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# Effects of Weight Reduction, Exercise, and Diet Modification on Lipids and Apolipoproteins A-1 and B in Severely Obese Persons*

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*We assessed the lipid and apolipoprotein effects of hypocaloric dieting, increased physical activity, and dietary modification in severely overweight adults (body mass index [BMI] 43.05 kg/m$^2$). The 34 women and four men enrolled in the ambulatory weight control program donated blood before, during, and after hypocaloric dieting (420 kcal/day).*

*Mean values before dieting included cholesterol of 223 mg/dL, high-density lipoprotein (HDL) cholesterol of 43 mg/dL, and cholesterol/HDL cholesterol of 5.90. This placed our subjects at high risk for coronary artery disease. Other values included triglycerides of 138 mg/dL, apolipoprotein A-1 of 152 mg/dL, and apolipoprotein B/apolipoprotein A-1 of 0.64. Significant reductions during hypocaloric dieting included mean cholesterol of 171 mg/dL, triglycerides of 99 mg/dL, and apolipoprotein A-1 of 120 mg/dL. During weight maintenance (mean BMI 36.08 kg/m$^2$), significant reductions compared to baseline included a mean cholesterol of 204 mg/dL and cholesterol/HDL cholesterol of 4.60. Also, a significant increase occurred in HDL cholesterol (51 mg/dL), but a nonsignificant elevation was observed in apolipoprotein A-1 (180 mg/dL). In four subjects, discordant ratios of cholesterol/HDL cholesterol or apolipoprotein B/apolipoprotein A-1 were seen, and one ratio improved in two subjects despite relapse of obesity. Changes in both HDL composition and HDL particle concentration may explain elevations of HDL cholesterol and apolipoprotein A-1 after dieting. Discordance between lipid and apolipoprotein ratios may occur. Improvement in lipids or apolipoproteins may be seen despite regained weight. (Henry Ford Hosp Med J 1987;35:216-20)*

Data from the National Health and Nutrition Examination Surveys (1,2) show that obese persons are more apt to have hypertension, diabetes mellitus, or hypercholesterolemia (1). Even children who are more obese have higher levels of the adverse lipids, low-density lipoprotein (LDL) cholesterol and triglycerides, and lower levels of the favorable lipids, high-density lipoprotein (HDL) cholesterol (2). The association of obesity with dyslipidemias, diabetes mellitus, and hypertension may be responsible for an increased risk of coronary artery disease (CAD) in obese adults (3).

An important question is to what extent will obese persons who attain a stable, lower weight manifest reduction in these risk factors? The effects of dietary weight reduction upon the dyslipidemia of the obese have been studied in 282 subjects of diverse degrees of adiposity, under various dietary regimes, and using several different measures of lipoprotein metabolism (4-15).

The stability of changes in lipoproteins is most important from the standpoint of whether reduced CAD risk may be obtained. A smaller number of obese persons (112) had their lipids evaluated after they attained a stable, lower weight (6-9,11-15). Measurement of apolipoproteins (Apo) A-1 and B, the major protein components of high- and low-density lipoproteins, respectively, is fundamental to better understand the effects of weight reduction on lipoprotein physiology. However, only one report has included Apo A-1 and B measurements in long-term follow-up (11). This report describes our experience in measuring serum lipids and Apo A-1 and B in a group of severely overweight adults who attended an ambulatory intensive weight reduction program. The objective was to assess the effects of acute weight loss brought on by a very low calorie diet (VLCD). Later, the same measures were repeated when the subjects were on a weight maintenance diet after a stable, lower weight had been achieved.

