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Genetics of Type A Behavior in Two European Countries: Evidence for Sibling Interaction

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Young male twins in The Netherlands and England completed the Jenkins Activity Survey (Dutch and English versions, respectively), a measure of Type A behavior. Separate model fitting analysis revealed a similar pattern of variance estimates and associated goodness of fit across the two countries. The data were then analyzed concurrently, with a scalar parameter included to account for differences in variance due to the disparity of the measurement scales. A model including additive genetic and individual environmental effects gave a good explanation to the data. The heritability estimate was 0.28. Models of social interaction and dominance explained the data even better, the former being preferred. The twins' parents were included in the analysis to examine population variation for Type A behavior intergenerationally. There was evidence for individual environmental experiences having a greater influence on Type A behavior in the older generation.

twins; parents; Type A behavior.

INTRODUCTION

The Type A behavior patterns has long been associated, with varying degrees of equivocation, with the development of coronary heart disease

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(Jenkins *et al.*, 1974; Jenkins, 1978; Matthews, 1988). There are said to be individual differences in "coronary-prone behavior" (Jenkins *et al.*, 1971). However, little research has been carried out to identify the possible etiology of these differences. The results that do exist are hampered by the fact that various methods have been used to measure Type A behavior: the Structured Interview (SI), Framingham Scale, Bortner Scale, and Jenkins Activity Survey (JAS). Cross-study comparisons are therefore not straightforward.

Although the SI is the preferred clinical method of Type A assessment, the JAS produces adequately concordant classification (Jenkins *et al.*, 1971). Its greater ease of administration makes it preferable as a screening tool. In the current context, since the JAS gives a continuum of scores, it should afford an easier means of detecting individual differences in variation than the dichotomous classification system of the SI. Indeed, this has been cited as one reason why, in an earlier study, no significant heritability for Type A behavior, measured by the SI, could be found (Rahe *et al.*, 1978; Matthews *et al.*, 1984).

Studies of parents and their offspring have afforded some indication of familial aggregation (Bortner *et al.*, 1970; Sweda *et al.*, 1986) for Type A behavior. These studies did not allow genetic and environmental influences to be distinguished. A twin study methodology has been used previously to determine the heritability of Type A behavior; however, until fairly recently, only limited forms of analysis, using correlations, have been applied (Matthews and Krantz, 1976; Rahe *et al.*, 1978; Koskenvuo *et al.*, 1981; Matthews *et al.*, 1984; Carmelli *et al.*, 1988; Meininger *et al.*, 1988).

The data can be more powerfully treated using path analysis and maximum-likelihood model fitting techniques. Eysenck and Fulker (1983) used this technique and found there to be a strong genetic contribution to individual differences in components of Type A behavior. Unfortunately, the use of their own scale makes comparison with other measures difficult.

Perhaps the most comprehensive analysis thus far has come from the SATSA (Swedish Adoption/Twin Study of Aging) researchers. They incorporated model fitting techniques to study genetic and environmental contributions to Type A and associated behavioral traits (Pedersen *et al.*, 1989). The SATSA project has the advantage of involving large numbers of twins, which greatly assists the power of analysis. Pedersen and colleagues report a heritability estimate of 27% for the Framingham Type A Scale. A significant genetic influence was also reported for associated Type A traits (pressure, hard driving and ambitious).

In the current study, we had the advantage of data from two coun-

tries, England and The Netherlands. The main aims were to verify the pattern of genetic influence upon Type A behavior and to determine whether there were any substantive differences across the two countries. Although both countries are European, it could be that there are cultural, environmental differences between them which are sufficient to alter population variance for Type A behavior.

Whereas the SATSA study provided information about elderly twins, we focused on young adult twins. By using younger twins, we had the added opportunity of being able to test their parents, to explore the possibility of developmental changes. In addition, the use of young adult twins, rather than children, gave an advantage over previous family studies (Bortner *et al.*, 1970; Matthews and Krantz, 1976; Sweda *et al.*, 1986) in allowing the same measure of Type A behavior, the JAS, to be used across generations. Type A traits have been demonstrated across all age groups. The present study allowed an initial investigation of whether the relative contribution of genes and environment is constant during life.

