PREVENTIVE MEDICINE 16, 616–625 (1987)

Aggregation of Blood Pressure in Anglo-American and Mexican-American Families[1]

THOMAS L. PATTERSON, PH.D.,*[,2] ROBERT M. KAPLAN,†,‡ JAMES F. SALLIS,* AND PHILIP R. NADER*

*Child and Family Health Studies, Division of General Pediatrics, and †Department of Community and Family Medicine, University of California at San Diego, La Jolla, California 92093, and ‡Center for Behavioral Medicine, San Diego State University, San Diego, California 92182

We investigated the aggregation of blood pressure within 95 Anglo-American and 111 Mexican-American families. Degree of genetic relatedness was evaluated by calculating separate correlations for spouses and for each spouse with both same-sex and opposite-sex offspring. In addition, sibling correlations were evaluated. These analyses were performed separately for Anglo-American and Mexican-American families. Replicating earlier findings, correlations between blood pressures of Anglo spouses were nonsignificant. However, there were significant spousal correlations in the Mexican-American group. For Anglo-American families, there were significant associations between blood pressures of fathers and sons and between blood pressures of mothers and daughters. Correlations between blood pressures for opposite-sex parent–child pairs were nonsignificant. In the Mexican-American group, there were significant correlations between fathers' blood pressures and those of both male and female offspring. For mothers, blood pressures were weakly correlated with all other family members. These relationships remained after adjustment for age, body mass index, and measures of dietary habits and activity levels. In general, blood pressure aggregation was greater in Mexican-American families.

INTRODUCTION

Epidemiologic studies have consistently demonstrated that elevated blood pressure (BP) is a significant predictor of coronary heart disease (CHD) and stroke (22). For this reason, studies of the determinants of BP variability are of great theoretical and practical importance. In this article, we consider factors associated with BP variation.

A significant number of studies suggest that BP aggregates within families (11, 12, 14–16, 18, 20, 25, 28, 29, 31, 36). In general, studies show that spousal correlations in BP tend to be low and that associations increase with genetic relatedness.

Although most studies support the genetic determinants of BP variability, several inconsistencies in the literature remain. For example, Staessen *et al.* (31) demonstrated that mother–offspring correlations in body mass were higher than father–offspring correlations. Since body mass is a strong correlate of BP, this suggests that mother–offspring BP correlations should exceed father–offspring

[1] Supported by Grant R18 HL 30872 from the National Heart, Lung, and Blood Institute to Philip R. Nader and K04 HL 00809 to Robert M. Kaplan.

[2] To whom reprint requests should be addressed at Child and Family Health Studies, Division of General Pediatrics, M-031-F, University of California at San Diego, La Jolla, CA 92093.

0091-7435/87 $3.00

correlations. Yet, studies in Belgium (31) and in the United States (14) indicate that same-sex parent–child correlations tend to be statistically significant, while opposite-sex parent–child correlations tend to be nonsignificant.

A second unresolved issue concerns spousal correlations. Studies of BP correspondence for married adult dyads have produced inconsistent results (28). Spouses may share the same home environment but different genetic backgrounds. After reviewing the literature on spousal BP concordance, Suarez *et al.* (33) noted that 7 of 12 published studies found spousal concordance in BP, while 5 failed to observe significant concordance. Although some studies show an apparent increase in concordance with duration of marriage, it appears this is an artifact of selective survival. In other words, data on spousal concordance are not entirely consistent. The Suarez *et al.* study showed that increases in spousal concordance as a function of marriage duration were largely influenced by the age of the wife. Spousal concordance tends to be low when the female partner is under 50 years old and higher for couples with older women. One complication in these findings is that there was higher concordance when the wife was between ages 20 and 29 years. This suggests assortative mating or similarities in behaviors for young couples, and behavioral determinants in older couples. The mid-life years, when there is greater spousal discordance, are also those in which greater disparities in diet and activity might be expected.

