to the previous obese state. The elevation of enzyme activity in the weight-reduced state and the fall with regained weight indicate that the enzyme activity in adipose tissue is under feedback control.

Others (22–24) who have looked at the change in AT-LPL activity with weight loss have found a decline. In their studies, however, subjects were evaluated while still hypocaloric. In two subjects studied while on the 600-cal diet in our protocol, a fall in AT-LPL activity below that in the basal obese state was confirmed (unpublished data), indicating that the hypocaloric state during weight loss is different from the eucaloric state after weight loss.

Genetically obese rats (fa/fa) restricted in their food

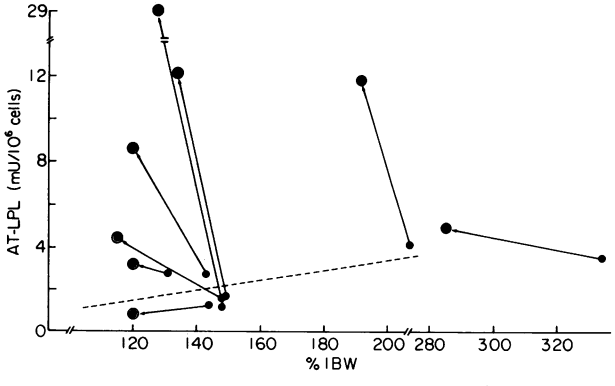

FIGURE 2 Study subjects before (●) and after weight loss (◉) are compared to the regression line (– – –) for the 17 weight-stable control subjects of Fig. 1. Significant correlations between AT-LPL activity and %IBW ($r = 0.71, P < 0.05, n = 7$) and between AT-LPL activity and fat cell size ($r = 0.81, P < 0.01, n = 8$) were present in the obese subjects before weight loss.

TABLE II
Long-term Follow-up

	Months	Weight	IBW	Fat cell size	AT-LPL
		kg	%	*μg TG/cell*	*mU/10⁶ cells*
2 Obese	0	103	148	0.576	1.44
Reduced	3.5	80	115	0.573	4.45
Obese	15	103	148	0.634	1.25
6 Obese	0	99	143	0.770	2.01
Reduced	4	83	120	0.678	8.42
Obese	18	103	149	0.770	2.59
7 Obese	0	95	148	0.484	1.12
Reduced	3.5	84	130	0.571	29.03
Obese	12	106	164	0.732	2.95
8 Obese	0	252	333	0.743	3.49
Reduced	7	213	285	0.560	4.97
Reduced	17	234	313	0.720	5.74

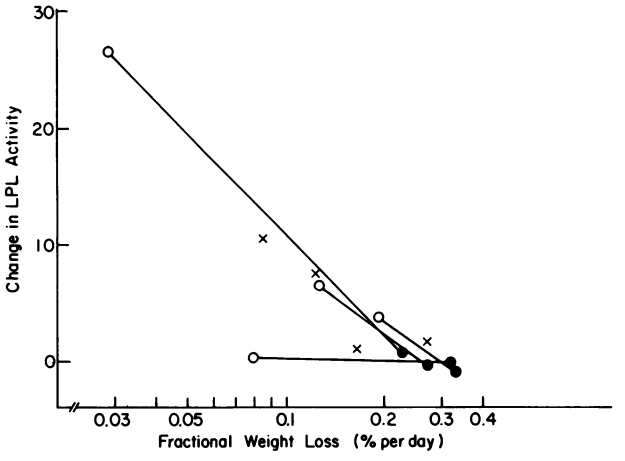

FIGURE 3 Comparison of the change in AT-LPL activity and the fractional rate of weight loss. Fractional weight loss was calculated as the change in weight divided by the initial weight times 100, divided by the number of days on the hypocaloric diet. Four subjects remained on the hypocaloric diet throughout the study (×). Four subjects were weight stabilized at the midpoint of the study to divide the periods of weight loss into an initial period of weight loss (●) and a second period of weight loss (○). The change in AT-LPL activity was inversely correlated with fractional weight loss (× and ●: $n = 8$, $r = -0.81$, $P < 0.01$; × and ○: $n = 8$, $r = -0.71$, $P < 0.05$).

