

Genetic determinants of individual differences in coping styles

BEATA KOZAK¹, JAN STRELAU², & JEREMY N.V. MILES³

¹*Silesian University, Poland*, ²*Warsaw School of Advanced Social Psychology, Poland*, ³*University of York, UK*

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Abstract

Genetic analyses of data from 612 adult Polish twin pairs demonstrated reasonable genetic contribution to variation in scores of Task-Oriented, Emotion-Oriented, Social Diversion and Distraction scales on Endler and Parker's Coping Inventory for Stressful Situations (CISS). Structural equation models were fitted to estimate heritability and showed the range to which individual differences in coping are influenced by genetic and environmental factors. The model was extended, using a Cholesky (triangular) decomposition, to examine genetic and environmental covariance. The environmental correlations were generally low (maximum $r = .24$), whereas some of the genetic correlations were considerably higher (maximum $r = .52$).

Keywords: *Coping style, stress, heritability, behavioural genetics*

The list of controversies concerning the concept of coping is almost as large as the collection of publications devoted to different aspects of this phenomenon. Undoubtedly, more than 20 years of research investigating coping has significantly contributed to our understanding of the successes and failures people have experienced when dealing with stressful situations. Yet, in spite of the commonly accepted belief that coping represents an interplay between environmental and personality factors, there are still many questions remaining regarding the exact nature of coping and the role of its biological, psychological, and environmental determinants (e.g., Endler & Parker, 1990; Heszten-Niejodek, 1996; Lazarus, 1999).

Many studies have attempted to specify whether coping should be thought of as a psychological and social process (e.g., Folkman, Lazarus, Dunkel-Schetter, DeLongis, & Gruen, 1986; Lazarus, 1993) or as a psychological trait (e.g., Bolger, 1990; Carver, Scheier, & Weintraub, 1989; Endler & Parker, 1994; Krohne, 1990; Lazarus, 1993). However, at the same time, many scholars reviewing empirical literature on responses to stressful life events have emphasized that both situation-based and person-based approaches to defining coping complement, rather than compete with, one another (e.g., Aldwin, 1994; Costa, Somerfield & McCrae, 1996; Hobfoll, 1998; Krohne, 1996).

This split over the fundamental essence of coping echoes many illuminating discussions concerning the “nature *versus* nurture” problem. Nevertheless, most researchers (e.g.,

Correspondence: Beata Kozak, Instytut Psychologii, ul. Grażyńskiego 53, 40-126 Katowice, Poland. E-mail: beata_kozak@katowice.home.pl

Kimble, 1993; Plomin & Colledge, 2001) agree that the phrase coined by Galton would more accurately reflect the findings of contemporary behavioural genetics if it read instead as “nature *and* nurture”. Given that the purpose of the present study was to investigate genetic determinants of individual differences in coping styles, we assumed that the trait-oriented approach would be most useful.

Notwithstanding the large number of studies of the genetics of personality (e.g., Borkenau, Riemann, Angleitner, & Spinath, 2001; Goldsmith, 1989; Heath, Neale, Kessler, Eaves, & Kendler, 1992; Loehlin, 1992; Oniszczenko, 1997; Saudino & Plomin, 1996; Strelau, 1998, 2000), it is somewhat surprising that only a few studies have paid close attention to the role of genetic factors in predicting coping behaviour.

Kendler and others (1991) studied female twins and identified three coping styles in their study: turning to others, problem solving, and denial. Obtained heritability estimates were about 30% for turning to others and problem solving, but no genetic effect was found for the denial. These results agree with those of the Mellins, Gatz, and Baker (1996) study where seven coping scales were examined in children aged from 9 to 16 years in 44 monozygotic (MZ) and 30 dizygotic (DZ) twin pairs. Five of seven scales were influenced by genetic factors. Following heritability estimates were found: distraction (.99), use of parents (.55), use of peers (.18), self-soothe (.53), problem focused (.57). For the remaining two scales, problem solving and emotion-focused coping, no genetic effects were found. Busjahn and others (1999) also showed that all of the examined coping styles were influenced by genetic factors. They examined 19 coping styles (and four secondary coping factors) in 117 MZ twin pairs and 95 DZ twin pairs.

Thus, the results of aforementioned examinations showed that genes, as well as environment, influence coping. In other words, although research in the behavioural genetics paradigm focuses on the influence of genetic factors, such studies also showed the role of environmental factors in explaining the variation within traits. Investigations into genotype-environment correlations support a current shift from using passive models describing how different environments affect individuals toward theoretical models emphasizing the active role of human beings in creating, modifying, and selecting their own environments in a process of adaptation.

