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Shaving, Coronary Heart Disease, and Stroke

The Caerphilly Study

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The relation between frequency of shaving and all-cause and cardiovascular disease mortality, coronary heart disease, and stroke events was investigated in a cohort of 2,438 men aged 45–59 years. The one fifth (n = 521, 21.4%) of men who shaved less frequently than daily were shorter, were less likely to be married, had a lower frequency of orgasm, and were more likely to smoke, to have angina, and to work in manual occupations than other men. Over the 20-year follow-up period from 1979–1983 to December 31, 2000, 835 men (34.3%) died. Of those who shaved less frequently than daily, 45.1% died, as compared with 31.3% among those who shaved at least daily. Men who shaved less frequently had fully adjusted hazard ratios (adjusted for testosterone, markers of insulin resistance, social factors, lifestyle, and baseline coronary heart disease) of 1.24 (95% confidence interval (CI): 1.03, 1.50) for all-cause mortality, 1.30 (95% CI: 0.99, 1.71) for cardiovascular disease mortality, 1.08 (95% CI: 0.61, 1.92) for lung cancer mortality, 1.16 (95% CI: 0.90, 1.48) for coronary heart disease events, and 1.68 (95% CI: 1.16, 2.44) for stroke events. The association between infrequent shaving and all-cause and cardiovascular disease mortality is probably due to confounding by smoking and social factors, but a small hormonal effect may exist. The relation with stroke events remains unexplained by smoking or social factors.

cerebrovascular accident; coronary disease; hair; hormones; men; mortality

Abbreviations: CI, confidence interval; HOMA, homeostasis model assessment.

Beard growth was considered a useful bioassay marker for androgen activity more than 40 years ago (1). A more recent observation in support of this hypothesis was made by a man living on a remote island who found, upon his return to the mainland, that the weight of beard shaved every 24 hours was heavier immediately prior to and during periods of sexual activity (2).

A case-control study comparing the frequency of shaving in 21 men under 43 years of age who had suffered a myocardial infarction and 21 controls found that nine of the cases but none of the controls shaved only every 2 or 3 days (3). Loss of libido prior to the myocardial infarction and breast tenderness were also observed in these men. It was concluded that hyperestrogenemia may be an important risk factor for myocardial infarction. The findings of this small study have not been replicated, and while the investigators

demonstrated a very strong association, it is quite possible that confounding by other unmeasured factors was responsible. We therefore examined the relation between frequency of shaving and subsequent risk of cardiovascular and all-cause mortality among men recruited in the Caerphilly Study.

MATERIALS AND METHODS

Full details on the Caerphilly Study are available elsewhere (4, 5). Briefly, in Caerphilly, a former mining town in southern Wales, electoral rolls for the defined area were used to identify a random sample of men, each of whom was contacted and asked his age. Those aged 45–59 years were invited to take part in the study. Of 2,818 men identified, 2,513 (89 percent) participated in the baseline survey (phase

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I) in 1979–1983. At recruitment, a clinic assessment comprising a standard medical history, including details on sexual behavior (6), weight, height, blood pressure, venipuncture for biochemical and hormonal assays, and a 12lead electrocardiogram was performed. Insulin resistance was estimated according to homeostasis model assessment (HOMA) as the product of fasting glucose level (mmol/liter) and insulin level (µmol/liter) divided by the constant 22.5; the higher the value, the greater the level of insulin resistance (7). All samples for hormonal analysis were frozen at -20°C and assayed within 3 months of collection. Levels of testosterone, estradiol, and cortisol were measured by radioimmunoassay; details have been previously published (8). Men were asked about their frequency of shaving by a medical interviewer during phase I. Responses were classified into categories ranging from twice daily to once daily, every other day, or less frequently. The 34 men with beards were not classified. These categories were dichotomized into once or twice per day and less frequently.

