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Associations of Diet and Alcohol Intake with High-Density Lipoprotein Subclasses

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Abstract

Nutritional components from three-day records were studied in association with plasma high-density lipoprotein (HDL) cholesterol, serum HDL2-mass, serum HDL3-mass, and plasma HDL apolipoproteins A-I, A-II, and D concentrations in a cross-sectional survey of 77 adult males. Correlation and regression analyses revealed that the serum concentrations of HDL3 were strongly associated with the intakes of various nutrients, whereas serum HDL2 concentrations showed only weak nutritional associations. Carbohydrate intake correlated negatively with HDL3 concentrations, and alcohol intake correlated positively with serum concentrations of HDL3 and apolipoproteins A-I, A-II, and D. These associations remained significant when adjusted for cigarette smoking, adiposity, and aerobic fitness. HDL2 did not correlate significantly with alcohol intake, total carbohydrates, or starch. HDL-cholesterol concentrations showed two distinct regions of inverse association with intake of sucrose, one involving HDL3 with sucrose between 0 and 10 g/1000 kcal and one involving HDL2 with sucrose above 25 g/1000 kcal. Alcohol, sucrose, and starch together accounted for 36% of the variance of HDL3 concentrations, but less than 5% of the variance of HDL2 concentrations. Thus, serum concentrations of HDL3 and HDL2 show different relationships to major dietary components.

High-density lipoprotein (HDL) cholesterol has been the focus of many clinical and epidemiologic investigations because its elevated concentration in plasma is associated with both reduced incidence of coronary heart disease (CHD) and various factors related to reduced CHD risk such as female gender, endurance physical activity, and alcohol consumption [1]. These associations have led to speculation that HDL may mediate the relationship between these CHD-related factors and the disease.

HDL-cholesterol is carried on two major subclasses of particles, a less-dense HDL2 subclass and a more-dense HDL3 subclass [1] (Fig 1). Apolipoproteins (apo) A-I and A-II account for most of the total HDL protein (approximately 66% and 21%, respectively) [2] with the A-I:A-II ratio tending to be higher in HDL2 than HDL3. The remaining HDL proteins are predominately apolipoproteins C (about 7%), D (approximately 1%, mostly confined to HDL3), and apolipoprotein E (about 1% to 2%) [1-4].

The clinical significance of HDL subclasses has been promoted by recent studies showing significant associations between CHD and specific HDL subclasses. Studies by Miller et al [5] and Ballantyne et al [6] suggest that CHD is related to HDL2-cholesterol concentrations

but is unrelated to HDL3-cholesterol concentrations. However, baseline coronary stenosis in participants of the National Heart, Lung, and Blood Institute (NHLBI) Coronary Intervention Study [7] were inversely related to HDL3-mass concentrations and not related to HDL2-mass concentrations in serums. Differences in the target populations and in laboratory methodology of the three studies may have contributed to these different study results. For example, analytic ultracentrifuge measurement of HDL3-mass in the NHLBI study includes the mass concentration of apolipoprotein A-I, a polypeptide that in men is found primarily in HDL3 [3] and which is reported to correlate with CHD [8], as well as phospholipid and other components whose relationships with CHD is unclear. Assessment of the relationship of HDL subclasses and apolipoproteins to CHD-related factors may further clarify the role of HDL as a mediating agent in CHD if it is confirmed that either HDL2 or HDL3 is preferentially related to CHD risk.

This report examines the interrelationships of diet and alcohol intake with fasting concentrations of plasma HDL-cholesterol, serum HDL2-mass, serum HDL3-mass, and plasma HDL-apolipoproteins A-I, A-II, and D in a cross-sectional sample of middle-aged men. The results of correlational and regression analyses suggest that variations in carbohydrate and alcohol intake were more strongly associated with interindividual differences in serum concentrations of HDL3 than HDL2.