The concept of heritability

When investigating individual differences, researchers are interested in latent variables, which are defined as variables that cannot be measured directly (see Bollen, 2002, for a fuller discussion and definition of latent variables). In behaviour genetics we are studying the ultimate latent variables—the genes and environment that have led to the current level of a trait for an individual. Yet, we can only assess behaviour—the phenotypic traits. Thus only observable, visible behaviour affords an opportunity to make inferences about latent traits. In cases where the heritability is relatively uncomplicated, such as in Huntington’s disease, this is relatively simple to work out. Huntington’s disease is caused by a single gene abnormality (on chromosome 4). If a person has the abnormal gene, she will get the disease, and there is a 50% chance that she will pass it on to each of her children (Bates, Harper, & Jones, 2002). The genotype is the presence of the form of the gene. In case of Huntington’s disease there is a one to one relationship between the genotype and the phenotype. If a person has the genotype, she will develop the condition.

Most cases of heritability are more complex than the case of Huntington’s disease, for three reasons. First, there are multiple genes, which affect the phenotype. Consider the case

of height. There are not just two conditions, tall and short. There are an (almost) infinite number of possible heights. If a person has tall parents, the probability that that person will be tall is increased, but we do not know exactly how tall this person would be. Second, there are environmental effects. Alterations in a person's diet will affect their adult height. If a person has a poor diet when young, it is likely that he will not grow as tall. Third, in many psychological variables there is the presence of measurement error. A person either has Huntington's disease or does not; theoretically there should not be a measurement error. Similarly, we can measure a person's height, with a substantial degree of accuracy and reliability. However, if we wish to measure coping styles we must rely on measures that are imperfect. Their reliability, whereas acceptable from a psychological standpoint, is considerably less than the reliability in measurement of height or in a diagnosis of Huntington's disease.

Instead of talking about the presence of a genotype, which causes a phenotype, behavioural geneticists talk about the proportions of variance. We know the total variance of the phenotype (this is the variance of our measure). We would like to know the source of the variance in this phenotype. In order to understand the concept of heritability, it is useful to introduce the sources of phenotypic variance, which are graphically depicted in Figure 1.

The total phenotypic variance in any population—measured differences between people (depicted as V_p in Figure 1)—comes from two sources: genetic variance (V_g ; differences in genes) and environmental variance (V_e ; differences in environment, e.g., diet). The genetic variance and environmental variance are possibly correlated.

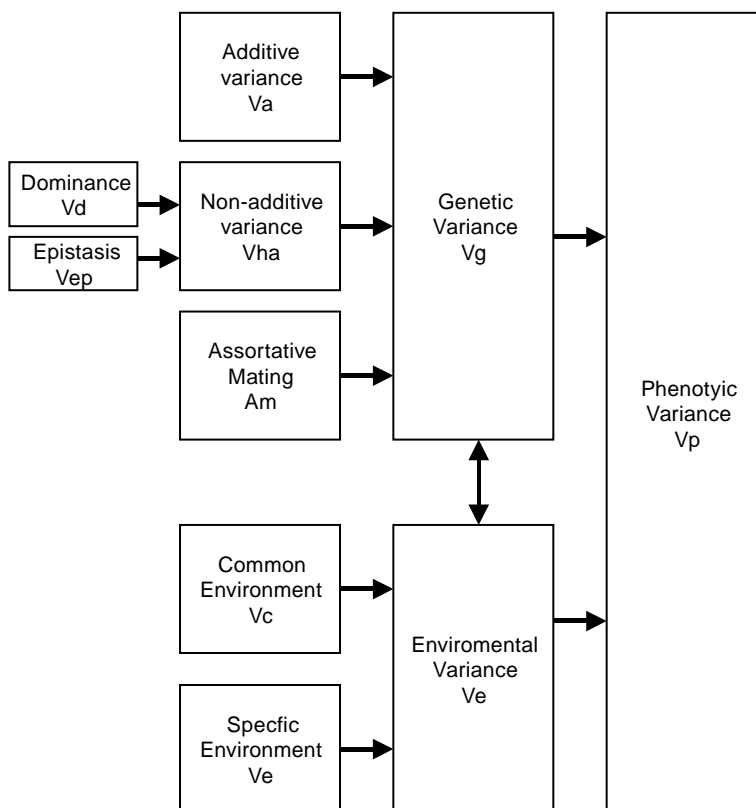


Figure 1. Sources of phenotypic variance.

of anxious coping, for example, may pass that characteristic onto their children, genetically. However, they may also raise their children differently than other parents, thus creating a different environment.