## Experimental Subjects

The original patient population consisted of predominantly white, middle-class adults who were severely overweight (1). The subjects recruited included 34 women (aged 25 to 60 years, mean 42 years) and four men (aged 32 to 50 years, mean 42 years). They agreed to follow the program and to donate blood specimens as necessary to monitor the metabolic effects of weight reduction. Of the women, all except three were premenopausal, four were on estrogen or progesterone prepara-

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**Table 1**
**Weight and Body Mass Index at Baseline, Phase 2, and Phase 3***

| | Baseline | Phase 2 | Phase 3 |
|---|---|---|---|
| Weight (kg) | 116.38 (88-191) | 103.04 (171-165) | 94.76 (74-127) |
| Body mass index ($kg/m^2$) | 43.05 (35-55) | 37.88 (29-56) | 36.08 (30-51) |
| Weeks | | 8.76 (2-16) | 33.04 (12-88) |
| kg lost | | 13.34 | 21.62 |

*Numbers are means with ranges in parentheses.

tions, and six were on a thiazide diuretic or beta blocker before dieting. Of the men, two were on a thiazide diuretic before dieting. Thiazide diuretics were discontinued in all patients after dietary treatment began.

## Materials and Methods

After a detailed history, physical examination, electrocardiogram, and Minnesota Multiphasic Personality Inventory, fasting serum was obtained and assayed for triglyceride, cholesterol, and HDL cholesterol using standard methods (16-18). Total serum Apo A-1 and B were assayed with a commercial assay by ligand combined with nephilometry (19,20). All subjects met the criteria for need and safety of a VLCD. The VLCD is defined as less than 800 kcal/day. All subjects signed an informed consent that outlined potential hazards and the need to comply rigidly with our instructions.

Dietary intervention occurred in three phases. Phase 1 was a 1,000 to 1,200 kcal/day balanced-deficit diet that lasted two to three weeks. Phase 2 was a VLCD diet including 420 kcal/day, 70 g protein, 30 g carbohydrate, 2 g fat, and vitamin and mineral supplementation (Optifast 70®, Sandoz Corporation, NY). The phase 2 diet was to be continued for 16 weeks or until goal weight was achieved. Goal weight was defined by the subject and physician as being a desirable and achievable weight, typically 10% to 20% over ideal body weight. Phase 3 was a 1,000 to 1,200 kcal/day balanced-deficit diet (20% protein, 50% carbohydrate, 30% fat with a P/S ratio of 1) that resulted in weight maintenance at the termination of rapid weight loss (phase 2). Blood specimens were obtained at baseline, at the termination of phase 2, and during weight maintenance (phase 3). Phase 3 blood was drawn when the subject was eating 1,000 or more kcal/day and had not lost weight for two or more weeks.

Several subjects had difficulty adhering strictly to this dietary program. Therefore, only baseline and phase 2 data are available for 12 subjects. Some subjects interrupted phase 2 for a variable time and then returned. Others continued the dietary therapy, without hiatus, for up to 36 weeks. In addition to dietary therapy, all subjects received a series of weekly lectures and support sessions to enhance compliance, to educate about healthy and unhealthy eating behaviors, and to encourage exercise in the form of walking 20 to 30 minutes three or more times weekly. Fasting serum was obtained in most subjects during phases 2 and 3. Several subjects donated blood six or more hours after their last meal rather than in the fasting state.

Statistical analysis included calculation of mean, range, and standard deviation. With paired *t* tests, comparisons were made between baseline to phase 2, baseline to phase 3, and phase 2 to phase 3. The Bonferroni method was used to minimize the chance of incorrectly claiming significance, and the P-value was set for groups of three tests at 0.017 and for two tests at 0.025.

**Table 2**
**Serum Lipids and Apolipoproteins Before, During, and After Acute Weight Loss***

| | Baseline | Phase 2 | Phase 3 |
|---|---|---|---|
| Cholesterol | 223 (37)† | 171 (27)‡ | 204 (26)‡ |
| Triglycerides | 138 (29) | 99 (22)‡ | 114 (18) |
| HDL Cholesterol | 43 (19) | 41 (8) | 51 (14)‡ |
| Apo A-1 | 152 (17) | 120 (10)‡ | 180 (12) |
| Apo B | 95 (17) | 61 (9) | 98 (11) |
| Cholesterol/ HDL Cholesterol | 5.90 (17) | 4.95 (9) | 4.60 (12)‡ |
| Apo B/Apo A-1 | 0.64 (17) | 0.52 (9) | 0.56 (11) |