MATERIALS AND METHODS

Subjects

Our report focuses on baseline dietary records and lipoprotein measurements of 81 sedentary but otherwise healthy men, 30 to 55 years old, who later participated in a 1-year exercise trial [9]. Subjects reported to our clinic in the morning after having abstained for 12 to 16 hours from all food and from any vigorous activity. Venous blood samples were drawn in evacuated tubes providing 1.5 mg/mL disodium EDTA and into empty serum tubes, while the subject remained in a sitting position. Plasma and serum were prepared from blood within two hours, and the blood, serum, and plasma were all kept at 4°C. Plasma lipid and lipoprotein cholesterol concentrations were determined by the methods of the Lipid Research Clinics [10]. The method remained “standardized” according to Lipid Research Clinic (LRC) criteria during all analyses. The concentrations of HDL2 and HDL3 in serum (as total mass) were determined by the Donner Laboratory of Medical Physics, University of California at Berkeley by computer analysis of the results of analytic ultracentrifugation [11]. This technique generates a schlieren curve, which describes the distribution of lipoproteins according to their flotation (F) rates, from which concentrations of lipoprotein classes are calculated using the areas under the curve for arbitrarily specified flotation intervals. For the present analysis, HDL2-mass was estimated as the sum of flotation intervals $F_{1.20}$ 3.5–9.0 and HDL3-mass as the sum of flotation intervals $F_{1.20}$ 0–3.5 [11]. Apolipoproteins A-I [12], A-II [13], and D [4] were determined in total plasma samples by radialimmunodiffusion methods.

Percent body fat was estimated by hydrostatic weighing and maximal oxygen uptake calculated from a graded treadmill exercise test [9].

Three-day food records were completed on consecutive days that were randomly assigned so as to insure a proportional number of week days and weekend days. A diet technician reviewed the written records and if unclear verified them with the participants. Records were coded using the Nutrition Coding Center (Minneapolis) code book and rules. Mean total calorie and nutrient intakes over the three days were determined using the computerized food composition tables of the Nutrition Coding Center and an analysis program.

Statistical Calculations

The strengths of the relationships between lipoprotein concentrations and nutrient levels are measured by Spearman's rho correlation coefficients and their forms graphically displayed by data-smoothing procedures. Spearman's correlation coefficients provide a nonparametric test for significant association, have high efficiency when the data are in fact normal, and are robust to outliers. The scatterplot smoothing procedures offer considerable flexibility over linear regression since they do not assume a simple straight-line fit between nutrient levels and lipoprotein concentrations, and may thus reveal important nonlinear relationships. The smoothing procedure applied to these data is described by Cleveland [14] and uses one-half of the data for fitting each point. Partial correlation coefficients are used to adjust these relationships for the potentially confounding effects of cigarette smoking, adiposity, and aerobic fitness.

RESULTS

Distribution of Nutrients and Lipoproteins

Complete three-day diet records were obtained for 77 of the 81 men who volunteered to participate in the study. Average total caloric intake for the group (mean $\pm$ 1 SD) was $2,486 \pm 586$ kcal/d. Their intakes of specific nutrients appear in Table 1 as g/1000 kcal, which corresponds to a diet consisting of $40.5 \pm 6.7\%$ of total calories from fat, $38.1 \pm 8.1\%$ from carbohydrates, $15.8 \pm 2.5\%$ from protein, and $5.6 \pm 5.9\%$ from alcohol, which is typical of the average American diet [15]. HDL-related lipoprotein and apolipoprotein concentrations were 48.7 ± 8.8 mg/dL for plasma HDL-cholesterol, 40.6 ± 31.8 mg/dL for serum HDL2-mass, 231.7 ± 36.8 mg/dL for HDL3-mass, 129.2 ± 15.8 mg/dL for plasma apolipoprotein A-I, 37.2 ± 5.2 mg/dL for apolipoprotein A-II, and 6.5 ± 1.0 mg/dL for apolipoprotein D. The distribution of lipoprotein particles by flotation rate for this group of men is displayed in Fig 1. Seventeen of the men smoked cigarettes.

Correlation Analysis

Although Table 2 examines a large number of correlations, the significant associations all correspond to the relationships predicted from the substantially larger LRC study of HDL-cholesterol and diet in 4,855 subjects [15]. Specifically, the associations between HDL levels and the dietary variables of total calories, protein, and fat (total, saturated, monounsaturated, and polyunsaturated fat) were too weak to produce Spearman's correlation coefficients significantly different from zero for this size sample. Plasma HDL-cholesterol concentrations were positively correlated with alcohol and negatively correlated with sucrose. The associations of HDL with carbohydrates and alcohol appear to be predominantly due to HDL3 rather than to HDL2. Whereas HDL3 concentrations were strongly correlated with total carbohydrates, starch, and alcohol, the correlations between HDL2 and these nutrients were all nonsignificant. Sucrose was negatively correlated with both HDL2 and HDL3; however, these associations are shown to be dose dependent (see following text).