Genetic variance includes additive and non-additive effects. Additive genetic effect (V_a) refers to that part of the genetic variation that is transmissible across generations and causes that offspring resemble their parents. Non-additive genetic effect (V_{na}) is not transmissible across generations and depends on the particular configurations of genes that are present: dominance (V_d) on the combination of genes present at a given chromosomal locus, and epistasis (V_{ep}) on configurations across loci (see: Loehlin, 1992; Plomin, DeFries, & McClearn, 1980).

Likewise, we can also divide the environment into different sources of variance. The common environment (V_c), also called shared or within-family environment, embraces all the environmental influences, which differentiate the given family from other families in the population. Aspects of the common environment include physical features such as the type of house the family lives in and the geographical location. The common environment factor also includes aspects related to the psychological or sociological environment that is shared by the family, e.g., the socio-economic status of the family, parents' attitudes to the children, or family traditions.

Specific environment (V_e), called also non-shared, unique, or between family environment, consists of all these factors which are responsible for differences among members of the same family. Relationships at school or at work, different ways of spending leisure time, daily life hassles, diseases, the birth order, can be examples of specific environmental factors because they cause differences among the members of the same family. One of the most important aspects of the unique environment is, of course, a presence of a sibling.

Elements of the common environment as well as specific environment can be prolonged and systematic or transient and incidental. Aspects of the common environment that affect different people in different ways are also included in the unique environment. A particular experience that is shared by siblings may have profoundly different effects on them, either as a result of other aspects of their environment or their genetic make up. For example, being brought up by a violent father may have different effects on two siblings. Unique environmental effects also include environment-environment interactions, and gene-environment interactions.

Assortative mating (am) assumes that spouses are not selecting each other with respect to the trait of interest. If mates are not matched randomly, non-additive genetic factors might be underestimated while input of the common environment may be overestimated.

Heritability (h^2) is a statistic that describes the ratio of genetic to phenotypic variance: the proportion of observed variance in a population that can be explained by genetic variance. In other words, heritability describes the extent to which genetic differences among individuals in a population make differences phenotypically.

Heritability can vary from population to population and from time to time. If the population changes, we can expect its parameters to change accordingly. During periods of strife, for example wars or serious economic depressions, it is likely that the characteristics of events people experience in their environments will vary greatly. Differences between rich and poor or urban and rural settings may be greater, and therefore, the proportion of variance attributable to the environment may increase. During more comfortable times, people's environments would be more similar, and thus the phenotypic variance due to environment may decrease. (Because we are always talking about proportions of variance, if

the environmental variance reduces, the proportion of variance due to genes will increase, even if the amount of variance due to genes remains constant.)

The term heritability can be conceptualized as having two facets: a broad one (h^2B) and a narrow one (h^2N). Broad sense heritability refers to the total proportion of the variation of the trait that is due to genes, and the narrow sense heritability, refers to that part of the genetic variation that is transmissible across generations. Both facets were a subject of many investigations and are widely described in the literature (Neale & Cardon, 1992; Oniszczenko, 1997; Plomin et al., 1980).

The present study

We used the classification of coping proposed by Endler and Parker (1990). These authors distinguished among emotion-oriented coping, task-oriented coping, and avoidance coping. The last style is considered to form two dimensions: avoidance by distraction coping and avoidance by social diversion coping. Consistently with the behaviour genetic approach, we conceptualized coping in terms of a style (rather than strategy or process). The bulk of prior research on the genetic causation of coping has found large (>50%) effects of unique environmental variance, moderate (19%–31%) additive genetic variance (e.g., Kendler et al., 1991), quite large non-additive genetic variance (23%–52%, Busjan et al., 1999), and small, or no, effects of common environment. These results from coping studies are consistent with results of research on other individual differences (e.g., Borkenau et al., 2001; Loehlin, 1992; Oniszczenko, 1997; Strelau, 1998) indicating that individual differences are caused by genetic as well as environmental factors.

Our study aimed to replicate and extend the findings of previous examinations of the heritability of coping using a twin-study design. MZ (monozygotic) twins share 100% of their genes, and 100% of their common environment. DZ (dizygotic) twins share 50% of their genes, on average, and 100% of their common environment. MZ twin pairs and DZ twin pairs were compared in terms of their similarity in coping styles. Comparison of the correlations between MZ and DZ twins enabled us to attribute proportions of variance to genetic causes, to unique environmental causes and to common environmental causes.

Method

Design of analyses

The analysis of broad sense heritability was based on the same-sex, monozygotic (MZ) and dizygotic (DZ) twins reared together method (Neale & Cardon, 1992), which is illustrated in Figure 2. (We used only same sex DZ twins to ensure equivalency with the MZ twins. MZ twins are obviously same sex, and DZ twins of different sex may share less environment than DZ twins of the same sex, thus producing an overestimation of the heritability.)

