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 hematlogical 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 ± S.D.)**

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

<table>
<thead>
<tr>
<th>Harvard stepscore</th>
<th>Bodyfat</th>
<th>Ponderal Index</th>
<th>Cigarettes per day</th>
<th>Months smoked</th>
<th>Height</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet count</td>
<td>-.344</td>
<td>.454</td>
<td>-.170</td>
<td>.106</td>
<td>-.337</td>
<td>-.172</td>
</tr>
</tbody>
</table>

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**

<table>
<thead>
<tr>
<th>Harvard stepscore</th>
<th>Bodyfat</th>
<th>Ponderal Index</th>
<th>Height</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet count</td>
<td>.197</td>
<td>-.430</td>
<td>.594*</td>
<td>.655*</td>
</tr>
</tbody>
</table>

*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.

**REFERENCES**


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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
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