Serum Lipid Levels and Steroidal Hormones in Women Runners With Irregular Menses

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Catalogue Data

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Mots-clés: estradiol, DHEAS, cholestérol, exercice physique, aménorrhée

Abstract/Résumé
This study compared the lipid profile of women runners with menstrual cycle irregularities with their normally menstruating counterparts. Relationships among selected steroid hormones and serum lipid levels in 10 eumenorheic (EU) and 8 oligo-amenorheic (O/A) women runners and 6 eumenorheic controls (CON) were examined. Serum 17β-estradiol (E2), progesterone (Prog), and dehydroepiandrosterone-sulfate (DHEAS) concentrations were determined in daily blood samples for 21 days, and integrated concentrations were calculated. Fasting blood samples were analyzed for total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), HDL2, HDL3, triglycerides (Trig), and apolipoproteins A-I, A-II, and B. The O/A group had significantly lower E2 and Prog than EU or CON groups. Women in the CON group had lower HDL-C and HDL2 than the runners. With all women grouped together, E2 was not significantly correlated with any measured blood lipid parameters. On the other hand, DHEAS was significantly correlated with HDL-C, HDL2, and apolipoprotein A-I. These data demonstrate that women runners, regardless of menstrual cycle status, exhibit higher HDL-C concentrations than CON and supports previous research reporting a positive association between DHEAS and HDL-C.

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Cette étude compare le profil lipidique de coureuses au cycle menstruel irrégulier à celui d'un groupe témoin au cycle normal. Les corrélations entre les concentrations de diverses hormones stéroïdiennes et le niveau de lipides sériques sont analysées chez 10 coureuses euménorrhéiques (EU), 8 coureuses oligo-aménorrhéiques (O/A), et 6 femmes euménorrhéiques (CON). Les concentrations sériques de 17β-estradiol (E2), de progestérone (Prog), et de déhydroépinandrosterone-sulfate (DHEAS) sont évaluées quotidiennement pendant 21 jours. La concentration de cholestérol total (TC), de cholestérol-lipoprotéines à faible densité (LDL-C), de cholestérol-lipoprotéines à forte densité (HDL-C), de HDL2, de HDL3, de triglycérides (Trig), et d’apolipoprotéines A-I, A-II, et B est aussi évaluée chez des femmes à jeun. Les valeurs de E2 et de Prog du groupe O/A sont significativement plus faibles que celles des groupes EU et CON. Les valeurs de HDL-C et HDL2 du groupe témoin sont plus faibles que celles des coureuses. Les valeurs de E2 de toutes les femmes réunies ne sont pas corrélées significativement aux mesures de lipides sanguins. Par contre, les valeurs de DHEAS sont significativement corrélées à celles de HDL-C, de HDL2, et d’apolipoprotéines A-I. Ces observations démontrent que, quel que soit l’état du cycle menstruel, les coureuses affichent des concentrations de HDL-C plus fortes que celles du groupe témoin; ces résultats confirment aussi des études antérieures au sujet de la relation positive entre le DHEAS et le HDL-C.

Introduction

Both habitual physical activity and exogenously administered estrogens alter the blood lipid profile. Prospective studies that have examined the impact of regular exercise on the blood lipid profile of women have shown increases in high-density lipoprotein cholesterol (HDL-C) (Hardman and Hudson, 1994; Lindheim et al., 1994; Rotkis et al., 1981), and apolipoprotein A-I (apo A-I; Despres et al., 1991; Lindheim et al., 1994; Tremblay et al., 1991), and decreases in total cholesterol (TC; Lamarche et al., 1992; Lindheim et al., 1994; Tremblay et al., 1991), low-density lipoprotein cholesterol (LDL-C; Lamarche et al., 1992; Lindheim et al., 1994; Tremblay et al., 1991), apolipoprotein B (apo B; Despres et al., 1991; Tremblay et al., 1991), and triglycerides (Trig; Lindheim et al., 1994). Similarly, exogenously administered estrogen alters the lipid profile by raising plasma HDL-C (Metka et al., 1992; Schaeffer et al., 1983) and apo A-I (Schaeffer et al., 1983), and by lowering LDL-C (Metka et al., 1992; Schaeffer et al., 1983). It has been suggested that estradiol elevates HDL-C by suppressing hepatic lipase activity (Tikkanen et al., 1982), which in turn slows the breakdown of HDL2.