High intrafamilial correlations in BP support two alternative explanations: First, intrafamilial correlations may indicate shared genetic background. The alternative explanation is that the correlations result from similar health habits. A recent article by Carmelli and Rosenman (6) noted the importance of considering a variety of health habits in family aggregation studies. For example, sex asymmetries in parent–child BP may indicate differences in activity patterns. In the present study, the San Diego Family Health Project data set allows us to determine the effect of behavioral variables on familial aggregation of BP. Detailed information is collected on dietary behaviors of each family member as well as detailed assessments of exercise patterns. Both dietary sodium intake (23) and exercise (35) habits are related to BP levels.

A second unique aspect of the Family Health Project data set is that it includes information on parallel samples of Anglo-American and Mexican-American families. In one study, hypertension rates in Mexican-American families were approximately one-third those of the U.S. average, but borderline hypertension tended to be somewhat higher in the Mexican-American population (13). It might be argued that genetic factors contribute to more severe cases of hypertension, while behavioral factors, including obesity, account for the observed high prevalence of borderline cases.

Another advantage of studying the Mexican-American population is that it has a unique cultural pattern of diet and exercise that differs significantly from that of the Anglo-American population. To date, there have been relatively few studies investigating BP aggregation in Mexican-American families. In one recent report, Stern and colleagues (32) showed that Mexican-American men and women had lower hypertension prevalence rates than Anglo-Americans from the same neighborhood. Despite these lower BPs, Mexican-Americans consumed more table salt than their Anglo-American counterparts. However, they still consumed less table salt than did black Americans (21). In summary, examination of BP aggregation in Mexican-American families is of some interest. This unique culture is characterized by several risk factors for high BP, including obesity (32) and increased use of table salt. Yet, both BP and CHD mortality appear to be lower in this group.

METHODS

Subjects

The data reported here were gathered as part of a cardiovascular risk reduction program known as the San Diego Family Health Project (26). Ninety-five Anglo-American (58 fathers, 87 mothers, 60 male offspring, 54 female offspring) and 111 Mexican-American families (42 fathers, 102 mothers, 65 male offspring, 78 female offspring) participated in the study. The Mexican-American group included many immigrants along with native-born participants. Tables 1 and 2 show the age distribution for these groups. In addition, the tables show means and standard deviations for body mass index, dietary sodium intake, exercise, and systolic (SBP) and diastolic (DBP) blood pressure. Families of 5th- and 6th-grade children were recruited from elementary schools in the San Diego Unified School District. Census tract data were used to identify schools with large numbers of Mexican- or Anglo-American families of lower socioeconomic status (SES). The Hollingshead (17) two-factor index of social position indicated that Anglo-American participants were predominantly middle class ($\overline{X}$ = 2.15), while the Mexican-American families tended to fall in the lower part of the distribution ($\overline{X}$ = 3.73).

TABLE 1
MEANS AND STANDARD DEVIATIONS FOR ALL MEASURES, BY FAMILY STATUS FOR ANGLO-AMERICANS

	Age	BMI	Sodium	KKD	SBP	DBP
Adult male						
$\overline{X}$	39.12	25.38	3,835.86	38.47	120.47	77.38
SD	5.89	4.48	1,869.70	6.42	12.11	8.26
N	58	58	58	58	58	58
Adult female						
$\overline{X}$	37.20	25.23	2,683.40	36.93	110.90	72.46
SD	5.29	7.24	1,324.93	4.24	11.20	9.28
N	87	87	83	84	87	87
Child male						
$\overline{X}$	11.47	18.29	3,330.19	39.43	101.54	67.19
SD	1.71	2.47	1,537.43	4.24	8.10	8.64
N	60	60	58	60	60	58
Child female						
$\overline{X}$	11.96	17.64	2,583.62	37.96	100.38	65.62
SD	1.85	2.66	985.83	3.46	9.26	9.34
N	54	54	52	54	54	43

Note. Age, in years; BMI, body mass index (weight kg/height cm^2); sodium, in mgs; KKD, kcal/kg/day; SBP, systolic blood pressure in mm Hg; DBP, diastolic blood pressure in mm Hg.

