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# Variations in Plasma Volume Affect Total and Low-Density Lipoprotein Cholesterol Concentrations During the Menstrual Cycle

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**Serum lipids are known to vary during the menstrual cycle. To determine if changes in plasma volume contribute to this effect, we determined serum lipids, lipoproteins, and estimated changes in plasma volume in 18 premenopausal women at the start of and at 5-day intervals after menstruation. Eleven men served as a comparison group. Changes in plasma volume were estimated from changes in hemoglobin and hematocrit. Total and low-density lipoprotein (LDL) cholesterol (mean ± SD) increased 15 ± 14 mg/dL (9% ± 10%) and 11 ± 13 (11% ± 14%) within 10 days after the start of menstruation ($P < .05$) and then decreased toward baseline during the rest of the cycle. High-density lipoprotein (HDL) cholesterol increased 3 mg/dL, or 5%, ($P < .05$) on days 10 and 15 after menstruation. Plasma volume decreased 4% ± 9% ($P < .06$) 10 days after the start of menstruation, and this maximum decrease in plasma volume coincided with peak increases in total, LDL, and HDL cholesterol. Except for an 8-mg/dL increase in LDL cholesterol at day 5, lipid changes were no longer significant after adjusting for changes in plasma volume. We conclude that alterations in plasma volume account for approximately half of the increase in total and LDL cholesterol during the menstrual cycle.**


FEMALE SEX HORMONES are thought to be responsible for the "antiatherogenic" lipid profile characteristic of premenopausal women.[1] Similarly, changes in ovarian hormone concentrations during the menstrual cycle have been assumed to account for much of the within-month variation in serum lipids noted in menstruating women.[1-4] The mechanism of this hormonal effect has not been clearly defined. Estrogen and progesterone are known to affect plasma volume, and an expansion in plasma volume contributes to the decrease in total cholesterol noted in the supine position[5] and after exercise training.[6] We hypothesized that changes in plasma volume may also contribute to the intramonthly variation in women's lipoprotein concentrations. In the present study, we determined serum lipids, lipoproteins, and estimated changes in plasma volume in 18 women to examine this hypothesis.

## SUBJECTS AND METHODS

### *Subjects*

Study subjects included 18 healthy premenopausal women aged 23 to 42 years with regular menstrual cycles. A cycle was considered regular if cycle lengths for the 3 prior months did not differ by more than 2 days. Only women who did not routinely take medication and who had not taken oral contraceptives for the preceding 4 months were recruited. A group of 11 men aged 22 to 43 years served as a comparison group. All subjects provided written informed consent.

Subjects were asked to maintain their habitual daily intake of alcohol and caffeinated beverages to avoid possible effects of these nutrients on dependent variables. No other dietary restrictions were imposed. Acetaminophen was permitted, but subjects were asked to refrain from using other medications. Medications deemed necessary by the subjects were recorded in daily diaries. Subjects were asked to delay medication use on the day of a blood sample until after phlebotomy. Smokers were not included if their daily consumption of cigarettes varied. Exercise was restricted for 12 hours before each blood sample.

### *Analytical Methods*

Venous blood samples in the women were drawn on day 1 or 2 of menstruation, day 5, and every 5 days thereafter through day 1 or 2 of the next cycle. This schedule resulted in seven samples for most women, but seven women provided eight samples. The extra sample was eliminated as follows. Samples 1 and 7 (day 1 or 2) were

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easily identified as menstrual phase. The sample corresponding to ovulation (sample 4) was next determined using a chart from a previous report by Adlercreutz and Tallqvist[7] indicating days of ovulation for any given cycle length. For women who provided seven samples, this left two samples in the follicular phase (samples 2 and 3) and two samples in the luteal phase (samples 5 and 6). Because of longer cycle lengths, five women had three luteal samples instead of two. This third luteal sample occurred in most cases just before the second menstrual period and was therefore eliminated. Likewise, two women with longer cycle lengths had three follicular samples instead of two. In these women, the third follicular sample was eliminated because of possible overlap near midcycle with ovulation. The seven samples correspond roughly to the menstrual (day 1 or 2), follicular (days 5 and 10), ovulatory (day 15), and luteal (days 20 and 25) phases. Blood samples in the men were obtained 5 days apart. Subjects were seated during phlebotomy, and samples were drawn in the morning after a 12-hour fast.

