

Seasonal changes in plasma and aortic lipids in young rats

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Summary. In young non-exercised rats, plasma triglyceride and plasma phospholipid levels increased in summer and low density lipoprotein cholesterol (LDL-C) increased in winter. As for lipids samples from the wall of the aorta, total cholesterol, cholesteryl ester and triglyceride decreased in summer and phospholipid decreased in winter. Exercise diminished the gain in body mass (Δm) and suppressed seasonal changes in the levels of LDL-C and plasma triglyceride. Seasonal changes in the aorta lipids in this case were similar to those found in non-exercised animals. The values of total energy intake (Q) and of $\Delta m \cdot Q^{-1}$ were found to change with season in both non-exercised and exercised rats. Seasonal changes in plasma and in aorta lipids observed in these animals ran in parallel with the respective levels of $\Delta m \cdot Q^{-1}$ and/or of Q. The training effect on the lipid values detected in summer and/or in winter was also found to be dependent on the reduction in $\Delta m \cdot Q^{-1}$ with exercise. In the non-exercised and in the exercised animals, plasma phospholipid was associated with aorta phospholipid and inversely related to aorta cholesteryl ester and aorta triglyceride. The relationship between these estimations suggests that an increase in the plasma phospholipid in summer would remove non-polar lipids from the walls of the aortae.

Key words: Young rats - Exercise - Season - Plasma phospholipid - Aorta lipids - Gain in body mass - Caloric intake

Introduction

Carlson and Lindstedt (1969) and Warnick and Albers (1976) found that serum triglyceride in human subjects increased in winter and decreased summer. In laboratory rats, Mikeska and Petrasek (1977) observed increases of plasma total cholesterol in winter and decreases in summer. On the other hand, in Norwegian reindeer kept in the open air, Larsen et al. (1985a) noted seasonal changes in plasma concentrations of total cholesterol, high density lipoprotein cholesterol [HDL-C] and plasma triglyceride. All of the values in the non-domesticated animals increased in summer and decreased in winter, thereby reflecting aspects of plasma lipid behaviour.

In the human circulatory system, lipid accumulation develops progressively with age (Smith et al. 1967). This would mask seasonal changes in the lipids, in the arteries if there are annual cycles of lipids. Thus, we speculate that samples of the aorta in the young would be the most appropriate ones in which to detect seasonal changes in vascular lipids.

We used young rats and estimated the levels of plasma and lipids in the wall of the aorta in summer and in winter. To take into consideration the physical activities of animals all the year round, we also tested for seasonal changes in the influence of exercise on the lipid levels. The correlation between aorta and plasma lipids was studied, the objective being to elucidate the causal relationship.

Methods

The procedure was similar to that described by Hashimoto and Masumura (1988). Briefly, female Wistar rats, 4-weeks old and about 50 g in mass, were divided into four groups of 14 to 20 rats each (Ns = rats non-exercised in summer; Nw = rats non-exercised in winter; Ws = rats exercised in summer; Ww = rats exercised in winter) and were individually caged at $23^{\circ} \pm 1^{\circ}$ C. All four groups were maintained on laboratory chow [Oriental Yeast Co., MF., Japan: carbohydrate, 530 g· kg⁻¹; lipids, 46 g· kg⁻¹; protein, 241 $g \cdot kg^{-1}$; energy: 3498 kcal \cdot kg⁻¹ (14639 kJ \cdot kg⁻¹)]. The Ws and Ww were exercised on running wheel cages. The winter groups were kept from January to April and the summer groups from June to August. The entire experiment in each season ran for 12 weeks. The animals were decapitated after a 24-h fast on the day of the last run. Blood samples were collected into tubes containing ethylenediaminetetraacetate $(0.1 \text{ g} \cdot 1^{-1})$ and centrifuged to separate the plasma. Thoracic and abdominal aortae were removed and perfused with chilled physiological saline (pH **7.2-**

