Relationship of Plasma Lipoprotein Lp(a) Levels to Race and to Apolipoprotein B


Lipoprotein Lp(a) is an atherogenic subfraction of plasma lipoproteins which has been studied predominantly in white populations. We quantified Lp(a) by electroimmunoassay in plasma from 105 black and 134 white healthy men and women. Results were correlated with clinical variables and plasma levels of lipids, other lipoproteins, and apolipoprotein (apo) B determined by radioimmunoassay. Black subjects had levels of Lp(a) that averaged twice those of whites (p < 0.001). Among blacks, Lp(a) levels showed a bell-shaped frequency distribution, while among whites the distribution was strongly skewed, with the highest frequencies at low levels. Contrary to previously published results, the apo B levels in our study correlated significantly, though weakly, with Lp(a) (r = 0.21, p = 0.001 among whites, and r = 0.15, p = 0.02 among blacks, Kendall rank correlation). The regression slopes and variances suggested that apo B in the Lp(a) lipoprotein could account for the correlation. Lp(a) levels did not correlate significantly with any other plasma lipoprotein or lipid levels.

The implications of this study are as follows: Despite the high levels of Lp(a) among blacks in the Houston area, these blacks do not experience greatly increased atherosclerotic progression and mortality. Thus, the atherogenicity of Lp(a) in blacks must be decreased or counterbalanced by other factors. The correlation between Lp(a) and apo B should be taken into account when analyzing atherogenic risk, but this correlation is not strong enough to dispute the independence of Lp(a) and apo B as risk factors. (Arteriosclerosis 5:265-272, May/June 1985)

Lipoprotein Lp(a) is a cholesterol-rich lipoprotein that can be identified by its characteristic Lp(a) antigenic activity in human plasma.1 2 The apoproteins of lipoprotein Lp(a) include apolipoprotein (apo) B and apo (a), the antigenic determinant.2 Lipoprotein Lp(a) migrates as a distinct prebeta, band on agarose gel electrophoresis1 and shows an apparent hydrated density in the range of 1.05 to 1.12 g/ml on ultracentrifugation.2 It is identical to the "prebeta," or the "sinking-prebeta" lipoprotein.3 4 Catabolic rates and pathways of lipoprotein Lp(a) resemble those of low density lipoprotein (LDL).3 5 Other metabolic aspects are obscure but appear to be distinct from those of LDL.7 9

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Despite the fact that lipoprotein Lp(a) typically carries less than 15% of the cholesterol in plasma, levels of this lipoprotein are strongly and positively associated with coronary heart disease.3 4 7 10 11 Plasma levels of apo B also are strongly associated with the risk for coronary heart disease, and in certain situations, apo B appears to impart risk independently of total plasma cholesterol or LDL cholesterol.12 15 However, there are few prevalence data in which both Lp(a) and apo B have been measured in the same group of individuals.4 Such data are needed to clarify the interactive roles of Lp(a) and apo B in lipid metabolism and atherogenesis.

Further understanding may be gained by measuring these putatively atherogenic entities within population groups that differ in their average risk of cardiovascular disease. As measured at autopsy, blacks showed less propensity for atherosclerosis than whites,16 17 and black men have relatively low mortality from coronary heart disease.18 19 Thus far, the comparison of atherogenic apolipoprotein levels in black versus white individuals is limited to qualitative Lp(a) determinations;20 21 we know of no relevant data for apo B.
We have performed a study of plasma concentrations of Lp(a), apo B, cholesterol, triglycerides, and high density lipoprotein (HDL) cholesterol in an urban biracial population group. Clinical background data were obtained from study participants. We found that: 1) black and white volunteer groups differed markedly in their patterns of Lp(a) plasma concentrations; and 2) Lp(a) and apo B levels were correlated independently of routine lipoprotein lipid levels.