METHOD

Subjects

The English twins were recruited from the population-based Birmingham Family Study Register, held at the University of Birmingham. There were 63 pairs of monozygotic (MZ) twins and 68 pairs of dizygotic (DZ) twins. They were all males, aged between 16 and 30 years (mean age of MZs = 19.8 years, SD = 3.5 years; mean age of DZs = 19.7 years, SD = 3.8 years). Zygosity was determined by means of visual inspection and a validated questionnaire (Kasriel and Eaves, 1976). All subjects participated in a research project which aimed to determine the genetic and environmental contributions to cardiovascular reactivity and hypertension. The details of the overall psychophysiological testing have been reported elsewhere (Carroll *et al.*, 1985).

The Dutch male twins were drawn from a population of twins and their families who had participated in a similar project on genetic aspects of cardiovascular risk factors at the Free University of Amsterdam. There were 35 MZ and 31 DZ pairs. They were aged between 14 and 21 years (mean age of MZs = 16.6 years, SD = 1.7 years; mean age of DZs = 17.2 years, SD = 1.7 years). Zygosity was determined by blood typing and, in some instances, by DNA fingerprinting (Jeffreys *et al.*, 1985).

Measures

The English twins completed the Jenkins Activity Survey (Jenkins *et al.*, 1979). In addition to providing a score of Type A behavior, scores can be calculated for three subscales, representing particular components of Type A behavior. These are Job Involvement (J), Speed and Impatience (S), and Hard-driving and Competitive (C).

The Dutch twins completed a Dutch translation of the JAS (Appels, 1985). The original English language version consists of 52 items; the Dutch translation has 24 items. It provides a Type A score but has no subscales.

Model Fitting

A series of alternative explanations for the pattern of variation in the observed data was compared with the aid of the computer program LISREL7 (Jöreskog and Sörbom, 1988), which incorporates a maximum-likelihood technique (Heath *et al.*, 1989). The principle of parsimony was adopted to choose an adequate model; a model with the fewest parameters is usually the most parsimonious explanation (Neale *et al.*, 1989). Once a full model fits the data, i.e., the observed data do not depart significantly from those predicted by the model, one can proceed to remove parameters from the model and see if this significantly worsens the fit. A difference chi-square tests the significance of the deleted parameters.

RESULTS

English Sample

The descriptive statistics for each of the Type A scales are given in Table I. The scores correspond to equivalent standardized scores reported for population samples (Bennett *et al.*, 1990; Jenkins *et al.*, 1979; Zyzanski, 1978). Table II shows the correlations for the observed data. The means were similar within twin pairs and across zygosities. For all scales, MZs had greater concordance than DZs, suggestive of the presence of a genetic contribution. Monozygotic twin correlations ranged from 0.56 to 0.34. In the case of the A and S scales, the DZ correlation was almost zero. When the MZ correlation is much larger than the DZ correlation, it may be that there is a nonadditive genetic influence, such as that created by genetic dominance. Another explanation for the large difference in MZ/DZ correlations is a social interaction model, namely, sibling competition (Eaves, 1976) or contrast (Carey, 1986). With this model, there

Table I. Mean Scores (and Standard Deviations) on the Jenkins Activity Survey for Twins and Their Parents

Family member	MZ twin		MZ mother		MZ father		DZ twin		DZ mother		DZ father	
English sample	(n = 63 pairs)		(n = 51)		(n = 46)		(n = 68 pairs)		(n = 57)		(n = 50)	
Scale												
A	200.82	(59.2)	190.06	(62.1)	203.37	(74.4)	206.93	(61.3)	208.74	(70.6)	200.98	(70.6)
J	201.74	(48.7)	150.08	(56.4)	188.20	(60.9)	188.31	(51.3)	166.46	(53.1)	175.50	(46.7)
S	159.16	(56.9)	151.73	(50.5)	152.22	(61.9)	172.26	(61.9)	157.35	(54.2)	165.42	(63.3)
C	114.60	(30.3)	128.31	(28.2)	136.78	(28.7)	118.71	(30.6)	141.11	(27.7)	138.88	(30.5)
Netherlands sample	(n = 35 pairs)		(n = 34)		(n = 31)		(n = 31 pairs)		(n = 31)		(n = 31)	
Scale												
A	10.15	(3.0)	10.41	(4.8)	11.39	(4.5)	10.18	(3.8)	11.32	(4.5)	13.68	(5.0)

Table II. Twin Correlations for the Jenkins Activity Survey

	English sample		Netherlands sample	
Scale	MZ	DZ	MZ	DZ
A	0.34	−0.10	0.39	0.02
J	0.53	0.38		
S	0.42	−0.06		
C	0.56	0.38		

is also a systematic difference between MZ and DZ phenotypic variances. The possibility of sibling competition was supported by the presence of a small negative covariance and larger variance in the DZs; this pattern is not in accordance with genetic dominance (Eaves *et al.*, 1978). A difference in MZ/DZ variance on the A scale of the JAS was also detected by Rahe *et al.* (1978), but its relevance was not tested.