MZ twins are genetically identical so the additive genetic factor is equal for them, while DZ twins share on average 50% of their genes. This implies that expected resemblance of MZ twins should be twice that of DZ twins if a trait is wholly genetically determined. The dominant genetic factor is fully shared by MZ twins and only in 25% by DZ twins. The common environment factor affects MZ and DZ twins the same way, hence it increases within pair resemblance and between pair differences. Specific environment causes dissimilarity between twins and it also contains a measurement error.

The data were corrected for age and sex before the statistical analyses were conducted. This correction was necessary because twins are unavoidably the same age and we used only

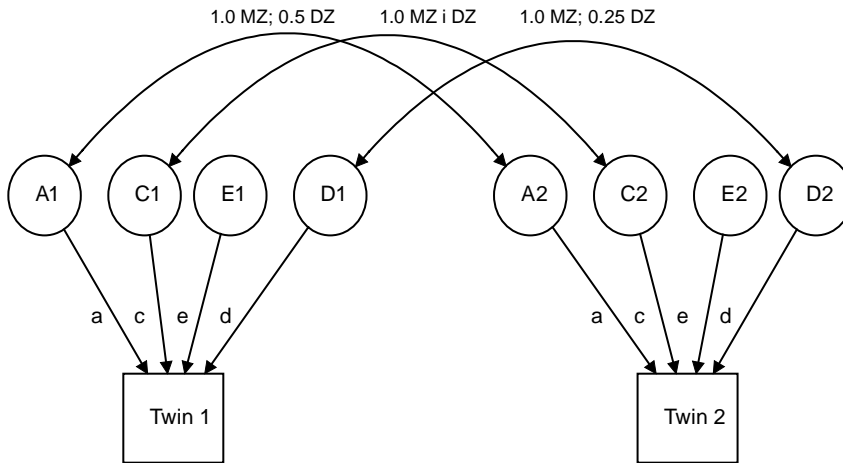


Figure 2. Basic path model used for univariate analysis for monozygotic MZ and dizygotic DZ twins reared together. Rectangles represent measurable phenotypic variables—here coping styles. Circles represent latent variables: A=additive genetic factor, C=common environment factor, E=specific environment factor, D=genetic dominance factor. 1 and 2 are designed for first and second twin; a, c, e, d=paths of loading of given factors for observed variables.

twins of the same gender. If a trait is related to either age or sex, and the data are not corrected, the correlation between twins would appear to be higher, and the heritability coefficients would be biased and high. Raw scores were therefore corrected by regressing them onto age, age² (to correct for a non-linear effect of age), sex and age x sex interaction (to correct for a non-linear effect of sex).

Participants and research procedure

The sample was randomly selected from the Polish National Twin Registry. The set of questionnaires was sent via mail to 3000 pairs of twins of unknown zygosity. The response rate was about 27%, 798 pairs of twins returned completed questionnaires. The low level of response can be explained by several factors. Often only one of a pair of twins responded, which meant that we could not make use of these data because we needed responses from both twins to estimate heritability. Unknown quantity of questionnaires failed to reach their intended destination because people moved, died, married and changed maid’s names. After excluding the twins with different genders, our final sample was composed of 612 same gender pairs. Table I illustrates the characteristics of the sample.

Table I. Sample characteristics.

Participants	N	Female	Male	Age	
MZ	324	196	128	34.26	(10.93)
DZ	288	165	123	35.48	(11.62)
Together	612	361	251	34.83	(11.27)

Note: MZ: monozygotic twins, DZ: dizygotic twins, N: number of pairs, Age in years and standard deviation in parenthesis.

Measures

Zygosity was determined by means of discriminative analysis of data obtained by The Physical Similarity of Twins Questionnaire (PSTQ), an instrument developed by Oniszczenko, Strelau and Angleitner (see, Oniszczenko & Rogucka, 1996). The first part of the questionnaire concentrated on general information about the twins, such as years of living together, their gender and age. The second part solicited information concerning physical similarity between twins: e.g., natural colour of hair, the shape of outer ear. Questions about whether twins have been mistaken by the family members and acquaintances were also included. Prior discriminant analyses showed that these three indicators correctly estimate zygosity in 100% of male pairs and in 95% of females (Oniszczenko, Bodunow, & Rogucka, 1995).

Coping styles were measured by Polish adaptation of Coping Inventory for Stressful Situations (CISS, Endler & Parker, 1994). Four coping styles were assessed: task-oriented, emotion-oriented, and avoidance which consists of social diversion and distraction. The Polish adaptation of CISS had been shown to have good psychometric properties (Szczepaniak, Strelau, & Wrzesniewski, 1996). In the present sample, reliability coefficients for four studied coping scales ranged from .75 (for distraction) to .91 (emotion-oriented coping).