Methods

Subjects

The study group contained 239 healthy volunteers 18 to 58 years old, recruited from employee groups at The Methodist Hospital and Baylor College of Medicine, Houston, Texas. Informed consent was obtained before blood was drawn. The study protocol was approved by the Institutional Review Boards for Human Research at both participating institutions. Race was determined by self-identification, skin color, and other phenotypic features. The definition of white race excluded persons with Spanish surnames and persons of Asian extraction. All subjects were questioned for history of symptoms of coronary and atherosclerotic diseases and were excluded if such a diagnosis was likely. Pregnancy and diabetes were other reasons for exclusion. Medication use included estrogen preparations in 19 subjects, thiazide diuretics in six, and beta blockers in two. These were distributed equally between racial groups. Patients were questioned about alcohol intake and cigarette smoking. Blood pressures in all subjects and height and weight in the last 190 subjects were recorded by a trained technician. Body mass index (BMI) was calculated as weight (kg) divided by height (m) squared.

Plasma Lipid and Lipoprotein Determination

Blood was obtained from all subjects after an overnight fast. Blood was drawn into tubes containing ethylenediamine tetraacetic acid (EDTA), and the plasma was separated without delay by low-speed centrifugation. Plasma cholesterol and triglycerides were determined the same day by an autoanalyzer technique according to the Lipid Research Clinics Program protocol. HDL cholesterol was determined by autoanalyzer after precipitation of plasma triglycerides by heparin-manganese according to the Lipid Research Clinics Program protocol. HDL cholesterol was determined by autoanalyzer after precipitation of plasma triglycerides by heparin-manganese according to the Lipid Research Clinics Program protocol. Since plasma triglyceride levels never exceeded 350 mg/dl, the values for LDL cholesterol were estimated according to the following formula:

\[ \text{LDL cholesterol} = \text{total cholesterol} - \text{HDL cholesterol} - \frac{\text{triglycerides}}{5} \] (1)

Determination of Lp(a) and Apo B

Antisera against Lp(a) were prepared from rabbits. Six rabbits were immunized with Lp(a) isolated from plasma by ultracentrifugation and gel filtration on a Bio-Gel A-15M column. Antigen (0.5 mg) was mixed with 1 ml complete Freund’s adjuvant to make a thick emulsion that was injected intradermally. The rabbits were given booster injections at 3-week intervals and were bled when adequate titers were achieved. To obtain an antiserum monospecific for Lp(a) antigen, the rabbit anti-Lp(a) serum was adsorbed with isolated LDL. The visible precipitate was removed by low-speed centrifugation. When the supernatant was tested by double diffusion against human plasma and purified lipoproteins, it gave a single precipitin line with plasma showing complete identity with Lp(a) but not LDL.

Electroimmunoassay was used to quantitate Lp(a) in plasma samples. Samples from black and white individuals were collected and analyzed concurrently, with racial identities unknown during analysis. Samples were stored at 4° C for a few hours or overnight before analysis. Adsorbed monospecific rabbit antiserum from the same bleeding was used throughout the study. Approximately 80 μl of antiserum were added to 25 ml 1% agarose to form the gel. Electrophoresis was performed on a water-cooled electrophoresis cell for 16 hours. A standard barbital electrophoresis buffer (pH 8.6) was used. Lp(a) concentrations were calculated by comparison of peak heights of sample and standard rockets. A serum sample from an individual with a high Lp(a) level was kept frozen at -60° C in small aliquots and used as a standard. This serum was standardized by electroimmunoassay against purified Lp(a) that had a known protein and lipid content. The lowest standard dilution always gave a peak higher than that of the samples, and the standard dilution curve was linear with a correlation coefficient of 0.99. Double determinations of the control serum in each run gave a between-day coefficient of variation of 4.2%.

The electroimmunoassay for LDL was performed on LDL purified by zonal ultracentrifugation from plasma pooled from 18 individuals. Less than 1% of the protein content of this preparation was detected as Lp(a) protein by electroimmunoassay. To determine the behavior of Lp(a) lipoprotein during routine HDL cholesterol determinations, Lp(a) electroimmunoassay was performed on plasma before heparin-manganese precipitation and on the supernatant afterwards. The Lp(a) lipoprotein was found to be totally precipitated by this technique.