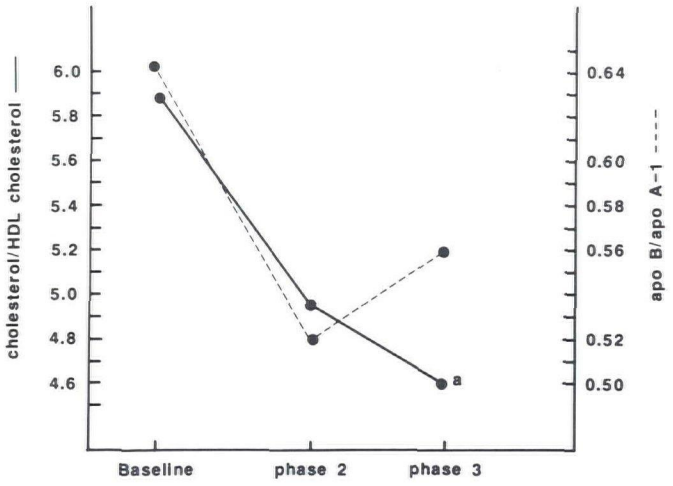
*All values are expressed as the mean in mg/dL.
†Number in parentheses indicates number of subjects.
‡$P < 0.017$ for baseline to phase 2 or baseline to phase 3.

## Results

Weight data, reviewed in Table 1, showed a severely overweight sample of subjects, mean BMI 43.05 $kg/m^2$. During rapid weight reduction (phase 2), a mean of 13.34 kg was lost over an 8.76-week period before phase 2 blood was drawn. When weight maintenance was achieved (phase 3), an average of 21.62 kg had been lost over a mean of 33.04 weeks.

Baseline lipid and apolipoprotein data for all subjects are listed in Table 2. Mean cholesterol was over the 75th percentile for both men and women, according to Lipid Research Clinics' normative data (21). Mean HDL cholesterol was within two standard deviations of Lipid Research Clinics' norms, but was below ideal for both men and women according to Framingham norms of 55 mg/dL for women and 45 mg/dL for men (22). A very high percentage of the women (6/17 [35%]) had HDL cholesterol values below 37 mg/dL (34 mg/dL is the fifth percentile). Mean cholesterol/HDL cholesterol of 5.90 placed our subjects in the same category as men with CAD (mean 5.8) or women with CAD (mean 5.3) (23). No normative data exist for Apo A-1 and B measurements. However, the mean Apo A-1 of 152 mg/dL would not be considered depressed, and thus increasing the CAD risk, when compared to men with CAD or female relatives of men with CAD (24,25). Likewise, the mean Apo B of 95 mg/dL was not elevated, and so indicative of increased CAD risk, compared to available comparison data with female relatives of CAD victims or putatively high-risk women with noninsulin-dependent diabetes mellitus (25,26).

The effect of a VLCD and a lower, stable weight on the same measures is also summarized in Table 2. Statistical analysis using paired *t* tests permitted comparison of changes in mean response from baseline to phase 2, baseline to phase 3, and phase 2 to phase 3. The VLCD (baseline versus phase 2) was associated with significant reductions in cholesterol, triglycerides, and Apo A-1. Statistically insignificant reductions were seen in Apo B, cholesterol/HDL cholesterol, and Apo B/Apo A-1. A stable, lower weight (baseline versus phase 3) was associated with statistically significant reductions in cholesterol and cholesterol/HDL cholesterol. A statistically significant increase was seen in HDL cholesterol.




*Figure—Changes in cholesterol/HDL cholesterol and apolipoprotein B/apolipoprotein A-1. (a = P<0.017.)*

Comparison of the cholesterol/HDL cholesterol ratio to that of Apo B/Apo A-1 revealed corresponding reductions in both during the VLCD (phase 2) (Figure). These reductions obtained statistical significance for cholesterol/HDL cholesterol only during weight maintenance (phase 3).

