

Stability, consistency, and heritability of electrodermal response lability in middle-aged male twins

ANDREW CRIDER,^a WILLIAM S. KREMEN,^b HONG XIAN,^c KRISTEN C. JACOBSON,^d
BRIAN WATERMAN,^e SETH A. EISEN,^{c,f} MING T. TSUANG,^{g,h} AND MICHAEL J. LYONS^{h,i}

^aDepartment of Psychology, Williams College, Williamstown, Massachusetts, USA

^bDepartment of Psychiatry and Behavioral Sciences, University of California, Davis, School of Medicine, Sacramento, California, USA

^cDepartment of Internal Medicine, Washington University School of Medicine, St. Louis, Missouri, USA

^dVirginia Institute for Psychiatric & Behavioral Genetics, Virginia Commonwealth University, Richmond, Virginia, USA

^eWaterman Research Solutions, St. Louis, Missouri, USA

^fDepartment of Psychiatry, Washington University School of Medicine, St. Louis, Missouri, USA

^gHarvard Department of Psychiatry at Massachusetts Mental Health Center, Boston, Massachusetts, USA

^hHarvard Institute of Psychiatric Epidemiology and Genetics, Boston, Massachusetts, USA

ⁱDepartment of Psychology, Boston University, Boston, Massachusetts, USA

Abstract

We examined individual differences in nonspecific electrodermal response (EDR) lability in terms of retest stability, cross-situational consistency, and heritability in a sample of 345 adult monozygotic and dizygotic twin pairs. We also examined the phenotypic and genetic relationships between EDR lability and speed of habituation of the specific EDR to a nonsignal stimulus. Individual variation in EDR lability showed substantial retest stability and cross-situational consistency and also predicted resistance to specific EDR habituation. Structural equation modeling showed that the covariation among EDR lability measures and resistance to specific EDR habituation operated through a single latent phenotype, which was influenced in approximately equal measure by genetic and unique environmental factors. We discuss these findings in terms of an information processing account of individual differences in phasic EDR activation.

Descriptors: Individual differences, Electrodermal response lability, Temporal stability, Cross-situational consistency, Behavioral genetics, Twin study

Electrodermal response (EDR) lability is most commonly defined by individual differences in the rate of emission of nonspecific (spontaneous) EDRs under resting conditions. Because resting assessments of nonspecific EDR activity are known to be stable on retest, EDR lability is often regarded as a psychophysiological trait (Crider, 1993; Dawson, Schell, & Filion, 2000). However, temporal stability does not sufficiently define a trait, which should also show cross-situational consistency. In the case

of nonspecific EDR lability, the consistency criterion requires the preservation of relative individual differences across tasks differing in their psychological demands, even though the tasks themselves may prompt different absolute rates of nonspecific activity.

The question of the cross-situational consistency of nonspecific EDR lability has received relatively little attention. An influential early study by Lacey and Lacey (1958) reported a substantial correlation between nonspecific EDR lability during rest and during performance of an effortful processing task. However, the Laceys were not able to replicate this finding in a second experiment, and Baugher (1975) reported only a modest relationship between nonspecific EDR lability at rest and during a vigilance task. The issue was revisited by O’Gorman and Horneman (1979), who addressed both retest stability and cross-situational consistency in a design that assessed nonspecific EDR lability on two separate occasions over three tasks varying in effortfulness (relaxation, vigilance, and mental arithmetic). Analysis of variance showed a large main effect for individual differences, which accounted for 50% and 58% of the total variance across occasions and tasks, depending on the EDR amplitude criterion employed. Although the O’Gorman and Horneman study essentially replicated the initial Lacey and Lacey

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Address reprint requests to: Michael J. Lyons, Department of Psychology, Boston University, 648 Beacon Street, 2nd Floor, Boston, MA 02215, USA. E-mail: mlyons@bu.edu.

finding of substantial cross-situational consistency in nonspecific EDR lability, further investigation of the reproducibility and size of the effect is warranted.

Stability and consistency in a psychological trait are congruent with a degree of genetic determination. In the case of major personality dimensions, for example, additive genetic influences typically account for 40% to 50% of total phenotypic variance (Plomin & Caspi, 1999). Genetic influences on nonspecific EDR lability under either resting or task conditions have not heretofore been investigated. Although the heritability of nonspecific EDR activity observed during the iterated presentation of innocuous stimuli has been examined, results are contradictory (Hume, 1973; Lader & Wing, 1966).

