PLATELET COUNT, HEART RATE, AND BODY COMPOSITION IN SMOKERS AND NONSMOKERS

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Introduction

The increased heart rate, blood pressure, cardiac output, and the concomitantly increased circulation velocity associated with physical activity appears to produce an appreciable and consistent rise in platelet count (1, 21). Cigarette smoking produces similar changes. The heart rate is increased 15 to 20 beats per minute by the smoking of a single cigarette. This is accompanied by an associated rise in systemic blood pressure, cardiac output (31, 26, 22, 30) and a transient increase in platelet count (24). These changes may last 10 to 30 minutes for each cigarette (11). It would appear, therefore, that chronic smoking may produce a chronic increase in platelet count. This becomes a matter of serious concern when one considers Wright's contention that young platelets are more adhesive than older platelets (33). Since subjects susceptible to thromboembolic disease have, on the average, a higher platelet turnover, it may be that the increased proportion of young platelets is a factor in the susceptibility of these individuals to thromboembolic disease. Most pertinent to the present study, however, is the evidence suggesting that platelet turnover and platelet adhesiveness are increased by cigarette smoking and hence may promote the formation of an arterial clot (17, 19). Adhesive platelets accomplish this by aggregating more readily and adhering to wettable surfaces on the vascular endothelium. Such contact results in platelet transformation and degranulation. This platelet disintegration promotes the release of ADP, lysosomal enzymes, and serotonin (34), all of which have been implicated in alteration of the physical properties or physiological reactivity of small vessels. Serotonin, liberated from disintegrated platelets when a vessel is torn, by its direct constrictor action on vascular smooth muscle, may pull the vessel walls together and assist in preventing further hemorrhage. When viscous metamorphosis is initiated by a wettable surface within an intact vessel, however, the vasoconstrictor properties of locally-liberated serotonin may cause spasm and reduce blood flow, especially in view of the fact that it has been incriminated as a potent precapillary constrictor. Consequently, when blood clots in a pulmonary, cerebral, or coronary vessel (either primarily or about an embolus) and additional clot forms on either side, serotonin so generated may produce additional, possibly dangerous, vascular obstruction (12). These considerations may have a significant bearing on the transient vascular accidents sometimes observed in

patients with vascular disease (27). Such observations have led some authorities to conclude that the reaction of the platelet to changes in certain vascular and hematological factors is the prime mechanism in thrombogenesis (30, 5).

The importance of platelet count *per se* in cardiovascular disease is indicated by several studies (18, 7, 33, 3, 23) which suggest that increased platelet counts frequently attend hypercoagulability and thrombosis. Some of the most significant findings with respect to pathological increases in the number of circulating platelets indicate that thrombocytosis facilitates the deposition of cholesterol on the rabbit aorta (9, 10). Increased counts may reflect greater platelet turnover and, as noted previously, an increased level of adhesiveness.

Several investigators have dealt with the relationship of morphological constitution and smoking. Included are studies of smokers and nonsmokers with regard to height, weight, overweight and underweight calculated from standard tables, ponderal index, skinfolds, and level of activity. One of the questions examined by these studies was whether smokers and nonsmokers may be considered as a single population. That is, are smokers and nonsmokers constitutionally different? Thomas concluded as the result of her observations of 297 nonsmokers and 321 smokers that differences observed in parental histories indicate that smokers and nonsmokers appear to have a somewhat different heritage and that at least some of the variations found in individual traits may be genetic in origin (31). Thomas also found that those subjects who were 40 percent or more overweight and exhibited unusually heavy body builds as reflected in ponderal indices were all regular smokers and the greatest percentage of those 10 percent or more underweight were nonsmokers. This is at variance with the findings of Damon (16). He concluded, as the result of his correlation of various measures of body type including subscapular skinfold, weight, and ponderal index that lean men smoke more than fat, but not muscular, men. Seltzer (28) obtained 12 measurements of 688 smokers and 234 nonsmokers from which he computed 10 body ratios. The smokers exhibited larger mean dimensions than the nonsmokers in every instance. Statistically significant superiority was exhibited by smokers in height, weight, and in the dimensions of the head, face, shoulders, chest, hip, leg, and hand. This conflicting information and the lack of

information available regarding the effect of smoking on platelet count prompted the present study. Its purpose was to examine differences between smokers and nonsmokers in the following variables: height, weight, bodyfat calculated from skinfolds, ponderal index, Harvard step test, and platelet count.

