# Role of Body Fat Loss in the Exercise-Induced Improvement of the Plasma Lipid Profile in Non–Insulin-Dependent Diabetes Mellitus

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**The study was designed to examine the impact of exercise training on the plasma lipid profile in non–insulin-dependent diabetes mellitus (NIDDM) and, more particularly, to determine the relationship between changes in body fat mass and changes in the lipid profile. Eleven men with NIDDM exercised for 1 hour thrice per week on an ergocycle over a 6-month period at 60% maximal oxygen uptake ($\dot{V}O_2$max). Diet and hypoglycemic agents were kept constant throughout this period. $\dot{V}O_2$max, body composition, fasting plasma glucose and insulin levels, glycosylated hemoglobin, and the lipid profile were determined at baseline, in the middle (3 months), and at the end (6 months) of the training program. When the subjects were considered as a group, the only significant effect of training was on $\dot{V}O_2$max, which increased from 32.2 ± 1.2 mL/kg/min at baseline to 38.0 ± 1.7 mL/kg/min at 6 months ($P < .001$). When the data were analyzed on an individual basis, significant associations were observed between changes in body fat mass and changes in the low-density lipoprotein (LDL) cholesterol/high-density lipoprotein (HDL) cholesterol ratio ($r = .62$, $P = .04$) or triglyceride (TG) levels ($r = .64$, $P = .03$). These data indicate that fat loss, not training per se, favorably alters the lipid profile of subjects with NIDDM who participate in an aerobic physical-conditioning program.**



CORONARY ARTERY DISEASE (CAD) stands as the major cause of death in subjects with non–insulin-dependent diabetes mellitus (NIDDM).[1] Although NIDDM may be considered an independent risk factor for CAD,[2] the increased prevalence of other cardiovascular risk factors in subjects with NIDDM also contributes to their higher mortality rate from CAD. Dyslipidemia is known to play a major role in the appearance and progression of CAD.[3] Moreover, dyslipidemia is frequently found in subjects with NIDDM.[4] Prevention or correction of alterations in the plasma lipid profile thus constitutes an important goal in the long-term management of subjects with NIDDM.[5]

In nondiabetic subjects, aerobic physical conditioning represents one of the available nonpharmacologic approaches to improve an altered plasma lipid profile[4]; in particular, exercise training may be beneficial by increasing high-density lipoprotein (HDL) cholesterol levels and decreasing low-density lipoprotein (LDL) cholesterol and triglyceride (TG) levels (see Després and Lamarche[6] for review). In non-obese individuals, the training-induced improvement in the lipid profile has been achieved in the absence of weight loss,[7,8] suggesting that exercise training per se plays a beneficial role in lipid metabolism. However, the well-randomized study by Wood et al,[9] comparing the effect of weight loss through diet or exercise in overweight men, led to the conclusion that weight loss, particularly the decrease in body fat mass, best explained the improvement in HDL cholesterol and TG levels observed during treatment.

Studies in subjects with NIDDM have led to controversial results. Studies in which exercise training was associated with a weight loss have always shown a concomitant improvement in the plasma lipid profile.[10-12] On the other hand, when no changes in body weight were present, exercise training was associated with either a slight improvement[13-15] or no change at all[16-19] in the plasma lipid profile. Although body fat mass has been measured[14,15,18] or estimated[11,16] in some of these studies, the relationship between training-induced changes in body fat mass and changes in plasma lipid subfractions in subjects with NIDDM has not yet been reported.

Therefore, this study was designed to evaluate in men with NIDDM the impact of a 6-month training program on the plasma lipid profile and body composition and, more specifically, to examine the relationship between the changes in plasma lipid subfractions and changes in body fat mass.

