# Biological, Psychological, and Social Characteristics of Men With Different Smoking Habits 

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[^0]THE POSSIBLE RELATIONSHIP of cigarette smoking to coronary heart disease, lung cancer, and various other illnesses has been investigated in a number of epidemiologic studies ( $1,1 a$ ). The same epidemiologic approach may provide useful knowledge concerning the distribution, and possibly the antecedents, of risk factors in disease. Previous studies directed toward investigation of the epidemiology of smoking habits have drawn attention to differences in social and cultural characteristics.

In the Tecumseh Community Health Study (2), for example, smoking habits were found to be associated with drinking habits, with smoking habits of other family members, and with other social characteristics. Lilienfeld (3) related smoking and selected emotional characteristics. In particular, smokers responding to questions concerning emotional status were found to express more "neurotic type" responses than nonsmokers. Similarly, the tendency of smokers to react to situations of stress with more anger has also been observed by Thomas (4), using a sample of medical students. Increased eating under stress was also characteristic of smokers in that study, although no differences were found in the Rorschach variables tested.

In the present study we examined selected biological, psychological, and social characteristics of men with different smoking habits. This, accordingly, is a limited epidemiologic study, not of a disease but of a characteristic believed to be associated with an increased risk of several diseases. Schuman has recently summarized the mortality associated with "smoking-related diseases," making apparent the importance to preventive medicine of the epidemiologic study of cigarette smoking behavior (5).

## Sample

Our research was carried out as part of the Western Collaborative Group Study (WCGS), a prospective investigation of coronary heart disease (CHD) conducted from 1960 to 1969 among employees of 11 corporations in California
(6). The present analysis covers the 2,318 men who entered the WCGS between the ages of 39 and 49 years. At intake a detailed medical history was taken, and interviews concerning past and current habits were conducted. In addition, an examination was conducted that included numerous laboratory determinations (6). The protocol of this study has been previously published ( $6 a$ ). From the data on smoking history, four categories of persons were defined:

1. Men who never smoked, called nonsmokers
2. Former smokers, including former pipe and cigar smokers
3. Light smokers, including men smoking less than 20 cigarettes daily, as well as pipe and cigar smokers
4. Heavy smokers, those smoking 20 or more cigarettes per day at the time of the report.

In the second portion of this paper we compare selected biological and psychological characteristics of persons who changed smoking habits (or maintained extreme habits) over a 4 -year period, 1961-65. The definitions of groups and methods of analysis will be presented subsequently.

## Methods

The serum cholesterol level was determined at the time of intake into the study (1960-61) by the method of Zak; lipoprotein analysis was done by the method of Straus and associates; the lipalbumin fraction was selected for specific consideration (6). Triglyceride determinations were made in the fasting state at the time of the first reexaminations (1962). Hematocrit determinations were performed in 1963. Blood pressure readings were obtąined, the coronary-prone behavior pattern was determined by interview, and data on the person's physical activity on the job, voluntary exercise, and various physical characteristics were gathered at the intake examinations in 1960-61.

## Results

Biological characteristics: The distribution of blood lipids for the four smoking categories are shown in table 1. Among the heavy cigarette smokers 16 percent fewer men had cholesterol
levels under 220 mg . per 100 ml . of blood serum and 9 percent more had levels in the " 260 mg . per 100 ml . and higher" bracket than did the men who had never smoked (nonsmokers). Light smokers and men who had quit smoking before intake were similar to each other in their cholesterol level distributions and intermediate between the heavy smoker and nonsmoker categories. The differences are highly significant. The correlation between serum cholesterol level and smoking habit found here is in agreement with the results of other investigators (4,7). Nevertheless, in other studies, such as the detailed Tecumseh study (8), no association was found between smoking status and serum cholesterol level.

The beta-alpha lipoprotein ratio was not found to have this relation with smoking, but the lipalbumin fraction of the lipoprotein determination showed a strong association. Lipalbumin, a conveyor of fatty acid, is a coronary heart disease risk factor whenever its values are low. The heavy cigarette smokers are distinct from the other three groups in their distribution of lipalbumin ; their distribution is in the direction condu-
cive to high CHD risk. Light smokers, persons who had quit smoking, and nonsmokers had lipalbumin distributions implying progressively lower CHD risk, in that order, but these groups were similar to each other in lipalbumin distribution.