Procedure

Twenty-four age-matched students, 12 smokers and 12 nonsmokers, were selected for the study. They were instructed to report in the post-absorptive state to the University of Toledo's Physiology of Exercise Research Laboratory where physical fitness, platelet count, and bodyfat were determined. Smokers were requested to abstain from smoking prior to their testing period on their test day. Subjects rested supinely for 5 minutes at the beginning of individual testing periods. They were then instructed to sit up for 30 seconds and then to stand, at which time a fingertip blood sample was obtained and the platelet count determined. Skinfolds were obtained by the method of Pascale (25) and converted to percent bodyfat with the Siri formula (29). Testing was concluded with the Harvard step test in a controlled environment room.

The fingertip blood sample was obtained and analyzed as described in the previous paper Marley and Linnerud 1973.

Results and Discussion

The ANOVA method employed for this study assessed between group differences with non-orthogonal linear contrasts between pairs of treatment combinations. No statistically significant differences were observed between smokers and nonsmokers (Table I). Analysis of covariance confirmed these findings. Some of the data, however, and factors which may have attenuated them and obscured their meaning require discussion.

TABLE I

GROUP PROFILES (mean $\pm$ S.D.)

	Nonsmokers (N=12)	Smokers (N=12)
Platelet count	178,950 $\pm$ 22,750	185,850 $\pm$ 30,400
Harvard step	74.17 $\pm$ 26.95	65.00 $\pm$ 21.74
Bodyfat	10.90 $\pm$ 5.15	11.38 $\pm$ 6.34
Ponderal index	12.84 $\pm$ .57	13.15 $\pm$ .74
Cigarettes/day	0.0	17.00 $\pm$ 6.09
Months smoked	0.0	37.67 $\pm$ 26.64
Height	68.48 $\pm$ 2.38	69.52 $\pm$ 2.50
Weight	153.13 $\pm$ 22.16	149.69 $\pm$ 23.29
Age (months)	230.00 $\pm$ 31.47	231.50 $\pm$ 33.60

Nonsmokers exhibited a lower mean platelet count, higher mean Harvard step test score, lower mean bodyfat, and a mean ponderal index which indicates a higher degree of mesomorphy than that of the smokers. The 12 smokers were not all of the same smoking classification, with the lowest individual consumption being 10 and the highest 30 cigarettes per day. Seven individuals were moderate smokers (10-19 cigarettes per day) and five were classified as heavy smokers (20-39 cigarettes per day). None were categorized as very heavy smokers (40 or more cigarettes per day). The length of time each of the smokers had smoked also varied. The shortest time as a smoker was 8 months, the longest 96 months.

This lack of homogeneity in smoking subjects may account for the lack of significance between group platelet counts. For instance, one smoker who averaged 10 cigarettes per day for 30 months exhibited the lowest platelet count (117,500) of all 24 subjects, far below the mean (185,850) for smokers in this study. Despite the care taken in counting platelets for this study, the disparity of this subject's cigarette consumption, 10 compared with a mean group consumption of 17, and his platelet count dramatizes a weakness of small sample research. It should be noted, nevertheless, that a subsequent investigation by Hawkins (20) appears to have substantiated the findings of the present study. She observed no significant difference between the platelet counts of nonsmokers, light smokers, and heavy smokers. She also employed a small sample, three groups of 10 subjects, but it was not possible to examine individual platelet counts as they were not listed.