intake have been shown to have elevated AT-LPL activity and greater fat mass when compared with lean (fa/fa) littermates of equal weight (25). In fact, food-restricted fat rats had AT-LPL activity similar to or greater than the *ad libitum* fed fat rats that had greater body weight and fat mass. The elevation in AT-LPL activity has been shown to be the first detectable metabolic abnormality in these genetically obese animals, occurring 2 wk before any detectable increase in body weight, fat cell size, or insulin concentration. These authors (25) speculate that the high AT-LPL activity can, by predisposing to excess triglyceride storage in fat tissue, cause relative obesity even without hyperphagia.

Because the uptake of lipoprotein triglyceride fatty acids by human adipose tissue has been shown to be correlated with the AT-LPL activity present in that tissue (26), it is likely that the elevated AT-LPL activity seen with weight loss would predispose to increased triglyceride storage. How the increase in enzyme activity and the increase in fat cell size, adipose mass, and the impressive tendency of patients to regain lost weight are related is not known. Preferential shunting of calories away from other tissues to adipose tissue during eucaloric intake, as occurs in the fa/fa rat (25), cannot be directly evaluated in the present study. The inverse relationship between the increase in AT-LPL activity and the fractional rate of weight loss in the subjects in the present study indirectly indicates that the enzyme is important in the maintenance of adipose mass. The increase in enzyme activity may account for some of the decreased rate of weight loss commonly seen with a prolonged hypocaloric diet.

To help determine the functional significance of the increase in AT-LPL activity seen in the weight-stable, reduced men in the present study, VLDL-TG and HDL cholesterol were measured before and after weight loss. It is well known that one of the primary effects of the AT-LPL enzyme is to clear circulating triglyceride from the plasma (12) and that individuals with low AT-LPL activity have elevated triglyceride levels (27, 28). Most subjects have lower triglyceride levels and rates of VLDL-TG synthesis after weight loss (7). In the present study VLDL-TG was lower in seven subjects and higher in one subject after weight loss. Both an increase in AT-LPL activity and a decrease in the rate of VLDL-TG synthesis (7) could account for a decrease in triglyceride levels after weight loss. Subjects with comparable obesity in the study of Olefsky et al. (7) had a decrease in the rate of VLDL-TG synthesis with weight loss. However, most of those subjects also had an increase in the rate of fractional removal of plasma triglyceride, compatible with an increase in AT-LPL activity. It is probable that both effects occur; which is predominant was not determined.

Plasma HDL cholesterol levels have recently been correlated with AT-LPL activity (29), and in the present study a significant rise in the HDL cholesterol was found for the group after weight loss, even though the response was quite variable. This rise in HDL cholesterol in the stable weight-reduced state confirms a previous report (30) and may be mediated by the concomitant change in AT-LPL activity (31).

We conclude that AT-LPL activity per cell is elevated in obesity and further increases after weight loss. The increase in AT-LPL persists in a stable reduced population 4–28 mo after initial weight loss. In subjects who regained their lost weight, the AT-LPL decreased to the previous obese level. We postulate that the increase in AT-LPL activity seen after weight stabilization at a reduced weight is a counterregulatory mechanism predisposing to increased triglyceride storage in adipose tissue and is involved in the reattainment of a "set point" for weight or fat cell size.

ACKNOWLEDGMENTS

We thank Martha Kimura and Steve Hashimoto for their expert technical assistance and Sharon Kemp for her skilled secretarial assistance.

These studies were performed in the Clinical Research Center at the University of Washington Hospital. Computational assistance was provided by CLINFO computer system. This work was supported by National Institutes of Health grants AM 02456 and HL 18687.

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