Results

Table II and Table III show the correlation matrices between the variables for the MZ twins and the DZ twins for the age and sex corrected data. Three sets of correlations can be discerned in these tables. First, on the top left and bottom right are the mono-twin hetero-trait correlations, where the correlations are of the same twins on different measures. In the bottom left block are the hetero-twin mono-trait measures (shown in bold) that show the correlations for the different twins on the same measures. Finally, there are the hetero-trait hetero-twin correlations, thus correlations for different twins on different measures.

It is interesting to note the differences in magnitude of the hetero-twin mono-trait correlations in the MZ and DZ twins. The MZ twin correlations tend to be higher (in the region of twice as high) than the DZ correlations. If there was only additive genetic variance, and no common environmental variance, the correlations for MZ twins should have been twice the correlations for MZ twins. If there was no additive genetic variance, and

Table II. Coping styles: Correlation matrix for MZ twins.

Twin 1	Emotion Oriented	1							
	Task Oriented	.099	1						
	Social diversion	-.151	.291	1					
	Distraction	.152	-.090	.318	1				
Twin 2	Emotion Oriented	.328	-.019	-.150	-.013	1			
	Task Oriented	.014	.352	.094	-.113	.034	1		
	Social diversion	-.017	.138	.327	.249	-.048	.276	1	
	Distraction	.004	-.077	.136	.388	.110	-.180	.237	1
		Emotion Oriented	Task Oriented	Social Oriented	Distraction	Emotion Oriented	Task Oriented	Social Oriented	Distraction
		Twin 1				Twin 2			

Table III. Coping styles: Correlation matrix for DZ twins.

Twin 1	Emotion Oriented	1							
	Task Oriented	-.003	1						
	Social diversion	-.007	.275	1					
	Distraction	.155	-.142	.274	1				
Twin 2	Emotion Oriented	.208	-.063	-.020	.050	1			
	Task Oriented	.023	.157	.093	-.035	-.009	1		
	Social diversion	-.005	.137	.177	.089	-.031	.310	1	
	Distraction	.022	-.002	.011	.103	.274	-.218	.283	1
		Emotion Oriented	Task Oriented	Social Oriented	Distraction	Emotion Oriented	Task Oriented	Social Oriented	Distraction
		Twin 1				Twin 2			

only common environmental variance, the correlations for MZ twins and DZ twins should have been equal.¹

Five alternative models were tested for each coping style.

1. The additive genetic model (AE), which assumed that, twins' similarity would be determined by an additive genetic (A) effect and a unique environmental effect (E).
2. Dominant genetic model (DE), which assumed that, twins' similarity would be caused by a dominance (D) effect and a unique environmental effect (E).
3. The full genetic model (ADE), which assumed both genetic (additive—A and dominant—D) effects as well as a unique environmental effect (E) on twins' similarity.
4. Full environmental model (ACE) assumed that similarity between twins would result from both genetic (additive—A), unique environmental (E) and common environmental (C) effects.
5. The environment only model, with common environmental and unique environmental effects only (CE).

All models were estimated using statistical modelling software Mx 1.3.61 (Neal, Boker, Xie, & Maes, 2004). Models were fitted using maximum-likelihood estimation. Because values of χ^2 depend on a sample size, other fit statistics were also used: Akaike's Information Criterion (AIC; see Bozdogan, 2000) and the Root Mean Square Error of Approximation RMSEA (Steiger & Lind, 1980, see also Browne & Cudeck, 1993; Neale & Cardon, 1992).

The first method used to assess the fit of a model is the value of χ^2 and its associated probability. A significant value of χ^2 is indicative of difference between the data and the model and suggests poor fit. If one model can be derived from another by freeing (adding) a parameter, the models are described as nested, for example the AE model and the ACE models are nested because the ACE model is simply the AE model, with the C parameter added to it. The ACE and the ADE models are not nested, because we would need to remove the C parameter and add the D parameter to convert one to the other. If models are nested, we can test the significance of the difference between the models. Adding a parameter to a model will always cause χ^2 to drop, that is to improve the fit. The question is, has it improved significantly? We can find out by finding the difference in the model χ^2 statistics. This is distributed as χ^2 , with df equal to the difference in df in the two models. If this result is non-significant, then adding the parameter does not significantly improve the

model (indeed, it worsens it, because it reduces its parsimony). If the drop is significant, then the model is significantly improved.

The RMSEA was also used to assess the fit of each model. A value of RMSEA of less than .05 is considered to show good model fit. Where models are not nested the AIC can be used to compare the fit of two models. The AIC is based on χ^2 , but penalizes models, which are more complex. Lower values of AIC are indicative of better fit. Results of these analyses are presented in Table IV. The best fitting models are indicated with the use of bold text.