Identification of coronary heart disease and stroke events

Over the ensuing 20 years (1979–1983 through December 2000), at follow-up examinations held every 4–5 years, men were asked about chest pain and about physician diagnosis of heart attack, stroke, and transient ischemic attack. Electrocardiogram-defined ischemia at baseline and at subsequent clinic visits was determined using Minnesota codes 1-1-1 to 1-2-5 or 1-2-7. Electrocardiogram-defined ischemia was given the date of clinic assessment in time-to-event analyses. Questions about hospital admission for severe chest pain, together with hospital activity analysis notifications of admissions coded 410-414 according to the International Classification of Diseases, Ninth Revision, were used as the basis for a detailed search of hospital notes to identify events that satisfied World Health Organization criteria for acute myocardial infarction and to identify persons who had suffered a stroke. Records of general practitioners were inspected for events that had not led to hospital admission. Radiology records of computed tomography scans were searched for every participant known to have been admitted to a hospital with a cerebral event. Summary information on each possible stroke was assessed by three independent physicians, who rated each event as: not a stroke, an ischemic event, a hemorrhagic event, a transient ischemic attack, or not known. Further details on the methods used for stroke ascertainment have been reported elsewhere (5). Deaths from coronary heart disease comprised all deaths coded 410-414, and deaths from stroke comprised all deaths coded 431-438, excluding code 430 (subarachnoid hemorrhage); data were obtained from the National Health Service central registry.

Statistical methods

Cox proportional hazards models were used to explore the relation between frequency of shaving and clinical events, with adjustment for age and a wide range of cardiovascular disease risk factors (social class, marital status, smoking,

alcohol drinking, prevalent angina, electrocardiogram ischemia, fibrinogen, systolic blood pressure, total cholesterol, high density lipoprotein cholesterol, triglycerides, fasting glucose, HOMA, and testosterone). Subgroup analyses examining effects among nonsmokers, those in nonmanual occupations, and those free of coronary heart disease at baseline were also conducted.

RESULTS

Of the 2,438 men who provided information on shaving, 521 (21.4 percent) shaved less frequently than daily. These men were shorter, more likely to be current smokers, more likely to report a diagnosis of angina, and more likely to work in manual occupations than other men. Men who shaved infrequently were also more likely to be unmarried and to report a lower frequency of sexual intercourse than other men. In addition, they tended to have higher blood fibrinogen levels, fasting glucose levels, and HOMA scores than men who shaved more frequently. No associations were found with serum testosterone, estrogen, cortisol, or cortisol: testosterone ratio (see table 1).

Over the 20-year follow-up period, 835 men (34.3 percent) died, of whom 312 died from coronary heart disease and 81 from lung cancer. There were 534 coronary heart disease events and 216 stroke events, 63 of them fatal, during the follow-up period. Of the 521 men who shaved less frequently than daily, 45.1 percent died during the follow-up period, as compared with 31.3 percent of men who shaved at least daily ($\chi^2 = 34.7$, p < 0.0001).

Cox proportional hazards analyses were confined to the 1,899 men with complete data for all variables included in the models. The age-adjusted hazard ratios demonstrate increased risks of all-cause, cardiovascular disease, and noncardiovascular-disease mortality and all stroke events among men who shaved less frequently. Coronary heart disease events were less strongly associated with shaving frequency. Men who shaved less frequently had age-adjusted hazard ratios of 1.49 (95 percent confidence interval (CI): 1.24, 1.78) for all-cause mortality, 1.52 (95 percent CI: 1.17, 1.97) for cardiovascular disease mortality, and 1.90 (95 percent CI: 1.34, 2.70) for stroke events (table 2). Lung cancer mortality risk was also increased, but confidence intervals were wide. These hazard ratios were not attenuated by adjustment for markers of the insulin resistance syndrome (HOMA, glucose, lipids, and systolic blood pressure) and testosterone. Further adjustment for height attenuated the associations with all outcomes. Adjustment for social class, marital status, smoking, high alcohol consumption, and evidence of angina or electrocardiogram evidence of ischemia attenuated the relations for all-cause and cardiovascular disease mortality, but less so for stroke events. Lung cancer hazard ratios were markedly attenuated by adjustments including smoking.

