Cigarette Consumption and Lipoprotein(a) Concentrations

To the Editor:

The habit of smoking cigarettes exerts various influences on lipid metabolism. It is well proved that smokers show lower concentrations of high-density lipoprotein (HDL) cholesterol but have higher concentrations of triglycerides and very-low-density lipoprotein (VLDL) [1–6]; an increase in peroxidation phenomena has also been described [7–11]. However, there is no unanimity concerning lipoprotein(a) [Lp(a)] concentrations. Some studies state that the smoking habit does not alter Lp(a) concentration; that is, neither would the consumption of cigarettes modify Lp(a) concentrations [12] nor would cessation of the tobacco habit mean any different in Lp(a) concentration shown by the same subjects during the previous stage of consumption [13]. However, a recent study comparing male and female smokers with nonsmokers found that the former presented lower Lp(a) concentrations than those of nonsmokers [14].

To try to clarify these findings, we studied the Lp(a) concentrations in 140 healthy male individuals belonging to the Academy of Cavalry in Valladolid, Spain after having obtained informed consent from each individual. This group was selected on the basis of a personal survey that included family or personal records of ischemic pathology or any other vascular disease, physical activity, and alcohol consumption. A standardized physical examination was also made which included height, weight, and blood pressure. The following inclusion criteria were used:

1) Males, both cigarette smokers for at least 5 years and individuals who had never smoked.
2) Healthy condition, checked through records, physical examination, and complementary analyses (electrocardiogram, thorax plates, and measurements of red and white blood cells, urinalysis, and blood chemistries, including creatine kinase).
3) Similar diets.
4) Similar physical activity.
5) Alcohol abstinence in all its forms.
6) No consumption of drugs at all.
7) Body mass index <27.
8) Blood pressure <140/90 mmHg.

The exclusion criteria were:

1) Consumption of alcohol.
2) Consumption of any kind of drugs.
3) Body mass index >27.
4) Blood pressure ≥140/90 mmHg.
5) Irregularities in nourishment.
6) Sedentary lifestyle.
7) Associated pathologies.

Using these criteria, we selected 140 men, who were classified in three groups: 76 nonsmokers, 21 light smokers (smoking ≤10 cigarettes per day), and 43 heavy smokers, who smoked >11 cigarettes daily. All participants fasted for 12 h, and venous blood was collected under standardized conditions, without stasis, into Vacutainer Tubes (Becton Dickinson, Rutherford, NJ) containing EDTA.

Lp(a) concentrations were determined in duplicate with a sandwich enzyme immunoassay using anti-Lp(a) monoclonal antibodies [15] (Terumo Medical Corp., Elkton, MD). The results were measured at 492 nm in an ELISA microplate spectrophotometer from SLT-Labinstruments Gesellschaft (Grödig-Salzburg, Austria).

The mean (±SD) Lp(a) concentrations for each group were as follows (mg/L): nonsmokers, 190 ± 110; all smokers (n = 64), 200 ± 120; light smokers, 205 ± 110; and heavy smokers, 190 ± 130.

The groups of all smokers, light smokers, and heavy smokers, were compared with each other and with the nonsmokers group: There were no significant differences between the mean concentrations of Lp(a) in each group.

Considering that the selection was very carefully performed to avoid other influences on Lp(a) concentrations inconsistent with the tobacco habit, and because the method of determination we used was sufficiently specific, we conclude that the different results obtained for Lp(a) so far in smokers are due to the biased distribution of Lp(a) in the population. To avoid this drawback, studies of a large number of very carefully selected cases are required.

References


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