## SUBJECTS AND METHODS

### *Subjects*

Eleven men with NIDDM with a mean age of 48 years (range, 37 to 59) provided written consent to participate in the protocol, which was approved by the Ethics Committee of Laval University. They had no clinical evidence of diabetic complications or renal, hepatic, or thyroid disease. Before engaging in the protocol, they had a negative treadmill exercise stress test supervised by a cardiologist. None of the subjects used insulin; their diabetes was treated with oral hypoglycemic agents (sulfonylureas alone or combined with metformin). They had not been involved in a regular exercise program for several months before entering the study. The known duration of diabetes ranged from 0.5 to 20 years. None of the subjects had severe dyslipidemia, but most of them had at least one parameter of their lipid profile outside the range of acceptable risk.[5]

### *Measures*

Maximal oxygen uptake ($\dot{V}O_2$max) was determined on an ergocycle beginning with a 5-minute warm-up at 50 W followed by progressive increases of 25 W every 3 minutes to the point of exhaustion. Peak oxygen consumption recorded for 1 minute was considered the $\dot{V}O_2$max. This variable was measured before, in the middle (3 months), and at the end (6 months) of the training

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program. Percent body fat was calculated from body density measured by hydrostatic weighing using the equation of Siri.[20]

### *Training Protocol*

Subjects underwent a 6-month aerobic exercise physical training program. They exercised thrice per week under the direct supervision of a graduate student in physical education. Each session consisted of exercise on an ergocycle (Monark, Stockholm, Sweden) at a workload corresponding to 60% $\dot{V}O_2$max. The intensity level was prescribed and monitored on the basis of heart rate. This was determined using values obtained during the $\dot{V}O_2$max test at the beginning and after 3 months of training. The duration of each exercise session was set at 30 minutes for the first 2 weeks, 45 minutes for the next 2 weeks, and 60 minutes for the remaining 22 weeks. The rate of adherence to the scheduled sessions was good throughout the program, with an overall 84% ± 3% attendance rate.

### *Blood Sample Collection*

Blood sampling was performed at 8 AM after an overnight fast (10 to 12 hours). The subjects were asked to abstain from vigorous exercise for 48 to 72 hours before blood sampling. For fasting plasma glucose[21] and insulin[22] determination, blood was drawn under EDTA, centrifuged immediately at 4°C, and stored thereafter at −20°C until subsequent analysis. Glycosylated hemoglobin was assayed on washed red blood cell hemolysates by the method of Trivelli et al.[23] The plasma lipid profile was determined with standardized methods.[24-26] LDL cholesterol was calculated with the formula of Friedewald et al.[27]

### *Statistical Analysis*

The results are expressed as the mean ± SE. A one-way ANOVA on repeated measures was used to evaluate the effect of training. The Pearson correlation coefficient was used to assess the relationship between variables. $P$ less than or equal to .05 was considered statistically significant.

## RESULTS

Physical and metabolic characteristics of the subjects at the beginning, in the middle, and at the end of the 6-month training program are shown in Table 1. There were no significant changes during the course of the study in body weight, percent body fat, lean body mass, or body fat mass. Similarly, fasting plasma glucose and insulin levels, as well as glycosylated hemoglobin levels, did not change in response to training. However, $\dot{V}O_2$max increased significantly by 8% ($P < .05$) at 3 months and by 18% ($P < .001$) at 6 months of training.

**Table 1. Physical and Metabolic Characteristics Before (baseline), in the Middle (3 months), and at the End (6 months) of the Training Program in 11 Men With NIDDM**

| Characteristic | Baseline | 3 Months | 6 Months |
|---|---|---|---|
| Weight (kg) | 83.1 ± 4.9 | 82.1 ± 4.9 | 82.3 ± 4.8 |
| Body fat (%) | 26.8 ± 1.6 | 26.6 ± 1.8 | 26.5 ± 1.7 |
| Lean mass (kg) | 60.6 ± 3.2 | 59.9 ± 3.1 | 60.2 ± 3.1 |
| Fat mass (kg) | 22.6 ± 2.3 | 22.2 ± 2.5 | 22.1 ± 2.4 |
| Glucose (mmol/L) | 11.1 ± 0.7 | 10.5 ± 0.8 | 11.2 ± 0.6 |
| Insulin (pmol/L) | 109 ± 22 | 111 ± 20 | 111 ± 14 |
| Hemoglobin $A_1$ (%)‡ | 9.9 ± 0.7 | 10.1 ± 0.6 | 10.2 ± 0.7 |
| $\dot{V}O_2$max (mL · kg$^{-1}$ · min$^{-1}$) | 32.2 ± 1.2 | 34.9 ± 1.4* | 38.0 ± 1.7† |

