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ABSTRACT

The purpose of this study was to investigate among postmenopausal women the relationship of dietary fat intake, tobacco smoking, alcohol use, physical activity, and body weight to total cholesterol, high density lipoprotein (HDL) cholesterol, and low density lipoprotein (LDL) cholesterol measures in order to assess the relative influence of each variable on approximating recommended lipid levels. Subjects were drawn from three age-segregated mobile home parks in a popular retirement area of South Texas. Exclusion of persons receiving lipid-lowering medications or hormone replacement therapy resulted in a sample of 238 white females ranging in age from 57 to 82 years with a mean age of 68.2 years. Data were gathered through a self-report health risk appraisal instrument and venipuncture drawn blood samples. Stepwise regression analysis indicated that the strongest modifying influences on total cholesterol were physical activity and alcohol nonuse. Physical activity was the primary modifier of LDL cholesterol, while desirable body weight exerted the greatest influence on HDL cholesterol. Findings suggest that for older women nontherapeutic serum lipid modification leading to reduced atherosclerotic risk is primarily a function of the positive health behaviors of tobacco nonuse and increased physical activity. (Author/LL)

Mediating Influences on Serum Lipids Among Postmenopausal Women*

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Mediating Influences on Serum Lipids Among Postmenopausal Women

This paper describes the modifying influences of selected lifestyle variables on serum total-, LDL-, and HDL-cholesterol levels among postmenopausal women.

Abstract

The purpose of this study was to investigate among postmenopausal women the relationship to total cholesterol, HDL cholesterol, and LDL cholesterol measures with the variables of dietary fat intake, tobacco smoking, alcohol use, physical activity, and body weight to assess each variable's relative influence on approximating recommended lipid levels. Subjects were drawn from three age-segregated mobile home parks located in a popular retirement area of South Texas. Exclusion of persons receiving lipid-lowering medications or hormone-replacement therapy resulted in a sample of 238 white females ranging in age from 57 to 82 years with a mean age of 68.2 years. Data were gathered through a self-report health risk appraisal instrument and venipuncture drawn blood samples. Stepwise regression analysis indicated the strongest modifying influences on total cholesterol were physical activity and alcohol nonuse. Physical activity was the primary modifier of LDL cholesterol, while desirable body weight exerted the greatest influence on HDL cholesterol. Findings suggest that nontherapeutic serum lipid modification is primarily a function of the positive health behavior of increased physical activity through its direct influence on total and LDL cholesterol and indirect influence on HDL cholesterol through affecting weight maintenance.

Coronary heart disease (CHD) is the leading cause of mortality in women; however, it does not become so until they reach their mid-sixties.[1] Atherosclerosis, an accumulation of lipid plaque within the arterial wall, is the most common type of CHD.[2] The importance of serum lipid levels as risk factors in atherosclerosis is well-documented.[3-6] There is also a large body of evidence indicating altered levels have beneficial effects in the secondary prevention of this CHD type.[7-10] The consensus of the literature is cholesterol-carrying lipoproteins are independent predictors of atherosclerotic risk. Serum total cholesterol and low density lipoprotein (LDL) cholesterol are positively correlated with CHD, whereas high density lipoprotein (HDL) cholesterol levels are inversely related.

It is widely reported that menopause affects occurrence of atherosclerosis through serum lipid changes induced by estrogen deprivation after the cessation of ovarian function. Studies have shown higher total and LDL cholesterol and lower HDL cholesterol in postmenopausal than premenopausal women.[11-14] The epidemiologic evidence of a protective effect of hormone replacement therapy against CHD is compelling.[15, 16] Nonetheless, while approximately 3 million post-menopausal women are receiving replacement therapy to alleviate symptoms associated with menopause, for an array of reasons, a significantly larger number are not.[17]

Given the powerful relationship between menopause and serum lipid changes, investigating the modifying effects of those variables affecting lipids not explained by hormone or other types of drug therapy could help provide a more appropriate alternative or adjunct to current medication regimens. Among the factors associated

with serum lipids are diet, smoking, alcohol intake, physical activity, and body weight.[3,18-20] Although it has been established beyond a reasonable doubt these links exist, of great importance would be explanation of the extent to which individual factors predict desired lipid levels. Information concerning strengths of contribution could impact efforts designed to help modify levels thus enhancing atherosclerosis intervention. The present study evaluated the relationship in postmenopausal women of total cholesterol, HDL cholesterol, and LDL cholesterol measures with selected risk variables to assess each variable's relative influence on modifying lipid levels.