First, univariate analyses were carried out. For the English data, across all four scales (A, J, S, and C), an additive genetic, individual environmental (AE) model best fitted the data. The χ^2 values were 5.21, 4.15, 9.24, and 4.40, respectively, for 4 degrees of freedom ($p > .05$). The associated heritabilities were 0.23, 0.56, 0.32, and 0.57 for A, J, S, and C, respectively.

Because of the large difference in MZ and DZ correlations for the A and S scales, the possibility of dominance and social interaction effects was tested for these scales. In the case of dominance models, the fit did not differ significantly from that of the two-parameter AE model (χ^2 differences of 1.98 and 2.92, respectively; n.s.). In addition, the estimate of additive genetic effects became zero; this effect was also seen by Pedersen *et al.* (1989) for other Type A traits.

The addition of a sibling interaction parameter to the AE model greatly improved its fit. The interaction model was therefore a most adequate alternative explanation for these data. For the A scale: $\chi^2 = 1.03$, df $= 3$, $p = .80$. The interaction parameter was significant and negative (−0.19; SE = 0.08), indicating competition or contrast between twin siblings. Estimates for additive genetic (h) and random environmental (e) factor loadings were 49.30 (SE = 6.74) and 38.72 (SE = 5.96). Similarly, for the S scale, a competition model produced a very good fit to the data ($\chi^2 = 3.42$, df $= 3$, $p = .33$). The interaction parameter was −0.21 (SE = 0.07) and the genetic and environmental factor loadings were 52.40 (SE = 6.74) and 34.65 (SE = 5.01).

Dutch Sample

As will be recalled, there was only an A score for the Dutch JAS. The mean scores for each zygosity are given in Table I. These correspond well to the population norms (Appels, 1985). The correlations are given in Table II. The MZ correlation of 0.39 was very similar to that seen for the English MZ twins on the A scale of the English JAS. The DZ correlation was again very low, at 0.02.

The same series of univariate models was applied to the Dutch data. The AE model gave a χ^2 of 5.52 (df = 4, p = .24). The heritability estimate calculated from this model was 0.37. Use of a dominance model failed to improve the goodness of fit (χ^2 = 4.24, df = 3, p = .24). The additive genetic parameter again became zero.

As in the English data, the data were suggestive of a sibling competition model. The DZ covariance was much less than the MZ covariance. In addition, the MZ variance was less than the DZ variance (the pooled variance difference reached significance at the 10% level). An AE model with a social interaction parameter of −0.22 (SE = 0.10) and genetic and environmental factor loadings of 3.18 (SE = 0.42) and 1.87 (SE = 0.36) had a χ^2 of 2.03 for 3 df (p = .57).

From the separate analyses it was apparent that the Dutch and English data sets followed a similar pattern in terms of genetic and environmental contributions to population variance for Type A behavior. Thus, a four-group univariate analysis was implemented (English and Dutch MZs and DZs, respectively). Because of the different scoring methods for the two questionnaires, there was a difference in the variance across countries, which was accommodated by the use of a scalar parameter. Model fitting results are given in Table III. As suggested by the previous

Table III. The Results of Fitting Genetic and Environmental Models to JAS (Type A) Data: English and Netherlands Sample

Model parameter[a]	χ^2	df	p
E	18.81	10	.043
EC	15.63	9	.075
EA	11.14	9	.266
EAC[b]	11.14	8	.194
EAD	7.97	8	.436
EAI	3.88	8	.868

[a] E, individual environmental variation; C, common environmental variation; A, additive genetic variation; D, dominance genetic variation; I, social interaction variation.
[b] C not identified.