The apolipoproteins A-I and A-II were negatively correlated with sucrose and dietary cholesterol and positively correlated with alcohol. In the case of sucrose, the magnitude of the correlations with apo A-I ($r = -0.26$) and apo A-II ($r = -0.29$) were similar to those obtained for HDL2 ($r = -0.23$), HDL3 ($r = -0.30$), and HDL-cholesterol ($r = -0.22$). Alcohol intake showed strong association with HDL3 ($r = 0.47$) but not with HDL2 ($r = 0.17$). The correlation that alcohol exhibits with apo A-I is intermediate ($r = 0.25$). While stronger correlation of alcohol intake with apo A-II than with apo A-I is consistent with the higher concentration of apo A-II in HDL3 than HDL2 [3], the ratio of apo A-II to apo A-I, which has been used as an index of the relative concentrations of HDL3 to HDL2 [3], was not significantly correlated with alcohol ($r = 0.08$) or with any other dietary variable.

Apolipoprotein D (not included in Table 2) correlated negatively with the proportion of total calories from starch ($r = -0.21$, $P = 0.06$) and positively with the proportion due to alcohol ($r = 0.25$, $P = 0.03$) but its concentrations were not significantly correlated with the reported intakes of other nutrients.

Partial correlation analyses (results not displayed) revealed that most of the nutrient-HDL associations of Table 2 and the nutrient-apo D associations cited previously remain significant when adjusted for smoking (cigarettes per day), adiposity (% body fat), and aerobic fitness (VO_{2max}). Only two of the correlations were made nonsignificant by their adjustment: apo A-I vs. dietary cholesterol and apo A-II vs. sucrose.

Graphic Analysis

The significant associations of Table 2 were further analyzed by scatterplot smoothing procedures to determine whether the form of the nutrient-HDL associations varies with the nutrient level. Figure 2 shows that the relationships of sucrose with HDL-cholesterol, HDL2-mass, and HDL3-mass concentrations are very different in form despite their similar correlation coefficients. The HDL-cholesterol plot shows two distinct regions of inverse association with sucrose, one between 0 and 8 g/1000 kcal and a second above 25 g/1000 kcal. These associations appear to be the consequence of two separate interactions—one involving HDL2-mass, which shows no association with sucrose below 25 g/1000 kcal and a negative association above 25 g/1000 kcal, and a second involving HDL3-mass, which exhibits a negative association with sucrose between 0 and 8 g/1000 kcal and no apparent association above 8 g/1000 kcal.

Figure 3 shows a linear association between alcohol intake (g/day) and HDL3-mass concentrations throughout the range of consumption levels, and no clear relationship between HDL2-mass concentrations and alcohol. The smoothed scatterplots of alcohol v HDL-cholesterol and apolipoproteins A-I, A-II, and D (not shown) were linearly increasing.

Regression Analysis

The dietary intake of alcohol, starch, and sucrose together accounted for 36% of the variance in HDL3-mass concentration but less than 5% of the variance in HDL2-mass concentration (Table 3). The proportions of the variance of HDL-cholesterol and apolipoproteins A-I and A-II accounted for by these nutrients are intermediate to the proportions observed for HDL2-mass and HDL3-mass, again possibly reflecting the origin of HDL-cholesterol and apo A-I and A-II from both HDL2 (which is weakly associated with these nutrients) and HDL3 (which exhibits strong nutritional associations).

The analyses further reveal the significance of the independent associations of alcohol ($P \leq 0.0001$), starch ($P \leq 0.05$), and sucrose ($P \leq 0.05$) with serum HDL3-mass concentrations. The remarkable similarity of the regression coefficients of starch and sucrose suggest that these two carbohydrates have very similar effects on HDL3-mass when adjusted for the effects of alcohol consumption.

