Lack of Association of Diagonal Earlobe Crease With Other Cardiovascular Risk Factors

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The relationship between diagonal earlobe crease and coronary risk factors, controlling for age and sex effects, was tested in 686 persons. A positive correlation ($\rho = .86$, $P < .001$) is obtained between age and percentage of persons with earlobe creases in each one-year age interval; no sex difference is seen. To test for associations between cardiovascular risk factors and earlobe creases, 67 persons with creases are compared with 67 controls (matched by age and sex) without creases, using the following variables: diastolic and systolic blood pressures, cigarette smoking, weight, height, scapular skinfold thickness, serum cholesterol level, high-density lipoprotein level, intracellular sodium, sodium-lithium countertransport, plasma renin level and the presence of diabetes and hypertension. None of these variables differs significantly between cases and controls, indicating that the previously documented association between earlobe crease and coronary heart disease may be independent of these risk factors. Although coronary heart disease has often been shown to aggregate in families, no familial aggregation is found for earlobe creases.

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A number of studies have shown a significant association between coronary heart disease and the presence of diagonal earlobe creases.[1-4] It is unclear whether this association is independent of other coronary risk factors or due to a mutual association of coronary risk factors with both coronary heart disease and earlobe creases. For example, in several studies a positive association was found between creases and hypertension,[4-7] while in others none was found.[2,3] In all but one of these studies[7] only patients with disease were selected (usually those who had had a myocardial infarction). This selection process could easily introduce biases and confounding effects in an evaluation of the association between risk factors and earlobe creases. In addition, most previous efforts have surveyed a limited number of risk factors.

We report findings on a group of persons who were not themselves selected because of a history of coronary heart disease. A large number of risk factors have been evaluated for their association with earlobe creases, several of which have not been considered in previous studies. Also, because coronary heart disease has often been observed to cluster in families, sib-sib aggregation of earlobe creases was measured.

Subjects, Data and Methods

Study subjects included 286 persons younger than age 20 years and 400 adults aged 20 years or older, equally divided between male and female subjects. They are members of pedigrees prone to hypertension, stroke or early coronary artery disease—that is, most of the adults had parents or grandparents with early coronary

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TABLE 1.—*Prevalence of Earlobe Creases By Age and Sex*

Age Interval	Sample Size ♂	Sample Size ♀	Crease on Either or Both Ears (%) ♂	Crease on Either or Both Ears (%) ♀
0-9	53	68	1.9	0.0
10-19	91	74	0.0	0.0
20-29	61	68	3.3	1.5
30-39	43	55	16.3	7.3
40-49	37	26	27.0	34.6
50-59	37	34	64.9	61.8
60-69	13	13	76.9	61.5
≥70	5	8	80.0	100.0
TOTAL	340	346	17.1	14.7

artery disease or stroke. Based on their family history of cardiovascular disease, the subjects were asked to participate in the clinical evaluation described below. Most of them were free of cardiovascular disease at the time of their evaluation. Hypertension was present in 68, diabetes in 20 and coronary artery disease manifestations in 10.

All subjects participated in a four-hour screening protocol at the Cardiovascular Genetics Research Clinic, University of Utah. Data collection included personal health and habits history, fasting blood samples, a standard 12-lead electrocardiogram, anthropometrics, 21 blood pressure measurements and physicians' history and physical examination. The presence of earlobe creases was evaluated in all subjects. The creases were identified in standing persons as a diagonal wrinkle extending across a third or more of the earlobe's surface. Sodium-lithium countertransport was determined using a modification of Canessa's method.[8] Plasma cholesterol, high-density lipoprotein and renin levels were assayed using standard clinical chemistry methods at the University of Utah Medical Center.

Familial aggregation of earlobe creases was tested using a subset of 82 adults older than age 30 in 29 sibships. Relative odds for siblings of persons with creases were calculated by adapting Altham's[9] method to deal with related pairs of persons rather than related persons. Essentially, the method consists of creating 2×2 contingency tables that compare presence and absence of creases for pairs of sibs. Older sib pairs tend to be concordant for earlobe creases due to the correlation of age with earlobe creases. To control for age effects, the sib pairs were stratified into several different age groups.