TABLE 2
MEANS AND STANDARD DEVIATIONS FOR ALL MEASURES, BY FAMILY STATUS FOR MEXICAN-AMERICANS

	Age	BMI	Sodium	KKD	SBP	DBP
Adult male						
$\bar{X}$	37.86	28.68	3,774.41	41.14	120.98	79.75
SD	6.85	4.18	2,357.35	8.92	12.09	12.31
N	42	42	41	42	42	42
Adult female						
$\bar{X}$	35.44	27.25	2,662.26	36.50	111.30	72.15
SD	6.04	5.94	1,500.97	3.23	12.95	10.42
N	102	102	97	101	101	101
Child male						
$\bar{X}$	11.89	20.17	3,029.19	38.60	103.28	66.70
SD	1.37	4.35	1,401.90	4.39	10.09	9.78
N	65	65	62	64	65	59
Child female						
$\bar{X}$	11.81	20.23	2,822.79	37.33	102.26	68.17
SD	1.56	3.61	1,462.77	3.25	10.06	9.24
N	78	78	73	76	78	61

Note. Age, in years; BMI, body mass index (weight kg/height cm^2); sodium, in mg; KKD, kcal/kg; SBP, systolic blood pressure in mm Hg; DBP, diastolic blood pressure in mm Hg.

Individuals were excluded from participation if there was some indication of high BP, defined in adults as taking BP medications, or a mean of three BP determinations of 150 systolic or 95 diastolic. Children were excluded if either systolic or diastolic determinations were above the 95th percentile for their age. Individuals were also excluded if there was preexisting cardiovascular disease, total cholesterol above the 95th percentile for that age and sex, or some chronic illness that would prohibit an individual from participating in the exercise/diet program promoted in the Family Health Project (30). Very few individuals in either ethnic group were excluded from the study for any of these reasons.

Procedures

The mean of three consecutive BP readings was used for this analysis. The measurement and staff certification procedures developed in the Hypertension Detection and Follow-up Program (9) were used for adults. For children, the cuff selection and fourth-phase determination procedures suggested by Berenson (3) and Kahn (19) were utilized. Adult cuffs were used for children with arm circumferences greater than 9 in. All BPs were taken on the right arm resting at heart level. In order to reduce observer bias, random-zero sphygmomanometers (10, 39) were used for all readings. The diaphram of the stethoscope was placed over the brachial artery. Children's BPs were sometimes faint, and in those cases the bell was used (27).

SBP was recorded at the appearance of the first regular Korotkoff sound. Fourth-phase BP was recorded for children at the point at which the Korotkoff sounds became muffled and was used as the DBP for children. Fifth-phase BP was recorded for children and adults at the disappearance of the Korotkoff sounds. At least 1 min elapsed between each of the three determinations. All measurements were taken between 7:00 and 9:30 AM. Subjects were asked to fast for the 12-hr period preceding measurement. The Quetelet Index (kg/m^2) was used as the measure of body mass. The amount of sodium in the diet was determined by a 24-hr diet recall, taken by trained nutrition graduate students. Additional questions regarding sodium intake supplemented the standard interview procedure. The Nutrition Coding Center (University of Minnesota) analyzed dietary histories and determined the total amount (in milligrams) of sodium in the diet. The number of kilocalories per kilogram of body weight expended per day was determined by a 7-day physical activity recall. This measure was developed for use in the Standard Five-City Project (5, 30) and has been validated with both adults (34) and children (37). Interviewers were trained exercise physiology graduate students. Information was gathered on length of time subjects engaged in moderate, hard, or very hard activities during the week prior to measurement.

RESULTS

Table 1 summarizes age, body mass, sodium intake, exercise, SBP, and DBP means and standard deviations for male and female adults and children from Anglo-American families. The same values for Mexican-American participants are given in Table 2.

The family aggregation results are displayed in Figs. 1 and 2. Figure 1 presents the results for Anglo-American families, while Fig. 2 focuses on Mexican-American families. The figures show zero-order correlations and relationships between family pairs with adjustments using partial correlations for body mass, exercise, and sodium intake. Sibling pairs include only those cases where both male and female children existed.