Serum was separated from blood cells within 2 hours of phlebotomy and stored at −70°C. Interassay variation was avoided by analyzing all samples from individual subjects in a single assay. Total cholesterol[8] and triglycerides[9] were quantified on a Gilford Impact 400 Clinical Chemistry Analyzer (Gilford Instrument Laboratories, Oberlin, OH) using enzymatic methods. The Lipid Research Clinics protocol was used for high-density lipoprotein (HDL) cholesterol determination.[10] $HDL_2$ and $HDL_3$ subfractions were determined by precipitation.[11] Low-density lipoprotein (LDL) cholesterol levels were estimated.[12] Apolipoprotein (apo) A-I,[13] A-II,[14] and B[15] levels were measured by radioimmunoassay. Hematocrit was determined by a microcapillary-tube technique. Hemoglobin level was measured using the cyanmethemoglobin method.[16] Hematocrit and hemoglobin levels were used to estimate changes in plasma volume[17] (see Appendix).

Body weight was measured at each sampling point. Chest, abdomen, and thigh skinfolds for men and triceps, hip, and thigh skinfolds for women were measured on the first sample day to estimate percent body fat.[18]

### *Statistical Analysis*

Data for men and women were analyzed separately using a two-way (method by time) repeated-measures ANOVA. Serum samples corrected and uncorrected for estimated changes in plasma volume were compared with sample 1 using a *t* test for dependent samples. The Wilcoxon signed-rank test for nonparametric data was used to evaluate estimated changes in plasma volume.[19] A Bonferroni correction was used for multiple comparisons to maintain an experiment-wise $\alpha$ level of .05.[20] Spearman correlation coefficients were used to examine the relationship between estimated changes in plasma volume and absolute and percent changes in total cholesterol.

## RESULTS

Mean ages for women (33.0 ± 5.7 years) and men (32.2 ± 6.7) were similar. Height, weight, and estimated percent body fat for women and men (mean ± SD) were 164.6 ± 7.4 versus 177.3 ± 7.4 cm, 59.8 ± 12.5 versus 76.5 ± 14.2 kg, and 22.9% ± 6.7% versus 16.0% ± 6.0%, respectively. Estimated percent body fat was typical of values for college-aged men and women.[21] Two women smoked cigarettes. Four women and six men drank alcohol, but none drank more than one alcoholic beverage per day during the study. Medication use occurred sporadically among study subjects despite our request that only acetaminophen be used. Ibuprofen (n = 1), Fiorinal (Sandoz Pharmaceuticals, East Hanover, NJ) (n = 1), naproxen (n = 2), antihistamines (n = 2), and aspirin (n = 4) were used by the women. Aspirin (n = 3), antihistamines (n = 2), and penicillin (n = 1) were used by the men.

Average initial values for triglycerides and total and LDL cholesterol (Tables 1 and 2) were within the normal range reported by the Lipid Research Clinics Program Prevalence Study.[22] HDL cholesterol for both groups was 5 mg/dL higher than respective population means,[22] possibly because subjects were generally lean nonsmokers with low triglycerides and eight of the women and three of the men exercised regularly (> two times per week). The amount of exercise appeared random throughout the study, and there was no pattern of exercise to suggest that any sample was affected differently because of more or less physical activity around that time point.

Average body weight in both groups varied less than 0.5 kg during the study (Tables 1 and 2). Plasma volume decreased 4.1% ± 8.9% ($P = .055$) 10 days after the onset of menstruation (Fig 1). This maximum decrease in plasma volume coincided with peak increases in total, LDL, and HDL cholesterol (Fig 2). Plasma volume in the men varied slightly, with a maximum estimated increase of 2.8% ($P > .05$) at day 5 (Fig 1).