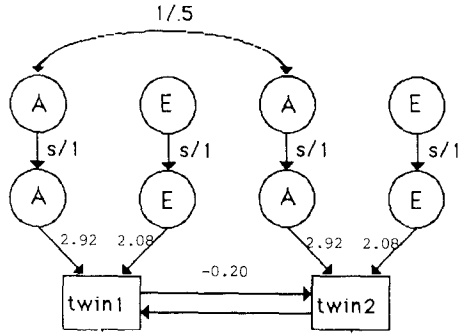

Fig. 1. Path diagram for a sibling interaction model. A and E are the latent additive genetic and unique environmental variables, respectively. Estimates for path coefficients are shown. The negative estimate for the sibling-effect parameter indicates a competition effect. *S* is the scalar parameter to account for the difference in JAS scoring in England and The Netherlands (this was found to be 17.7 for the English sample).

univariate analyses, an AE model provided an adequate fit ($\chi^2 = 11.14$, df $= 9, p = .27$). The estimate for the scalar parameter was 17.7 (SE $= 1.35$). The parameter estimates were 1.80 and 2.92 for h and e, respectively. The heritability estimate was 0.28. The fit of the dominance model was similar to that of the AE model (χ^2 difference $= 3.17$; ns). The EC model was significantly poorer in fit than the AE model.

Finally, scalar models including an estimate of sibling interaction were tested. These provided a significantly better fit to the data than the simpler models. The χ^2 for the AEI model was 3.88 (df $= 8, p = .87$) (Fig. 1). The parameter estimates were 2.92 and 2.08 for h and e, respectively. The social interaction parameter was -0.20 (SE $= 0.06$). Overall, the pattern of correlation seen in the English and Dutch MZs and DZs could be best explained by a social interaction model.

Parent–Offspring Models

The inclusion of parents in the analysis allowed us to compare the genetic and environmental contribution to population variation on the JAS across generations. Parental data were not available for all the twins previously tested. There were 45 MZ and 50 DZ complete English families. The parents were all middle-aged [mean age of MZ mothers, 48.2 years (SD, 6.2); MZ fathers, 50.7 years (SD, 7.1); DZ mothers, 49.5 years (SD, 7.3); DZ fathers, 52.4 years (SD, 7.1)]. There were 31 complete MZ and DZ Dutch families, respectively (mean age of MZ mothers,

44.5 years (SD, 6.0); MZ fathers, 46.2 years (SD, 6.7); DZ mothers, 46.3 years (SD, 5.4); DZ fathers, 49.3 years (SD, 5.8). The parent–offspring model is represented in Fig. 2.

English Sample. The parent–offspring correlations tended to be small and nonsignificant (Table IV). Hence there was little evidence of familial aggregation, of either a genetic or an environmental nature. A lack of assortative mating was suggested by the low spousal correlations. In the DZ group, the twin correlation was lower than the parent–offspring correlation. This weakens the evidence for the existence of dominance effects.

Biometrical analyses confirmed the correlational findings. Although the full (AEC) model fitted the data ($\chi^2 = 22.19$, df $= 17$, $p = .18$), closer examination showed large standard errors for the additive genetic and common environmental parameter estimates.

These particular data were best explained by a model where all variation was attributed to individual environmental (E) effects ($\chi^2 = 23.31$, df $= 19$, $p = .22$). Furthermore, a model where parent and offspring loadings were allowed to take different values for the environmental variance gave a significantly better fit than a model where variance in the two generations was assumed to be equal (χ^2 difference $= 5.27$, df $= 1$, $p < .05$).

Dutch Sample. The parent–offspring correlations in the Dutch sam-

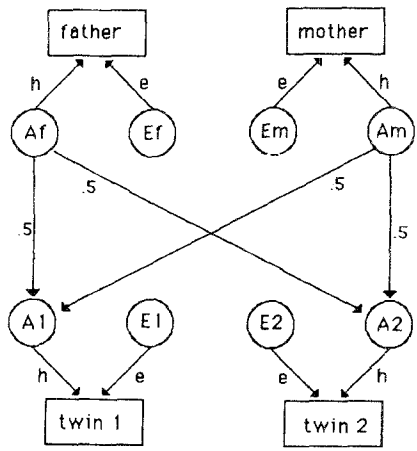

Fig. 2. Path diagram for parent–offspring model. A and E are the latent additive genetic and unique environmental variables respectively. Subscripts *m* and *f* refer to mother and father. To simplify the diagram, only one family group is represented and the scalar parameter is omitted.