Subsequent to the determinations that formed the basis for this report, an anti-Lp(a) antibody purified by affinity chromatography using an Lp(a)-Sephrose column has become available (JW Gaubatz and JD Morrisett). The electroimmunoassay determinations for Lp(a), using the affinity-purified antibody, were compared with the determinations using antisera prepared as described above. Plasma samples from two black and two white individuals served as unknowns, and plasma from a third black individual served as the reference material. Measured Lp(a) levels showed small discrepancies due...
to the antibody preparation (no greater than 19%, averaging 6% higher for gels made with affinity-purified antibody). The apo B content of plasma frozen at -60° C was measured by radioimmunoassay25 using a specific polyclonal antibody directed against LDL. LDL used for immunization, for preparation of tracer, and as a standard was prepared by zonal ultracentrifugation. More than 98% of the protein moiety was insoluble in tetramethylurea.26

**Statistical Analysis**

Student's t-test, analysis of variance, and parametric and nonparametric regression analyses were performed using the Statistical Package for the Social Sciences. For nonparametric statistics, both Kendall and Spearman rank correlation procedures were performed with similar results. Because of frequent tie scores, the Kendall statistics are reported.

**Results**

**Clinical Characteristics**

Table 1 summarizes the clinical characteristics of the subjects by race and sex groups. White subjects were significantly older than blacks, but this study clearly deals with a predominantly young adult population in every race-sex group. Calculations of body mass index showed white women to be significantly leaner than black women. A positive history for smoking was obtained in only 37 of 232 subjects (16%) whose responses to this question were recorded. Smoking was particularly rare (6%) among the white women. Alcohol usage was more common among whites than among blacks. This tendency was significant for the comparison of all whites to all blacks (p = 0.02), but not for comparisons within sex.

**Plasma Determinations**

The plasma lipids and Lp(a) and apo B levels are summarized in Table 2. The total cholesterol levels tended to be lower in blacks than in whites, and this difference was significant (p = 0.04) for the comparison of all blacks to all whites. The lipoprotein fractions responsible for the lower total cholesterol among blacks were different between men and women. HDL cholesterol was significantly lower in black women compared to white women (p = 0.006). However, covariance analysis adjusting for differ-
ences in clinical characteristics (see below) negated this finding. Among men, LDL cholesterol was significantly lower in blacks compared to whites ($p = 0.03$), whereas HDL cholesterol tended to be higher in black men compared to whites ($p = 0.13$). Mean triglyceride levels did not differ significantly between races for either sex.

The most striking and statistically significant racial differences were found in the mean levels of plasma Lp(a), which were twice as high in black subjects as in whites ($p < 0.001$ for men, $p < 0.001$ for women). There was no apparent effect of gender on plasma Lp(a). The racial difference in mean Lp(a) levels could be attributed to a markedly different profile for the frequency distribution of Lp(a) levels, as shown in Figure 1. Two-thirds of the white subjects had Lp(a) concentrations below 16 mg/dl, and the frequency distribution was highly skewed. Only 18% of Lp(a) plasma concentrations in blacks were below 16 mg/dl, and a bell-shaped distribution was apparent. In all subjects, 19% (31% of whites, 5% of blacks) had Lp(a) levels below the limit of detection of the assay (about 5 mg/dl).

The estimated LDL cholesterol levels shown in Table 2 may be reconsidered in light of the racial difference in Lp(a). Although Lp(a) is found mostly in the HDL range by ultracentrifugation, it behaves like LDL in the heparin-manganese precipitation technique for measuring HDL cholesterol. Thus Lp(a) cholesterol is included in the LDL cholesterol estimates in Table 2. Considering that 30% of the mass of Lp(a) is composed of cholesterol, the true reduction in LDL cholesterol in black men compared to whites would be close to 16% rather than 12%. Similarly, in black women compared to whites the true reduction in LDL cholesterol may be approximately 6% instead of 1.5%.

Apo B levels showed no significant differences due to race. However, the trends in the mean levels were consistent with the racial effects noted for LDL cholesterol.

Because of the influence of age, body mass index, smoking, and drinking on plasma lipoproteins, the differences in Table 2 were also tested by a covariance analysis which adjusted for these clinical variables. The higher levels of Lp(a) in blacks ($p = 0.01$ for men, $p = 0.001$ for women) and the significantly lower LDL cholesterol level in black men as compared to white men ($p = 0.02$) were confirmed. Covariance analysis also suggested that the difference in HDL cholesterol between black and white men is significant ($p = 0.03$). However, the significant difference in HDL cholesterol due to race among women disappeared ($p = 0.69$), suggesting that the difference shown in Table 2 was due to acquired clinical characteristics.