The interpretation of heart rate response to submaximal work tasks presents some difficulty. Pulse rate can, however, be an especially good index under conditions in which the physical work is heavy and of relatively short duration (13). That is, heart rates ranging from 120 to 140 (6) maintained for three to five minutes would be required to demonstrate differences in the post-exercise heart rates of smokers and nonsmokers. The Harvard step test satisfied these criteria for the present study. Post-exercise heart rates for both smokers and nonsmokers averaged approximately 140 one minute after the completion of stepping. This would appear to add credence to the lack of significance observed between the Harvard step test scores of the two groups. The difference between the mean post-exercise heart rates taken in conjunction with the Harvard test for smokers, 142, and nonsmokers, 138, was also not significant. This finding is especially curious in view of the results presented by Cunningham and colleagues (14) in which male smokers had significantly higher ($P < 0.05$) heart rates one minute following a submaximal step test than nonsmokers. Female smokers in the same study exhibited significantly higher ($P < 0.05$) heart rates both at rest and following exercise. This is consonant

with the data of Chevalier *et al.* (8) and Blackburn *et al.* (2) in which pulse rates averaged about five beats per minute faster for smokers than for nonsmokers at the same intensity of exercise. This latter figure approximates the mean difference for post-exercise heart rates of smokers and nonsmokers in the present study.

The lack of significance between ponderal indices for smokers and nonsmokers is spurious in somewhat the same manner as post-exercise heart rate. Cureton (15), in

a study of 585 college freshmen, found the average ponderal index score to be 12.80 for subjects presenting mesomorphic somatotypes and 13.03 for ectomorphs. The cause of greater or lesser ponderosity can be determined by further examination of body musculature, density, and fat. The close similarity of the group bodyfat means in the present study would seem, therefore, to indicate that the nonsmokers are primarily mesomorphs with the smokers tending to ectomorphy.

TABLE II
CORRELATION COEFFICIENTS FOR SMOKERS

	Harvard stepscore	Bodyfat	Ponderal Index	Cigarettes per day	Months smoked	Height	Weight
Platelet count	-.344	.454	-.170	.106	-.337	-.172	.129

No significant correlations were found for smoker variables (Table II). Two significant relationships, platelet count and ponderal index and platelet count and height, were obtained between nonsmoker variables

(Table III). These merely support the known relationship between formed elements and body dimensions.

TABLE III
CORRELATION COEFFICIENTS FOR NONSMOKERS

	Harvard stepscore	Bodyfat	Ponderal Index	Height	Weight
Platelet count	.197	-.430	.594*	.655*	-.088

*Significant at the .05 level.

These analyses permit the following conclusions:

1. There is no difference between the chronic platelet counts of smokers and nonsmokers.

2. Post-exercise heart rates for smokers are higher than for nonsmokers.
3. There is no difference between the amount of bodyfat in smokers and nonsmokers.

4. Nonsmokers are predominantly mesomorphs with smokers tending to ectomorphy.

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OBITUARY

Mr. T. A. Mason

Every society or association depends for its running not only upon the work of its officers and committees, but upon a section of its Ordinary Members, who attend meetings, take part in discussions, and stimulate the committees to action. The B.A.S.M., will feel the sad death of Tom Mason as a severe loss of one of its most faithful and enthusiastic members. He joined our Association in 1960, attended most of the meetings since then, even those outside London after his retirement from work and living on a small income. He was a weight-lifting coach, and brought a great deal of expert knowledge of the subject to the B.A.S.M., and his regard for the possible hazards of this activity to the adolescent was a decided factor leading to our Symposium in 1970 on the risks of weight training and lifting upon the young (*Brit.Journ.Sports Med.* Vol. V. No 1). That the dangers of spinal hyper-extension during presses is now accepted in medical circles as hazardous is probably the best memorial we can give Tom Mason.

H. E. Robson