Table IV. Familial Correlations for Type A Behavior

Relationship	English MZ twins (n = 45 families)		English DZ twins (n = 50 families)		Dutch MZ twins (n = 31 families)		Dutch DZ twins (n = 31 families)	
Mother with Twin 1, 2	0.009,	−0.119	−0.036,	0.342	0.12,	0.14	0.29,	−0.03
Father with Twin 1, 2	0.003,	−0.074	−0.058,	−0.055	0.30,	0.32	−0.03,	0.34
Mother/father	0.112		0.235		0.02		−0.07	

ple were somewhat larger than in the English sample. The father–twin correlations were significant ($p < .05$) in the MZ sample but were not consistently so in the DZ sample. Spousal correlations were almost zero. As in the English sample, parent–offspring correlations tended to be greater than the DZ correlation, reducing the possibility of a dominance effect.

The AE model gave a parsimonious solution to the data ($\chi^2 = 23.20$, df = 18, $p = .18$). The heritability estimate calculated from this model was 0.47. The alternative, purely environmental models did not fit the data. The dominance (AED) model also gave an adequate fit ($\chi^2 = 21.85$, df = 17, $p = .19$). An AE model with different individual environmental variance for twins and parents fitted the data significantly better ($\chi^2 = 12.66$, df = 17, $p = .76$). The environmental variation was greater in the parental generation ($e = 2.62$ in offspring and 4.05 in parents). The heritability estimate was reduced from 0.46 in the twins to 0.26 in the parents.

Finally, a series of models was fitted jointly to the four groups. A scalar parameter was again included to account for the difference in variance arising from the two scoring systems. The results are shown in Table V. Maintaining equivalent variance in the two generations, the significance levels were rather low. Both two-parameter models were not significantly poorer than the three parameter models, although only the AE model reached the 5% significance level ($\chi^2 = 52.15$, df = 37, $p = .05$). The scalar parameter was approximately 16. The heritability estimate was calculated to be 0.19. This is smaller than the heritability estimate using twin data alone.

Table V. The Results of Fitting Genetic and Environmental Models to JAS (Type A) Data: English and Dutch families

Model parameter[a]	χ^2	df	p
E	58.58	38	.02
EC	53.00	37	.04
EA	52.15	37	.05
EAC	51.96	36	.04
EAD	49.34	36	.07
EAI	51.89	36	.04
E_TE_P	43.52	37	.21
E_TE_PA	36.18	36	.46
E_TE_PAI	35.25	35	.46
E_TE_PC	37.75	36	.39

[a] E, individual environmental variation; C, common environmental variation; A, additive genetic variation; D, dominance genetic variation; I, social interaction variation; subscript T, twins; subscript P, parents.

A plausible alternative focuses upon the changing impact of environmental influences upon the expression of Type A behavior as we age. The separate models indicated that there was a different degree of variance for individual environmental influences in parents and offspring; specifically, the parental portion of the population had larger variances. A feasible hypothesis is that the genetic contribution to Type A behavior remains constant across generations and that individual environmental factors have an increasing impact upon its expression. The model for this hypothesis was wholly adequate for the data ($\chi^2 = 36.18$, df = 36, $p = .46$). This model was significantly better than the simple AE model. The heritability esimate decreased from 0.21 for the twin offspring to 0.13 for their parents, owing to the greater environmental variation in the parents. The alternative, purely environmental model (E_T E_P C) was also adequate ($\chi^2 = 37.75$, df = 36, $p = .39$).

The inclusion of a reciprocal social interaction parameter for twins in the AE model gave a nonfitting model ($\chi^2 = 51.89$, df = 36, $p = .04$). The coefficient for social interaction was small and not significant. Including both sibling interaction and different environmental variances for the two generations into the same model gave a fitting model, but here also the interaction parameter was not significant.

DISCUSSION

A genetic contribution to individual differences in Type A behavior was demonstrated here using the JAS. This heritable influence was observed in data from two countries and across two generations. The degree of heritability was similar to that reported by Pedersen *et al.* (1989) using the Framingham A scale.