Total cholesterol was higher on days 5 and 10 ($P < .05$) after the onset of menstruation (Fig 2). Other total cholesterol values for the women did not differ from the initial sample. Adjusting for estimated changes in plasma volume reduced these increases from 11 to 7 mg/dL on day 5 and from 15 to 6 on day 10 (Fig 2). In contrast, total cholesterol in the men unexpectedly decreased 6 to 10 mg/dL ($P > .05$) during the study.

LDL cholesterol concentrations in each group paralleled changes in total cholesterol (Fig 2). However, after correction for plasma volume, day 5 LDL cholesterol levels in women remained 8 mg/dL higher ($P < .05$) than baseline. This increase in LDL cholesterol was accompanied by a 7-mg/dL increase in apo B (Fig 2). Plasma volume correction reduced the apo B increase only slightly (by 2 mg/dL), suggesting that the increases in early follicular-phase LDL cholesterol and apo B are only partly due to changes in plasma volume. Apo B in men did not vary and was not affected by changes in plasma volume.

Absolute changes in HDL cholesterol during the menstrual cycle were less than 3 mg/dL and were unaffected by adjustments for plasma volume (Fig 2). Peak values occurred at days 10 and 15 or during the late follicular and ovulatory phases, respectively ($P < .05$ for both). Changes in HDL subfractions and HDL apoproteins in women were also small and were not affected by corrections for plasma volume. Similarly, concentrations of HDL cholesterol, HDL subfractions, and apoproteins in men varied little during the study (Fig 2). Serum triglycerides did not change significantly in either group.

## DISCUSSION

Several investigations have observed higher total[1,2,23,24] and LDL[1,3,4] cholesterol concentrations during the follicu-

**Table 1. Uncorrected and PVcorr Lipid, Lipoprotein, and Apolipoprotein Concentrations (mean ± SD) in 18 Women During the Menstrual Cycle**

| | Sample Day | | | | | | |
|---|---|---|---|---|---|---|---|
| | 1-2 M | 5 | 10 | 15 | 20 | 25 | 1-2 M |
| Total cholesterol | | | | | | | |
| Uncorrected | 170 ± 21 | 181 ± 21* | 185 ± 24* | 178 ± 25 | 176 ± 25 | 174 ± 26 | 170 ± 26 |
| PVcorr | 170 ± 21 | 177 ± 18 | 176 ± 17 | 177 ± 20 | 168 ± 23 | 168 ± 19 | 175 ± 20 |
| LDL | | | | | | | |
| Uncorrected | 97 ± 23 | 108 ± 26* | 108 ± 26* | 102 ± 25 | 101 ± 25 | 101 ± 28 | 98 ± 27 |
| PVcorr | 97 ± 23 | 105 ± 23* | 102 ± 22 | 101 ± 21 | 97 ± 23 | 98 ± 24 | 101 ± 25 |
| Apo B | | | | | | | |
| Uncorrected | 80 ± 20 | 87 ± 22* | 84 ± 22 | 79 ± 19 | 83 ± 18 | 82 ± 20 | 79 ± 23 |
| PVcorr | 80 ± 20 | 85 ± 20 | 79 ± 19 | 79 ± 18 | 79 ± 17 | 79 ± 17 | 81 ± 20 |
| Triglycerides | | | | | | | |
| Uncorrected | 66 ± 17 | 64 ± 20 | 71 ± 21 | 64 ± 21 | 65 ± 18 | 64 ± 17 | 63 ± 20 |
| PVcorr | 66 ± 17 | 63 ± 19 | 68 ± 20 | 63 ± 20 | 62 ± 16 | 62 ± 14 | 64 ± 18 |
| HDL | | | | | | | |
| Uncorrected | 60 ± 14 | 60 ± 15 | 63 ± 14* | 63 ± 15* | 61 ± 16 | 60 ± 14 | 59 ± 14 |
| PVcorr | 60 ± 14 | 59 ± 15 | 60 ± 13 | 63 ± 16 | 59 ± 16 | 58 ± 14 | 61 ± 14 |
| $HDL_2$ | | | | | | | |
| Uncorrected | 28 ± 12 | 26 ± 10 | 29 ± 11 | 30 ± 11 | 26 ± 11 | 26 ± 11 | 27 ± 11 |
| PVcorr | 28 ± 12 | 26 ± 10 | 28 ± 11 | 30 ± 11 | 25 ± 11 | 26 ± 11 | 28 ± 12 |
| $HDL_3$ | | | | | | | |
| Uncorrected | 32 ± 5 | 34 ± 7 | 33 ± 5 | 33 ± 6 | 35 ± 7* | 33 ± 6 | 32 ± 6 |
| PVcorr | 32 ± 5 | 33 ± 7 | 32 ± 4 | 34 ± 6 | 33 ± 7 | 32 ± 6 | 33 ± 4 |
| Apo A-I | | | | | | | |
| Uncorrected | 129 ± 27 | 131 ± 32 | 134 ± 31 | 133 ± 31 | 130 ± 29 | 128 ± 26 | 126 ± 26 |
| PVcorr | 129 ± 27 | 128 ± 33 | 128 ± 31 | 133 ± 32 | 125 ± 30 | 124 ± 26 | 129 ± 25 |
| Apo A-II | | | | | | | |
| Uncorrected | 29 ± 5 | 30 ± 5 | 31 ± 5* | 30 ± 5 | 30 ± 6 | 30 ± 5 | 29 ± 6 |
| PVcorr | 29 ± 5 | 29 ± 4 | 29 ± 5 | 30 ± 5 | 29 ± 5 | 29 ± 5 | 30 ± 5 |
| Body weight (kg) | 60.5 ± 13.1 | 60.2 ± 13.4 | 60.3 ± 13.4 | 60.7 ± 13.5 | 60.3 ± 13.0* | 60.2 ± 13.2* | 60.3 ± 13.0 |