As shown in Fig. 1, BP tended to be uncorrelated in spouse pairs for Anglo-

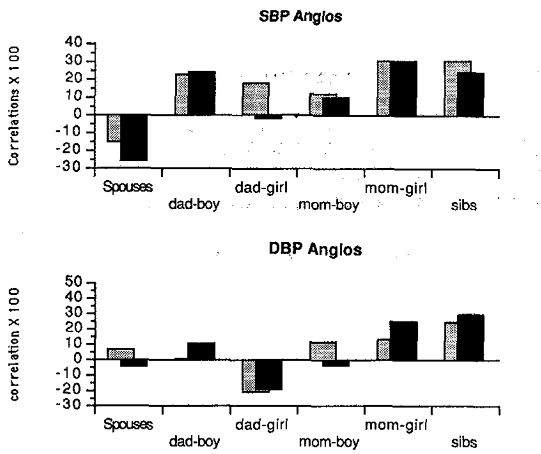

FIG. 1. Correlations of blood pressures in Anglo-American families ($N = 95$). For spouse pairs, father–son (dad–boy), father–daughter (dad–girl), mother–son (mom–boy), mother–daughter (mom–girl), and siblings. The left bar of each pair is the zero-order correlation and the right bar is the partial correlation after adjusting for age, body mass, sodium intake, and exercise habits.

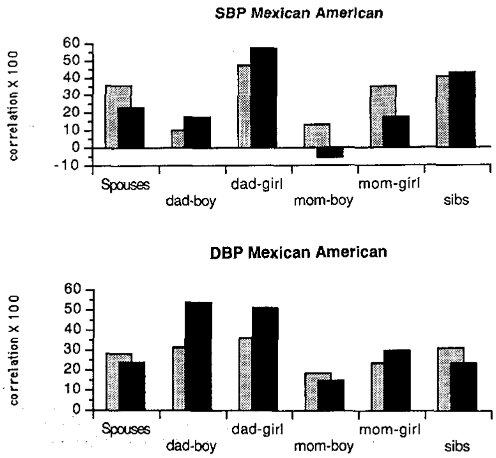

FIG. 2. Correlations of blood pressures in Mexican-American families ($N = 111$). For spouse pairs, father–son (dad–boy), father–daughter (dad–girl), mother–son (mom–boy), mother–daughter (mom–girl), and siblings. The left bar of each pair is the zero-order correlation and the right bar is the partial correlation after adjusting for age, body mass, sodium intake, and exercise habits.

American participants. The relationship for DBP was near zero, while it was slightly negative for SBP ($r = -0.15$). Adjustments for a variety of different factors did not influence the nonsignificant relationship among spousal BPs.

The relationship between SBP of fathers and sons was nonsignificant, although there was a trend in the positive direction ($r = 0.23$, $P < 0.08$). Father–son correlations for DBP were nonsignificant either as zero order or with adjustments for other factors.

All associations between SBP or DBP between fathers and daughters were nonsignificant as were relationships between mothers and sons. However, the association between BPs of Anglo-American mothers and daughters was statistically significant both without ($r = 0.31$, $P < 0.01$) and with ($r = 0.31$, $P < 0.01$) adjustments for other risk factors. Sibling correlations were significant for SBP ($r = 0.26$, $P < 0.05$) but not for DBP.

The relationships for Mexican-American participants did not fit the same pattern. First, as shown in Fig. 2, there were significant spousal correlations in the Mexican-American group ($r = 0.28$, $P < 0.04$ for DBP; $r = 0.36$, $P < 0.01$ for SBP). These relationships were attenuated by adjustment for body mass ($r = 0.21$ and $r = 0.22$ for DBP and SBP, respectively). However, the adjustment for body mass and other risk factors had only small effects on spousal correlations.

Father–son correlations were statistically significant in the Mexican-American group for DBP. These relationships were accentuated by adjustments for other variables ($r = 0.54$, $P < 0.02$). The father–son correlation for SBP was nonsignificant. In contrast to the Anglo-American families, there was also a significant association between BPs of fathers and daughters. With adjustments for other factors, this relationship was highly significant for both DBP ($r = 0.51$) and SBP ($r = 0.57$). Mother–daughter correlations were statistically significant for both SBP and DBP as zero-order relationships ($r = 0.24$ and $r = 0.25$, respectively). Although there was some attenuation, these relationships remained after adjustment for other risk factors. All mother–son correlations were nonsignificant. Finally, there were strong sibling correlations for both DBP ($r = 0.41$) and SBP ($r = 0.31$). These relationships were relatively unaffected by adjustment for dietary sodium and exercise habits.