The serum triglycerides after fasting also showed a significant and stepwise increase of high concentrations ( $\geqslant 177 \mathrm{mg}$. per 100 ml . of blood serum), progressing from nonsmokers (19 percent), former smokers ( 22 percent), light smokers ( 23 percent), to heavy smokers ( 28 percent). The two groups not currently smoking had a onethird greater frequency of men with concentrations of triglycerides under 100 mg . per 100 ml . Triglycerides, however, relate very much to previous dietary intake, including alcohol, and the Tecumseh study (2) has shown that smokers are likely to drink more alcohol. The higher concentration of triglycerides reported here among smokers thus could possibly be associated with higher alcohol intake rather than with smoking. Unfortunately, no data on alcohol were collected concurrent with these triglyceride determinations.

In brief, men aged 39-49 years who smoked a

Table 1. Percentage of men 39-49 years in each smoking group with "high" serum lipids, Western Collaborative Group Study
$\left.\begin{array}{ccccc}\hline \text { Variable } & \begin{array}{c}\text { Group 1 } \\ \text { Never } \\ \text { smoked } \\ (\mathrm{N}=546)\end{array} & \begin{array}{c}\text { Group 2 } \\ \text { Quit smoking } \\ \text { before intake } \\ (\mathrm{N}=359)\end{array} & \begin{array}{c}\text { Group 3 } \\ \text { Light } \\ \text { smokers } \\ (\mathrm{N}=547)\end{array} & \begin{array}{c}\text { Group 4 } \\ \text { Heavy smokers- } \\ \text { 20 cigarettes } \\ \text { daily }\end{array} \\ (\mathrm{N}=866)\end{array}\right]$
${ }^{1}$ Expressed as mg . per 100 ml . of blood serum.
2 Expressed as percentage of total lipoprotein.

Table 2. Percentage of men 39-49 years in each smoking group at each level of selected biological variables, Western Collaborative Group Study

| Variable | Group 1 <br> Never smoked $(\mathrm{N}=546)$ | $\begin{aligned} & \text { Group } 2 \\ & \text { Quit } \\ & \text { smoking } \\ & \text { before } \\ & \text { intake } \\ & (\mathrm{N}=359) \end{aligned}$ | Group 3 <br> Light smokers $(N=547)$ | Group 4 Heavy smokers $20+$ cigarettes daily $(\mathrm{N}=866)$ |
| :---: | :---: | :---: | :---: | :---: |
| Systolic blood pressure (1960-61): |  |  |  |  |
| $\leq 119$. | 28 | 26 | 26 | 25 |
| 120-149. | 65 | 66 | 65 | 67 |
| $\geq 150$. | 7 | 8 | 9 | 8 |
|  | $\mathrm{X}^{2}=3.20, \mathrm{df}=6, P<.90$. |  |  |  |
| Diastolic blood pressure (1960-61): |  |  |  |  |
|  | 90 | 92 | 91 | 94 |
| $\geq 95$. | 10 | 8 | 9 | 6 |
|  | $\mathrm{X}^{2}=6.82, \mathrm{df}=3, .05<P<.10$. |  |  |  |
| Hematocrit ${ }^{1}$ (1963): |  |  |  |  |
| $\leq 43$. | 16 | 13 | 13 | 7 |
| 44-50. | 81 | 81 | 78 | 79 |
| $\geq 51$. | 3 | 6 | 9 | 14 |
|  | $\mathrm{X}^{2}=62.69, \mathrm{df}=6, P<.001$. |  |  |  |

${ }^{1}$ Red cell volume-percent of red cells per blood column.
pack of cigarettes or more per day were most likely to have blood lipid patterns conducive to coronary heart disease. These differences are all significant at the $P=.001$ level, but no time sequence or causal relation can be established from this particular set of tables.

Analyses of other biological characteristics are shown in table 2. Systolic blood pressure readings were similar in all smoking groups, but heavy smokers tended less often to have an elevated diastolic blood pressure. This inverse relationship has been documented consistently in U.S. and European studies (4,7-9). Reid and associates (9) and others further observed that the association between blood pressure levels and smoking status disappeared when weight was controlled. Although the association of diastolic blood pressure and smoking groups was only suggestive in our results, it too was further diminished when weight was controlled by means of the minimum modified chi-square procedure (10). The hematocrit, however, was much higher in smokers; nearly five times as many heavy smokers as "never smokers" had hematocrits greater than 50 percent. This correlation of elevated hematocrit with cigarette usage has also been observed elsewhere, for example, in unpublished data on the Georgia Food Store Workers group, studied by Dr. C. D. Jenkins and Dr. C. G. Hames.

