Male pattern baldness is not associated with established cardiovascular risk factors in the general population

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ABSTRACT

A number of studies have shown an association between male pattern baldness (MPB) and cardiovascular disease. Few of these studies, however, have examined whether MPB is a novel risk factor, or is associated with abnormalities of established coronary risk factors. We have therefore performed an analysis of MPB and cardiovascular risk factors in the general population. A total of 1219 male participants aged 18–70 years from the Victorian Family Heart Study were surveyed using a validated questionnaire for degree and pattern of baldness. Carefully standardized measures of height, weight, blood pressure, pulse rate, total and high-density lipoprotein cholesterol, and plasma fibrinogen were made. Subjects were grouped according to the degree and pattern of baldness as: no baldness, frontal baldness and vertex baldness. Bald men were older than non-bald men (P < 0.0001). Age was also associated with increased levels of coronary risk factors (P < 0.0001). When multiple regression was used to adjust for age differences, the levels of coronary risk factors were not significantly different between the bald and non-bald groups. The lack of association between baldness and established coronary risk factors implies that baldness may predispose to coronary heart disease through novel mechanisms yet to be defined.

INTRODUCTION

In a recent examination of male pattern baldness (MPB) and coronary heart disease, Lotufo et al. [1] showed an association between severity of baldness and coronary heart disease. This report supported a number of previous studies which had indicated that MPB may be a risk factor for cardiovascular disease. A case-control study by Lesko et al. [2] suggested that baldness on the vertex of the scalp was associated with myocardial infarction, and the Framingham Heart Study [3] showed an association between progression of hair loss and coronary disease in men aged under 55 years. The Copenhagen City Heart Study [4] showed an association between frontal baldness and myocardial infarction, and the First National Health and Nutrition Examination Survey (NHANES I) [5] uncovered a link between severe baldness and coronary disease mortality.

Although these studies suggest an association between cardiovascular disease and MPB, the explanation remains unknown. It seems logical that androgens may be involved, given the androgen-dependent nature of MPB [6] and the increased risk of cardiovascular disease in males when compared with females [7]. It remains to be defined whether MPB modifies known risk factors or operates independently through some novel mechanism to influence cardiovascular risk.

Key words: blood pressure, body mass index, cholesterol, hair loss, height, weight.
Abbreviations: HDL, high-density lipoprotein; MPB, male pattern baldness.
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Few studies have examined individual risk factors in relation to MPB in detail. A study by Trevisan et al. [8] suggested a link between MPB and both cholesterol levels and diastolic blood pressure. The Physicians’ Health Study [1] also demonstrated that increased risk of cardiovascular disease in balding men was further augmented in those who also had high blood pressure or high serum cholesterol.

In the present study, we further investigate the association between MPB and established cardiovascular risk factors in a general population sample of 1219 male participants of the Victorian Family Heart Study.

METHODS

Recruitment and data collection
The Victorian Family Heart Study is a population-based study of cardiovascular risk. A total of 2959 healthy Caucasians were recruited between 1991 and 1996. This group contained 783 families, consisting of two parents, aged 40–70 years, and at least one natural offspring aged 18–30 years [9].

Cardiovascular risk profiles were recorded at the time of recruitment. In specially arranged clinics, trained research nurses measured coronary risk factors, including height, weight, blood pressure, pulse rate, cholesterol (total and high-density lipoprotein (HDL)) and plasma fibrinogen. Height was recorded, with shoes removed, to the nearest half centimetre. Body weight was measured in street clothes with shoes removed. Body mass index was calculated from height and weight (kg/m²). The average of the second and third lying and standing systolic and diastolic blood pressures were recorded. Lying and standing pulse rates were counted for 1 min. Blood was collected for assessment of serum total cholesterol and HDL cholesterol, and for fibrinogen assay by the ‘clot opacity’ method [10]. All current medication was recorded, including the use of treatments for hypertension and hyperlipidaemia.

Degree of baldness was assessed in male participants of the Victorian Family Heart Study by way of a self-assessment questionnaire. This questionnaire asked participants to indicate the degree of baldness, if any, using the Hamilton Baldness Scale, as modified by Norwood [11]. In this classification, hair loss was graded progressively from Type I (no loss) to Type VII (hair loss is complete at the crown). Both frontal recession and vertex loss patterns were described. To validate this approach, a research nurse independently assessed a subgroup of participants who had already completed questionnaires. There was strong agreement between the assessment of the participant and the research nurse [12]. For the purpose of analyses, the baldness groups were further simplified into none (Type I), frontal-only (Types II and III) and vertex (Types III vertex, IV, V, VI and VII).