NOTE. Data are the mean ± SE.
*$P < .05$, †$P < .001$: $v$ baseline.
‡Normal range, 5.8% to 7.9%.

**Table 2. Lipid Profile Before (baseline), in the Middle (3 months), and at the End (6 months) of the Training Program in 11 Men With NIDDM**

| Parameter | Baseline | 3 Months | 6 Months |
|---|---|---|---|
| Cholesterol (mmol/L) | 5.1 ± 0.3 | 4.9 ± 0.3 | 5.0 ± 0.3 |
| LDL cholesterol (mmol/L) | 3.81 ± 0.24 | 3.63 ± 0.26 | 3.72 ± 0.23 |
| HDL cholesterol (mmol/L) | 0.85 ± 0.03 | 0.90 ± 0.04 | 0.86 ± 0.04 |
| $HDL_2$ (mmol/L) | 0.30 ± 0.03 | 0.35 ± 0.03 | 0.38 ± 0.04 |
| $HDL_3$ (mmol/L) | 0.56 ± 0.03 | 0.55 ± 0.03 | 0.49 ± 0.03 |
| TG (mmol/L) | 2.24 ± 0.29 | 2.12 ± 0.20 | 2.26 ± 0.24 |
| Apo A1 (g/L) | 1.35 ± 0.06 | 1.44 ± 0.04 | 1.41 ± 0.07 |
| Apo B (g/L) | 1.09 ± 0.06 | 1.05 ± 0.05 | 1.10 ± 0.06 |
| Cholesterol/HDL cholesterol | 6.06 ± 0.35 | 5.60 ± 0.37 | 5.92 ± 0.35 |
| HDL/LDL | 0.23 ± 0.01 | 0.26 ± 0.02 | 0.24 ± 0.02 |
| $HDL_2/HDL_3$ | 0.57 ± 0.07 | 0.67 ± 0.08 | 0.82 ± 0.11 |
| Apo A1/apo B | 1.26 ± 0.06 | 1.40 ± 0.08 | 1.33 ± 0.10 |
| Apo B/LDL cholesterol | 0.29 ± 0.01 | 0.29 ± 0.01 | 0.30 ± 0.01 |

NOTE. Data are the mean ± SE.

The impact of training on the lipid profile is shown in Table 2. There were no significant changes in plasma total cholesterol, LDL cholesterol, total HDL cholesterol or $HDL_2$ and $HDL_3$ subfractions, and TGs. There were also no significant changes in plasma concentrations of apolipoprotein (apo) A1 and apo B. To investigate more subtle changes in the lipid profile, the ratios between some of these variables were also calculated. There were no significant changes in any of the following ratios: total cholesterol/HDL cholesterol, LDL cholesterol/HDL cholesterol, $HDL_2/HDL_3$, apo B/LDL cholesterol, and apo A1/apo B (Table 2).

Although the mean values for these ratios at the end of the training program were comparable to baseline levels, they improved in some subjects and did not improve in others. Changes in the LDL cholesterol/HDL cholesterol ratio were significantly correlated with changes in body fat mass during the training program (Fig 1A). A trend toward a relationship between the changes in this ratio and the change in total body weight was also observed ($r = .587$, $P = .058$). However, such a relationship was not observed between changes in the LDL cholesterol/HDL cholesterol ratio and changes in $\dot{V}O_2$max (Fig 1B). No association was found between the changes in this ratio and changes in glycosylated hemoglobin levels ($r = .212$, $P = .531$) or in fasting plasma levels of glucose ($r = .432$, $P = .185$) or insulin ($r = .228$, $P = .499$).