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7.4). After blotting to remove adherent fluid, adipose tissues on the outer wall of the aortae were excised. Aorta lipids were extracted by the method of Folch et al. (1957). Organic solvents (chloroform and methanol) in these extracts were evaporated by bubbling with N_2 gas at room temperature. Each of the dried preparations was dissolved in 0.5 ml of a solution containing 10% propanol and 10% polyethylene glycol monododecyl ether (Boehringer Mannheim Yamanouchi Co, Japan) to estimate aorta lipids. Triglyceride and phospholipid contents in the aorta samples and in the plasma were determined using a Trigly-Quick BMY Kit (Boehringer Mannheim Yamanouchi Co., Japan) (Takeda et al. 1985) and a PL Kit (Nippon Shoji Co., Japan) (Takayama et al. 1977), respectively. Total and free cholesterol contents in the aortae were measured, using cholesterol and free cholesterol E-test kits, respectively (Wako Pure Chemical Industries Ltd., Japan), with a slight modification of the methods of Allain et al. (1974). Both kits contained 3,5-dimethoxy-N-ethyl-N-(2-hydroxy-3-sulphopropyl)-anilin-Na instead of phenol. The absorbance of the chromogen developed in the assay was estimated at 600 nm, using a spectrophotometer. The [HDL-CI and [HDL-C] plus concentration of low density lipoprotein cholesterol [LDL-C] were determined by the method of Noma et al. (1979). The amounts of LDL-C were calculated from the difference between the values of [HDL-C] and ([HDL-C]+[LDL-C]). Total cholesterol concentration in the plasma was measured, using the same reagents (Noma et al. 1979). Aorta cholesteryl ester contents were calculated from the difference between the levels of aorta total and free cholesterol. The statistical significance between these results was determined using Student's t-test.

Results

The gain in mass (Δm) in each animal was determined from the difference between the initial and final masses. The values decreased by 8.3% in Ws and by 9.4% in Ww, compared to Ns, Nw, respectively. The total energy intake (Q) in each animal was measured to determine the level of $\Delta m \cdot Q^{-1}$. The total number of revolutions (RN) of each exercise wheel in $Ws + WW$ was assessed to calculate the corresponding values of $RN\cdot Q^{-1}$.

Each value of $RN \cdot Q^{-1}$ was used as an indicator of the efficiency in the conversion of the energy intake to the level of exercise. Here, we found a negative correlation between the values of $\Delta m \cdot Q^{-1}$ (g $\cdot kJ^{-1}$) and $RN \cdot Q^{-1}$ ($RN \cdot kJ^{-1}$), thereby suggesting that an augmentation of exercise leads to a decreased level of $\Delta m \cdot Q^{-1}$. The relationship between these estimations in Ws and Ww is as follows:

$$
\Delta m \cdot Q^{-1} = -1.28 \cdot 10^{-4} \cdot \text{RN} \cdot Q^{-1} + 8.24 \cdot 10^{-3}
$$

r = -0.790, P < 0.005

There were seasonal changes in the values of Q and $\Delta m \cdot Q^{-1}$ between Ns and Nw, and between Ws and Ww (Table 1): in all cases higher values of the total caloric intake and lower values of $\Delta m \cdot Q^{-1}$ were observed in summer as compared to winter. If energy consumption in these animals is in inverse proportion to the $\bar{\Delta}m \cdot Q^{-1}$ values, energy output in the non-exercised and in the exercised rats should be higher in summer than in winter because the levels of $\Delta m \cdot Q^{-1}$ decreased in the order of Nw, Ns, Ww and Ws (Table 1).

In Ns and Nw, there were seasonal changes in LDL-C, plasma triglyceride, and plasma phospholipid. Seasonal change in the $[LDL-C] \cdot [HDL-C]^{-1}$ values was also evident. However, the concentrations of other lipids measured in the plasma were unchanged, (Table 2A). The plasma concentrations of LDL-C in Nw increased by 28.6%, whereas those of triglyceride and of phospholipid in this group decreased by 16.4% and by 12.4%, respectively, as compared with the values estimated in Ns. The $[LDL-C] \cdot [HDL-C]^{-1}$ values in Nw increased by 30.2% of the levels determined in the Ns.