For emotion-oriented coping style, the AE model has the lowest RMSEA and AIC – RMSEA was low enough to indicate good fit. In addition, the value of χ^2 was not significant, indicating good fit. However, the other models also showed good fit according to these criteria. It can be seen that both the RMSEA and the AIC were lower in the AE model than in the other models. Because the AE model was nested within both the ACE and ADE models, we could test whether the ACE or the ADE models were significantly better than the AE model. First, for the C parameter, the difference in the values of χ^2 between the AE and the ACE models was .29 (3.58–3.29), which was not significant. Therefore, adding the C parameter to the AE model did not lead to a significant improvement. Using a similar procedure for the D parameter, the χ^2 difference was zero.

Table IV. Results of data fitting to univariate MZ and DZ twins, same-sex, reared together models in coping styles.

Model	e ²	a ²	c ²	d ²	df	χ^2	p	RMSEA	AIC
Emotion-oriented coping style									
ACE	.67	.27	.06		3	3.29	.35	.013	–2.71
ADE	.65	.35		.00	3	3.58	.31	.018	–2.42
AE	.65	.35			4	3.58	.46	.000	–4.42
CE	.73		.27		4	6.72	.15	.034	–1.28
DE	.65			.35	4	7.27	.06	.037	–.73
Task-oriented coping style									
ACE	.66	.34	.00		3	2.61	.45	.000	–3.39
ADE	.66		.29	.05	3	2.58	.46	.000	–3.42
AE	.66	.34			4	2.61	.62	.000	–5.39
CE	.73		.27		4	8.42	.07	.043	.42
DE	.65			.35	4	3.95	.41	.000	–4.05
Distraction									
ACE	.67	.32	.01		3	5.34	.15	.036	–.66
ADE	.67	.33		.00	3	5.36	.15	.036	–.64
AE	.67	.33			4	5.36	.25	.024	–2.64
CE	.75		.25		4	9.83	.04	.049	1.83
DE	.65			.35	4	7.69	.10	.039	–.31
Social diversion									
ACE	.64	.36	.00		3	7.35	.06	.049	1.35
ADE	.61	.02		.37	3	5.09	.16	.034	–.91
AE	.64	.36			4	7.35	.12	.037	–.65
CE	.74		.26		4	20.25	<.01	.08	12.25
DE	.61			.39	4	5.10	.27	.021	–2.90

e²=variance attributed to non-shared environment, a²=variance attributed to additive genetic factor, c²=variance attributed to common environment, d²=variance attributed to non-additive genetic factor, df=degree of freedom, χ^2 =value of statistic, p=significance of χ^2 , AIC=Akaike's Informational Criterion. Model the best fitted to data bold.

ACE: full environment and additive genetic model, ADE-full genetic and specific environment model, AE-reduced genetic and environment model, CE-reduced environment model, DE-reduced non-additive genetic and specific environment model.

Hence, again there was no significant improvement in the model by adding the D parameter.

For the task oriented coping style, the AE model had the lowest RMSEA and AIC, and a non-significant χ^2 . Carrying out the same procedure as for emotion oriented coping, we found that the ADE and ACE models did not offer any statistically significant improvement (for the ADE, $p = 1.00$, and for the ACE, $p = .86$).

For distraction coping style, again, the best fitting model was the AE model, with the lowest RMSEA and AIC. The differences between this model and the ACE and ADE were not statistically significant ($p = .85$ and 0.95 , respectively).

For the social diversion coping style, the DE model (which has dominance effects, but no additive genetic effect) was the best fitting model. It had the lowest value of AIC and RMSEA, and the ADE model was not a significant improvement over it ($p = .92$).

These results indicated that genetic factors contributed a considerable proportion of the phenotypic variance in coping styles. In our models, genetic variance accounted for 33% to 39% of the variance and unique environmental effects and measurement error accounted for the remainder. These findings showed that heritability of all coping styles measured by CISS did not vary a lot, and all styles were inherited to a similar extent. The additive genetic model (AE) accounted best for the variance of all coping styles excluding social diversion, for which the dominant genetic model (DE) offered the best fit. This finding suggested that individual differences concerning this coping style might be caused by different factors than the factors causing differences in other styles. It is also interesting to note that the coefficient of heritability for social diversion coping style was the highest (.39) of all obtained coefficients.

A genetic source of individual differences in coping was also indirectly confirmed by rejecting the reduced environmental model (CE). Based on these findings, a general conclusion might be proposed. Our analyses indicated a genetic effect and confirmed expectation that genes contribute to individual differences in coping.