DISCUSSION

Our data suggest that familial aggregation of BP is greater in Mexican-American than in Anglo-American families. As noted earlier, results of studies on spousal concordance in BP have been mixed. In general, population studies of white Americans have typically failed to demonstrate significant spousal BP concordance. Some of the studies that have demonstrated associations had large sample sizes and the capacity to detect very small deviations from zero (33). For example, the Framingham Study (28) was able to demonstrate a significant relationship between BPs of husbands and wives with a 0.054 coefficient. This coefficient was statistically significant, because the sample size was 1,026. In general, those studies observing significant associations have tended to observe coefficients below 0.15 (cf. Table 3).

Against these findings, observations of substantial spousal correlations are of prime interest. It is possible that the close-knit nature of Mexican-American families allows similar determinants to affect each spouse. The study in the literature showing the highest spousal correlations was for a population of Taiwanese agricultural workers (36). However, our adjustments for body mass, exercise, and other factors had little impact on the observed correlations.

Our observation of greater parent–child correlations for like-sex pairs is consistent with studies in both Europe (31) and the United States (14) (see Table 3 for a summary of a number of aggregation studies). However, the Mexican-American families did not follow the typical pattern. BPs of fathers were significantly correlated with those measured from offspring of either sex. BP correlations of Mexican-American mothers tended to be slightly higher with daughters than with sons. For the Anglo–American families, the sex synchrony in BP correlation was observed for SBP but not for DBP. It is interesting that in the Staessen *et al.* study (31), the father–daughter pairs were associated with the lowest correlations. In our data, there were virtually no correlations between SBPs of fathers and daughters ($r = -0.01$, adjusted for age, body mass, exercise, and sodium intake). However, father–daughter correlations in the Mexican-American group were among the strongest in the data set (SBP adjusted for other factors, $r = 0.57$, $P < 0.001$).

In summary, our data suggest that studies of BP aggregation based on white and European populations may not be predictive of patterns of family aggregation in Mexican-American families. BP determinants in Mexican-American families are of particular interest for several reasons. First, this group has several risk factors for high BP including high prevalence of obesity and high sodium intake. Nevertheless, the prevalence of high BP and of CHD tends to be low. Our data also suggest that adjustment for high BP risk factors such as sodium intake, obesity, and exercise has little effect on the estimates of family BP aggregation for either Anglo- or Mexican-American families. These known influences upon BP are each measured with significant error. Measurement error can attenuate the

TABLE 3
COMPARISON OF BLOOD PRESSURE AGGREGATION STUDIES
(SYSTOLIC CORRELATION/DIASTOLIC CORRELATION)