With regard to the aortae in the non-exercised animals, we found seasonal changes in all the measured values, except for the level of aorta free cholesterol (Table 2A). The levels of total cholesterol, cholesteryl ester and triglyceride in the aortae of Nw were higher than those in Ns. These increasing percentages in Nw rats amounted to 20.5% for aorta total cholesterol, 269% for aorta cholesteryl ester and 43.2% for aorta triglyceride. The aorta phospholipid contents in Nw diminished by 32.3% compared with Ns.

In exercised rats, there was seasonal change in plasma phospholipid and in the $[LDL-C] \cdot [HDL-C]^{-1}$. However, the levels of other lipids determined in the plasma were unchanged (Table 2 B). The values of [LDL- C [HDL-C]⁻¹ and of plasma phospholipid in Ww rats increased by 26.8% and decreased by 20.9%, respective-

Table 1. Data from non-exercised rats in summer (Ns), non-exercised rats in winter (Nw), exercised rats in summer (Ws) and exercised rats in winter (Ww)

	п		Δm (g)	$(10^4 \cdot kJ)$	$\Delta m \cdot Q^{-1}$ $(10^{-3} \cdot g \cdot kJ^{-1})$	$RN\cdot Q^{-1}$ $(RN \cdot kJ^{-1} \cdot 12 \text{ weeks}^{-1})$
Ns	20	Mean	121 ^a	1.79 ^a	$6.75^{\rm a}$	
		SD.	8.96	0.0782	0.395	
Nw	20	Mean	128 ^b	1.51 ^b	8.50 ^b	
		SD.	11.1	0.0753	0.494	
Ws	14	Mean	111 ^c	2.21 ^c	5.03 ^c	22.5°
		SD	11.8	0.0978	0.530	5.20
Ww	15	Mean	$116^{a,c}$	$1.85^{\rm a}$	6.30 ^d	17.7 ^b
		SD	9.95	0.0871	0.477	3.48

 $\Delta m =$ gain in mass, Q = total caloric intake in each rat; RN = total number of revolutions of each wheel in the exercised group (Ws + Ww); a, b, c, d: means in any column not sharing a common superscript are significantly different $(p < 0.05)$

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ly, as compared with findings in Ws. Thus, physical training apparently suppressed seasonal changes in the levels of LDL-C and triglyceride in the plasma.

Seasonal changes in aorta lipids in the exercised rats were similar to those in the non-exercised group (Table 2). The levels of aorta free cholesterol were unchanged. The aorta total cholesterol, cholesteryl ester and triglyceride contents in Ww animals increased by 11.5%, 104.4% and 59.0% respectively in comparison with Ws. The aorta phospholipid levels in Ww decreased by 33.0%.

To test for the occurrence of seasonal change in the influence of physical training on plasma and aorta lipids, the values seen with each season in exercised rats (Table 2B) were compared with the values of similar lipids in the non-exercised groups (Table 2A). In this assessment, it was noted that exercise in winter reduced plasma phospholipid levels, but exercise in summer had hardly any effect. In both seasons, exercise decreased plasma total cholesterol, HDL-C and plasma and aorta triglyceride. The other lipids determined in the study were little affected by exercise, irrespective of the season.

To elucidate the role of biorhythm in lipid metabolism, we examined the relationship between the values of $\Delta m \cdot Q^{-1}$ and each lipid in the plasma and in the aor**tae of the Ns and Nw and Ws and Ww groups. Table 3A shows these results. The [LDL-C] in Ns and Nw rats** were associated with the levels of $\Delta m \cdot Q^{-1}$. The $\Delta m \cdot Q^{-1}$ ratios in the non-exercised and exercised **groups negatively correlated with the respective values of plasma phospholipid. Thus, it would be expected that seasonal changes in [LDL-C] or plasma phospholipid in the non-exercised and/or in the exercised animals originated from seasonal variation in the values of** $\Delta m \cdot Q^{-1}$. The levels of total cholesterol, cholesteryl es**ter and triglyceride in the aortae in the non-exercised and in the exercised group were related to the corre**sponding values of $\Delta m \cdot Q^{-1}$. These groups had an inverse relationship between the values of $\Delta m \cdot Q^{-1}$ and **aorta phospholipid. We tentatively conclude that seasonal changes in aorta lipids found in the non-exercised and in the exercised animals were the results of** seasonal changes in the levels of $\Delta m \cdot Q^{-1}$.