NOTE. Values are in mg/dL; n = 16 for body weight.

Abbreviations: PVcorr, plasma volume–corrected; M, menstruation.

*Significantly different from day 1 ($P < .05$) using $t$ test and Bonferroni correction for multiple comparisons.

lar versus luteal phase of the menstrual cycle. Reported increases from prospective studies are approximately 8%[23] to 10%[2,24] for total and 10% for LDL[3,4] cholesterol. In the present study, both total and LDL cholesterol, as well as apo B, concentrations were consistently greater during the follicular phase of the menstrual cycle. Total and LDL cholesterol increased 15 and 11 mg/dL, respectively, or 9% ± 10% and 11% ± 14%, 10 days after the onset of menstruation. Individual increases in total cholesterol between day 1 and day 10 varied from 1 to 46 mg/dL, and only two women had a decrease of total cholesterol (−3 and −23 mg/dL) between these sampling days. Individual increases in LDL cholesterol at day 10 varied from 1 to 36 mg/dL, with only two subjects failing to show an increase in LDL cholesterol concentration (−10 and −14 mg/dL). Twelve of 18 women had a maximum total cholesterol level during the follicular phase.

In contrast, men demonstrated a more even distribution of maximum values over the sampling period. Total cholesterol unexpectedly decreased 10 mg/dL in the men. This decrease observed in the present study may be partly attributed to high initial values in three men who entered the study after the Thanksgiving and Christmas holidays and who subsequently had a decrease of total cholesterol level of 20 to 40 mg/dL. There is considerably less variation in the group's mean values when considering all but the initial sample (Fig 2).

The mechanisms responsible for within-month variation in serum lipids of premenopausal women are not entirely clear. Such variations have been attributed primarily to estrogen-induced alterations of cholesterol metabolism. Exogenous estrogen increases hepatic LDL receptor activity[25] and LDL uptake[26] in rats, suggesting that higher estrogen secretion could reduce cholesterol levels by augmenting LDL clearance. Indeed, Mattson et al[3] noted increases in 17β-estradiol and decreases in LDL cholesterol levels between the follicular phase and midcycle. However, estrogenic effects alone are unlikely to account fully for the changes in serum lipids.