Although premenopausal women are generally considered at low risk for developing cardiovascular disease because of the estrogen-induced elevation in HDL-C concentration (Barrett-Conner and Bush, 1991), the relationships among endogenous gonadal steroid hormones and lipid levels in premenopausal women are not well defined. In nonexercising eumenorrheic women, Gorbach et al. (1989) demonstrated that follicular phase levels of bound estradiol were significantly correlated with HDL-C and very-low-density lipoprotein cholesterol (VLDL-C). Additionally, those authors found significant partial correlations between bound estradiol and HDL-C and VLDL-C, and a significant negative partial correlation between bound estradiol and LDL-C (Gorbach et al., 1989). In addition to the
relationship between estradiol and HDL-C, recent investigations (Barrett-Connor and Goodman-Gruen, 1995; Slowinska-Srzednicka et al., 1995) report that women with higher dehydroepiandrosterone-sulfate (DHEAS) levels also exhibit elevations in HDL-C.

Irregular menstes are more frequent in women exercisers than in nonathletic populations (Loucks et al., 1992). Exercise-induced menstrual cycle irregularities are characterized by decreased plasma levels of follicle-stimulating hormone (FSH), luteinizing hormone (LH) and E₂ (Baer, 1993; Marcus et al., 1985), as well as diminished LH pulsatility (Veldhuis et al., 1985); however, the impact of menstrual cycle irregularities (and thus lower integrated concentrations of the gonadal steroid hormones) on the blood lipid profile is not well defined. Lamon-Fava et al. (1989) reported the level of apo A-I and the apo A-I/apo B ratio were higher in the eumenorrheic runners compared with amenorrheic runners, but TC and Trig did not differ between groups. In contrast, Friday et al. (1993) reported that amenorrheic runners had higher TC, Trig, LDL-C, HDL-C, and HDL₃. Thus, it is not yet clear to what extent hypoeostrogenemia, or other hormonal alterations characteristic of subgroups of women runners, might affect the circulating lipid profile.

The purpose of the present study was to investigate the relationships among steroidal hormone profiles (specifically estradiol and DHEAS) and fasting blood lipid parameters in eumenorrheic nonexercising women, eumenorrheic runners, and oligo-/amenorrheic runners.

**Methods**

**SUBJECTS**

All subjects provided written informed consent approved by the Human Investigation Committee at the University of Virginia. The Caucasian women runners recruited for this study met the following criteria:

1. Were between 18 and 40 years old
2. Ran 32 kilometers or more per week for at least the previous 6 months
3. Had not used oral contraceptives within the previous 12 months
4. Did not smoke
5. Were without metabolic disorders

Six eumenorrheic (10–13 menses per year) women (all characteristics as above except none engaged in regular aerobic training) served as controls (CON). Subjects completed the Eating Disorders Inventory (EDI; Garner et al., 1983) and recorded their food intake for 7 days. Based on dietary intake (in comparison with reported physical activity) and the EDI subscales, none of the women were suspected of suffering from eating disorders. Analyses revealed that the groups scored similarly on seven of eight EDI subscales and were clearly different from a comparison group of women suffering from anorexia nervosa. Additional information about the relationships between reproductive hormones, dietary patterns, alcohol
intake, eating behaviors, and bone mineral density in these women has been reported previously (Snead et al., 1992a, 1992b).