**Relationship of Lp(a) to Other Variables**

Parametric and nonparametric correlation coefficients were obtained in the total subject population and within racial groups for crosswise comparison among the continuous variables of age, body mass index, diastolic blood pressure, lipids, lipoprotein cholesterol fractions, Lp(a) and apo B. Many expected associations, such as a positive relationship between age and total plasma cholesterol, were found. In this comparison, plasma Lp(a) did not correlate significantly with age, total cholesterol, LDL cholesterol or any other continuous variable except apo B. The deviation from normal of the Lp(a) distribution in whites dictated that nonparametric correlation with apo B be performed. The correlation coefficients for blacks (0.15) and for whites (0.21) showed that the correlation was weaker than that of apo B with LDL cholesterol (0.48 for blacks, 0.46 for whites). The relationship between apo B and Lp(a) was, nevertheless, significant among blacks ($p = 0.02$) and whites ($p = 0.001$). Because a simple linear relationship between apo B and Lp(a) might be postulated on theoretical grounds, a linear, parametric regression of apo B on Lp(a) was also performed. Correlations were similar to the nonparametric statistics just given, except that in blacks the $r$ value was of borderline significance ($p = 0.06$). The slopes of the least square regression lines, 0.24 ± 0.13 (SE) for blacks and 0.34 ± 0.11 for whites, were not significantly greater than a theoretical slope of 0.18 based on the composition of the Lp(a) lipoprotein (see Discussion).

Multiple stepwise regression within race was performed to test the possible confounding effects of gender, LDL cholesterol, HDL cholesterol, and triglycerides on the relationship of apo B to Lp(a). Apo B levels in whites were found to be associated with the following (in order of decreasing variance fractions): LDL cholesterol, triglycerides, and Lp(a). All these associations were significant at $p < 0.001$. The change in $r^2$ associated with the entry of successive variables indicates the fractions of the total variance of apo B that may be attributed to the variables. These fractions ($r^2$) were 0.62 for LDL cholesterol, 0.04 for triglycerides, and 0.04 for Lp(a). The distri-

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**Figure 1.** Frequency distributions of Lp(a) levels in whites and blacks.
bution of these variance fractions is similar to the expected distribution of apo B within lipoprotein classes, i.e., LDL, very low density lipoproteins, and Lp(a). Among the black subjects, Lp(a) was not significantly associated with apo B by multiple regression.

The only clinical variable possibly associated with higher Lp(a) levels was smoking history. Analysis of variance using race and smoking class (<10 versus ≥10 cigarettes per day) as independent variables showed the effect of smoking to be positive and significant (p = 0.02). However, when a dose-response effect was sought by reassigning smokers to four classes (0, 0 < x < 10, 10 ≤ x < 20, and ≥20 cigarettes per day) the effect of smoking was no longer significant (p = 0.11). A history of alcohol intake was not associated with Lp(a) levels, and neither smoking nor alcohol history affected apo B levels.

**Discussion**

The major new findings in this study are the higher levels of Lp(a) lipoprotein in black individuals and the correlation between Lp(a) and apo B plasma levels.

Although the immunologic basis for detection of Lp(a) was discovered in 1963, the development of good quantitative assay techniques in several laboratories is more recent. Our mean Lp(a) plasma level for whites, 16.3 mg/dl, is close to that (140 mg/dl) for Washington State residents determined by a radial immunodiffusion technique on plasma by Albers and Hazzard. Using rocket immunoassay, Enholm et al. found mean plasma Lp(a) levels of 21.3 mg/dl in European volunteers. Kostner et al. found similar levels (21.4 mg/dl) in Europeans. These levels are close to our results in white subjects. The subsequent development of radioimmunoassay by Albers et al. clarified the status of individuals with Lp(a) levels too low to detect by gel and immunoelectrophoresis techniques. These persons all had detectable, though low, levels of Lp(a), except for one person who had abetalipoproteinemia.