A total of 1219 out of 1461 male participants completed and returned the questionnaire.

Statistical analysis
All statistical analyses were carried out using the SPSS (Macintosh version 6.1) statistical software program. Associations between baldness groups and risk factors were explored by the use of ANOVA procedures that adjusted for the effects of age. To allow for multiple comparisons, \( P < 0.01 \) was accepted as evidence of statistical significance. In addition, degree of baldness, as determined by the Hamilton scale, was used as a ranking score in non-parametric Spearman correlations with age-adjusted risk phenotypes.

RESULTS

The prevalence and distribution of baldness is shown in Table 1. Overall, 42.7% of participants reported no baldness, 25.4% reported frontal-only baldness and 31.9% reported vertex baldness. These frequencies are similar to those reported by other groups [1,5,8].

Age and cardiovascular risk factor profiles in each of the baldness groups are summarized in Table 2. The average age of men with no baldness was 32 years, with frontal-only baldness 42 years and with vertex baldness 54 years of age. With the exception of pulse rate and HDL cholesterol, all measured coronary risk factors were significantly higher \( (P < 0.0001) \) in bald compared with non-bald men (Table 2). However, these variables also showed significant association with age per se. When the risk phenotypes were adjusted for differences in age (Table 2), none of the cardiovascular phenotypes was significantly different between the three baldness groups.

Additionally, correlation of degree of baldness, using the Hamilton Baldness Scale [11] as a ranking score, with risk factor phenotypes adjusted for age revealed no significant associations.

<table>
<thead>
<tr>
<th>Baldness group</th>
<th>Baldness type</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>I</td>
<td>520 (42.7)</td>
</tr>
<tr>
<td>Frontal-only</td>
<td>II</td>
<td>234 (19.2)</td>
</tr>
<tr>
<td></td>
<td>III</td>
<td>76 (6.2)</td>
</tr>
<tr>
<td>Vertex</td>
<td>III vertex</td>
<td>115 (9.4)</td>
</tr>
<tr>
<td></td>
<td>IV</td>
<td>101 (8.3)</td>
</tr>
<tr>
<td></td>
<td>V</td>
<td>69 (5.7)</td>
</tr>
<tr>
<td></td>
<td>VI</td>
<td>64 (5.3)</td>
</tr>
<tr>
<td></td>
<td>VII</td>
<td>40 (3.3)</td>
</tr>
</tbody>
</table>
DISCUSSION

That male pattern baldness may be a risk factor for cardiovascular disease was first suggested in 1972, when Cotton et al. [13] demonstrated an association between the occurrence of cardiovascular disease and hair loss. Several subsequent studies appeared to support the early findings. However, there were discrepancies regarding the nature of this relationship. Some indicated that the rate of progression, rather than the presence or degree of baldness, was important [3]. Others suggested that the pattern of baldness was relevant, but disagreed on the relative importance of frontal baldness [4] versus vertex baldness [2,5]. Nevertheless, few studies have investigated the mechanisms by which MPB might increase cardiovascular risk, and whether this involves established risk factors. Only two studies addressed this question indirectly and demonstrated a possible interaction between MPB and both blood pressure and cholesterol levels in determining risk [1,8]. The explanation for these discrepancies is not immediately obvious, but differences in study design might be relevant. In our study, we used a validated instrument to achieve reliable self-reporting of baldness within no more than 4 years (and typically within 1 year) of other cardiovascular risk factor phenotype measurement. In the analysis by Trevisan et al. [8], the relative timing of baldness assessment and phenotype measurement is not stated, but may have been up to 13 years different. The Physicians’ Health Study [1] relied on data from males, aged 51–95 years, who were asked to recall their degree of baldness at age 45 years. Neither of the two previous studies was on a representative sample of general population, based instead on factory employees [8] and physicians from whom those with a history of cardiovascular disease were excluded [1].

In summary, our analysis of MPB and established cardiovascular risk factor phenotypes in over 1200 healthy male participants of the population-based Victorian Family Heart Study has demonstrated no associations between MPB and other phenotypes. If MPB is indeed predictive of cardiovascular disease, then it is likely to operate through some pathway that fails to influence known risk factors. These studies provide impetus to identify such novel mechanisms of increased cardiovascular risk.

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REFERENCES


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