The subjects were examined by a physician and were subsequently classified based on the number of menses they had in the previous year. Runners with 10–13 spontaneous menses in the previous year were classified as eumenorrheic runners (EU, n = 10). Subjects were classified as oligo-/amenorrheic runners (O/A, n = 8) if they had 0–9 menses in the previous year. Subjects classified as O/A underwent further testing (i.e., measurement of appropriate circulating hormones and physical examination) in order to rule out thyroid dysfunction, androgen excess, hyperprolactinemia, and premature ovarian failure.

REPRODUCTIVE HORMONE MEASUREMENTS

Daily blood samples (10 cc) were collected at the General Clinical Research Center of the University of Virginia Health Sciences Center. Subjects were instructed to have the blood samples collected at the same time of day, but were allowed to choose the time that best suited their daily schedules. Subjects were requested to avoid exercise and food intake 6–12 hours prior to blood collection. Eumenorrheic women had blood sampled daily from Day 9 of their menstrual cycle to the last day of the cycle. The number of days sampled ranged from 19 to 23. O/A runners had daily blood samples collected for 21 consecutive days, during which time no subject experienced menses. From the daily blood sampling, 21-day integrated area under the curve values were calculated for serum 17 β-estradiol (E₂), progesterone (Prog), and dehydroepiandrosterone-sulfate (DHEAS).

Serum E₂ and Prog were measured by radioimmunoassay using antibody-coated tubes and ¹²⁵I-labeled ligand (Diagnostic Products Corp., Los Angeles, CA). The minimum detectable concentrations for E₂ and Prog were 0.8 ng · dl⁻¹ and 5 ng · dl⁻¹, respectively. Intra-assay coefficient of variation (CV) for both E₂ and Prog was less than 5%. The antisera utilized in E₂ and Prog assays were highly specific for these hormones and exhibit low crossreactivity to other naturally occurring steroids or therapeutic drugs.

DHEAS was measured from serum by radioimmunoassay. ³H-labeled DHEA (New England Nuclear, Boston, MA) served as the ligand, and the antibody to DHEAS was obtained from ICN Biomedicals, Inc. (Costa Mesa, CA). The minimum detectable concentration was 8 μg · dl⁻¹. Intra-assay CV was <5%, and interassay CV was 14.0%. Crossreactivities with other steroids were extremely low (<0.03%), with the exception of androstenedione (12.5%).

CHARACTERIZATION OF LIPOPROTEINS, APOLIPOPROTEINS, AND PLASMA LIPIDS

TC, Trig, VLDL-C, LDL-C, HDL₁, HDL₂, apo A-I, apo A-II, and apo B were measured in fasting plasma samples according to standard methods employed by the Lipid Research Center at Washington University School of Medicine (National Heart, Lung, and Blood Institute, 1974). Samples were obtained after a 12-hour fast without regard for timing of the menstrual cycle.
MEASUREMENT OF MAXIMAL OXYGEN CONSUMPTION

Maximal oxygen consumption (\(\dot{V}O_2\max\)) was determined with an incremental treadmill protocol. Subjects ran at a velocity of 160 m \cdot min\(^{-1}\) (140 m \cdot min\(^{-1}\) for CON) with a 0\% grade for 2 min. Thereafter, the percent grade was increased by 2.5\% every 2 min until subjects reached volitional exhaustion. The highest oxygen uptake observed (averaged over 1 min) was designated as \(\dot{V}O_2\max\). Oxygen uptake was measured with standard open-circuit spirometry techniques, and heart rate was measured electrocardiographically (Snead et al., 1992a).

BODY COMPOSITION ASSESSMENT

Body density (\(D_B\)) was assessed by hydrostatic weighing (Katch et al., 1967), and residual lung volume was measured by the oxygen dilution technique described by Wilmore (1969). The revised formula of Brozek et al. (1963) was used to compute the percent body fat (%BF) from \(D_B\).