Results

In Table 1 is listed the age and sex distribution of earlobe creases (on either or both ears) in this population. Among the 109 subjects with creases, 77 (70.6%) had creases of both ears. The prevalence of creases increases greatly with age. The product-moment correlation between age and the proportion of persons with creases in each one-year age interval is .866 ($P<.0005$). A rank-order correlation test (Spearman's ρ) gave a similar value: .863 ($P<.001$). The difference between sexes in percent with creases is statistically nonsignificant and shows no consistent pattern across age groups.

In this population it was possible to match (by five-year age interval and sex) 67 persons with creases on either or both earlobes (cases) with an equal number who had no crease on either earlobe (controls). In Table 2 are given the mean values of a number of coronary artery disease and hypertension risk factors for the cases and controls, as well as significance levels for t tests. There was no significant difference between cases and controls for any of these risk factors. Of the 67 cases, 19 (28.4%) had a diagnosis of hypertension, as did 19 of the controls. One (1.5%) of the cases had had a myocardial infarction, whereas three (4.5%) of the controls had had a myocardial infarction. Diabetes had been diagnosed in six (9.0%) of the cases and in four (6.0%) of the controls. Application of a test for the equality of two percentages[10] shows the difference between the latter two pairs of percentages to be nonsignificant. Because of the small numbers involved, however (especially for the cases of myocardial infarction), this result should not be regarded as conclusive.

Eleven of the cases and 17 of the controls were receiving medication for hypertension. The t tests were run on a sample in which these persons were removed.

TABLE 2.—*Mean Values for Coronary Risk Factors in 67 Cases and 67 Controls*

Coronary Risk Factors	Cases Mean	Cases Sample Size	Controls Mean	Controls Sample Size	Significance
Diastolic blood pressure (mm/mercury)* ..	75.4	67	75.8	67	NS
Systolic blood pressure (mm/mercury)* ...	114.4	67	115.9	67	NS
Years of cigarette smoking	4.1	67	2.9	67	NS
Weight (kg)	79.9	67	77.7	67	NS
Height (cm)	167.8	67	168.6	67	NS
Scapular skinfold thickness (mm)	267.6	67	252.7	67	NS
Cholesterol (mg/dl)	214.5	66	205.8	67	NS
High-density lipoprotein (mg/dl)	50.6	47	54.2	51	NS
Intracellular sodium (mM)	50.6	67	54.9	67	NS
Sodium-lithium countertransport†	288.0	62	282.1	63	NS
Renin (ng/ml/hour×100)	160.6	65	175.9	65	NS

*Average of six resting sitting measurements.
†μmol an hour per liter of erythrocytes according to a modification of Canessa's[8] method.

The mean values for each risk factor were very similar to those of the full sample, and again no differences were statistically significant.

In the analysis of familial aggregation, 32 of the 82 adults had earlobe creases. Calculation of relative odds (Table 3) shows that the probability of observing an earlobe crease in a person is not statistically raised when the person's sibling has an earlobe crease. As expected, older siblings were usually concordant for the presence of crease, and younger siblings were usually concordant for the absence of crease.

Discussion

While most studies have shown a positive association between earlobe creases and coronary heart disease, at least one showed no association.[11] Because our population was not selected on the basis of disease, the number of cases of myocardial infarction was quite small. However, there were 15 persons who had had a myocardial infarction (average age, 55.5 years). Of these, 11 (73.3%) had earlobe creases. The incidence of creases in the 50- to 59-year-old age group of our clinic population was 63.4%. The difference between these two percentages is not statistically significant ($P>.4$; test for the equality of two percentages). However, the sample size for the myocardial infarction group is small, and the difference of the two percentages is in the expected direction. In another study in the Salt Lake City area, Blodgett[12] compared 77 patients with myocardial infarction and 77 controls (matched for age and sex) for the presence of earlobe creases. The average age in both groups was 61.5 years. Among the infarction cases, 75% had earlobe creases, whereas 35% of the controls had creases. This difference is highly significant. These data and our own both lend further support to the relationship between coronary artery disease and earlobe creases.

It is interesting that while the percentage of creases among Blodgett's cases of myocardial infarction was similar to that of our study, the percentage among the controls was substantially lower. This could reflect the fact that many of the persons in our study population are members of families prone to coronary artery dis-

TABLE 3.—*Frequency of Earlobe Creases in Sibling Pairs*

	Number of Pairs With Crease in			
	Both Persons	*One Person*	*Neither Person*	*Relative Odds**
Both age 50+	22	9	4	1.1
Younger pairs	10	27	82	1.1

*None statistically significant.