The relationship between changes in plasma triglyceride levels and changes in physical and metabolic characteristics was also examined. There was a significant correlation between the change in triglyceride levels and change in fat mass (Fig 2A). There was also a trend toward such a relationship between changes in plasma triglyceride levels and changes in body weight ($r = .565$, $P = .070$). Such a relationship was not found with the changes in $\dot{V}O_2$max (Fig

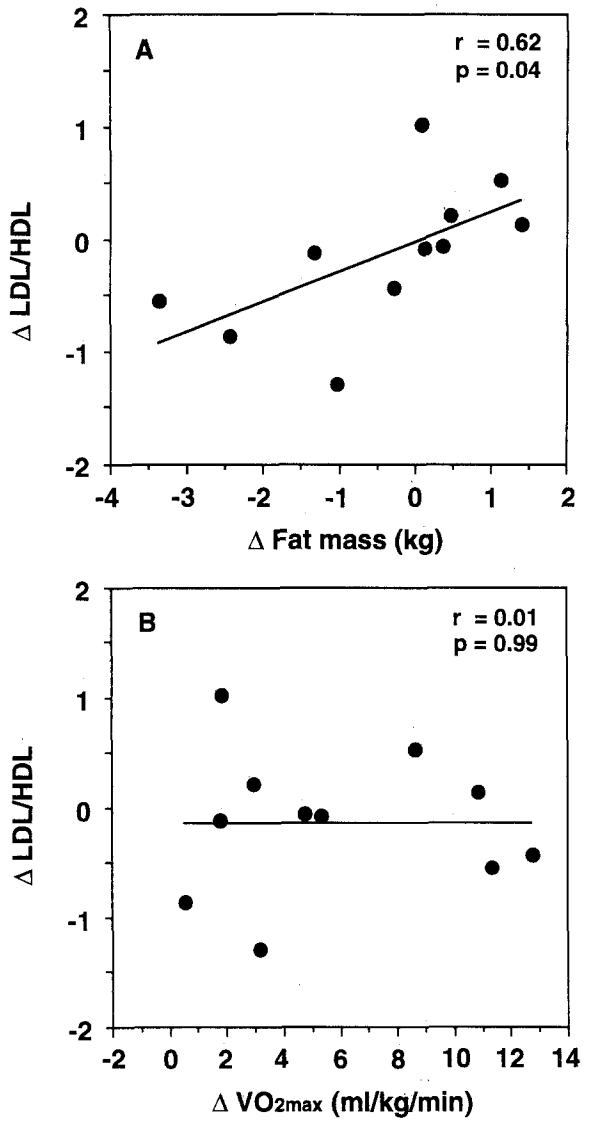


**Fig 1. Relationship between changes (△) in body fat mass (A) or $Vo_2max$ (B) and changes in the LDL/HDL cholesterol ratio.**

2B) or with indicators of glucose homeostasis such as glycosylated hemoglobin levels ($r = .145, P = .670$) or fasting plasma levels of glucose ($r = .434, P = .182$) or insulin ($r = .159, P = .639$).

## DISCUSSION

This study examined the impact of a 6-month training program on the plasma lipid profile of 11 men with NIDDM treated with diet and oral hypoglycemic agents. Diabetic subjects were instructed at the beginning of the program not to modify their usual dietary habits and medication. They were regularly evaluated throughout the program, and no adjustment in medication was needed. Theoretically, the increase in energy expenditure induced by exercise should have led to a loss in body weight or body fat if food intake and nonexercise physical activity had been kept constant. However, such a decrease in fat mass was observed only in five of 11 subjects (Figs 1A and 2A), with the mean value for fat mass at the end of the program being nonstatistically different from that at baseline. This is not very surprising from a clinical standpoint, since it would appear impossible to maintain the same caloric intake over a long period unless strict external dietary supervision is applied. Moreover, it has been shown by Goran and Poehlman,[28] using the doubly labeled water technique, that the direct energy cost of a training program can be offset by a compensatory decline in energy expended in other daily physical activities. Thus, our study is comparable in many respects to those reported in the literature in which no changes in mean body weight have resulted from the training program.[13-19]