STATISTICAL ANALYSES

One-way analysis of variance (ANOVA) was utilized to investigate mean differences among the groups. A significance level of \(p < .05\) was chosen a priori. When significant difference was found, a least squares means (LSM) post hoc procedure was conducted to determine which groups differed. The Pearson product-moment correlation \((r)\) was used to examine the relationship between each hormone and blood lipids concentrations. All subjects were combined for these analyses.

Results

The descriptive characteristics and training practices of the groups are summarized in Table 1. There were no significant differences in age, height, or weight among the groups. The CON group had a significantly higher \((p < .05)\) body fat percentage (26.5 \pm 4.1\%) than the EU (21.5 \pm 2.7%) and O/A (19.8 \pm 3.1%) runners. The CON and EU groups had a similar number of menses per year (11.7 \pm 0.5 and 12.1 \pm 0.3 cycles, respectively), but the O/A group had significantly fewer cycles during the previous year (3.4 \pm 3.2 cycles). \(\dot{V}O_2\max\) was significantly higher in the EU (56.9 \pm 4.2 ml \cdot kg\(^{-1}\) \cdot min\(^{-1}\)) and O/A (54.8 \pm 6.4 ml \cdot kg\(^{-1}\) \cdot min\(^{-1}\)) groups than in the CON group (40.9 \pm 6.9 ml \cdot kg\(^{-1}\) \cdot min\(^{-1}\)) \((p < .05)\). EU and O/A runners reported similar training patterns, with no significant differences in any measured parameter. As expected, the CON and EU groups had higher integrated values for both E\(_2\) and Prog than the O/A group \((p < .05)\). No significant differences were observed among the groups for DHEAS.

Table 2 contains the results of the blood lipid analyses. No differences among groups were observed for TC, LDL-C, HDL-C, or Trig. The CON women had lower HDL-C and HDL\(_c\) values than either of the running groups \((p < .05)\). Apolipoprotein concentrations were not significantly different among groups.

Table 3 illustrates the correlations between selected blood lipid parameters and integrated DHEAS and E\(_2\) with all subjects grouped together. Correlations
Table 1  Characteristics of Subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n = 6)</th>
<th>Eumenorrheic (n = 10)</th>
<th>Oligo/amenorrheic (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>28.9 ± 6.7</td>
<td>32.5 ± 4.1</td>
<td>26.1 ± 6.7</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>165.4 ± 8.2</td>
<td>164.7 ± 6.4</td>
<td>170.6 ± 8.6</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>60.8 ± 4.4</td>
<td>58.5 ± 3.2</td>
<td>59.6 ± 8.0</td>
</tr>
<tr>
<td>% body fat</td>
<td>26.5 ± 4.1*</td>
<td>21.5 ± 2.7</td>
<td>19.8 ± 3.1</td>
</tr>
<tr>
<td>No. of menses*</td>
<td>11.7 ± 0.5</td>
<td>12.1 ± 0.3</td>
<td>3.4 ± 3.2**</td>
</tr>
<tr>
<td>VO₂max (ml·kg⁻¹·min⁻¹)</td>
<td>40.9 ± 6.9*</td>
<td>56.9 ± 4.2</td>
<td>54.8 ± 6.4</td>
</tr>
<tr>
<td>Kilometers run per week</td>
<td>0</td>
<td>53.1 ± 17.9</td>
<td>44.3 ± 17.2</td>
</tr>
<tr>
<td>Min running per day</td>
<td>0</td>
<td>54.5 ± 13.6</td>
<td>53.1 ± 10.8</td>
</tr>
<tr>
<td>Weeks of training per year</td>
<td>0</td>
<td>50.2 ± 1.4</td>
<td>51.1 ± 2.0</td>
</tr>
<tr>
<td>Years of training</td>
<td>0</td>
<td>7.3 ± 5.9</td>
<td>8.8 ± 4.6</td>
</tr>
<tr>
<td>17 β-Estradiol (ng·dl⁻¹·21d)</td>
<td>230.6 ± 60.6</td>
<td>283.3 ± 102.0</td>
<td>114.4 ± 102.3**</td>
</tr>
<tr>
<td>Progesterone (ng·dl⁻¹·21d)</td>
<td>14.8 ± 5.2</td>
<td>13.1 ± 2.8</td>
<td>1.8 ± 1.5**</td>
</tr>
<tr>
<td>DHEAS (µg·dl⁻¹·21d)</td>
<td>4.165 ± 2.510</td>
<td>4.433 ± 2.933</td>
<td>5.613 ± 3.198</td>
</tr>
</tbody>
</table>