Study	Spouse	Father/Son	Father/ Daughter	Mother/ Son	Mother/ Daughter	Sibs
Anglo-American (present study)	−0.15/0.07	0.23/0.01	0.18/−0.21	0.12/0.12	0.31/0.13	0.31/0.39
Mexican-American (present study)	0.36/0.28	0.10/0.31	0.47/0.36	0.13/0.19	0.25/0.24	0.16/0.31
Staessen *et al.* (31) (two Belgian towns)	0.22/0.40	0.45/0.39	0.09/0.18	0.49/0.56	0.44/0.40	0.40/0.59
Johnson *et al.* (18)[a] (Tecumseh, MI)	0.06/0.04	0.14/0.10	0.18/0.08	0.12/0.19	0.13/0.17	0.17/0.10
Miall and Oldham (25)[b] (Wales)	−0.01/−0.14	0.16/0.20	0.25/0.11	0.23/0.09	0.25/0.23	0.30/0.20
Sackett *et al.* (29)[c] (Framingham)	0.09/0.05	ND	ND	ND	ND	ND
Hayes *et al.* (16) (Evans Co., GA)						
Whites	0.13/0.08	0.13/0.40[d]	ND	ND	ND	0.20/0.17
Blacks	−0.03/−0.05	0.26/0.17[d]	ND	ND	ND	0.14/0.19
Beresford (4)[d] (Harrow, London)	0.07/0.03	ND	ND	ND	ND	ND
Havlik *et al.* (14)[e] (Framingham)	0.15/0.14	0.18/0.18	0.13/0.17	0.17/0.21	0.25/0.22	0.18/0.20
Tseng (36)[f] (Taiwan rural)	0.15/0.12	0.18/0.18	0.15/0.22	0.11/0.18	0.23/0.09	ND
Miall *et al.* (24)[g] (Jamaica)	ND	0.08/−0.02	0.29/0.26	0.22/0.19	0.08/0.17	0.12/0.08
Bengtsson *et al.* (2)[h] (Sweden)	0.04/ND	−0.03/ND	−0.06/ND	0.17/ND	0.18/ND	0.32/ND
Connor *et al.* (8)[i] (Portland)	0.12/0.14	0.30/0.14	ND	−0.01/−0.10	ND	−0.07/−0.02
Annest *et al.* (1)[j] (Montreal Adoption Survey)	0.15/0.18	0.24/0.21	ND	0.27/0.26	ND	0.38/0.53
Clarke *et al.* (7)[k] (Muscatine, Iowa)	ND	0.09/0.14	ND	0.15/0.16	ND	0.16/0.16

Note. ND = No data.

[a] Johnson *et al.* reported correlations blocked by age of wife. We chose the range 30–39 for comparison purposes, since this is closest to our sample.

[b] Data are regression coefficients which should approximate correlation coefficients, since the method employed for score computation gives equal age–sex specific standard deviations. Correlation for sibs is "male propositi with brothers" as reported in Miall, W. E., Heneage, P., Khosla, T., Lovell, H. G., and Moore, F. Factors influencing the degree of resemblance in arterial pressure of close relatives. *Clin Sci.* 33, 271–283(1967).

[c] Data are expressed as Kendall's Tau-A.

[d] Correlations between parent and offspring.

[e] All correlations are zero order. Authors also report partial correlation coefficients. Sibling correlation was reported as correlations between brothers and sisters.

[f] Correlations were reported after correcting for age and sex.

[g] As in footnote [b], data are regression coefficients. Tabled data for sibs are for brothers; sisters' data are 0.20/0.27.

[h] No diastolic blood pressures were recorded.

[i] The correlations listed under father/son are actually father–mean of children and under mother/son are actually mother–mean of children.

[j] Correlations listed under father/son are actually father/natural child and under mother/son are mother/natural child.

[k] All correlations are adjusted for age. Correlations under father/son are actually father/child and under mother/son are actually mother/child.

impact of adjusting for these covariates. Thus, failure of the covariate adjustment to modify the effect does not necessarily mean that these variables have no effect on estimates of family aggregation.

Any interpretation of the difference in aggregation patterns between Anglo- and

Mexican-American families must be highly speculative. At least some component of BP in these families must be determined by genetic make-up. This would explain the higher correlations between genetically similar (siblings and parent–child pairs) than between unrelated (spouses) individuals. Nevertheless, spousal associations remain substantial among Mexican-Americans. This suggests that assortative mating or similar health habits contribute to the observed similarities. Yet, adjustments for relevant health behaviors did not reduce the observed associations. It should be noted that our sample was more homogeneous in SES than that found in most epidemiologic studies. Thus, some correlations may have been attenuated due to restricted range.

Mexican and Mexican-American populations remain largely underrepresented in epidemiologic studies. Further investigation of the determinants of BP and other CHD risk factors is clearly indicated.