On the other hand, substantial genetic influence has been reported for the speed of habituation of the specific EDR elicited by a nonsignal stimulus (Lykken, Iacono, Haroian, McGue, & Bouchard, 1988). Lykken et al. estimated that additive genetic influences accounted for about 40% of the variance in the number of trials required to habituate the specific EDR in a sample of mostly adult, mixed-gender monozygotic (MZ) and dizygotic (DZ) twin pairs. This finding is pertinent to the present investigation because specific EDR habituation speed is robustly correlated with resting nonspecific EDR lability, such that more labile subjects show slower specific EDR habituation. Because the correlation between the two measures approaches their respective reliabilities, Crider (1993) suggested that nonspecific EDR lability and specific EDR habituation speed can be regarded as alternative indices of a common central mechanism regulating phasic EDR activation. Therefore the reported genetic influence on specific EDR habituation speed implies a genetic influence on nonspecific EDR lability.

Evidence of the traitlike nature of phasic EDR activity has important implications for the interpretation of anomalies of such activity in various forms of psychopathology. Anomalies of nonspecific EDR lability and/or specific EDR habituation speed have been observed in a variety of psychological disorders, including childhood conduct disorder (e.g., Herpertz et al., 2001), criminal psychopathy (e.g., Raine, Venables, & Williams, 1995; Schalling, Lidberg, Levander, & Dahlin, 1973), and schizophrenia (e.g., Schell, Dawson, Nuechterlein, Subotnik, & Ventura, 2002). Such anomalies may be either a secondary consequence of a given disorder or, in contrast, a marker of psychophysiological dysfunction integral to the disorder. The first possibility usually requires that the anomaly be present during active phases of the disorder but show normalization in periods of remission. The second possibility usually requires that the anomaly be present during both active and remitted phases of the disorder. By implication, a marker for psychopathology should show reasonable stability over time and consistency across situations. Further evidence that genes that influence phasic EDR activity are related to genes that influence a particular disorder would indicate that the marker functions as a mediating endophenotype between the genetic diathesis for the disorder and aspects of its clinical expression. Therefore a demonstration that phasic EDR activity acts as a stable, consistent, and genetically influenced trait is an important step in identifying such activity as an endophenotype associated with various forms of psychopathology.

The data of the present investigation are derived from the psychophysiological assessment of a large sample of adult male MZ and DZ twin pairs. The assessment included recordings of phasic EDR activity during two rest periods bracketing a specific EDR habituation procedure and an effortful cognitive task. This

design allowed us to investigate the intrasession stability of nonspecific EDR lability under resting conditions, the cross-situational consistency between nonspecific EDR lability under resting and task conditions, and the relationship between nonspecific EDR lability and resistance to specific EDR habituation. In addition we employed structural equation modeling techniques to investigate the extent to which environmental and additive genetic factors contributed to variation within measures of nonspecific EDR lability and specific EDR habituation speed, as well as to the anticipated covariation across these measures.

Method

Participants

Participants were drawn from the Vietnam Era Twin Registry, a national register of several thousand male twin pairs in which both members served in the military between 1965 and 1975. Zygosity was determined using questionnaire and blood group methods with 95% accuracy. A complete description of the register's construction has been previously reported (Eisen, Ture, Goldberg, Henderson, & Robinette, 1987).

The present sample included 345 twin pairs and 3 unpaired twins without participating co-twins, for a total of 693 individuals. This sample was selected from a larger random sample of more than 3300 pairs surveyed in the Harvard Drug Study (Tsuang, Bar, Harley, & Lyons, 2001). Participants were recruited from these 3300+ pairs for participation in a subsequent twin study of vulnerability to alcoholism, which included a psychophysiological assessment. The twins were not selected on the basis of alcohol or drug use; however, only twins without service in Vietnam were recruited for the present study to avoid the potential confounding influence of combat exposure. To be included, both members of a pair had to agree to participate. Participants were flown in from around the country for a day-long series of assessments at either the University of California, Davis, School of Medicine, Sacramento, California, or Harvard Medical School, Boston, Massachusetts. Participants were given their choice of study site. In most cases twin pairs reported together to the same site on the same day. Assessments were conducted on 77 MZ and 79 DZ twin pairs reporting together to the Sacramento site, on 91 MZ and 75 DZ twin pairs reporting together to the Boston site, and on 1 MZ twin pair in their hometown. In addition, members of 7 MZ and 15 DZ twin pairs were assessed separately, one at each site.