Methods

Subjects were taken from a universe of three age-segregated mobile home parks located in a popular retirement area, Hidalgo County, located in the Lower Rio Grande Valley region of Texas. The three parks were each randomly selected from one of five county areas in which parks were stratified by geographic location based on city size and/or proximity.[21] Following a prearranged presentation by the investigator in the park recreation hall, a one-page flier describing the study and seeking volunteers was distributed. Through this method the convenience sample was drawn. Data were collected during December, 1992 and January, 1993.

Information on serum lipid variables was obtained through a self-report instrument dealing with dietary fat intake, tobacco smoking, alcohol use, physical activity, and body weight. The dietary fat index, developed by Knapp and others,[22] is

a 6-item Likert-type scale indicating the extent to which the respondent specifically avoids food sources high in saturated fats and cholesterol. Instrument component item validity was determined by t-tests performed between fat avoidance responses and dietary recall. A significant relationship ($p < .01$) existed between lipid consumption responses and actual intake. Chronbach's alpha of internal consistency (.50) indicated each of the six items contributed to the scale's reliability; i.e. deletion of an item resulted in a lower alpha coefficient. While the coefficient falls below the .60 considered necessary for scales designed to measure individual traits,[23] it appears to be adequate for measuring group traits. A more detailed discussion of instrument validation with recall methodology is presented elsewhere.[22,24]

The index inquired how often skin was removed from chicken; the type of milk most often used; the type of hamburger meat eaten most often; the type of fat used most often in cooking; how often fat was trimmed from meat; and the number of eggs eaten per month. Scale scores for all six items could range from 0 to 6.0, with 6.0 indicating maximum fat avoidance. The remaining demographic and lifestyle variables were assessed through a health risk appraisal instrument developed by the Centers for Disease Control[25] measuring Likert-type frequencies, ranging from 1 to 5, with which one engages in each of the subscales. A pilot test involving 28 elderly females (elderly defined as 60 years or older) indicated the instrument appropriate and suitable in terms of reliability, comprehension, ease of administration, and intended use.

A woman was designated postmenopausal if she reported no menstrual period in the previous six months or described herself as being in menopause. To ascertain

for drug therapy, the respondent was asked to indicate (Yes or No) if she were receiving hormone replacement treatment and/or lipid-lowering medications. An affirmative reply to either item excluded the respondent from the study. If any subject was unsure as to her status or there were questions regarding other medications, use or non-use and/or possible contraindications was ascertained by having prescriptions or pill containers brought during drawing of blood samples where the declaration was made by a certified technician. Additionally, age and ethnicity were appraised by single item measures.

Blood samples were obtained by venipuncture following a 12-hour fast with subject fasting compliance discussed prior to testing. Lipid and lipoprotein levels were measured according to Lipid Research Clinics[26] procedures. Total cholesterol was measured by enzymatic techniques with a biochromatic analyzer and a high performance cholesterol reagent (Technicon Instruments, Tarrytown, NY). HDL cholesterol was measured by precipitating the other lipoproteins with heparin and manganese chloride according to standardized methods. LDL cholesterol was calculated using the Friedwald formula.[27] All data collection was conducted at a prearranged single session within each park and no subject was represented more than once.

Pearson zero-order correlation coefficients were used to investigate the general relationship between total-, LDL-, and HDL-cholesterol measures and the risk variables of dietary fat intake, smoking, alcohol use, physical activity, and body weight. The hypothesis of no significance of correlates was tested at the .01 and .05 levels of confidence. Stepwise regression analysis was performed to determine the

relative contribution of each variable to lipid levels approaching recommended measures. Measures (total cholesterol ≤200 mg/dl; LDL ≤130 mg/dl; HDL ≥35 mg/dl) established by the National Cholesterol Education Program were used as cutoff points in the regression.[28]

Using lipid levels as the dependent variable and risk factors as predictor or independent variables, standardized beta coefficients were computed to reflect the amount of expected change in levels for a unit change in the independent risk factor with the remaining factors held constant. The SAS[29] statistical package was used for all data analyses.