REFERENCES

1. Annest, J. L., Sing, C. F., Biron, P., and Mongeau, J. Familial aggregation of blood pressure and weight in adoptive families. II. Estimation of the relative contribution of genetic and common environmental factors to blood pressure correlations between family members. *Amer. J. Epidemiol.* **110**, 492–503 (1979).
2. Bengtsson, B., Thulin, T., and Schersten, B. Familial resemblance in casual blood pressure—A maternal effect? *Clin. Sci.* **57**, 279s–281s (1979).
3. Berenson, G. S. "Cardiovascular Risk Factors in Children: The Bogalusa Heart Study." Oxford Univ. Press, New York, 1980.
4. Beresford, S. A. Concordance of blood pressure in spouses. *Rev. d'Epidemiol.* **24**, 395–403 (1976).
5. Blair, S. N. How to assess exercise habits and physical fittness, *in* "Behavioral Health: A Handbook of Health Enhancement and Disease Prevention" (J. D. Matarazzo, S. M. Weiss, J. A. Herd, and N. E. Miller, Eds.), pp. 424–447. Wiley, New York, 1984.
6. Carmelli, D., and Rosenman, R. Family resemblance on the Thurstone activity scale, systolic blood pressure, and total cholesterol among first degree relatives of subjects with and without coronary heart disease. *Genet. Epidemiol.* **2**, 55–70 (1985).
7. Clarke, W. E., Schrott, H. G., Burns, T. L., Sing, C. F., and Lauer, R. M. Aggregation of blood pressure in the families of children with labile high systolic blood pressure. *Amer. J. Epidemiol.* **123**, 67–80 (1986).
8. Connor, S. L., Connor, W. B., Henry, H., Sexton, G., and Keenan, E. J. The effects of familial relationships, age, body weight, and diet on blood pressure and the 24-hour urinary excretion of sodium, potassium, and creatinine in men, women, and children of randomly selected families. *Circulation* **70**, 76–85 (1984).
9. Curb, J. D., Labarthe, D. R., Cooper, S. P., Cutter, G. R., and Hawkins, C. M. Training and certification of blood pressure observers. *Hypertension* **5**, 610–614 (1983).
10. DeGaudemaris, R. D., Folsom, A. R., Prineas, R. J., and Luepker, R. V. The random-zero versus the standard mercury sphygmomanometer: A systematic blood pressure difference. *Amer. J. Epidemiol.* **121**, 282–290 (1985).
11. Friman, G., and Waern, U. Blood pressure and blood lipids in members of families with a heavy aggregation of essential hypertension. *Acta Med. Scand.* **196**, 11–16 (1974).
12. Gerson, L. W., and Foder, J. G. Family aggregation of high blood-pressure groups in two Newfoundland communities. *Canad. J. Public Health* **66**, 294–298 (1975).
13. Hanis, C. L., Ferrell, R. E., and Schull, W. J. Hypertension and sources of blood pressure variability among Mexican-Americans in Starr County Texas. *Int. J. Epidemiol.* **14**, 231–238 (1985).
14. Havlik, R. J., Garrison, R. J., Feinleib, M., Kannell, W. B., Castelli, W. P., and McNamara, P. M. Blood pressure aggregation in families. *Amer. J. Epidemiol.* **110**, 304–312 (1979).