Other investigators have mentioned but de-emphasized the possible effects of plasma volume on serum cholesterol variation in women.[7,27] Adlercreutz and Tallqvist[7] did note a weak but statistically significant correlation between hematocrit and total cholesterol in 29 women. Hematocrit varied by only 1.6%, leading these investigators to minimize the effects of plasma volume on serum lipids. However, hematocrit alone can underestimate changes in plasma volume,[28] and the present results suggest that plasma volume partly contributes to the variation of serum cholesterol in women. Nevertheless, correlations between change

**Table 2. Uncorrected and PVcorr Lipid, Lipoprotein, and Apolipoprotein Concentrations (mean ± SD) in 11 Men**

| | Sample Day | | | | | | |
|---|---|---|---|---|---|---|---|
| | 1-2 | 5 | 10 | 15 | 20 | 25 | 1-2 |
| Total cholesterol | | | | | | | |
| Uncorrected | 201 ± 37 | 195 ± 31 | 195 ± 25 | 193 ± 23 | 190 ± 31 | 191 ± 27 | 191 ± 28 |
| PVcorr | 201 ± 37 | 201 ± 34 | 194 ± 26 | 194 ± 22 | 188 ± 31 | 188 ± 26 | 187 ± 29 |
| LDL | | | | | | | |
| Uncorrected | 132 ± 22 | 130 ± 19 | 129 ± 16 | 128 ± 12 | 124 ± 21 | 125 ± 14 | 123 ± 17 |
| PVcorr | 132 ± 22 | 134 ± 23 | 128 ± 18 | 129 ± 12 | 123 ± 23 | 124 ± 17 | 121 ± 20 |
| Apo B | | | | | | | |
| Uncorrected | 96 ± 8 | 95 ± 8 | 94 ± 6 | 94 ± 5 | 93 ± 8 | 94 ± 7 | 97 ± 9 |
| PVcorr | 96 ± 8 | 98 ± 11 | 93 ± 8 | 95 ± 8 | 93 ± 14 | 94 ± 11 | 96 ± 12 |
| Triglycerides | | | | | | | |
| Uncorrected | 89 ± 40 | 80 ± 28 | 88 ± 44 | 78 ± 22 | 79 ± 24 | 85 ± 39 | 86 ± 32 |
| PVcorr | 89 ± 40 | 82 ± 26 | 86 ± 39 | 79 ± 24 | 78 ± 25 | 84 ± 38 | 84 ± 29 |
| HDL | | | | | | | |
| Uncorrected | 51 ± 19 | 49 ± 15 | 49 ± 16* | 49 ± 15 | 50 ± 16 | 49 ± 17 | 50 ± 18 |
| PVcorr | 51 ± 19 | 50 ± 16 | 48 ± 16 | 49 ± 13 | 49 ± 13 | 47 ± 15 | 49 ± 16 |
| $HDL_2$ | | | | | | | |
| Uncorrected | 18 ± 17 | 17 ± 15 | 16 ± 14 | 15 ± 14 | 17 ± 16 | 16 ± 16 | 17 ± 16 |
| PVcorr | 18 ± 17 | 17 ± 15 | 16 ± 14 | 15 ± 13 | 17 ± 14 | 15 ± 14 | 16 ± 14 |
| $HDL_3$ | | | | | | | |
| Uncorrected | 34 ± 4 | 32 ± 3 | 32 ± 3 | 34 ± 4 | 33 ± 2 | 33 ± 3 | 34 ± 4 |
| PVcorr | 34 ± 4 | 33 ± 3 | 32 ± 4 | 34 ± 4 | 33 ± 4 | 33 ± 4 | 33 ± 4 |
| Apo A-I | | | | | | | |
| Uncorrected | 123 ± 26 | 120 ± 26 | 123 ± 26 | 121 ± 22 | 123 ± 25 | 119 ± 26 | 121 ± 25 |
| PVcorr | 123 ± 26 | 123 ± 28 | 122 ± 25 | 122 ± 20 | 120 ± 20 | 116 ± 21 | 118 ± 23 |
| Apo A-II | | | | | | | |
| Uncorrected | 31 ± 4 | 30 ± 3 | 30 ± 2 | 32 ± 3 | 31 ± 3 | 31 ± 3 | 31 ± 3 |
| PVcorr | 31 ± 4 | 31 ± 3 | 30 ± 2 | 32 ± 3 | 31 ± 5 | 31 ± 4 | 30 ± 3 |
| Body weight (kg) | | | | | | | |
| Uncorrected | 79.0 ± 14.8 | 78.7 ± 14.7 | 79.0 ± 14.5 | 79.0 ± 14.7 | 78.9 ± 14.7 | 78.8 ± 14.9 | 78.7 ± 14.6 |