Nutritionally Defined HDL Subclasses

A more refined analysis of the association of HDLmass with alcohol and starch intake is provided in Table 4 by correlating the intake of these nutrients with the serum concentrations of each of the 15 individual HDL flotation intervals measured by analytic ultracentrifugation, (Fig 1). For comparative purposes, Table 4 also includes correlations for changes in HDL flotation intervals with exercise level (miles run per week) and exercise-induced body fat loss from data collected as part of a 1-year exercise trial [9]. The correlations reveal very specific associations that exist between the flotation intervals of greater and lesser density and alcohol

and starch intake, change in exercise level, and fat loss. In particular, the cross-sectional correlations with starch and alcohol intake are individually significant for densities between $F_{1,20}$ 0–3.5 and longitudinal correlations with exercise level or exercise-related weight loss are mostly significant for changes in HDL-mass between $F_{1,20}$ 3.0–9.0. The results suggest a physiologic approach to defining HDL subclasses by combining flotation intervals on the basis of their similar associations with nutrients or exercise.

DISCUSSION

Comparison With Other Studies

We have already noted the agreement of our results with the findings of the much larger LRC Visit 2 prevalence study [15] on diet and HDL-cholesterol, ie, a positive correlation with intake of alcohol and negative correlations with sucrose, starch, and total carbohydrates. The LRC also reported a weak association between HDL-cholesterol and dietary cholesterol, which concurs with our finding. The correlations given by the LRC report are generally weaker than those reported by us, which may be due to less-precise nutritional assessment by one-day diet recall questionnaires as compared with our three-day diet records.

The smoothed scatterplots of Fig 2 suggest that different lipoprotein effects may occur at different nutrient levels. Dose-dependent nutritional effects may also partly explain the different results obtained from our cross-sectional studies and three metabolic ward studies. Whereas we found no significant relationship between P/S ratio and HDL-cholesterol concentration, Shepard et al [16] found plasma HDL-cholesterol concentrations to be higher for participants on diets high in saturated fats (P/S ratio = 0.25) v diets high in polyunsaturated fats (P/S ratio = 4.0). Other dietary manipulations by Gonen et al [17] (80% of calories from carbohydrates v normal diet) and Kashyap et al [18] (65% carbohydrate v 15% carbohydrate diets) were interpreted to show that serum concentrations of HDL2- mass were significantly lowered by carbohydrate-enriched diets. Alterations in HDL-cholesterol or HDL subclass levels induced by these extreme dietary perturbations would not necessarily predict the effects of the more modest variations in diet on HDL levels in free-living populations. For example, the P/S ratios of the 77 men of our study were all less than 1.6, and the percentage of total calories derived from carbohydrates was no less than 19% and no greater than 53%. These ranges do not include the nutrient levels studied by Shepard, Gonen, Kashyap and their colleagues.

Alcohol and HDL Subclasses

The positive association between alcohol consumption and elevated HDL-cholesterol has been established from population studies [1,15]. However, it has only recently been shown that serum HDL3-mass concentration is decreased with alcohol cessation in moderate drinkers and increased with resumed drinking, whereas serum HDL2-mass appears to be unaffected by these dietary perturbations [19]; the present cross-sectional correlations are in accordance with these experimental findings and reinforce the generalization of the conclusions.

The observed effect of moderate alcohol intake in elevating HDL3-mass concentrations without apparent influence on HDL2-mass suggests that either HDL3 is not inert in the development of CHD, or the association between reduced incidence of CHD and moderate alcohol intake is due to factors unrelated to HDL2. Taskinen et al [20] reported that in ten male alcoholic subjects who were free of cirrhosis or other alcoholic liver disease, abstinence resulted in a decrease in HDL primarily in the HDL2 fraction; as measured by preparative ultracentrifugation, relative to their initially elevated level. These results, compared with those reported here and elsewhere [19], suggest that physiologic mechanisms that elevate HDL in

alcoholics may differ from those that affect moderate drinkers, although differences in methodology for measuring HDL2 and HDL3 may also be involved.