Berg compiled data on the results of qualitatively testing the plasma Lp(a) antigen in various population groups. Among Norwegians, the most extensively studied group, 35% of the population were positive and the remainder, negative. Among 242 black individuals from the Washington, D.C. area, 34% were positive. In our study, 35% of the white subjects and those studied by Berg.

Plasma levels of Lp(a) exhibit a very high degree of heritability. In a family study, genetic factors accounted for 74% of the variance in levels. The data were thought to be more consistent with polygenic inheritance, although the possibility of a single major gene suggested by earlier work could not be ruled out. In either case, it seems likely that black-white racial differences in Lp(a) levels will be explained largely by genetic rather than environmental factors. However, this supposition remains to be confirmed.

The association between coronary heart disease and a lipoprotein migrating at the prebeta, position on cellulose acetate or agarose gel electrophoresis was reported in 1972. Lp(a) lipoprotein has prebeta, electrophoretic mobility, and Lp(a) antigen in individual plasma samples correlates highly with a prebeta, band on electrophoresis. Studies involving both of these qualitative assays have confirmed the association of Lp(a)/prebeta, lipoprotein with a recent personal history of myocardial infarction, with a family history of coronary heart disease, and with angiographic evidence of coronary artery disease. On the basis of radioimmunoassay for Lp(a) in survivors of myocardial infarction, Albers et al. confirmed the association of high levels of Lp(a) with premature coronary disease. Kostner et al. also used a quantitative Lp(a) assay in myocardial infarction survivors and controls and suggested that in individuals with normal plasma cholesterol and triglycerides, Lp(a) levels higher than 30 mg/dl may signify 1.75 times the normal risk for myocardial infarction. The magnitude of this estimate is emphasized by their finding that 25% of their control group possessed these high levels of Lp(a). If these estimates are accurate, then the coronary risk imparted by Lp(a) would have major public health implications.

It should be noted, however, that the most extensive population studies of coronary risk due to Lp(a) have been conducted in Sweden, Finland, Seattle, and Austria, all regions with relatively few blacks. We recently reported the correlation between Lp(a) levels measured by electroimmunoassay and coronary heart disease measured by a semiquantitative angiographic assessment in a white population; there was a significant correlation in persons aged 55 years or younger.
Studies comparing black and white persons in the United States have suggested that coronary heart disease, but not cerebrovascular disease, develops more slowly and leads to fewer clinical events in black men. Autopsy data\(^1\) suggest that atherosclerotic fibrous plaques in the coronary arteries develop 10 years later in black men and women compared to whites. In Evans County, Georgia, black men had one-half the incidence of coronary heart disease than white men had, but rates in women were equal.\(^4\) In Texas between 1969 and 1971, the age-adjusted death rates from coronary heart disease were 13% lower in black men than in white men, but were 25% higher in black women than in white women.\(^1\) United States national statistics for 1970 were similar.\(^3\) Thus, coronary heart disease has been found consistently decreased in black men, but there have been variable results in women. Our finding of higher Lp(a) levels among blacks sheds no light on the apparent protection from coronary heart disease enjoyed by black men. It is possible that the atherogenicity of Lp(a) is decreased in black men or that other racial factors override the atherogenic effects of Lp(a). Further work is needed to decide between these possibilities.

Several large studies have compared lipoprotein and lipid levels between black and white populations. Higher HDL cholesterol levels and lower LDL cholesterol levels have been found in blacks as compared to whites, with greater differences generally found between black and white men.\(^3\) Our data for men are consistent with these earlier results. Among women, we actually found significantly higher HDL cholesterol levels in whites. However, closer examination using covariance analysis suggested that clinical characteristics, including lower body mass index and higher frequency of alcohol usage among the white women, may have accounted for this result. Thus the LDL and HDL cholesterol differences in this study are consistent with lower coronary risk in black men as compared to white men. The higher coronary risk among black women compared to white women may be attributed partly to the lack of such lipoprotein cholesterol differences due to race in women.

In this study, apo B was the only parameter measured in plasma that correlated with Lp(a) levels. It is unlikely that cross-reactivity between assay systems can account for this correlation. The Lp(a) electroimmunoassay detected less than 1% of the protein in LDL purified by zonal ultracentrifugation. This minimal value represents the sum of effects of cross-reactivity of the anti-Lp(a) serum and the actual presence of Lp(a) particles in the LDL preparation. Similarly purified LDL was used to induce antibodies for the apo B radioimmunoassay and to produce tracers for this assay.