NOTE. Values are in mg/dL; n = 9 for body weight.

in plasma volume and cholesterol were not impressive. On day 5, the Spearman rank correlation for absolute change in total cholesterol and percent change in plasma volume was −.47 ($P$ = .054). For percent change in total cholesterol and percent change in plasma volume, $r$ was −.39 ($P$ = .11). On day 10, the correlation between absolute change in cholesterol versus percent change in plasma volume was −.35 ($P$ = .14) and was −.34 ($P$ = .17) for percent change in cholesterol versus percent change in plasma volume. Given that influences on both total cholesterol and plasma volume are multifactorial, it is not surprising that the correlations are not stronger. Nevertheless, some association is suggested, and these results are consistent with the finding of a weak association ($r$ = .32) between total cholesterol and hematocrit.[7]

It is likely that intramonthly variation in ovarian and other hormones affects plasma volume. Both plasma renin activity and aldosterone levels are higher in the midluteal than in the midfollicular phase,[29] an effect possibly related to progesterone secretion.[30] Estrogen may also contribute to plasma volume expansion, since estradiol injections decrease urine output and renal excretion of sodium and chloride.[31,32]

Only women who reportedly did not routinely use medications were recruited for the present study. Nevertheless, five women used acetaminophen and nine women used a medication other than or in addition to acetaminophen. Three of these nine used nonsteroidal antiinflammatory agents, which are known to expand plasma volume.[33] We did not detect differences in the total cholesterol response

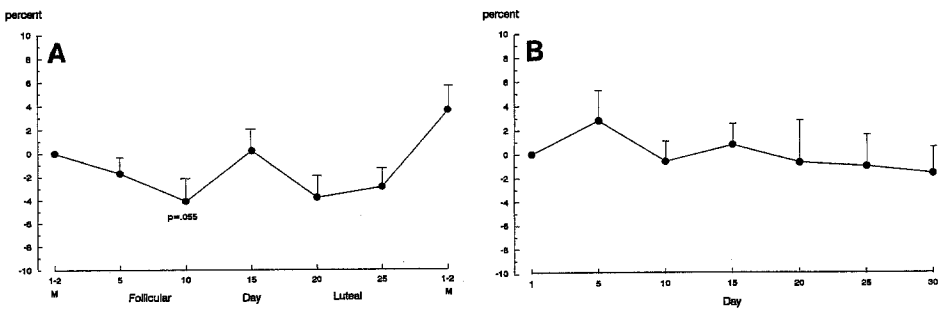


**Fig 1. Mean estimated changes in plasma volume (%) for (A) women (n = 18) and (B) men (n = 11). M, menstruation; value in women at day 10 is different from day 1 ($P$ = .055) using Wilcoxon signed-rank test for nonparametric data. Bars represent SEM.**