Note. Values are M ± D, DHEAS = dehydroepiandosterone-sulfate.
*Significantly different from running groups (p < .05). **Significantly different from the normally menstruating groups (p < .05).

between integrated E₂ and the blood lipid parameters were nonsignificant with correlations ranging from r = -.27 to -.03. On the other hand, integrated DHEAS was significantly correlated with HDL-C (r = .59, p = .01), HDL₂ (r = .58, p = .01), and apo A-I (r = .51, p = .01).

Discussion

The most important observation of the present study was that in spite of pronounced differences in E₂ and Prog between EU and O/A runners, no differences were found in serum lipids, lipoproteins, or apolipoproteins. HDL-C and HDL₂ levels were higher in both O/A and EU runners than in the nonexercisers (Table 2). It can be hypothesized that, in the O/A women, the impact of exercise on hepatic lipase, lipoprotein lipase, or both is able to offset the lack of endogenous estradiol. These data suggest that exercise and E₂ exert independent effects on serum lipids and that, in premenopausal O/A women, the exercise-induced elevation in HDL-C overrides the effects of low E₂. This conclusion supports other findings that exercise
Table 2  Comparison of Blood Lipid Variables Among Subjects

<table>
<thead>
<tr>
<th>Variables</th>
<th>Controls</th>
<th>Eumenorheic</th>
<th>Oligo/amenorheic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>4.42 ± 1.37</td>
<td>4.68 ± 0.85</td>
<td>4.68 ± 0.67</td>
</tr>
<tr>
<td>(mmol)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL-C (mmol)</td>
<td>2.64 ± 1.34</td>
<td>2.66 ± 0.83</td>
<td>2.46 ± 0.52</td>
</tr>
<tr>
<td>HDL-C (mmol)</td>
<td>1.32 ± 0.16*</td>
<td>1.66 ± 0.26</td>
<td>1.81 ± 0.31</td>
</tr>
<tr>
<td>HDL_{2} (mmol)</td>
<td>0.39 ± 0.28</td>
<td>0.39 ± 0.16</td>
<td>0.47 ± 0.18</td>
</tr>
<tr>
<td>HDL_{3} (mmol)</td>
<td>0.93 ± 0.16*</td>
<td>1.27 ± 0.18</td>
<td>1.34 ± 0.18</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>1.05 ± 0.36</td>
<td>0.81 ± 0.21</td>
<td>0.88 ± 0.25</td>
</tr>
<tr>
<td>(mmol)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apolipoprotein A-I</td>
<td>55.0 ± 12.1</td>
<td>63.2 ± 7.9</td>
<td>66.8 ± 9.6</td>
</tr>
<tr>
<td>(µmol)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apolipoprotein A-II</td>
<td>19.6 ± 2.9</td>
<td>17.3 ± 3.5</td>
<td>19.6 ± 5.8</td>
</tr>
<tr>
<td>(µmol)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apolipoprotein B</td>
<td>1.09 ± 0.25</td>
<td>1.47 ± 0.65</td>
<td>1.22 ± 0.22</td>
</tr>
<tr>
<td>(µmol)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. Values are M ± SD. LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol. *Value is significantly lower ($p < .05$) than for either running group.