Similar patterns were seen for nonsmokers, those in nonmanual occupations, and those who were free of coronary heart disease at baseline. Among nonsmokers (n =1,082), 83.6 percent shaved once or twice a day. Of these men, 11.4 percent (n = 103) died of cardiovascular disease as compared with 16.4 percent (n = 29) of nonsmokers who

TABLE 1. Distribution of cardiovascular disease risk factors among men aged 45-59 years, by shaving frequency, Caerphilly Study, 1979-2000

	No. of men					
Risk factor			or twice day	Less frequence	<i>p</i> value	
		Mean	SD*	Mean	SD	
Age (years)	2,438	52.08	4.41	52.37	4.55	0.19
Height (m)	2,400	1.71	0.06	1.69	0.07	< 0.001
Body mass index†	2,398	26.13	3.44	26.39	4.12	0.15
Systolic blood pressure (mmHg)	2,437	140.82	19.12	140.79	18.74	0.98
Fibrinogen level (g/liter)	2,397	3.75	0.82	3.92	0.87	< 0.001
Glucose level (mmol/liter)	2,392	4.97	1.15	5.17	1.79	0.003
Insulin level (µmol/liter)	2,158	6.07	2.22	6.40	2.49	0.22
Homeostasis model assessment	2,075	1.26	2.25	1.29	2.45	0.68
Testosterone level (nmol/liter)	2,366	22.58	7.46	22.82	7.35	0.52
Estradiol level (pmol/liter)	1,726	247.92	63.10	248.09	56.39	0.96
Cortisol level (mmol/liter)	2,345	4.37	1.38	4.26	1.46	0.15
Cortisol:testosterone ratio	2,341	0.21	0.10	0.20	0.09	0.11
Total cholesterol level (mmol/liter)	2,363	5.71	1.12	5.69	1.19	0.73
HDL* cholesterol level (mmol/liter)	2,358	1.12	0.34	1.09	0.32	0.09
Triglyceride level (mmol/liter)	2,354	1.99	1.32	2.04	1.23	0.44
	-	No.	%	No.	%	=
Married	2,438	1,720	89.7	440	84.5	0.001
Sexual intercourse less often than once per month	872	149	20.6	49	33.0	<0.001
Manual occupation	2,378	1,158	61.6	466	93.6	< 0.001
Alcohol user (>21 units‡ per week)	2,438	676	35.3	195	37.4	0.36
Current smoker	2,438	1,012	52.8	344	66.0	< 0.001
Angina	2,438	120	6.3	67	12.9	< 0.001
Electrocardiogram ischemia	2,438	253	13.2	83	15.9	0.11

^{*} SD, standard deviation; HDL, high density lipoprotein.

shaved less frequently (p = 0.06). This difference was more marked for stroke events: 7.6 percent (n = 69) of those who shaved once or twice a day suffered a stroke as compared with 12.4 percent (n = 22) of the less frequent shavers (p =0.04). Of 754 men in nonmanual occupations, 95.8 percent shaved once or twice a day. Of these, 12.7 percent (n = 92)died of cardiovascular disease, whereas 18.8 percent (n = 6)of men in nonmanual occupations who shaved less frequently died from this cause (p = 0.32); however, numbers of events were small. Finally, of those men who were free of coronary heart disease at baseline (n = 1,929), 80.3 percent shaved once or twice a day. Of these, 11.9 percent (n = 184) died of cardiovascular disease, while 16.3 percent (n = 62) of those who shaved less frequently died of this cause (p = 0.02).

