A STUDY OF PLATELET COUNT, BODYFAT, AND HARVARD STEP TEST SCORE

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ABSTRACT

The relationship between platelet count, bodyfat, and Harvard step test score was examined in 15 post-absorptive male subjects. Subjects rested for five minutes at the beginning of the testing period. A fingertip blood sample was then obtained and the platelet count determined by the method of Brecher and Cronkite. One-percent ammonium oxalate was employed as the dilution fluid. Bodyfat was calculated with the Siri formula on the basis of skinfolds obtained by the method of Pascale. Testing was concluded with the Harvard step test in a controlled environment room. Pearson product-moment correlation calculations yielded no significant relationship (P = > .05) between platelet count and bodyfat or platelet count and Harvard step test score. Subjects were equated by age matching into lean (under 9.9%), average (10-14.9%), and overweight (above 15%) bodyfat groups. An analysis of variance (ANOVA) revealed no significant difference (P > .05) between the platelet counts of these groups.

Introduction

One authority (61) has estimated that approximately 70 million American adults suffer from overweight and the increased morbidity and mortality rates associated with excessive weight underscore the significance of this major health problem. Indeed, some findings indicate that death rates among obese men average 50% higher than normal (3). More specifically, obese individuals incur a greater risk of developing hypertension, atherosclerosis, coronary heart disease (CHD), diabetes mellitus, gall bladder disease, degenerative arthritis, and kidney disease (33, 39, 19, 52). Further, the obese hypertensive experiences a greater risk of CHD than the non-obese hypertensive. Marked obesity has also been linked with another complication, reactive polycythaemia, and the concomitantly increased haematocrit and blood viscosity may lead to development of thromboembolic structures (56, 30).

Some investigators have indicated that the CHD risk attributed to overweight is accounted for primarily by its relationship to hypertension (57). Other research implicates the relatively high level of serum lipids associated with overweight (22, 29, 14, 11). In this respect, hyperlipaemia appears to accelerate blood clotting (10, 41) by increasing blood viscosity, platelet aggregation and adhesiveness, and red blood cell aggregation (59, 20).

Physical activity, by contributing to weight control, may indirectly reduce the risk of circulatory disease. This contention is reinforced by the work of Ahrens, which indicates that a decrease in weight is accompanied by a decrease in serum lipids (1). Physical activity might also have a direct stabilizing influence on lipid "risk" factors of consequence to CHD (25). For instance, vigorous cardiorespiratory activity appears to reduce serum lipids if it is sufficiently regular (15, 46, 12, 4, 31, 40, 54). This apparent effect of exercise on blood lipids and the increased body density associated with training has resulted in attention being directed to the relationship of coagulation factors, bodyfat, and somatotype. Burt et al. (9) found no significant relationship between bodyfat and blood coagulation time. Goldrick (27), however, demonstrated that fibrinolysis was inversely related to bodybuild. Fibrinolytic activity in Australian males decreased with increasing bodyfat, while in New Guinea natives it was associated with increasing muscularity. This inverse relationship between fibrinolysis and obesity was confirmed both in European males (23, 51) and females (47, 5) and observed to be more profound than significant changes in various disease states (43, 45, 24). This same phenomenon has been recorded in Asians but not in lean Africans with a narrow range of skinfolds (50).

The aetiology of the fibrinolytic defect in obese subjects is unknown. While it is due to neither known inhibitors of the fibrinolytic system nor standard lipid or carbohydrate parameters, it may be related to genetic-metabolic aspects of obesity or lack of exercise in the obese. Some investigators have observed that weight loss by dieting is accompanied by a return of fibrinolysis to normal levels. This led them to conclude that obesity per se may induce the fibrinolytic mechanism defect (28). Others have presented evidence of a relationship between free fatty acid (FFA) and fibrinogen synthesis which appears to provide a metabolic link between hyperlipaemia and coagulation in the genesis of arteriosclerosis (49).
The platelet plays a major role in a number of physiological and pathological processes in the blood of mammals (34, 35). Of particular interest is its assumed role in the generation of thromboembolic disease (32). The interaction of platelets and lipid factors may play a significant role in atherogenesis, especially in view of the following: lipids appear to be unmasked within degenerating platelets (2), chylomicra have been observed in contact with platelet aggregates (6, 26), and the known potential of fatty acids, released concomitant to trauma, for aggregating platelets (58). In addition, platelet disintegration promotes the release of ADP, lyso-somal enzymes, and serotonin (62), 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 wett able surface within an intact vessel, however, the vasconstrictor 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 (18). Reid has commented on the significance of this in peripheral vessels (18). Such observations have led some authorities (55) to conclude that the prime mechanism in thrombogenesis is the reaction of the platelet to changes in certain vascular and hematological factors.