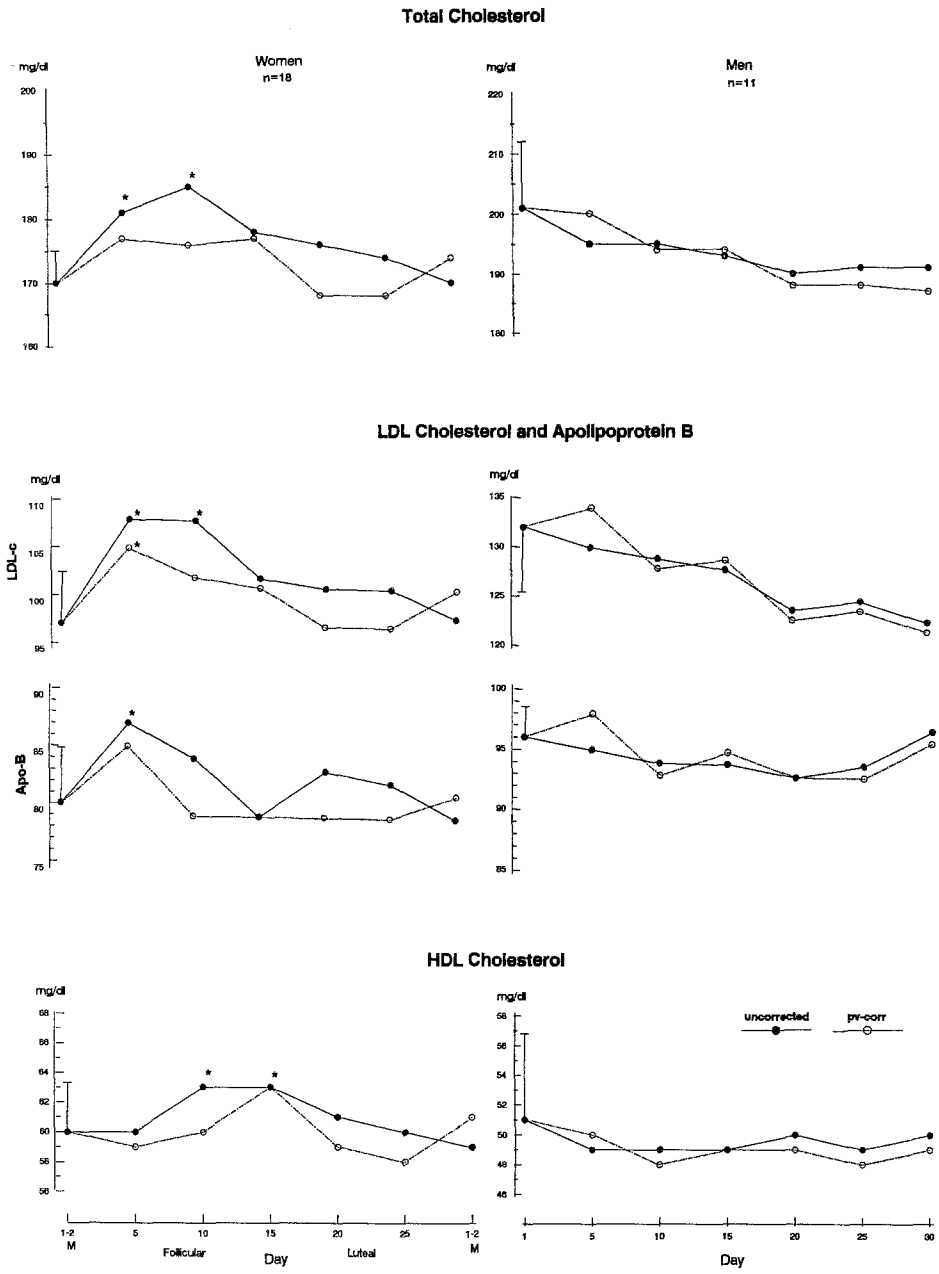


**Fig 2. Total cholesterol LDL cholesterol and apo B, and HDL cholesterol before and after correcting for plasma volume (pv-corr) for women (n = 18) and men (n = 11). *Statistically different from sample 1 ($P < .05$). Bars represent SEM.**

when reexamining the four subjects who did not use a medication and those that used a medication other than acetaminophen. Total cholesterol in both groups peaked 5 to 10 days after menstruation, and the cholesterol curves were similar between the groups both before and after correction for estimated changes in plasma volume (Fig 3).