Table 3  Correlations Between Blood Lipid Parameters and 17 β-Estradiol and DHEAS

<table>
<thead>
<tr>
<th>Variable</th>
<th>17 β-estradiol</th>
<th>DHEAS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$r$</td>
<td>$p$</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>-.03</td>
<td>.898</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>-.22</td>
<td>.301</td>
</tr>
<tr>
<td>LDL-C</td>
<td>-.18</td>
<td>.390</td>
</tr>
<tr>
<td>HDL-C</td>
<td>-.14</td>
<td>.514</td>
</tr>
<tr>
<td>HDL$_2$</td>
<td>-.05</td>
<td>.824</td>
</tr>
<tr>
<td>HDL$_3$</td>
<td>-.15</td>
<td>.496</td>
</tr>
<tr>
<td>Apolipoprotein A-I</td>
<td>-.27</td>
<td>.195</td>
</tr>
<tr>
<td>Apolipoprotein A-II</td>
<td>-.08</td>
<td>.703</td>
</tr>
<tr>
<td>Apolipoprotein B</td>
<td>-.12</td>
<td>.584</td>
</tr>
<tr>
<td>Apo B/Apo A-I</td>
<td>-.05</td>
<td>.802</td>
</tr>
</tbody>
</table>

*Note. $n = 24$. DHEAS = dehydroepiandrosterone-sulfate; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; Apo B/Apo A-I = ratio of apolipoprotein B to apolipoprotein A-I. *$p < .05$. 
and exogenous estrogen have independent effects in postmenopausal women (Lindheim et al., 1994).

The precise mechanism through which exercise elevates HDL-C is unclear. Exercise decreases hepatic lipase activity (Thompson et al., 1991) while increasing lipoprotein lipase activity in both muscle and adipose tissue (Kiens and Lithell, 1989; Nikkilä et al., 1978). These changes are thought to increase HDL-C in aerobically trained individuals. It has also been hypothesized that the elevated HDL-C observed in trained individuals is due to an increase in reverse cholesterol transport (Gupta et al., 1993). In a recent paper, Berg and Nilsson-Ehle (1994) demonstrated that exogenously administered corticotropin and glucocorticoids elevate HDL-C. Although these pharmacologically elevated cortisol levels were much higher than those induced by exercise or measured in amenorrheic runners (Loucks et al., 1989), it seems reasonable to hypothesize that increases in HDL-C in runners may be related to the frequent elevations in cortisol during exercise. Another possible reason that endurance training raises HDL-C is the reduction in percentage of body fat. Williams (1990) theorized that elevated HDL-C levels in runners are strongly related to reduced levels of adiposity due to higher adipose tissue lipoprotein lipase activity. In the present study, the runners were leaner than the controls and had higher levels of HDL-C. Although the present study did not test any of these means through which exercise may alter HDL-C, it appears that several mechanisms may act to elevate HDL-C concentrations in aerobically trained persons.

In agreement with Hetland et al. (1995), the present study did not find any significant differences in blood lipid variables when comparing EU and O/A runners. The finding of no difference in apolipoprotein levels in the present study also compares favorably with the data of Friday et al. (1993). In contrast, however, other researchers have reported a trend for lower apo A-I levels and a significantly lower apo A-I/apo B ratio in amenorrheic runners (Lamon-Fava et al., 1989). Also in agreement with the current findings, the data of Lamon-Fava et al. (1989) reported that amenorrheic and eumenorrheic runners have similar TC and Trig. On the other hand, Friday and colleagues (1993) reported that amenorrheic athletes have higher TC, Trig, LDL-C, HDL-C, and HDL2. The inclusion of both oligomenorrheic and amenorrheic women in the present study may help explain some of the discrepancies with previous findings. The use of the 21-day integrated area for hormonal analysis in this study provides a distinction from the previous research. This type of hormonal profile is used to provide information about the general hormonal milieu and the cumulative hormonal influence that may be impacting the blood lipid profile. Additional studies comparing eumenorrheic, oligomenorrheic, and amenorrheic runners are needed to clarify the effect that menstrual cycle dysfunction has on the blood lipid profile. Careful examination of dietary practices and training habits will be critical in future studies.