To examine any possible role attributable to the influence of exercise on lipid metabolism, we studied the relationship between the levels of $\Delta m \cdot Q^{-1}$ and each **lipid in the plasma and in the aortae of both the nonexercised and exercised groups in summer and in** winter (Table 3B). The values of $\Delta m \cdot Q^{-1}$ measured in **summer and in winter were associated with those of plasma total cholesterol, [HDL-C] and plasma and aorta triglyceride. Thus, it follows that the training effect on the lipid values depends on the corresponding levels** of $\Delta m \cdot Q^{-1}$ in exercise. In addition, the levels of $\Delta m \cdot Q^{-1}$ estimated in winter correlated with those of **plasma phospholipid.**

Within the limits of the experiments, however, seasonal change in plasma triglyceride in the non-exercised rats was not associated with the seasonal change in $\Delta m \cdot Q^{-1}$, but did relate to Q. A similar relationship

Table 3. Relationship between the values of the energy conversion coefficient ($\Delta m \cdot Q^{-1}$) and plasma lipids as well as those of $\Delta m \cdot Q^{-1}$ and aorta lipids

			Plasma				Aorta				
			Total cholesterol	HDL-C	LDL-C	Tri- glyceride	Phospho- lipid	Total cholesterol	Cholesteryl ester	Tri- glyceride	Phospho- lipid
	A $Ns + Nw$ $(n=40)$	$\Delta m \cdot O^{-1}$			$r = 0.343$ P < 0.05		$r = -0.414$ $r = 0.325$ P < 0.01	P < 0.05	$r = 0.410$ P < 0.01	$r = 0.512$ P < 0.005	$r = -0.583$ P < 0.05
	$Ws + WW$ $(n=29)$	$\Delta m \cdot Q^{-1}$					$r = -0.455$ $r = 0.522$ P < 0.05	P < 0.005	$r = 0.585$ P < 0.005	$r = 0.474$ P < 0.01	$r = -0.465$ P < 0.025
	B $Ns + Ws$	$\Delta m \cdot Q^{-1}$	$r = 0.438$	$r = 0.441$		$r = 0.422$				$r = 0.380$	
	$(n=34)$		P < 0.01	P < 0.01		P < 0.01				P < 0.05	
	$Nw + Ww$	$\Delta m \cdot O^{-1}$	$r = 0.565$	$r = 0.587$		$r = 0.421$	$r = 0.613$			$r = 0.529$	
	$(n=35)$		P < 0.01	P < 0.01		P < 0.05	P < 0.01			P < 0.005	

 A = rats non-exercised (Ns + Nw) and exercised (Ws + Ww); B = rats non-exercised and exercised in summer (Ns + Ws), and non-exercised and exercised in winter (Nw + Ww); r = correlation coefficient; for other definitions see Table 2

between Q and lipid value was also observed in the case of plasma phospholipid in the non-exercised and exercised groups, as shown in Table 4.

We found no relationship between aorta free cholesterol and [HDL-C]. The plasma phospholipid was associated with aorta phospholipid and inversely related to aorta lipids such as cholesteryl ester and triglyceride as shown in Table 5.

Throughout the study, seasonal changes in the plasma and aorta lipids in the young rats were found to be expressed in terms of the biorhythm of $\Delta m \cdot Q^{-1}$ and/or Q. The effect of training on the lipid values detected in summer and/or in winter were also found to be dependent on the values of $\Delta m \cdot Q^{-1}$ in exercise. Moreover, seasonal changes in phospholipid, cholesteryl ester and triglyceride in the aortae were coupled with the seasonal variation in plasma phospholipid.