A second purpose of the study was to examine correlations between the genetic and the environmental components of each measure. Generally the measures of coping are correlated, which implies a common causation. That is, something similar is affecting these constructs. Therefore we examined the extent to which such a correlation was due to genetic factors, as well as the extent to which it was due to environmental factors.

Consider a simple example. Children with higher levels of reading skill (ability) are likely to read a lot of books, which may cause them to become nearsighted. Therefore, there is a correlation between nearsightedness and a volume of book reading. This is a phenotypic correlation. The reason that this occurs is because of an environmental correlation. Children who read more books may become more nearsighted. This is not a genetic correlation because there is no genetic tendency for nearsightedness, which if it were, would also lead to book reading. On the other hand, haemophilia is an inherited condition. It is due to a defective gene, which is carried on the x-chromosome. Women have two copies of the x-chromosome and thus are very unlikely to inherit the defective copy of the gene from both parents. Hence haemophilia is very rare in women. On the other hand, men have only 1 x-chromosome. Thus if they inherit a defective gene from their parents, they will suffer from this condition (approximately 1 in 10,000 males has haemophilia). Males also tend to be taller than females, and consequently there is a correlation between haemophilia and height. However, this correlation is entirely genetic—males inherit genes that make them taller, and males inherit genes that make the vulnerable to haemophilia.

The analysis to be carried out is shown in Figure 3 (for one twin only). For each of the four measures, the genetic and environmental variances are correlated. We conducted a Cholesky, or triangular, decomposition (see Loehlin, 1996, for technical details) using the computer program Mx. This model was fitted to the data, and provided a good fit, $\chi^2 = 48$, $df = 52$, $p = .62$, $RMSEA = .00$.

The correlations of the unique environmental variances are shown in Table V, and the correlations of the genetic variances are displayed in Table VI. The correlations of the unique variances were generally smaller than the genetic variances. None of the unique factor correlations approached or exceeded Cohen's (1988) criteria for a medium strength correlation (.3). Among the genetic correlations, three of them exceeded the value of .3. The correlation between distraction and social diversion exceeded .5 (the criterion for a large correlation). The correlation between task orientation and social diversion ($r = .37$) was greater than the criterion for a moderate correlation, and the correlation between distraction and task orientation was moderate and negative ($r = -.28$). Comparing these values with the correlations shown in Table II and Table III, it can be seen that the majority of correlations between the variables seems to be due to genetic factors, with little of the overlap being due to unique environmental factors.

Discussion

The first important finding in our study was that four coping styles: task-oriented, emotion-oriented, social diversion, and distraction, showed evidence of genetic variance. The second important point demonstrated in conducted analyses was that no single genetic factor was shared by all different coping styles. In other words, there were both specific and shared genetic influences for different coping styles.

As in earlier studies (Busjahn et al., 1999; Kendler et al., 1991; Mellins et al., 1996) we did not find a homogeneous heritability pattern. However, all examined coping styles showed some evidence of genetic variance. Three of the coping styles, emotion-oriented,

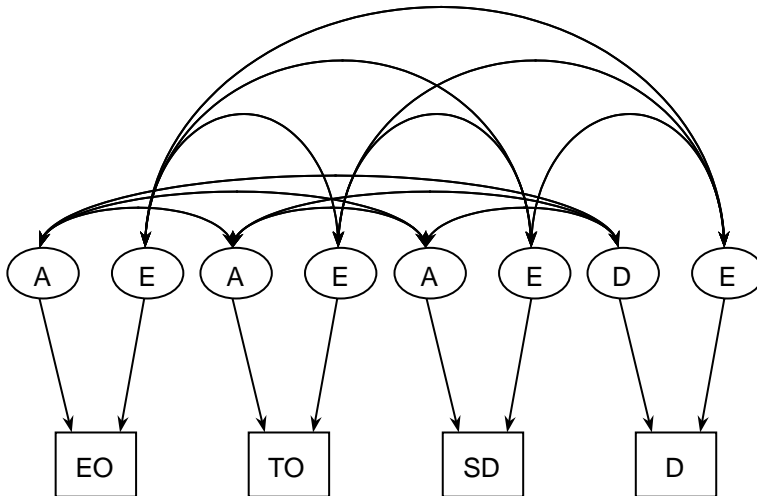


Figure 3. Multivariate model to be estimated. Curved arrows represent correlations between variables. Note that all genetic variables are correlated, and all environmental variables are correlated (for distraction the genetic variance was dominant). EO-emotion oriented coping, TO-task oriented coping, SD-social diversion, D-distraction, A-additive genetic factor, E-specific environment factor.

Table V. Correlations of unique environmental variances.