Results

A total of 308 individuals were tested. Deletion of incomplete response instruments, fasting non-compliance, or hyperlipidemia medication/hormone replacement resulted in a study sample of 238 white postmenopausal females ranging in age from 57 to 83 years with a mean age of 68.2 years. Table 1 presents sample lipid means, ranges, and standard deviations. Pearson correlation coefficients between lipid measurements and the risk variables of diet, tobacco smoking, alcohol intake, physical activity, and body weight are contained in Table 2. Observance of lipid levels with risk variables revealed relationships in the expected direction. Serum measurements were significantly related to alcohol use, physical activity, and body weight. Total-, LDL-, and HDL-cholesterol were positively associated with higher alcohol consumption. Increased physical activity lowered total and LDL cholesterol

elevated HDLwith the inverse occurring with tendency toward overweight. No statistically significant relationships were found between lipid levels and either dietary fat intake or tobacco smoking.

Regression analysis performed to determine the relative contribution of all variables in the prediction of approximating recommended lipid levels is presented in Table 3. The strongest modifying influence on total cholesterol was physical activity followed by alcohol nonuse. Sixteen percent of the variance was explained by a combination of these two variables. Physical activity and nonsmoking (15% of the variance combined) were the primary modifiers of LDL cholesterol while desirable body weight and nonsmoking (26% of the variance combined) exerted the greatest influence on HDL cholesterol modification.

Discussion

The present study provides relationships between cholesterol-carrying lipoproteins and selected risk variables in postmenopausal women. The strong correlations between alcohol, physical activity, and body weight variables and serum lipids are consistent with the results of other reports.[18, 19, 30]

Of particular interest was alcohol consumption; not only were recommended total and LDL cholesterol levels elevated through increased consumption, more than one drink per day, but HDL also. Alcohol's ability to elevate HDL notwithstanding, its profoundly negative effects on the body and strong association with many disease states far outweigh any perceived benefit.

In terms of the zero-order relationships reported here, several risk variables were significantly related to the subjects' serum lipid levels. The findings, however, indicate a number of the observed general relationships disappeared when the variables were introduced individually with the remaining variables held constant to control for potential confounding. Among the subjects studied, alcohol use exerted no significant effects on LDL and HDL cholesterol; physical activity had no effect on HDL cholesterol, and increased body weight had no effect on LDL cholesterol.

All variables investigated undoubtedly contribute in some degree to atherosclerotic risk, however, the most potent individual modifiers appear to be tobacco nonuse and increased physical activity due to their predictive influence on total and LDL cholesterol. Assuming higher body weight, the most powerful predictor of HDL cholesterol, to be a corollary of decreased energy expenditure, increased activity could subsequently also act as a HDL modifier.

Even though heart disease is the primary cause of death among postmenopausal women and those receiving hormone replacement therapy typically have half the risk of nonusers,[15, 31] there is a growing body of evidence indicating behavioral changes, even in old age, can benefit health and quality of life. This research suggests that for elderly females, nontherapeutic serum lipid modification leading to reduced atherosclerotic risk is primarily a function of the positive health behaviors of tobacco nonuse and increased physical activity. Additional study should be conducted to further clarify the relationships between health practices and serum lipids. Of particular importance would be investigation into

lipid response rates to selected practices over time thus enhancing modifying applicability.

Table 1. Serum Lipid Distributions

	Mean	Standard Deviation	Range
Total cholesterol (mg/dl)	237.9	53.8	150-608
LDL cholesterol (mg/dl)	158.6	44.7	42-465
HDL cholesterol (mg/dl)	46.1	10.7	21- 74

Table 2. Coefficients of Lipid Measurements With Risk Variables

	Risk Variable				
	Diet	Smoking	Alcohol Intake	Physical Activity	Body Weight
Total cholesterol	-.101	.076	.126*	-.280**	.085
LDL cholesterol	-.095	.019	.137*	-.254**	.146*
HDL cholesterol	.047	-.044	.230**	.148*	-.237**

**p<.01
*p<.05

Table 3. Stepwise Regression Analyses With Lipid Levels as Dependent Variables

Lipid	Standardized Beta Coefficients				
	Diet	Smoking	Alcohol Intake	Physical Activity	Body Weight
Total cholesterol (≤200 mg/dl)	-.093	.108	.141*	-.339**	.101
LDL cholesterol (≤130 mg/dl)	-.067	.173*	.138	-.332**	.073
HDL cholesterol (≥35 mg/dl)	.081	-.297**	.088	.144	-.387**

**p≤.01
*p ≤.05

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