There was no improvement with training in any of the plasma lipid fractions or lipoprotein levels (Table 2). This is in agreement with the conclusion reached in some previous studies.[16-19] However, as mentioned before, a few stud-

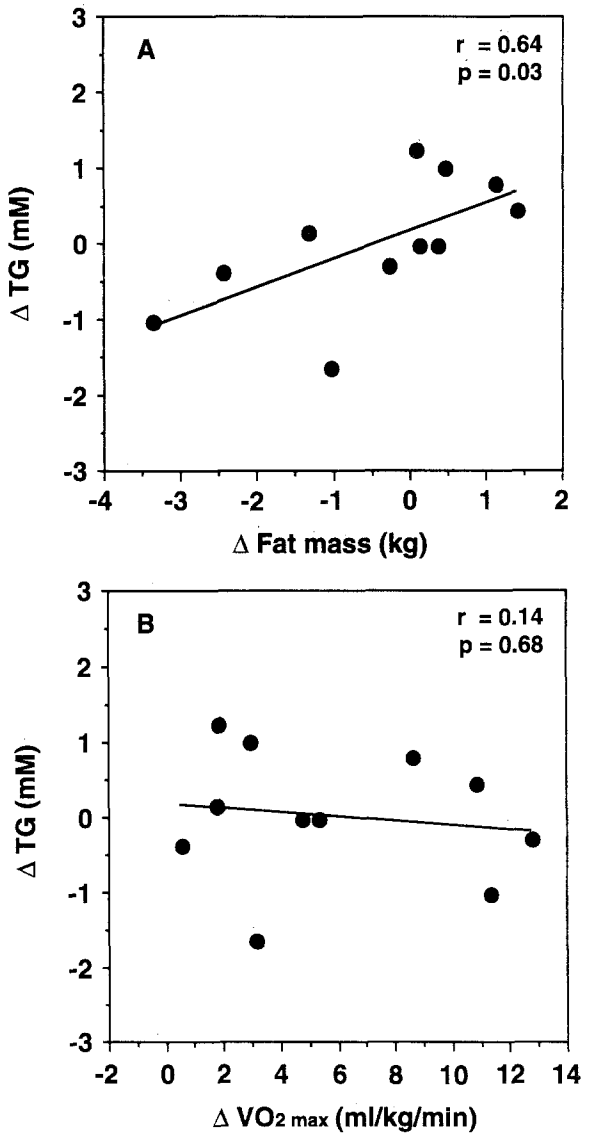


**Fig 2. Relationship between changes (△) in body fat mass (A) or $Vo_2max$ (B) and changes in TG levels.**

ies[13-15] have shown a slight improvement in plasma lipid levels after training in subjects with NIDDM in the absence of weight loss, and this deserves further comment. The first study by Ruderman et al[13] was performed in five subjects and showed a modest 15% decrease in TG levels in four subjects but a large 272% increase in the other one. There was also a modest 8% decrease in total cholesterol levels in these five subjects, but the change was statistically significant only if the data of one subject in whom an 8% increase was observed were excluded from the analysis. In the study by Schneider et al,[14] 20 non-obese men with NIDDM were studied before and after 6 weeks of a physical training program. Lipid data were reported for 16 subjects. No changes in total cholesterol were noted, but a significant improvement in plasma TG levels was found. In a report[29] most likely obtained from the same cohort of subjects, no change in body fat as estimated by skinfold measurements was observed after training, whereas an improvement in metabolic control as assessed by glycosylated hemoglobin $A_1$ levels was found. The possibility that the improvement in TG levels was due to the improvement in glucose homeostasis rather than to a direct effect of training must be considered.[30] Finally, Verity and Ismail[15] have reported data obtained in 10 obese women with NIDDM. These subjects were assigned to either an exercise or a control group; the exercisers were submitted to a fitness program consisting mainly of walking three times per week over a 4-month period, whereas the control group was assessed weekly and received informal communication. There were no changes in body weight or percent body fat in either group. Interestingly, total cholesterol levels decreased significantly and similarly ($\sim 12\%$) in the two groups, presumably because of a decreased fat content in the diet. However, HDL cholesterol levels remained stable in the exercising group, whereas a significant 17% decrease was observed in the control group. Overall, the currently available data, including those reported herein, would not strongly support the hypothesis that training per se has a beneficial impact on the plasma lipid profile of subjects with NIDDM. Indeed, the only trend that can be observed resides in the $HDL_2/HDL_3$ cholesterol ratio, which increased from $0.57 \pm 0.11$ at baseline to $0.82 \pm 0.11$ at the end of training ($P = .077$). This ratio has previously been suggested to be useful in the estimation of CAD risk.[31] In a previous study[32] in obese nondiabetic women, a 14-month training program was also associated with a similar, albeit significant, increase in this ratio. Interestingly, changes in the cholesterol content of these two HDL subfractions or in their ratio were not correlated with body fat loss in either study. The 44% increase in this ratio probably would have reached statistical significance in a larger cohort of subjects. However, the data in Table 2 do not suggest that increasing the number of subjects would have modified the overall interpretation of the impact of training on any of the other lipid subfractions or ratios.