TABLE 4.—*Percentages of Ear Creases by Age in This and Other Studies**

Study / *Age*	*Lichstein, et al[3]* CHD	Control	*Schoenfeld, et al[4]* CHD	Control	*Petrakis and Koo[13]* Control	*Haft, et al[14]* CHD	Control	*Christiansen, et al[15]* CHD	Control	*Rhoads, et al[16]* CHD	Control	*This Study* Control
0-9	...	...	...	...	...	...	...	...	...	...	...	1
10-19	...	...	...	...	...	...	...	...	...	...	...	0
20-29	...	...	...	...	18	11	0	...	...	...	...	2
30-39	15	14	73	25	23			0	4	...	...	11
40-49	46	39				52	27	0	2	...	...	30
50-59	48	28	80	40	51			47	14	26	30	63
60-69	55	40	74	38		63	53	43	24	28	35	69
≥70	45	37	83	44				47	35	42	26	92

CHD = coronary heart disease

*Brackets indicate different grouping of ages.

TABLE 5.—*Summary of Relationships Between Earlobe Creases and Coronary Risk Factors From Other Studies*

Study	*Hypertension*	*Diabetes*	*Smoking*	*Obesity*	*Cholesterol*	*HDL*	*Triglyceride*
Kaukola, et al[2]	0	0	0	0	0	0	0
Lichstein, et al[3]	0	0	0	...	...	...	...
Schoenfeld, et al[4]	1	...	0	...	0	...	...
Kristensen[6]	1	...	...	...	...	...	...
Moncada, et al[7]	1	0	...	...	0	...	0
Haft, et al[14]	0	...	0	1	0	...	0
This study	0	0	0	0	0	0	...

HDL = high-density lipoprotein

*1 = significant association found, 0 = no association found, ... = no information.

ease and may thus present ear creases as an early sign of a predisposition to coronary disease. However, the lack of familial aggregation of ear creases noted above militates somewhat against this conjecture.

A positive correlation between age and ear creases has been observed in most other studies,[1-3,11-15] but at least two studies[4,16] failed to show this relationship. In addition, as Table 4 shows, the percentages of creases in each age class varies widely from study to study. These inconsistencies may reflect differences in sampling procedures—that is, differences among patients with myocardial infarction, hospital patients in general and healthy persons—methods of diagnosing earlobe creases, age intervals sampled (only older men were sampled in one study[16]) and ethnic composition of the study populations.[13] Our study population is comprised mostly of persons of northern European descent, and our distribution of creases by age corresponds fairly well with that obtained by Petrakis and Koo[13] for their "white" population sample. Generally, however, the frequencies in our study population tend to be higher than those of the controls from other populations. Again, this may reflect the ascertainment factor mentioned above.

The lack of a sex difference in earlobe crease prevalence is consistent with the findings of two other studies,[4,17] though one other survey found a significant difference.[14] In our study, it is especially interesting that premenopausal women, who have lower rates of myocardial infarction than men of comparable ages, do not show lower percentages of earlobe creases.

The relationships found between creases and coronary risk factors in other studies are summarized in Table 5. The study of Frank[5] was not included in this table because it did not use a control population. The table indicates that most risk factors are not associated with ear creases, a finding that agrees with the results of our study. Three studies did show an association with hypertension, however, whereas ours did not. (It should be noted that Rhoads and co-workers[16] found a small but significant difference in systolic blood pressure but no difference in diastolic pressure.) In one of these studies,[4] the comparison for earlobe creases was done only on patients who had had a myocardial infarction; this could well account for their result. The study most comparable to ours is that of Moncada and associates[7] because they studied healthy persons. They did find a positive association between hypertension and earlobe creases. These conflicting results could be due to different sampling schemes (we sampled families at risk for hypertension) or the dissimilar ethnic backgrounds of the two populations, or both.

A frequently voiced concern is that the earlobe crease may be associated with coronary heart disease because of a mutual association with some other coronary risk factor.[3] Our results indicate that earlobe creases are not associated with another major risk factor and may therefore be an independent diagnostic sign for coronary heart disease.

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