DISCUSSION

We have demonstrated an increased risk of all-cause and cardiovascular disease mortality among men who shave infrequently. However, such men had other characteristics that put them at increased risk of cardiovascular disease death: They smoked more and were more likely to suffer from angina; they more often worked in manual occupations; they were shorter; and more of them were unmarried. Risks for all-cause and cardiovascular causes of death were attenuated by similar amounts following adjustment for smoking, social class, marital status, and prevalent cardiovascular disease. Modest increases in risk were still evident for allcause mortality (hazard ratio = 1.24, 95 percent CI: 1.03, 1.50) and stroke events (hazard ratio = 1.64, 95 percent CI: 1.14, 2.38). The crude association with lung cancer mortality was markedly attenuated in the fully adjusted models that

[†] Weight (kg)/height (m)2.

[‡] One unit of alcohol represents an average drink (e.g., 150 ml of wine, 250 ml of beer, and 30-50 ml of spirits) and contains 7-10 g of ethanol.

Model	All-cause mortality (n = 614)		Cardiovascular disease mortality (n = 292)		Non-cardiovascular- disease mortality (n = 322)		All coronary heart disease events (n = 390)		All stroke events (n = 148)		Lung cancer mortality (n = 65)	
	HR*	95% CI*	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
Adjustment for age	1.49	1.24, 1.78	1.52	1.17, 1.97	1.45	1.13, 1.87	1.31	1.04, 1.65	1.90	1.34, 2.70	1.51	0.87, 2.63
Model I†	1.47	1.23, 1.77	1.50	1.15, 1.95	1.46	1.13, 1.87	1.33	1.05, 1.68	1.93	1.36, 2.75	1.52	0.87, 2.65
Model II‡	1.24	1.03, 1.50	1.28	0.98, 1.68	1.22	0.94, 1.58	1.13	0.88, 1.44	1.64	1.14, 2.38	1.06	0.60, 1.88
Model III§	1.24	1.03, 1.50	1.30	0.99, 1.71	1.23	0.95, 1.60	1.16	0.90, 1.48	1.68	1.16, 2.44	1.08	0.61, 1.92

TABLE 2. Hazard ratios for mortality, coronary heart disease events, and stroke events over a period of 20 years, by shaving frequency (less than daily vs. daily or more often) (n = 1,899), Caerphilly Study, 1979-2000

included smoking, the dominant causal factor, which suggests that the relation between frequency of shaving and cardiovascular disease mortality is also likely to be confounded by smoking. However, the relation with stroke events remained strong and was little changed by full adjustment, making residual confounding by measured risk factors an unlikely explanation for this relation. Early life circumstances appear to be more strongly associated with stroke than with coronary heart disease (9), and it is possible that lower shaving frequency, itself associated with adult height—a marker of childhood growth—is due to a developmental process linked with early life exposures.

Our assessment of shaving frequency as a measure of beard growth was qualitative rather than quantitative. Beard growth has been related to high levels of androgens (10) and appears to be increased by acute illness, which suggests that cortisol and other hormones may also play a role (11). In our data, however, there was no association between cortisol or testosterone level and frequency of shaving. Although the case-control study that first put forward the hypothesis found signs and symptoms suggestive of hyperestrogenemia (3), we found no relation with serum estrogen levels. However, we did find an association between low frequency of sexual intercourse and infrequent shaving.

Frequency of shaving, as a secondary sex characteristic, may be a more relevant marker of hormonal status than a single casual testosterone measurement. Adjustment for blood testosterone in our analyses did not attenuate hazard ratios for any clinical event, but this finding in itself cannot exclude the possibility that the residual association between frequency of shaving and these outcomes is explained by hormonal status. Analyses confined to men who were not exposed to major confounders (i.e., those who were nonsmokers, worked in nonmanual occupations, and were free of coronary heart disease at baseline) showed similar associations between frequency of shaving and cardiovascular disease, which provides support for frequency of shaving as a marker of hormonal status. However, in our more powerful analyses using all of the data, the attenuation in shaving frequency and lung cancer hazard ratios—a condition caused by smoking—suggests that the attenuation seen in the shaving frequency-cardiovascular disease association is, at least in part, similarly explained by smoking and social class. These factors, in this population, are powerful determinants of shaving frequency, and the small observed association with cardiovascular disease remaining after adjustment may represent residual socioeconomic confounding or a small effect of hormonal status.