Because some of the available evidence obtained in nondiabetic subjects suggests that fat loss is an important determinant of changes in lipid levels following training,[9] this possibility was tested in our cohort of subjects with NIDDM. Changes in the ratio of LDL cholesterol to HDL cholesterol levels were significantly correlated with changes in body fat mass (Fig 1A). A similar finding was also observed with TG levels (Fig 2A). Such findings are particularly of significance since the changes in body fat mass in the present study were small, ranging from $-3.4$ to $+1.4$ kg. Moreover, the lack of a relationship between these changes in plasma lipid subfractions and changes in $\dot{V}O_2max$, hemoglobin $A_1$, or fasting plasma glucose suggests that this improvement in the lipid profile could not be explained by changes in aerobic capacity or by improved glucose homeostasis. Furthermore, the changes in body fat mass or lipid subfractions could not be explained by differences in body fat mass or other characteristics at baseline (data not shown).

From a clinical standpoint, our data indicate that it is not exercise training per se but rather the fat loss that training may help to produce that best explains the improvement in the LDL/HDL cholesterol ratio and TG levels observed in subjects with NIDDM. Therefore, to obtain these beneficial effects of training, the exercise-induced increase in energy expenditure should be associated with control of food consumption.[33] It is thus not surprising that the more favorable changes in the lipid profile of subjects with NIDDM have been reported when both dietary restriction and exercise conditioning were prescribed and closely monitored.[34,35] Indeed, from the regression line shown in Fig 1A and B, it can be seen that if no change in fat mass occurs, no changes in the LDL/HDL cholesterol ratio or TG levels are expected to be obtained from the training program. This supports the recommendation of the National Institutes of Health Consensus Development Conference[36] that weight loss through dieting is more important than exercise in obese NIDDM subjects.

In summary, this study in men with NIDDM treated with oral hypoglycemic agents has shown that, taken as a group, there was no improvement in the plasma lipid profile after 6 months of exercise training even though aerobic capacity had increased significantly. There were also no changes in mean body weight or fat mass after training. However, when individual changes in the LDL/HDL cholesterol ratio or TG levels were correlated with changes in total body fat mass, statistically significant relationships were observed. These data indicate that fat loss, not training per se, favorably alters the lipid profile of subjects with NIDDM who participate in an aerobic physical-conditioning program.

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