The four smoking groups differed substantially in their reported history of weight gain since age 25 , as shown in table 3 . Here 39 percent of the former smokers reported having gained 20 or more pounds since age 25 , whereas only 29 percent of the heavy smokers gained that much weight. The nonsmokers and the light smokers are intermediate. Eighteen percent of the heavy smokers actually reported a net loss of weight in the 14 to 24 years since they were 25 ; more than one-third of these losses were 10 pounds or more.

The implications of this kind of weight loss for general health have not been studied as thoroughly as have the implications of weight gain. One is led to believe that these differences in weight accumulation occurred a long time before initiation of the study, specifically when the men were between the ages of 25 and 35 , inasmuch as the determined variable "weight gained since age 35 " did not show these significant differences. Other weight-based variables, such as the currently measured percent relative body weight (ratio of observed weight to ideal weight derived from life insurance tables), percent fat (calculated by established formulas from skinfold and bone measurements), and the ponderal index, did not differ significantly by smoking category.

Current behavior. Two variables dealing with physical activity relate to smoking, but in oppo-
site directions. Voluntary physical exercise, including all sports, jogging, and the taking of walks, was not regularly practiced by most of this population. Heavy smokers were obvious in their avoidance of exercise. The other three groups were similar to each other. The negative effects of heavy smoking on a person's physical condition may make regular exercise more difficult to maintain. Smokers (heavy and light), however, reported more physical exertion on the job than did former smokers and nonsmokers. Although only about 6 percent more smokers than nonsmokers were in the moderate activity category; the difference was statistically significant. On-the-job exertion is closely correlated with occupation, a variable discussed under Social Characteristics.

Recent reports from the Western Collaborative Group Study have shown a strong relationship between coronary heart disease and the coronaryprone type A behavior pattern (11). In brief, the type A behavior pattern is characterized by enhanced competitiveness, aggressiveness, and feelings of being under the pressure of time and the challenge of responsibility (6). Heavy smokers are type A significantly more often ( 53 percent) than men of any other smoking category. Men
who have never smoked are least likely to be type A (table 3). Nevertheless, previous work has shown that these differences in rates of coronary, heart disease between men in the Western Collaborative Group Study who are of type A and men of the converse type, B, cannot be accounted for simply by differences in their smoking habits. Similarly, the association of coronary heart disease with cigarette smoking is not attributable to an excess of "coronary-prone" men in this category (12).

Social characteristics. Men of different educational backgrounds developed sharply different smoking histories by middle age (table 4). Whereas the majority of men in this total study population had gone to college, a larger percentage of the heavy smokers had not. The nonsmoker group had the highest proportion of college men. This observation agrees with the reports of Hinkle and associates regarding Bell Telephone System employees (13). Income was also related to smoking habits; the two groups in our study not currently smoking earned the most money. Heavy smokers had the lowest percentage of men in the annual income bracket of more than $\$ 15,000$. The association of cigarette smok-

Table 3. Percentage of men 39-49 years in each smoking group with selected personal characteristics, Western Collaborative Group Study

| Variable | Group 1 <br> Never smoked $(\mathrm{N}=546)$ | Group 2 Quit smoking before intake ( $\mathrm{N}=359$ ) | Group 3 <br> Light smokers $(\mathrm{N}=547)$ | Group 4 Heavy smokers$20+$ cigarettes daily $(\mathrm{N}=866)$ |
| :---: | :---: | :---: | :---: | :---: |
| Weight change since age 25 : |  |  |  |  |
| Lost weight. | 11 | 11 | 15 | 18 |
| Gained $\leq 19$ lbs . | 56 | 50 | 52 | 53 |
| Gained $\geq 20 \mathrm{lbs}$. | 33 | 39 | 33 | 29 |
|  | $\mathrm{X}^{2}=24.83, \mathrm{df}=6, P<.001$. |  |  |  |
| Voluntary exercise: |  |  |  |  |
| None. | 10 | 13 | 10 | 16 |
| Occasional. | 62 | 59 | 62 | 62 |
| Regular. | 28 | 28 | 28 | 22. |
|  | $\mathrm{X}^{2}=21.37, \mathrm{df}=6, P<.01$. |  |  |  |
| Physical activity on the job: |  |  |  |  |
| None to light. | 88 | 89 | 84 | 82 |
| Medium. | 12 | 11 | 16 | 18 |
|  | $\mathrm{X}^{2}=12.41, \mathrm{df}=3, P<.01$. |  |  |  |
| Coronary-prone behavior pattern: ${ }^{1}$ |  |  |  |  |
| Type A. . . . . . . . . . . . . . . . . | 41 | 45 | 47 | 53 |
| Type B...................... | 59 | 55 | 53 | 47 |
|  | $\mathrm{X}^{2}=20.10, \mathrm{df}=3, P<.001$. |  |  |  |