The current study did not control the timing (i.e., menstrual cycle phase) of the blood lipid measurements. Controversy exists over the impact of menstrual cycle phase on blood lipids. Both Demacker et al. (1982) and Brideau et al. (1992) reported that no blood lipid parameters are affected by menstrual cycle phase. On
the other hand, others have concluded that triglycerides are elevated during the follicular (Mendoza et al., 1979) or ovulatory (Woods et al., 1987) phases. Kim and Kalkhoff (1979) provided evidence that TC, LDL-C, and apo B are lower during the luteal phase. Jones et al. (1988) and Tangney et al. (1991) demonstrated lower TC during the luteal phase; however, Tikkanen and colleagues (1986) reported that only LDL-C was lower during the luteal phase. From the inconsistencies in the literature, it is unclear whether the specific changes in blood lipids may be predicted based on menstrual cycle phase. The only differences found in blood lipid variables in the present study were higher HDL-C and HDL₄ values for the runners in comparison to the nonrunning controls. Although it is possible that controlling for menstrual cycle phase could have affected the correlational data, it seems unlikely that the clear distinction in HDL-C between runners and nonrunners would have been changed by an alteration in methodology.

This study found no significant relationship between integrated E₂ concentration and HDL-C ($r = -0.14, p = 0.51$). In contrast, DHEAS levels were found to be significantly ($p < 0.01$) correlated with HDL-C ($r = 0.59$), HDL₄ ($r = 0.58$), and apo A-I ($r = 0.51$) (Table 3). The relationship between DHEAS and HDL-C is in contrast to a previous study (Herrington et al., 1990) reporting no significant relationship between DHEAS and HDL-C. This discrepancy may be related to the different subject populations studied. Herrington et al. (1990) utilized subjects (males and females) with known cardiovascular disease and a median age of 52 years. This is in sharp contrast to the healthy younger women tested for the present study. Additionally, the present study examined the correlation between integrated DHEAS and blood lipid parameters, but Herrington et al. (1990) obtained only a single DHEAS sample from each subject. Similar to the present findings, Barrett-Conner and Goodman-Gruen (1995) and Slowinska-Srzednicka et al. (1995) found a positive association between DHEAS and HDL-C. Barrett-Conner and Goodman-Gruen (1995) found this relationship when studying postmenopausal women, while premenopausal women were studied by Slowinska-Srzednicka et al. (1995).

Studies utilizing women subjects have reported mixed findings concerning the relationship between DHEAS and cardiovascular disease (Nafziger et al., 1991). In men, there appears to be an inverse relationship between DHEAS and the development of cardiovascular disease (Barrett-Conner et al., 1986; Herrington et al., 1990). This decrease in cardiovascular disease may be the result of an inverse relationship between TC and DHEAS or a positive correlation between HDL-C and DHEAS (Nafziger et al., 1991). Recently, Barrett-Conner and Goodman-Gruen (1995) reported a positive association between DHEAS and HDL-C but found no association between DHEAS and death from cardiovascular disease. The correlation between DHEAS and HDL-C found in the present study suggests a relationship between the variables; however, the small number of subjects necessitates that this relationship be viewed with caution. The relationship between DHEAS and blood lipids parameters in all women and specifically in women of varied menstrual status deserves further investigation.

In conclusion, women runners, without regard to menstrual cycle status, exhibit higher plasma HDL-C than age-matched, inactive, eumenorrheic women.
The elevated HDL-C levels in the O/A runners occurred in spite of suppressed plasma E₂ concentrations. It can be hypothesized that the impact of exercise on HDL-C is able to override the influence of reduced E₂ levels. Although no relationship was observed between E₂ and HDL-C, a significant positive correlation was found between DHEAS and HDL-C. This finding supports previous research reporting a positive association between DHEAS and HDL-C in women.

References


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