Nutrient-Sensitive HDL, and Nutrient-Insensitive HDL

Our analyses characterize HDL3 as a nutritionally associated subclass and HDL2 as having a weaker association with the nutritional variables studied here. Variations in dietary intake of carbohydrates and alcohol in the population were chiefly related to variations in HDL3-mass concentrations, and were relatively independent of variations in HDL2-mass concentrations. Alcohol, starch, and sucrose intake together accounted for 36% of the variance for serum HDL3-mass but less than 5% of the variance for serum HDL2-mass. Moreover, correlations computed with respect to the 15 individual HDL subfractions singled out those particles with flotation rates $F_{1.20}$ 0–3.5 as having the common characteristic of positive association with alcohol intake and negative association with starch. Particles of $F_{1.20}$ 3.0–9.0 were distinguished from particles of greater density by their positive correlation with exercise level and negative correlation with changes in body fat. Thus the grouping of HDL flotation intervals on the basis of their specific associations with diet and exercise variables shows agreement with the traditional approximation of HDL3 and HDL2 concentrations. However, there may be further heterogeneity of HDL particles [21] within these two subclasses not distinguished by their associations with these variables. Other studies have reported that female gender, estrogen use, weight loss, and aerobic exercise are primarily associated with HDL2 levels and are independent of, or show less-defined interactions with, HDL3 levels [1]. The differential associations of the HDL subclasses with diet, gender, weight loss, exercise, and sex steroid use suggest that physiologic functions and metabolic pathways may be different for HDL2 and HDL3.

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Fig 1.

Distribution of serum HDL mass concentrations by flotation rates. The curve is drawn by averaging the heights of the individual curves of the 77 men, and the shaded area under the curve is equal to the average serum HDL mass concentration for the group. The predominately HDL₃ and HDL₂ portions of the particle distribution are indicated on the horizontal axis.

Fig 2.

Smoothed scatterplots showing cross-sectional association of plasma HDL-cholesterol, serum HDL2-mass, and serum HDL3-mass concentrations with dietary sucrose. HDL-cholesterol shows two distinct regions of inverse association, one involving HDL3 with sucrose between 0 and 8 g/1,000 kcal, and one involving HDL2 with sucrose above 26 g/1,000 kcal.

Fig 3. Smoothed scatterplot showing cross-sectional association of serum HDL2-mass and HDL3-mass concentrations with alcohol. Alcohol intake (g/d) appears to be linearly related to HDL3-mass but shows no clear association with HDL2-mass concentrations.

Table 1

Average Daily Nutrient Intake for 77 Middle-Aged Men *

Nutrient	Mean $\pm$ SD
Total protein (g/1000 kcal)	39.6 $\pm$ 6.2
Total fat (g/1000 kcal) [†]	45.0 $\pm$ 7.4
Saturated fat (g/1000 kcal)	16.2 $\pm$ 3.3
Monounsaturated fat (g/1000 kcal)	17.4 $\pm$ 3.9
Polyunsaturated fat (g/1000 kcal)	8.1 $\pm$ 2.6
Total carbohydrates (g/1000 kcal)	95.2 $\pm$ 20.2
Sucrose (g/1000 kcal)	15.3 $\pm$ 10.8
Starch (g/1, 000 kcal)	42.5 $\pm$ 13.4
Crude fiber (g/1000 kcal)	1.9 $\pm$ 0.7
Other carbohydrates (g/1000 kcal)	35.6 $\pm$ 13.3
Alcohol (g/1000 Kcal)	8.0 $\pm$ 8.4
Cholesterol (mg/1000 kcal)	189.5 $\pm$ 82.3
P/S ratio	0.5 $\pm$ 0.2

* Three-day diet records were coded using the Nutrition Coding Center (Minneapolis) code book and rules. Mean nutrient intakes were determined using the computerized food composition tables of the Nutrition Coding Center.

[†] Saturated, monounsaturated, and polyunsaturated fat do not sum to the total fat due to the presence of unsaponifiable matter and emulsifiers

Table 2
Cross-Sectional Spearman's Correlations of High-Density Lipoproteins vs. Dietary-Variables in 77 Middle-Aged Men.