Table 4. Relationship between total caloric intake (O) and plasma lipid in rats non-exercised $(Ns + Nw)$ and exercised $(Ws + Ww)$

		Plasma triglyceride	Plasma phospholipid
$Ns + Nw$ $(n=40)$ $Ws + Ww$ $(n=29)$	υ o	$r = 0.337$ P < 0.005	$r = 0.503$ P < 0.01 $r = 0.641$ P < 0.01

r: correlation coefficient

Table 5. Relationship between plasma and aorta lipids in the nonexercised $(Ns + Nw)$ and exercised $(Ws + Ww)$ groups

		Aorta ester	Aorta	Aorta cholesteryl triglyceride phospholipid
$Ns + Nw$ $(n=40)$ $Ws + Ww$ $(n=29)$	Plama phospholipid $P < 0.01$ Plasma phospholipid $P < 0.05$		$r = -0.445$ $r = -0.475$ $r = 0.313$ P < 0.01 $r = -0.428$ $r = -0.482$ $r = 0.707$ P < 0.01	P < 0.05 P < 0.01

r: correlation coefficient

Discussion

Mikeska and Petrásek (1977) have detected seasonal changes in total protein, glucose, esterified fatty acids and total cholesterol in the blood serum of female Wistar rats, when their animals were kept at a constant temperature. This finding together with our results suggests that seasonal fluctuations of plasma and aorta lipids in mammals are independent of the annual cycle of temperature. In contrast to these findings for adult laboratory rats (Mikeska and Petrásek 1977) and for humans (Carlson and Lindstedt 1969; Warnick and Albers 1976), we found no seasonal change in plasma total cholesterol in young animals, but did note a contradictory result regarding plasma triglyceride. The levels of plasma triglyceride in human subjects have been shown to be greater in winter than in summer (Carlson and Lindstedt 1969; Warnick and Albers 1976). In our young non-exercised rats, a higher value for this lipid was evident in summer.

Larsen et al. (1985a, b) reported that serum total cholesterol, [HDL-C] and serum triglyceride in reindeer increased in summer with the enhancement of food intake. However, these findings are not necessarily applicable to the non-exercised and exercised rats because the increased levels of the Q in this season were not accompanied by any increase of total cholesterol and [HDL-C] in the plasma. We found the levels of plasma triglyceride were associated with those of Q in only the non-exercised animals, thereby suggesting that a high Q in summer is responsible for the increase of plasma triglyceride. In the non-exercised and in the exercised group, seasonal change in plasma phospholipid depended on the seasonal variation in Q and in $\Delta m \cdot Q^{-1}$ as follows for the non-exercised animals,

[plasma phospholip- \vec{a} [e] = 2.13.10⁻³ \cdot \hat{Q} - 2.90 \cdot 10³ \cdot $\Delta m \cdot Q^{-1}$ + 1.01 \cdot 10²

and for the exercised group,

[plasma phospholip- $\vec{a}d = 2.38 \cdot 10^{-3} \cdot \vec{Q} - 4.24 \cdot 10^{3} \cdot \Delta m \cdot \vec{Q}^{-1} + 8.01 \cdot 10$ Accordingly, the increased levels of Q and reduction in $\Delta m \cdot Q^{-1}$, as observed in summer, would favour an increase in values of plasma phospholipid to maintain the annual cycle of this lipid in the plasma.

Referring to the findings on non-exercised (NE) and exercised (WE) young rats in winter (Hashimoto and Masumura 1988), we could not find seasonal changes in the variables $(Q, \Delta m \cdot Q^{-1})$. That is, the levels of O and $\Delta m \cdot O^{-1}$ in NE were very close to those measured in Ns. Regarding WE animals, only O diminished by 11.9% $(P<0.05)$ in comparison with those of Ws. In these groups (Ns and NE, Ws and WE), neither plasma triglyceride nor plasma phospholipid concentrations were associated with the levels of Q and $\Delta m \cdot Q^{-1}$, because these variables did not simultaneously oscillate between the seasons.

Ahlers et al. (1982) observed that serum total cholesterol in young male rats was the least affected by season. Considering this finding and taking into account the unchanged [HDL-C] in both the non-exercised and exercised groups (see Table 2 A, B), we assumed that [HDL-C] in young animals interfered with the seasonal behaviour of plasma total cholesterol. A lack of seasonal change in plasma triglyceride and [LDL-C] in the trained group shows that exercise affected metabolic rates of these plasma lipids. Thus, care should be taken when attempting to extrapolate seasonal variation in plasma lipids in trained animals from the results of a sedentary group.