Review of individual values of cholesterol/HDL cholesterol and Apo B/Apo A-1 before and after a stable, usually lower, weight had been achieved (baseline versus phase 3) showed that one or both ratios fell in most subjects (9/12 [75%]) (data not shown). In subjects 1 and 2, substantial weight reduction was associated with reductions in cholesterol/HDL cholesterol while Apo B/Apo A-1 increased (Table 3). In subjects 3 and 4, cholesterol/HDL cholesterol increased and Apo B/Apo A-1 decreased after weight reduction. In subject 4, reductions in Apo B/Apo A-1 occurred even though she regained weight and increased her ratio of cholesterol/HDL cholesterol. In subject 5, a reduction in cholesterol/HDL cholesterol was observed although all the weight lost had been regained.

## Discussion

The dyslipidemia of a group of severely overweight, middle-class Americans and the effect of both rapid weight reduction with a VLCD and maintenance of a lower weight upon this dyslipidemia have been described. In addition to diet, the therapy included behavior change instruction to encourage more healthy eating behaviors and moderate exercise. Most subjects were very sedentary before the program, but a majority reported the equivalent of 20 minutes of walking three or more times weekly by the period of weight maintenance. Also, most subjects were eating a diet high in fat and calories before the program, but dietary interviews revealed efforts to restrict fat and total calories by the time they had entered weight maintenance. Because of the complex changes that occurred in subjects' weight, physical activity, and the composition of the diet, our results must be interpreted as the consequence of all these changes, rather than loss of body weight per se.

At baseline, serum lipid values suggested that the sample as a whole may have a higher-than-average CAD risk. The mean cholesterol of 223 mg/dL was over the 75th percentile by Lipid Research Clinics' norms (21), and would warrant dietary treatment according to the Consensus Conference on Lowering Blood Cholesterol (27). The mean HDL cholesterol of 43 mg/dL placed both our women and men subjects at increased CAD risk according to Framingham data (22), and 35% of the women had HDL cholesterol values below 37 mg/dL (the fifth percentile is 34 mg/dL) (21). The mean cholesterol/HDL cholesterol ratio of 5.90 corresponded to studies of men and women subjects who have CAD (23).

During rapid weight reduction, significant drops were observed in mean cholesterol (223 to 171 mg/dL or 23%) and triglycerides (138 to 99 mg/dL or 28%) without a corresponding drop in HDL cholesterol. Significant reductions in cholesterol and LDL cholesterol have been frequently reported immediately after a period of hypocaloric diet-induced weight loss (2,4,8, 11,14,15). Only two studies have reported VLDL and LDL concentrations of triglycerides and cholesterol after acute weight reduction. Wolf and Grundy (14) described a significant reduction in cholesterol and VLDL cholesterol, but not LDL cholesterol. Avogaro et al (4) described reduction in VLDL, LDL, and total cholesterol. Therefore, without direct measures we cannot specify whether reductions in VLDL or LDL cholesterol, or both, contributed to the dramatic reduction in cholesterol. Significant reductions in triglycerides have also been frequently reported immediately after a period of hypocaloric diet-induced weight loss (2,4,13-15). The reduction in triglycerides could be due to increased adipose tissue lipoprotein lipase activity (28), perhaps associated with a reduction in VLDL triglyceride synthesis (29). Reduction in cholesterol may be related to a reduction of dietary fat and cholesterol on the VLCD (30). Reduced cholesterol and LDL cholesterol may then be a consequence of increased hepatic LDL receptors (31).

**Table 3**
**Discordant and Unexpected Changes in Ratios Before and After Weight Reduction**

| | Cholesterol/HDL Cholesterol | | Apo B/Apo A-1 | | Body Mass Index (kg/m²) | |
|---|---|---|---|---|---|---|
| Subject (Age/Sex) | Baseline | Phase 3 | Baseline | Phase 3 | Baseline | Phase 3 |
| 1 (52/F) | 3.17 | 3.00 | 0.36 | 0.38 | 39 | 31 |
| 2 (32/M) | 6.58 | 4.20 | 0.25 | 0.50 | 55 | 35 |
| 3 (58/F) | 2.86 | 2.98 | 0.46 | 0.30 | 51 | 41 |
| 4 (49/F) | 4.02 | 4.31 | 0.75 | 0.50 | 38 | 37 |
| 5 (25/F) | 7.20 | 4.49 | | | 43 | 43 |

Phase 3 means after rapid weight reduction.