[^1]Table 4. Percentage of men 39-49 years with selected social characteristics at intake into Western Collaborative Group Study

| Variable | Group 1 <br> Never smoked $(\mathrm{N}=546)$ | Group 2 Quit smoking before intake ( $\mathrm{N}=359$ ) | Group 3 <br> Light smokers $(\mathrm{N}=547)$ | Group 4 Heavy smokers$20+$ cigarettes daily $(\mathrm{N}=866)$ |
| :---: | :---: | :---: | :---: | :---: |
| Education |  |  |  |  |
| High school or less. | 35 | 43 | 42 | 52 |
| College or more.... | 65 | 57 | 58 | 48 |
|  | $\mathrm{X}^{2}=40.00, \mathrm{df}=3, \mathrm{P}<.001$. |  |  |  |
| Annual income |  |  |  |  |
| <\$10,000. | 41 | 42 | 46 | 48 |
| \$10,000-\$15,000. | 43 | 41 | 39 | 40 |
| > \$15,000. | 16 | 17 | 15 | 12 |
|  | $\mathrm{X}^{2}=13.22, \mathrm{df}=6, P<.05$. |  |  |  |
| Occupation |  |  |  |  |
| Professional and technical. | 44 | 40 | 43 | 35 |
| Managers and administrators. | 43 | 47 | 41 | 50 |
| All others. | 13 | 13 | 16 | 15 |
|  | $\mathrm{X}^{2}=19.69, \mathrm{df}=6, P<.01$. |  |  |  |
| Marital status |  |  |  |  |
| Single. | 3 | 4 | 3 | 3 |
| First marriage. | 92 | 89 | 86 | 87 |
| Divorced-remarried, widowed. | 5 | 7 | 11 | 10 |
| $\mathrm{X}^{2}=23.00, \mathrm{df}=6, P<.031$. |  |  |  |  |

ing with income is weaker than the other associations reported here, but it is nevertheless statistically significant ( $P=.05$ ).

In terms of type of occupation, heavy smokers were the least likely to be in professional and technical positions. In this study group, heavy smokers and former smokers were most likely to be listed as managers and officials. Men in clerical and sales positions, skilled workers, and men in all other occupations did not have different smoking distributions than the total study group. There were few service workers or laborers in this study population.

It is not known whether differences in educational background, stresses on the job, opportunities for tobacco use on the job, or some selective factors based on personality are responsible for the observed occupational differences in smoking. It should be emphasized that this study group is not a cross section of the employed U.S. population, since it is deficient in clerks, technicians, laborers, and other low to middle income groups.

The smoking groups also differed in marital status. Both heavy and light smokers had higher proportions of men who were divorced, widowed, or remarried (table 4). While these differences are small in magnitude, they are nevertheless.
highly significant statistically. This study population contained so few men who had never married that important differences among single men may have been overlooked.

The results presented have implications for future research as to the effects of cessation of cigarette smoking on the risk of coronary heart disease. Several studies, but certainly not all, have shown that persons who have stopped smoking have a lower risk of coronary heart disease, but in general these studies have not made concurrent comparisons of the other risk factors in either the groups who have stopped or who have continued smoking (14). The reason why cessation of smoking appears to reduce CHD risk in some groups and not in others can best be understood if the other differences in risk factors between the various smoking groups are monitored simultaneously with alterations in smoking habits and changes in CHD incidence.

The present report shows substantial differences between persons with different smoking histories in such factors as serum cholesterol, triglycerides, lipalbumin, hematocrit, weight gain since age 25 , voluntary exercise, physical activity on the job, presence of a coronary-prone behavior pattern, education, income, occupation, and mari-
tal status. The different smoking categories appear to represent somewhat different populations of persons, at least with regard to these factors.