15. Havlik, R. J., Garrison, R. J., Katz, S. H., Ellison, R. C., Feinleib, M., and Myrinthopoulos, D. C. Detection of genetic variance in blood pressure of seven-year old twins. *Amer. J. Epidemiol.* **109**, 512–516 (1979).
16. Hayes, C. G., Tyroler, H. J., Cassell, J. C., and Hill, C. Family aggregation of blood pressure in Evans County Georgia. *Arch. Intern. Med.* **128**, 965–975 (1971).
17. Hollingshead, A. B. "Two Factor Index of Social Position." Yale Univ. Press, New Haven, CT, 1965.
18. Johnson, D. C., Epstein, F. H., and Kjelsberg, M. O. Distributions of familial studies of blood pressure and serum cholesterols in a total community—Tecumseh, Michigan. *J. Chronic Dis.* **18**, 147–160 (1965).
19. Kahn, H. S. Personal communication, 1983.
20. Kass, E. H., Zinner, S. H., Margolius, H. S., Lee, Y. H., Rosner, B., and Donner, A. Familial aggregation of blood pressure and urinary kallikrein in early childhood, *in* "Epidemiology and Control of Hypertension" (O. Paul, Ed.), pp. 359–369. Symposia Specialists Miami, FL, and Thieme, Stuttgart, 1975.
21. Kerr, G. R., Amante P., Decker, M., and Callen, P. W. Ethnic patterns of salt purchase in Houston, Texas. *Amer. J. Epidemiol.* **115**, 906–916 (1982).
22. Labarthe, D. R. The rationale for treatment of "mild" hypertension, *in* "Behavioral Epidemiology and Disease Prevention" (R. M. Kaplan and M. H. Criqui, Eds.), pp. 135–159. Plenum, New York, 1985.
23. MacGregor, G. A. Dietary sodium and potassium intake and blood pressure. *Lancet* **1**, 750–753 (1983).
24. Miall, W. E., Kass, E. H., Ling, J., and Stuart, K. L. Factors influencing arterial pressure in the general population in Jamaica. *Brit. Med. J.* **2**, 497–506 (1962).
25. Miall, W. E., and Oldham, P. D. The heredity factor in arterial blood-pressure. *Brit. J. Med.* **1**, 75–80 (1963).
26. Nader, P. R., Sallis, J. F., Rupp, J., Atkins, C., Patterson, T., and Abramson, I. San Diego Family Health Project; accessing families through the schools. *J. Sch. Health* **56**, 227–231 (1986).
27. Prineas, R. J., and Jacobs, D. Quality of Korotkoff sounds: Bell vs diaphram, cubital fossa vs brachial artery. *Prev. Med.* **12**, 715–719 (1983).
28. Sackett, D. L. Studies of blood pressure in spouses, *in* "Epidemiology and Control of Hypertension" (O. Paul, Ed.), pp. 21–35. Symposia Specialists, Miami, FL and Thieme, Stuttgart, 1975.
29. Sackett, D. L., Anderson, G. D., Milner, R., Feinleib, M., and Kannel, W. B. Concordance for coronary risk factors among spouses. *Circulation* **52**, 589–595 (1975).
30. Sallis, J. F., Haskell, W. L., Wood, P. D., Fortmann, S. P., Rogers, T., Blair, S. N., and Paffenbarger, R. S. Physical activity assessment methodology in the Five-City Project. *Amer. J. Epidemiol.* **121**, 91–106 (1985).
31. Staessen, J., Dulpitt, C. J., Fagard, R., Joossens, J. V., Lijnen, P., and Amery, A. Familial aggregation of blood pressure, anthropometric characteristics and urinary excretion of sodium and potassium—A population study in two Belgium towns. *J. Chronic Dis.* **38**, 397–407 (1985).
32. Stern, M. P., Haffner, S. N., Hazuda, H. P., and Rosenthal, M. "Cardiovascular Disease in Mexican-Americans." Presented at the Woodlands Conference on Chronic Diseases among Mexican-Americans, San Antonio, Texas, December 2–6 (1984).
33. Suarez, L., Criqui, M. H., and Barrett-Connor, E. Spouse concordance for systolic and diastolic blood pressure. *Amer. J. Epidemiol.* **118**, 345–351 (1983).
34. Taylor, C. B., Coffey, T., Berra, K., Iaffaldano, R., Casey, K., and Haskell, W. L. Seven-day activity and self-report compared to a direct measure of physical activity. *Amer. J. Epidemiol.* **120**, 818–824 (1984).
35. Tipton, C. M. Exercise training and hypertension, *in* "Exercise and Sport Sciences Reviews" (R. L. Terjung, Ed.), Vol. 12, pp. 245–305. Collamore, Lexington, MA, 1984.
36. Tseng, W. P. Blood pressure and hypertension in an agricultural and fishing population in Taiwan. *Amer. J. Epidemiol.* **86**, 513–525 (1967).
37. Wallace, J., McKenzie, T., and Nader, P. R. Observed vs. recalled exercise behavior: A validation of the Blair seven day exercise recall for boys 11 to 13 years old. *Res. Q. Exercise and Sport* **56**, 161–165 (1985).
38. Winkelstein, W., Kantor, S., Ibrahim, M., and Sackett, D. M. Familial aggregation of blood pressure: A preliminary report. *JAMA* **195**, 848–850 (1966).
39. Wright, B. M., and Dore, C. F. A random zero sphymomanometer. *Lancet* **1**, 337–338 (1970).