Emotion Oriented	1.000			
Task Oriented	.078	1.000		
Social Diversion	.001	.241	1.000	
Distraction	.228	-.091	.146	1.000
	Emotion Oriented	Task Oriented	Social Diversion	Distraction

task-oriented, and distraction, showed evidence of additive genetic factors (where the AE model fitted the best). This result suggests that coping styles are transmissible between generations. On the other hand, the findings with social diversion were suggestive of non-additive genetic factors (DE model fitted the best). This non-additive genetic influence might point to interactions between genes in the complex genetic background of different psychological traits, including coping styles as well. Such non-additive genetic influences (dominance) have been found in other studies on genetic determinants of coping (Busjahn et al., 1999; Kendler et al., 1991). It is important to mention that the interpretation of dominance effects is difficult because the present study did not have a great deal of power to distinguish between additive genetic and dominance effects.

Specific environmental factors were important for all coping styles. Such results indicate that coping styles could be shaped both by genetic as well as by environmental factors. The importance of specific environment factors in the present study suggests that coping behaviour in spite of genetic determination could be learnt.

We did not find evidence of shared environment effects on coping styles. In that, our results are not consistent with the results of Busjahn and colleagues (1999) who showed that shared environmental factors were important for coping styles such as distraction from situation, situational control, avoidance, self-pity, and aggression. Kendler and his collaborators (1991) also indicated the importance of shared environment factors for denial coping style. Nevertheless, our failure to find a statistically significant common environmental effect should not be taken as evidence that such effect do not exist. Borkenau et al. (2001) carried out a power analysis in their twin study and determined that their study would have required 1870 participants to have 75% power to detect a common environmental effect of .27. This fact, of course, implicates our study as suffering from low statistical power.

As mentioned before, heritability can be understood both in a broad and a narrow sense. The broad sense heritability (h^2B) refers to the total proportion of the variation of the trait that is due to genes, and the narrow sense heritability (h^2N) refers to that part of the genetic variation that is transmissible across generations. For three of four studied coping styles (emotion-oriented, task-oriented and distraction) narrow sense heritability was sufficient to

Table VI. Correlations of genetic variances.

Emotion Oriented	1.000			
Task Oriented	-.063	1.000		
Social Diversion	-.170	.373	1.000	
Distraction	.087	-.284	.522	1.000
	Emotion Oriented	Task Oriented	Social Diversion	Distraction

explain phenotypic variation in these variables. For social diversion the broad sense heritability was more useful.

The heritability estimates of coping styles were 35% for emotion-oriented coping, 34% for task-oriented coping, 33% for distraction, and 39% for social diversion, respectively. These values were consistent with previous estimates of heritability of coping styles. Broad sense heritability estimates of four secondary coping factors identified by Busjahn and others (1999) were as follows: defence (.52), emotional coping (.23), and substitution factor (.41). Narrow sense heritability of active coping was (.21). In Kendler and his collaborators' (1991) work the heritability estimates for the styles "turning to others" and "problem solving" were near .30. The heritability coefficient for the denial coping denial was close to .00.

The final part of our research plan involved the Cholesky decomposition that estimated correlations between the genetic components of the measures and the correlations between the environmental components of the measures. Again, our results were broadly in line with those reported by Busjahn et al. (1999). We found that both the genetic and environmental correlations varied. There was no consistency in these correlations, implying that there is no one underlying genetic trait, which influences each of the coping styles. In addition, and consistent with the reports of Busjahn, the genetic correlations ranged from being close to zero to values of approximately .5. However, the results of our environmental Cholesky model differed from those of Busjahn et al. who reported higher mean correlation (.30) than the mean correlation (.10) found in a present study. Undoubtedly, it is reasonable to assume that differences observed between heritability coefficients obtained in the present study and those reported by prior studies were simply due to the population, geographical and historical (time) differences. Nevertheless the stability of heritability estimates across studies seems to be fairly consistent (see Zawadzki, 2002, for a discussion of cross-cultural issues in estimating heritability of temperament).

It is important to remember that coping is often considered as a "secondary" factor, which may be influenced by "primary" factors, such as temperament, or the personality super-factors (the Big 5, Eysenck's Big 3). Thus a further avenue for an empirical exploration may be to extend the Cholesky model to consider the genetic and environmental correlations between coping styles and temperamental and personality traits. This direction of research was also suggested by other researchers examining genetic determinants of coping (Busjahn et al., 1999; Kendler et al., 1991; Mellins et al., 1996).

As a final comment, it is worth stating that although the results of our study provided evidence for a genetic influence on coping styles, it must be remembered that genetic determination does not imply fixedness. Hence, coping styles, just as many other human characteristics, are changeable during the life span.

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Note

1 It was not possible to estimate the model, which contained both dominance and additive genetic variance and common environmental variance, as these models were not statistically identified.

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