Several limitations on the interpretation of these data should be pointed out. First, the observed strong gradients of difference between smoking groups appeared in men 39 to 49 years; the differences were weaker or even insignificant for many of the variables among men aged 50 59. This result is not surprising inasmuch as the sharply defined associations of risk variables with coronary heart disease found in young men are often blurred among older populations, and the comparisons herein have analogous properties. Second, the cross-sectional nature of these observations makes it impossible to determine whether smoking preceded or followed the observed biological differences. To help clarify this issue, groups that changed their smoking habits during the first 5 years of the longitudinal study were identified and compared with respect to selected biological and psychological characteristics.

Concomitants of change in smoking habits. What changes in smoking habits occurred between intake in 1960 or 1961 and the fifth followup examination in 1965? Among the 546 men 39-49 years of age who had never smoked before intake, only 3 took up cigarette usage to the degree of 10 cigarettes per day by 1965 , a rate of 6 per 1,000 . The corresponding rate of going back to smoking ( 10 or more cigarettes per day) among former cigarette smokers was 84 per 1,000. Thus, to the extent that these results can be generalized, health workers interested in smoking prevention campaigns for men over age 40 should concentrate their efforts on former smokers of cigarettes, in addition of course to getting current cigarette smokers to stop. Of men smoking cigarettes at intake, more than 68 percent were still smoking at least 10 per day in 1965. Nevertheless, between intake and 1965 more than four times as many men in the study stopped smoking cigarettes as took up the habit.

The next stage of the data analysis was designed to examine the time relationships between change in smoking habits and change in biological and psychological parameters. It should be kept in mind that the gathering of laboratory data was part of a longitudinal study of coronary heart disease so that the timing and frequency of repeated measures were not optimal for the purposes of our study. The changes in smoking habits oc-
curred at different times for different men across the 4 years examined here. The timing of these changes relative to the blood chemistry and blood pressure determinations may also have influenced the results reported.

Four smoking groups were defined on the basis of their habits of tobacco use from 1960 through 1965:

1. Men who had not smoked any form of tobacco (cigarette, pipe, or cigar) before intake and who remained nonsmokers through 1965the group called nonsmokers
2. Men who were smoking cigarettes at some time during the period 1960 to 1963 and whose total cigarette usage during 1964 and 1965 was zero-the group called "quit cigarettes"
3. Men who were not smoking cigarettes at intake (1960) but who were smoking cigarettes in 1964 or 1965-the group called "resumed cigarettes"
4. Men smoking at least 10 cigarettes per day both at intake and in 1965 and who averaged at least 20 cigarettes per day for the whole 5 -year period, called "heavy smokers."

This categorization is not exhaustive in that it omits smokers of only pipes or cigars and also omits persons who changed their habits according to other timetables or who smoked a small amount consistently. The four groups have mean ages all within 0.5 of a year-not significantly different by analysis of variance.

In table 5 scores on the Jenkins Activity Survey (JAS) (15) for the four groups are compared. This objective test to measure coronary-prone behavior patterns was taken by the entire WCGS population in 1965, that is, at the end of the period of observation reported here. Men who had resumed smoking cigarettes scored significantly more in the type A direction on the A-B scale than any other group. The other three groups were similar to one another and in the direction of type B , or easygoing behavior. Overall, these differences were statistically significant.

Table 3, however, identifies heavy smokers as being more type $A$ than were nonsmokers. The differences are influenced by the fact that table 3 presents results for type A men identified by the clinical interview, whereas in table 5 the type A determinations are based on scores on the 1965 JAS. In addition, it is possible that selective changes may have occurred in these smoking