During rapid weight reduction, a significant drop was also observed in Apo A-1 (152 to 120 mg/dL or 21%), but nonsignificant reductions occurred in Apo B (95 to 61 mg/dL or 36%) and in the ratio of Apo A-1/Apo B (0.64 to 0.52 or 19%). The reduction in Apo A-1, the major protein component of HDL, occurred without a corresponding reduction in HDL cholesterol. This suggests that HDL particles may have increased their carriage of cholesterol. Two prior studies have shown an increase in both Apo A-1 and HDL cholesterol during or immediately after hypocaloric diet-induced weight loss (4,11). By contrast, Apo B, the major protein component of LDL, and cholesterol both fell in our subjects. This observation is consistent with prior observations of the acute effects of hypocaloric dieting on Apo B and cholesterol. During weight maintenance at a lower weight, cholesterol, triglyceride, and HDL cholesterol rose from their nadirs during the weight loss phase. Cholesterol remained significantly lower than baseline (223 to 204 mg/dL or 9% lower). Triglycerides were not significantly lower than baseline (139 to 114 mg/dL or 17% lower). HDL cholesterol rose significantly over baseline (43 to 51 mg/dL or 19% higher). Reductions in cholesterol after weight loss have frequently been reported, but statistical significance, compared to baseline, has only been reported once before (8). Triglycerides also commonly fall, but significance has been achieved in only three reports (12,14,15). HDL cholesterol elevation, to a similar, significant degree, has been more commonly reported (6-9,11,12,14).

We obtained reports of increased physical activity and dietary reductions in total and saturated fat, compared to baseline. These factors may have acted in concert with weight reduction to elevate HDL cholesterol in our subjects. In obese, sedentary men, increased physical activity or weight loss has been shown to increase HDL cholesterol (32). In hypercholesterolemic men, reduction in total and saturated fat has been associated with increased HDL cholesterol (33). We did not observe an inverse relationship between triglycerides and HDL cholesterol during weight reduction. Others have shown that elevations of HDL cholesterol with weight reduction in the obese are independent of triglycerides (6). The elevation of both HDL cholesterol and Apo A-1 during refeeding suggests that HDL particle concentration may increase after hypocaloric dieting in the obese.

During weight maintenance at a lower weight, Apo A-1 and B rose from their nadirs to levels not significantly different from baseline levels. Nonetheless, a more favorable apolipoprotein profile was suggested by the increase in Apo A-1, baseline 152, to phase 3, 180 mg/dL or 18% higher, and reduction in the ratio of Apo B/Apo A-1, baseline 0.64, to phase 3, 0.56 or 13% lower. Sorbis et al (11) described a significant elevation of Apo A-1 two to three weeks after a five-week period of weight loss. We are aware of no similar reports and conclude that available data support a modest increase in Apo A-1 levels after a stable, lower weight has been obtained in obese persons.

Ratios of cholesterol/HDL cholesterol and the like are useful in assessing risk for CAD (23). Two studies have shown that apolipoproteins may be even more useful (24,34). In four subjects, we observed discrepancies between cholesterol/HDL cholesterol and Apo B/Apo A-1. For the same subjects, the cholesterol/HDL cholesterol ratio fell when Apo B/Apo A-1 rose or vice versa. Which ratio should we believe, and how should we counsel our patients? More data are needed about the effects of risk factor intervention on both lipids and apolipoproteins.

Measures of lipoprotein metabolism during hypocaloric dieting may support a clinical impression that the patient is complying with the dietary regimen. However, these measures are not stable and may not correspond to values obtained after a lower weight has been achieved and maintained. The present data have shown reductions in cholesterol, increases in HDL cholesterol, and reductions in the ratio of cholesterol/HDL cholesterol after a stable, lower weight had been achieved. These favorable changes should argue well for our subjects' CAD risk if they persist over time. Two of our subjects showed improvement in the ratios of cholesterol/HDL cholesterol or Apo B/Apo A-1 despite their having regained most of the weight lost. If this last observation can be confirmed, a more favorable outlook in terms of CAD risk may be expected for the obese person who returns to weight reduction therapy even after repeated failures to keep the weight off.

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