	HDL 3-mass (mg/dl)	HDL 2-mass (mg/dl)	HDL-cholesterol (mg/dl)	Apolipoproteins mg/dl	
				A-I	A-II
Calories	-0.12	-0.13	-0.10	-0.16	-0.06
Protein (g/1000 kcal)	-0.01	-0.06	-0.13	-0.01	-0.12
Total fat (g/1000 kcal)	-0.05	-0.01	-0.04	-0.08	-0.16
Saturated fat (g/1000 kcal)	0.01	-0.11	-0.14	-0.16	-0.09
Monounsaturated fat (g/1000 kcal)	-0.16	-0.10	-0.12	-0.16	-0.17
Polyunsaturated fat (g/1000 kcal)	-0.05	0.13	0.13	0.10	-0.06
Total carbohydrates (g/1000 kcal)	-0.30 [‡]	-0.06	-0.12	-0.12	-0.07
Sucrose (g/1000 kcal)	-0.30 [‡]	-0.23 [*]	-0.22 [*]	-0.26 [*]	-0.29 [‡]
Starch (g/1000 kcal)	-0.42 [§]	0.00	-0.13	-0.14	-0.05
Crude fiber (g/1000 kcal)	-0.02	0.14	0.06	0.12	-0.03
Other carbohydrates (g/1000 kcal)	0.19	0.18	0.12	0.19	0.18
Alcohol (g/1000 kcal)	0.47 [§]	0.17	0.31 [‡]	0.25 [*]	0.37 [‡]
Cholesterol (mg/1000 kcal)	-0.09	-0.10	-0.16	-0.26 [*]	-0.26 [*]
P/S ratio	-0.04	0.15	0.17	0.14	0.01

^{*} P<0.05;

[‡] P<0.01;

[‡] P<0.001;

[§] P<0.0001.

Multiple regression analysis of high-density lipoprotein subclasses, cholesterol, and apolipoprotein concentrations as functions of alcohol, sucrose, and starch in 77 middle-aged men.

Table 3

	HDL3-mass (mg/dl)	HDL2-mass (mg/dl)	HDL-cholesterol (mg/dl)	Apolipoproteins mg/dl	
				A-I	A-II
Intercept	257.5	54.8	49.7	134.8	34.0
Alcohol (g/1000 kcal)	1.63	0.04	0.23	0.40	0.24 [†]
Sucrose (g/1000 kcal)	-0.67 [*]	-0.13	-0.01	-0.09	0.05
Starch (g/1000 kcal)	-0.67 [*]	-0.58	-0.15	-0.31	-0.04
Percentage of variance explained	36.15 [§]	4.5	10.9 [*]	13.8 [†]	15.8 [†]

Significance levels for individual coefficients and for the entire model are coded:

- * P<0.05;
- [†] P<0.01;
- [‡] P<0.001;
- [§] P<0.0001.

Table 4

Spearman's correlational analyses of individual HDL flotation intervals in 77 middle-aged men

Intervals (F _{1,20})	Nutritional associations*		Associations with exercise [†]	
	Starch (g/1000 kcal)	Alcohol (g/1000 kcal)	Miles run per week	Δ Percent body fat
8.0–9.0	−0.19	0.14	0.28	−0.48 [‡]
7.0–8.0	−0.03	0.08	0.31 [‡]	−0.45 [‡]
6.0–7.0	−0.02	0.15	0.31 [‡]	−0.48 [‡]
5.5–6.0	0.01	0.16	0.37 [‡]	−0.47 [§]
5.0–5.5	0.02	0.15	0.38 [‡]	−0.43 [§]
4.5–5.0	0.02	0.17	0.35 [‡]	−0.47 [§]
4.0–4.5	0.01	0.16	0.36 [‡]	−0.52 //
3.5–4.0	−0.03	0.18	0.33 [‡]	−0.54 //
3.0–3.5	−0.13	0.25 [‡]	0.32 [‡]	−0.47 [§]
2.5–3.0	−0.29 [§]	0.34 [§]	0.24	−0.37 [‡]
2.0–2.5	−0.37 //	0.42 [¶]	0.11	−0.21
1.5–2.0	−0.34 [§]	0.48 [¶]	−0.15	0.05
1.0–1.5	−0.33 [§]	0.41 //	−0.41 [‡]	0.31
0.5–1.0	−0.35 [§]	0.35 [§]	−0.20	0.19
0.0–0.5	−0.28 [‡]	0.11	0.12	0.07

* N = 77. Cross-sectional correlations for HDL flotation intervals (mg/dL) versus nutrient intake of the present report.

[†] N = 41. Longitudinal correlations for 1-year changes in the HDL flotation intervals (Δmg/dL) v average miles run per week and 1-year changes in percent body fat in men who participated in a 1-year exercise training study:[‡] P<0.05;[§] P<0.01;

// P<0.001;

[¶] P<0.0001.