In the analysis of the English twin data, where it was possible to analyze subscales of the JAS, the highest degree of heritability was found for C, the Hard-driving/Competitive scale. This is the only scale which has been reliably related to an increased risk for coronary heart disease in retrospective and prospective studies (Jenkins *et al.*, 1974; Rosenman *et al.*, 1976). Matthews and Krantz (1976) also report a genetic component for this scale, albeit based only upon a MZ/DZ correlational difference. This scale was the only scale that showed a significant parent–offspring correlation in their study (mother–son $r = .36$). In contrast, Rahe *et al.* (1978) found a significant heritability only for the Speed-Impatience Scale. They noted a significant Holzinger's heritability for the A scale also, but in correcting it for the heterogeneity of MZ/DZ variance differences, the result was no longer significant.

Concurrent testing of the data sets from the two countries lent greater

power to the social interaction model. The present results are one of the few examples (Eaves *et al.*, 1978) of a good-fitting interaction model. The results indicate that social interaction between twins may explain the large difference in MZ/DZ correlations. It was noted earlier that Rahe *et al.* (1978) also found a difference in MZ/DZ variance for the A scale of the JAS. A reanalysis of their reported mean squares was carried out. The social interaction model proved to be the best explanation for their data also ($\chi^2 = 1.43$, df $= 1, p = .23$).

Eaves' (1976) definition of competition is where an increasing allele produces a decreasing environmental effect on a sibling. (Put more simply, the genes that increase trait expression in one twin concurrently generate an environment that reduces trait expression in the cotwin.) This approach emphasizes the genetic influence of one twin upon their cotwins' environment. There is also the possibility that environmental interplay occurs. For this to be tested successfully, a family density design is required, to distinguish general environmental effects from those which are "sibling" effects. Sibling effects can represent a form of gene–environment interaction, where the expression of environmental differences is genetically controlled. Gene–environment covariance occurs if the same genes influence the trait in one twin while indirectly influencing the environment of the cotwin. However, without using singletons, it is not possible to determine whether the genes responsible for sibling interaction differ from those directly responsible for the trait, the two effects being confounded in twins alone. Applying Eaves' model for competition (1976), a significant difference between MZ and DZ variance would be predicted. However, Eaves *et al.* (1989) have stated that this test is a rather weak one: "the differences are not expected to be very great, even in the presence of competition." Therefore the current finding remains a feasible explanation of the observed variation for Type A behavior. Normally, when only twins are available, extremely large samples are required to resolve the effects of dominance and competition. The findings here will therefore benefit from validation using a larger sample, including singletons. In this design, twins would expect to have larger variances than singletons if there was competition, since the cotwin affords a supplementary source of environmental variance and genotype–environment covariance, absent in singletons (Eaves, 1976).

Interestingly, a social interaction model has also been suggested for extraversion (Eaves *et al.*, 1989). In their paper, Eysenck and Fulker (1983) discussed the possibility that Type A behavior may be subsumed under the more well established extraversion–neuroticism personality domain. Subsequent research has related neuroticism and, less frequently, extraversion to Type A scores (e.g., Langeluddecke and Tennant, 1986;

Llorente, 1986). Nevertheless, May and Kline (1987) have recently argued that the Type A behavior pattern is only partially related to extraversion and neuroticism, and cannot be totally accounted for by these factors. It could be useful in future studies to examine the genetic and environmental covariance of Type A behavior and extraversion–neuroticism characteristics.

Population variation for Type A behavior was similarly explained in both The Netherlands and England. A scalar parameter satisfactorily accommodated the differences in variance. Without using identical questionnaires, it is not possible to determine whether the English population are inherently more variable in their expression of Type A behavior.

Although Type A behavior has a genetic component, the results show that much of the population variation can be explained in terms of individual environmental experience. Formerly, it was thought that Type A behavior was consequent upon the stressors of adulthood. While studies incorporating children have shown this not to be the case (Meininger *et al.*, 1988; Matthews and Angulo, 1980), the current results indicate that the environment plays a more important role in determining the expression of Type A as we get older.

Given that the contribution of individual environmental experiences to population variation in Type A behavior is substantial, it may be possible to allay coronary heart disease via behavioral modification therapy. Rahe and his colleagues (1978) mistakenly suggested that if the behavioral trait were largely heritable, it would be difficult to alter. However, if the trait were established relatively early, as seems to be the case (Meininger *et al.*, 1988), it may prove not so straightforward to modify. The acquisition of more detailed information concerning genetic and environmental interaction may help to determine which components of Type A behavior prove most amenable to intervention.

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