Eight women exercised during the study, but there was no consistent pattern to the exercise that would explain the plasma volume and lipid changes noted during the menstrual cycle. In addition, the pattern of changes in total cholesterol and percent change in plasma volume for the exercisers resemble patterns for the entire group. Total cholesterol increased 11 mg/dL at day 5 and 18 mg/dL at day 10 in the exercisers (compared with respective increases of 11 and 15 mg/dL for the entire group). As for the entire group, this peak increase in total cholesterol at day 10 in the exercisers was accompanied by a peak decrease in plasma volume (−5.7% ± 7.3%, mean ± SD). After correction for percent change in plasma volume, the increase in total cholesterol at day 5 was reduced by 3 mg/dL in the exercisers (versus a 4-mg/dL reduction in the entire group), and the increase at day 10 was reduced by 12 mg/dL (compared with a 9-mg/dL reduction in the entire group).

Published changes in HDL cholesterol during the menstrual cycle are not consistent, with different studies reporting peak changes during both the follicular[4,34] and luteal[2,23,35] phases. The 5- to 7-mg/dL increases observed by Mattson et al[3] are among the largest reported, with the highest concentrations occurring in the late follicular and late luteal phases. Most reported changes in HDL choles-

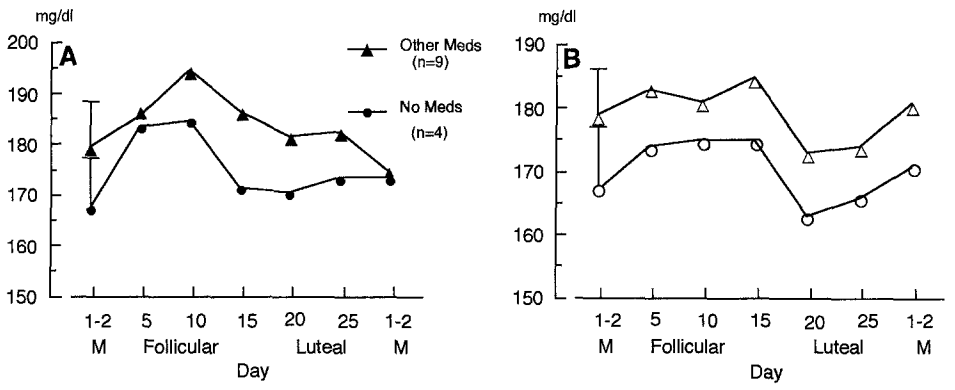


**Fig 3. (A) Mean total cholesterol and (B) corrected for plasma volume for women using no medication (●, ○; n = 4) and women using medication besides acetaminophen (▲, △; n = 9). Bars represent SEM.**

terol are smaller and comparable to the 3-mg/dL increase in the present study. Consequently, despite small intramonthly changes, HDL cholesterol remains persistently elevated in premenopausal women throughout the menstrual cycle.

The major finding in the present study was the increase in total (15 mg/dL, or 9%) and LDL (11 mg/dL, or 11%) cholesterol within 10 days after the onset of menstruation. Adjustment for estimated changes in plasma volume reduced these increases to 7 and 8 mg/dL, respectively. These results suggest that approximately half of the increase in total cholesterol during the follicular phase is due to changes in plasma volume, and that this factor contributes to the intramonthly variation of serum cholesterol in premenopausal women.

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## APPENDIX

### Formula for Estimating Percent Change in Plasma Volume

NOTE. Blood volume pre is assumed to be 100.

Blood volume post = blood volume pre · (hemoglobin pre/hemoglobin post)

Cell volume = blood volume/(hematocrit)

Plasma volume = blood volume − cell volume

Percent change (%Δ) blood volume = 100(blood volume post − blood volume pre)/blood volume pre

%Δ cell volume = 100(cell volume post − cell volume pre)/cell volume pre

%Δ plasma volume = 100(plasma volume post − plasma volume pre)/plasma volume pre

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