Concerning the aorta lipids in non exercised and in exercised rats, total cholesterol, cholesteryl ester, triglyceride and phospholipid changed with season, the levels following the respective functions of $\Delta m \cdot Q^{-1}$. In all these cases, higher levels of aorta triglyceride were observed, when compared with the findings of previous studies (Masumura et al. 1985; Hashimoto and Masumura 1988), thereby suggesting that aorta triglyceride levels in young rats are variable. In non-exercised groups (Ns and NE) and exercised groups (Ws and WE), we have failed to detect any relationship between $\Delta m \cdot Q^{-1}$ and aorta lipid, owing to the lack of differences in the levels of $\Delta m \cdot Q^{-1}$ between Ns and NE as well as Ws and WE.

The $\Delta m \cdot Q^{-1}$ values in the exercised rats decreased as compared with those found in the non-exercised animals. These percentage decreases amounted to 25.8% for Nw and Ww, 25.5% for Ns and Ws and 21.2% for NE and WE. When this percentage exceeded 25%, the decreased levels of plasma and aorta lipids in exercise paralleled the decrement of $\Delta m \cdot Q^{-1}$.

In an earlier study (Hashimoto and Masumura 1988), we have found that young rats exercised from January to April have a diminished gain in mass, [HDL-C] is reduced from 3.05 to 2.07 $(g \cdot \hat{1}^{-1}) \cdot 10^{-1}$ and aorta free cholesterol is increased, findings suggestive of a reduction in [HDL-C] which in turn stimulates aorta lipid accumulation. The young exercised rats had a diminished gain in mass and a reduced [HDL-C], but there was no increase in the aorta free cholesterol values, in comparison with the respective values of the sedentary animals. There was no further change in this aorta lipid, when [HDL-C] exceeded $3.05 \cdot (g \cdot 1^{-1}) \cdot 10^{-1}$.

Schierf et al. (1983) pointed out that lower levels of $[LDL]$. $[HDL]^{-1}$ in humans should reduce cardiovascular risk. Thus, the question arises as to whether or not such lower values of $[LDL-C] \cdot [HDL-C]^{-1}$ as observed in summer would hamper an increment of cholesteryl ester in the aortae. However, the values of [LDL- C [HDL-C]⁻¹ in non-exercised and in exercised animals were not related to those of aorta cholesteryl ester. Instead, the levels of plasma phospholipid were inversely related to those of the aorta esters, under almost the same [HDL-C]. In these cases, aorta phospholipid was increased when the plasma phospholipid was augmented in the summer, which in turn led to a decrease in the aorta cholesteryl ester and triglyceride contents. Thus, the aorta cholesteryl ester, triglyceride and phospholipid contents appear to oscillate between summer and winter, a situation probably reversible in the young. However, in cases of advanced atherosclerosis, we suggest there is an irreversibility of the lipid deposit with age. Stafford and Day (1975) have shown that phospholipid infusion removed arterial cholesterol. This may suggest the possibility that seasonal elevation of plasma phospholipid in young rats prevents vascular tissues from non-polar lipid deposits.

Exercise acts as a prophylactic against atherosclerotic lesions (Hasler et al. 1984). In the aortae of weanling exercised rats, we have observed a reduction only in triglyceride. As triglyceride in the aortae of these rats changed seasonally and decreased in the summer, a decrease of this fat would be amplified by physical training from an early age, during the summer season.

The most remarkable finding in the present study is that seasonal changes in plasma and aortic lipids in the young non-exercised and exercised rats were expressed in terms of Q and/or $\Delta m \cdot Q^{-1}$. In many instances, the levels of Q and/or $\Delta m \cdot Q^{-1}$ changed seasonally and the values of $\Delta m \cdot Q^{-1}$ reduced in exercise were correlated with the corresponding lipid values simultaneously fluctuating in the plasma and aortae. We raise the question as to whether this is the case in the human beings. However, this question is as yet unsolved as there has been no previous work on the subject. We consider that estimations of Q and $\Delta m \cdot Q^{-1}$ in young human subjects could serve as a measure of plasma lipid behaviour. In this respect, we need to investigate further the annual cycles of plasma lipids in human subjects, with reference to the response of lipid values to exercise and Q.

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