Albers and co-workers\(^6\) reported no correlation between Lp(a) and apo B. The most important difference between their study and this one is that one-third of their study sample were patients who had been referred for hyperlipidemia, while less than 10% of our subjects were even minimally hyperlipidemic (plasma cholesterol >250 mg/dl or triglycerides >180 mg/dl). This apparent discrepancy might be explained as follows: A correlation between Lp(a) antigen and apo B antigen could be due to the coexistence of the two within the Lp(a) lipoprotein. Lp(a) may correlate poorly with the levels of other apo B-containing lipoproteins such as LDL and very low density lipoproteins (VLDL). When changes in the levels of LDL or VLDL account for an increased proportion of the variance of apo B as in diverse hyperlipidemic states, then the proportion of variance of apo B due to Lp(a) is reduced, and a correlation between apo B and Lp(a) is less detectable. When LDL and VLDL are more constant, as in this study, then the proportion of variance of apo B due to Lp(a) is increased, and the correlation becomes evident.

The slope and intercept of our correlations in both blacks and whites are consistent with the possibility that these correlations are entirely due to the apo B contained in Lp(a) lipoprotein. The intercepts of about 70 mg/dl of apo B are close to the expected mean plasma concentrations of apo B contained in LDL and VLDL. The slopes, which represent approximately 24 mg to 34 mg of apo B per 100 mg of Lp(a) lipoprotein, are close to an estimated concentration of 18 mg apo B per 100 mg of isolated Lp(a) lipoprotein. This estimate is derived from radioimmunoassay results showing that apo B comprises 61% of the total apoprotein mass in Lp(a) lipoprotein (W. Patsch and J. Gaubatz, unpublished observations) and from compositional studies\(^2\) showing that protein comprises approximately 30% of total lipoprotein mass. Thus, our results are consistent with the hypothesis of Albers et al.\(^4\) that the plasma level of Lp(a) lipoprotein is under metabolic control separate from the control of LDL and VLDL levels. It is noteworthy that clinical manipulations that affect plasma LDL levels (including cholesterol feeding to raise LDL\(^4\)) or administration of cholestyramine to lower LDL\(^6\) appear to have no effect on plasma Lp(a). Conversely, it was recently shown\(^9\) that the administration of stanozolol, an anabolic steroid, lowered plasma Lp(a) levels with little effect on apo B. This is further evidence of the metabolic independence between LDL, which is the major plasma reservoir of apo B, and Lp(a) lipoprotein.

Interest in apo B has been heightened recently by reports\(^12\) that apo B levels may predict coronary heart disease with greater discrimination than total or LDL cholesterol. The weak, though significant, correlation between plasma apo B and Lp(a), demonstrated here, implies a small degree of interdependence between these two coronary risk factors, but the major portion of risk from each probably is imparted independently.

We found a significant association between smoking and elevated levels of Lp(a), but there was no dose-response effect. Past cross-sectional studies on smoking and Lp(a) levels have yielded mixed,
mostly negative results. Most recently, plasma Lp(a) levels followed longitudinally in a group of successful smoking quitters showed no trend either downward or upward. Thus, the relationship between smoking and Lp(a) seen in the present study may be due simply to chance.

As the atherogenic potential of lipoprotein Lp(a) is increasingly confirmed, the clinical and biochemical correlates of this lipoprotein must be identified and its metabolic role must be defined. The demonstration of a mean two-fold increase in Lp(a) levels in blacks broadens our understanding of racial differences in lipoprotein metabolism, but does not help to explain the protection against coronary heart disease enjoyed by black men. Whether blacks experience substantial atherogenic risk from Lp(a) remains a question for future study. Knowledge of the physical association between apo B and Lp(a) antigens within the Lp(a) lipoprotein is extended by finding a significant, though weak, correlation between total plasma concentrations of these antigens. This correlation should be anticipated in future multivariable studies of atherogenic risk, but it will probably play only a minor role in the assignment of independent risk to apo B and Lp(a).

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References


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