Table 5. Means of the biological and psychological variables for groups changing their smoking habits, Western Collaborative Group Study

| Variable | Group 1 Never smoked $(\mathrm{N}=543)$ | Group 2 Resumed cigarettes after 1961 and before $\begin{gathered} 1964 \\ (\mathrm{~N}=44) \end{gathered}$ | Group 3 Quit cigarettes after 1961 and before 1964 $(\mathrm{~N}=200)$ | Group 4 Continuing heavy smokers $(\mathrm{N}=599)$ | F ratios |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Age in years. | 43.3 | 43.2 | 43.1 | 43.6 | 1.39 |
| Average daily cigarettes for 4 years (1962-65)..... . | . 0 | 11.0 | 13.8 | 36.5 |  |
| $1965 \text { JAS scales } 1$ <br> A-B scale (behavior type) | -1.1 | 3.9 | -1.1 | -. 1 | 23.90 |
| Speed and impatience factor. | -. 3 | 3.4 | -. 2 | . 2 | 1.87 |
| Hard-driving factor. . . . . . . | -1.5 | . 5 | -. 8 | . 1 | 2.52 |
| Job involvement factor | . 9 | 1.2 | -. 3 | . 9 | . 71 |
| Blood chemistry and pressure 1961 cholesterol (mg. per 100 mil.)...... . | 217.2 | 226.2 | 227.6 | 233.6 | 214.04 |
| 1963 cholesterol (mg. per 100 ml .).. | 219.7 | 235.2 | 231.7 | 237.0 | 214.45 |
| 1962 triglycerides (mg. per 100 ml .). | 135.4 | 133.0 | 154.9 | 155.9 | 26.02 |
| 1961 lipalbumin (percent of total lipoprotein)..... . | 21.1 | 19.2 | 19.5 | 19.4 | 26.82 |
| 1961 systolic blood pressure. . . . . . . | 126.5 | 124.3 | 127.3 | 128.4 | 2.49 |
| 1961 diastolic blood pressure . . . . . . . . . . . . . . . . | 82.1 | 80.7 | 80.2 | 80.6 | 3.37 |
| 1963 hematocrit (percent volume of red cells in blood column). | 46.3 | 47.4 | 46.8 | 47.8 | 222.67 |

[^2]groups from the time data were collected in 1961 (table 3) to the time JAS scores were obtained in 1965.

In addition to the scale measuring the type $\mathbf{A}$ coronary-prone behavior pattern, the Jenkins Activity Survey also is scored for three independent traits derived by factor analysis. The group that resumed smoking was distinctly higher on the factor score for speed and impatience, but this result was not statistically significant. Inasmuch as the "resumed smoking" group was not distinctive on the other two factor scores related to the coronary-prone pattern, the main components of the type A pattern that appear to relate to resumption of cigarette usage are the traits of speed and impatience.

The biological data were considered in terms of whether they supported the hypothesis that elevated levels of risk followed cigarette usage or the hypothesis that persons already high on these variables selected themselves into certain smoking groups ("the selection hypothesis"). If the pattern of means of biological variables was such that persons who never smoked (group 1) and those not smoking at intake who later resumed
smoking (group 2) were both low, whereas smokers at intake who subsequently quit (group 3) and continuing heavy smokers (group 4) were both high, the results could be construed to be consistent with the hypothesis that the smoking habit was prospectively associated with the level of the biological variable. On the other hand, if the means of the biological variable were such that groups 2 and 4 were high and groups 1 and 3 were low, the results would be consistent with the selection hypothesis, in that those who resumed smoking already had an elevated risk before their change of habit and those who were about to quit were already lower on the biological variable than those who continued smoking.

At least two additional possibilities exist. One is that smoking relates to serum lipids on a longterm basis so that former smokers (who comprise almost all those in this sample who resumed smoking) might have mean risk factor values still partially influenced by their prior history of smoking. Similarly, biological measures taken shortly after resumption of the habit may still retain some aftereffects from the nonsmoking period.

Another problem applies to group 3, those who quit cigarettes after intake and before 1964. It is not known whether their average cigarette use per day at intake was equivalent to that of the continuing heavy smokers. If, for example, a consumer of 10 cigarettes per day is more likely to quit completely than a consumer of 40 cigarettes per day, it would follow that groups 3 and 4 were not equivalent in cigarette consumption at intake and that the differences observed between them may relate to their current levels of cigarette consumption and not to some selective factor predisposing one group to quit smoking.

With these hypotheses in mind, let us consider the results reported in table 5. Significant differences existed among these four smoking groups in mean levels of three blood lipids. The cholesterol level at intake was highest in those who continued to smoke heavily, lowest in lifetime abstainers, and intermediate in the groups who quit or resumed cigarette usage. This pattern of results does not clearly support either of the two main hypotheses.