The importance of platelet count per se in cardiovascular disease is indicated by several studies (21, 13, 60, 7, 42) 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 are those of Cohen and McCombs (16, 17) which indicate that thrombocytosis facilitates the deposition of cholesterol on the rabbit aorta.

The senior author has reviewed the acute and chronic effects of exercise on platelet count (36) and, in a pilot study (unpublished data) of platelet count, bodyfat, and maximal oxygen consumption, found no relationship between these variables. The purpose of the present study, then, was to reexamine the relationship of platelet count, bodyfat, and physical fitness.

Procedure

Twenty-nine male students volunteered for the study. Fifteen age-matched individuals were selected and 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. 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 (48) and converted to percent bodyfat with the Siri formula (53). Testing was concluded with the Harvard step test in a controlled environment room.

The fingertip blood sample was obtained and analyzed in the following manner. A lancet was utilized and the first drop of blood wiped away. The blood sample was then drawn into an oval bead pipette and immediately mixed with a 1% ammonium oxalate solution. The pipette was then inserted in a pipette shaker for 5 minutes, after which the first 4 drops were expelled and both cells of a counting chamber filled in the usual manner. The loaded chamber was then allowed to stand for 15 minutes in a moistened Petri dish. Platelet counts were made with the 43X objective of a phase microscope according to the method of Brecher and Cronkite (8). Two platelet counts were made for each of the original 29 subjects. A Pearson product-moment correlation coefficient was then calculated between these split-sample counts for the purpose of examining reliability. A coefficient of 0.901 was obtained and paired t analysis revealed no difference between the split-sample means (P > 0.05).

### TABLE I

<table>
<thead>
<tr>
<th>SAMPLE VARIABLES (MEAN ± S. D.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet count</td>
</tr>
<tr>
<td>Bodyfat</td>
</tr>
<tr>
<td>Harvard step</td>
</tr>
<tr>
<td>Height</td>
</tr>
<tr>
<td>Weight</td>
</tr>
<tr>
<td>Age</td>
</tr>
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</table>

Results

The means and standard deviations for the variables observed in this study are presented in Table I. Pearson product-moment correlations were calculated between platelet count and bodyfat (-0.095) and then platelet count and step test score (-0.112). They were not significant (P > 0.05).

The subjects were then divided into lean (9.9% and under), average (10-14.9%), and overweight (above 15%) bodyfat groups on the basis of their skinfold measurements. The criteria for these groups were based on a search of the literature. Table II presents the platelet count, bodyfat, and Harvard step test means of
these three groups. Table III is an analysis of variance (ANOVA) for the group platelet counts. The obtained value is not significant (P > 0.05).

The present findings support the conclusion of an earlier study by the senior author (unpublished data) that there is no relationship between platelet count and body fat. This is in accordance with Burt et al. (9) and their demonstration of a lack of relationship between body fat and coagulation time, but at variance with the data of Goldrick (27) and others (23, 51, 47, 5).

Previous studies have shown significant (37), nonsignificant (38), and curvilinear (44) relationships between platelet count and fitness. The present data give further indication that there is no relationship between physical fitness and platelet count.

Acknowledgements

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TABLE II

<table>
<thead>
<tr>
<th>GROUP</th>
<th>N</th>
<th>BODYFAT (MEAN ± S.D.)</th>
<th>PLATELET COUNT (MEAN ± S.D.)</th>
<th>HARVARD STEP TEST SCORE (MEAN ± S.D.)</th>
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<tbody>
<tr>
<td>Lean</td>
<td>5</td>
<td>8.62 ± .52</td>
<td>175,500 ± 16,250</td>
<td>88.00 ± 25.15</td>
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<tr>
<td>Average</td>
<td>5</td>
<td>12.22 ± 1.36</td>
<td>176,750 ± 15,450</td>
<td>75.00 ± 21.51</td>
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<tr>
<td>Overweight</td>
<td>5</td>
<td>21.16 ± 5.89</td>
<td>174,000 ± 34,850</td>
<td>54.00 ± 27.48</td>
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TABLE III

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<td>Between</td>
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<td>.317</td>
<td>.014</td>
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<tr>
<td>Within</td>
<td>12</td>
<td>274.800</td>
<td>22.900</td>
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<tr>
<td>Total</td>
<td>14</td>
<td>275.433</td>
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<td></td>
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