Depression is a risk factor for myocardial infarction (12), and it is possible that the hypothalamic-gonadal axis is disturbed in depressed men, resulting in reduced testosterone secretion and less beard growth. However, it has been reported that beard growth in depressed men with loss of libido and diminished nocturnal penile tumescence does not differ from that in nondepressed men (13), which suggests either that beard growth is a poor marker of testosterone level or that the pituitary-gonadal axis is not disturbed in depression.

In contrast to beard growth, several studies have examined the relation between baldness and coronary heart disease risk (14). In the Physicians' Health Study, vertex baldness but not frontal baldness was associated with increased risk of coronary heart disease events (for severe vertex baldness, relative risk = 1.36, 95 percent CI: 1.11, 1.67) (15). Similar findings have been reported for other cohorts (16, 17). The relation between male-pattern baldness and coronary heart disease is particularly strong among those who become bald at younger ages, which tends to be associated with elevated dihydrotestosterone:testosterone ratios (18). In addition to elevated androgen levels, baldness might result in increased risk of coronary heart disease through association with other coronary heart disease risk factors, although this does not appear to be the case (19). The Caerphilly Study did not record information on baldness.

It seems paradoxical that a marker of elevated androgen levels-baldness-and a marker of depressed androgen or increased estrogen levels—slow beard growth—should both be associated with an increased risk of coronary heart disease mortality. In a small nested case-control study using Framingham data, serum estradiol level but not testosterone

^{*} HR, hazard ratio; CI, confidence interval.

[†] Data were adjusted for age, homeostasis model assessment, glucose level, testosterone level, total cholesterol level, high density lipoprotein cholesterol level, triglyceride level, and systolic blood pressure.

[‡] Data were adjusted for age, social class, marital status, smoking, alcohol drinking, prevalent angina, electrocardiogram ischemia, and fibrinogen level.

[§] Data were adjusted for all of the above factors.

level was increased among men with myocardial infarction (20). These findings could not be replicated in the Honolulu Heart Study (21). Low levels of testosterone have been shown to predict development of diabetes mellitus (22) and are associated with components of the insulin resistance syndrome (8). It is possible, therefore, that these contradictory findings between the Framingham Study and the Honolulu Heart Study are explained by differences in susceptibility to insulin resistance between the populations. However, in the Caerphilly population, the insulin resistance syndrome did not predict 10-year ischemic heart disease risk above and beyond that expected by the individual components (23), but plasma testosterone was associated with the insulin resistance syndrome.

The role of sex hormones in cardiovascular disease risk, particularly when contrasting coronary heart disease and stroke, merits further elucidation. Study designs capable of controlling for the powerful confounding effects of smoking and social class—such as studies using cohorts of university alumni or physicians—would be valuable. Better characterization of hormonal status is required, perhaps by means of repeated measures of hormones, age at sexual maturity, age at first shave, and sexual potency. It would also be important to examine potentially important covariates such as insulin resistance, mood, and other relevant lifestyle factors.

In conclusion, we have demonstrated an association between infrequent shaving and all-cause and cardiovascular disease mortality that is probably due to confounding by smoking and social class. The relation with stroke events remains unexplained by smoking and social class, but hormonal factors may play a role. Low levels of testosterone and high levels of estrogen result in slow beard growth, and these hormones are implicated in the development of insulin resistance. However, adjustment for these factors in our study did not attenuate the association observed, which suggests that any relation between beard growth and cardiovascular disease is not mediated through insulin resistance in our study population.

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