The selection hypothesis finds some support in that group 2 is higher than group 1 and group 3 is lower than group 4, but it is weakened by the fact that group 2 is not higher than group 3. Serum cholesterol was also measured in 1963, after the greater part of the observational period considered in table 5 had passed. The mean cholesterol levels for all four groups increased slightly, as would be expected from the general tendency in the United States for cholesterol to increase with age. The group that resumed smoking cigarettes after 1961 (group 2) showed the greatest increase in cholesterol levels, but the group that stopped smoking cigarettes (group 3) did not show the decrease that would be expected if in fact cholesterol levels were responsive to changes in cigarette usage.

Serum triglycerides drawn after fasting were highest among continuing heavy smokers and among the smokers at intake who later quit smoking. Triglycerides were lowest among those who had never smoked and among former smokers who resumed the habit after intake. This pattern of means is clearly consistent with the first hypothesis offered, namely, that triglyceride levels are associated with current smoking habits and not with the propensity to change these habits. These results, however, must be considered with caution, as pointed out earlier, because no ac-
count has been taken of dietary factors, particularly alcohol consumption.

A different picture was presented by the fraction of the beta-alpha lipoprotein spectrum identified as lipalbumin. Nonsmokers had the highest average values, an indication that they were at the least risk from this source. The other three groups were similar, the group that had resumed smoking having values suggestive of the highest coronary risk. The differences are highly significant statistically but small in absolute magnitude. The pattern of means does not support the hypothesis of selective movement of men with low lipalbumin values into higher risk groups. The pattern further implies that lipalbumin levels may not be sensitive to changes in smoking habits.

Systolic and diastolic blood pressures did not differ significantly among the four groups.

The differences in mean hematocrit levels were also highly statistically significant but small in absolute magnitude; the continuing heavy smokers and those who resumed smoking demonstrate the higher mean hematocrit levels. These means are not inconsistent with the selection hypothesis. The late date of the hematocrit determinations (1963), however, allows the possibility that some men in the group that resumed smoking cigarettes were already smoking at the time of this one determination.

This complex set of significant relationships suggests the need for careful study of profiles of lipid concentrations at various densities, both immediately before and promptly after sharp changes in smoking habits. Future research should also be designed to test the possibility that a prior common factor influences both lipid concentrations and smoking. The work of Seltzer raises the possibility of a constitutional factor functioning in this way (16).

These results also raise the question of how much of the decline in serum lipid levels observed in diet-heart studies may be due to changes in smoking habits and not to dietary changes per se. Most dietary studies have failed to consider cigarette smoking as a co-variable in explaining lipid changes.

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Data from medical examinations and interviews of 2,318 men 39 to 49 years, gathered in a prospective study of cardiovascular disease, were analyzed to determine whether men with different histories and different habits of tobacco use differed systematically in other characteristics as well. Compared with nonsmokers, heavy smokers of cigarettes showed higher levels of serum cholesterol, higher concentrations of triglycerides after fasting, and higher hematocrits. They reported less weight gain since age 25, less often engaged in voluntary exercise, and more often manifested a "coronaryprone behavior pattern." In this
group, heavy smokers tended to have less education and lower incomes and were more likely to be in managerial rather than in technical or professional occupations.
Longitudinal analysis showed that men who were nonsmokers at intake but who later resumed cigarette smoking had higher test scores on the coronary-prone behavior pattern, with an emphasis on the attributes of haste and impatience. Longitudinal analysis also revealed systematic serum lipid differences between groups before they changed their smoking habits; the overall pattern of these differences did not appear
to be the result of elevated lipid levels influencing smoking patterns. The data do not prove, but are consistent with either the hypothesis that a common prior factor increases both triglycerides and smoking or the hypothesis that smoking raises triglycerides.

The data for serum cholesterol and lipalbumin did not consistently support either of the major hypotheses offered. Further research is needed that will measure characteristics, such as the full lipid spectrum, the hematocrit, and the person's behavior type, both immediately before and promptly after radical changes in smoking habits.


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[^1]:    ${ }^{1}$ Based on interview conducted at intake, 1960-61.

[^2]:    ${ }^{1}$ Sample sizes for Jenkins Activity Survey (13) analyses were 497 for group 1, 40 for group 2, 181 for group 3, and 517 for group 4.
    ${ }_{2}$ Significant at 0.01 level.
    Note: Table is based on all men 39-49 years at intake who qualified for the groups as defined in the text. The period of observation was from intake into the study in 1960-61 through the 1965 annual reexamination.