The mean age of all participants was 47.8 years ($SD = 3.3$, range, 41–58); 92.2% were Caucasian, 5.5% were African-American, 1.9% were Hispanic, and 0.4% were of other racial origin.¹ In addition, 96.7% were high school graduates and 33% were college graduates; 79.1% were married, 12.1% divorced, and 8.8% widowed, separated, never married, or refused response. Among participants reporting full-time (92.2%) or part-time (1.6%) employment, 33.5% held service or manual labor positions, 24.4% held clerical or semiprofessional positions, and 41.1% held professional positions. Median household income category was \$60,000–\$70,000 (range, <\$10,000–\$100,000+). There were no significant differences at the $\alpha = .05$ level between MZ and DZ twins on any of the demographic measures.

¹Analyses were redone using Caucasian twins only. Results did not differ from those presented here, suggesting that any potential racial and ethnic variation in phasic EDR activity did not bias our findings.

Procedure

Psychophysiological assessments at both study sites employed identical equipment and followed a written protocol designed and supervised by the senior author. In the typical case in which both members of a twin pair reported together, one twin was assessed in the late morning and the second in the early afternoon of the same day. Participants sat upright in a lounge chair shielded from visual contact with the adjacent recording and stimulus presentation equipment. Ambient noise was attenuated with padded headphones worn throughout the procedure.

All participants underwent an identical testing sequence consisting of an initial rest period, a habituation procedure, a digit transformation task, and a final rest period. Participants were initially instructed to assume a comfortable position but to remain alert and refrain from movement. Following a 4-min rest period, a series of 1000-Hz tones was presented binaurally via audio tape at varying intervals of 30, 45, or 60 s. Tone duration was 1.0 s, with rise and fall times of 25 ms and an amplitude of 80 dB at the earpiece. The tones continued for 10 trials and thereafter for a maximum of 18 trials or until a habituation criterion was reached.

The digit transformation task was presented via audio tape and consisted of 20 trials requiring the participant to add a specified number to each of a string of four random digits held in memory and to report the result. Each trial was introduced by a ready signal followed by the presentation of the four digits at a 1-s rate. Following a 4-s rehearsal pause, participants were instructed to add either 0, 3, or 4 to each digit. After a brief pause, the participant was then cued to write out the transformed series on an answer sheet. A 10-s intertrial interval ensued. This paced task is known to produce a variety of sympathetic-like responses, including electrodermal arousal (Kahneman, 1973).

Immediately following the digit transformation task, participants were again asked to assume a relaxed posture for the final 3-min resting period.

EDR Recording and Measurement

Phasic skin conductance responses (SCRs) were recorded with a Grass Model 7 polygraph via a Model SCA1 signal conditioner, which imposed a constant 0.5 V across two electrodes placed on the thenar and hypothenar eminences of the nondominant hand. Participants washed and dried their hands prior to the attachment of disposable Ag-AgCl foam electrodes having a 1.0 cm² recording surface and filled with Grass EC33 isotonic conducting medium. The signal was amplified by a Model 7P1 preamplifier set at an input time constant of 0.8 s and recorded on paper at a sensitivity of 0.5 μ S/cm. An SCR was defined as a phasic increase in conductance of 0.05 μ S or greater or, in the case of cascading responses, from a minimal recovery of the same magnitude.

Five SCR frequency measures were derived from manual scoring of the paper record. Resting SCR liability was taken as the frequency of criterion SCRs during the initial rest period (Rest 1), during the final rest period (Rest 2), and during the two rest periods combined (Rest 1+2). Task SCR liability (Task) was taken as the frequency of criterion SCRs during 10 consecutive minutes of the digit transformation task between the onset of Trial 2 and the onset of Trial 20. Because of the varying lengths of the measurement periods, the frequency counts were converted to rates per minute by dividing through by the number of minutes in each period. Trials to specific SCR habituation (Trials) was measured as the trial number preceding three consecutive failures

to elicit a criterion SCR within 3s of tone onset during the habituation procedure.

Scoring reliabilities for the Rest 1, Rest 2, Task, and Trials measures were computed as intraclass correlations among four trained judges who independently scored a sample of 10 records (Rosenthal & Rosnow, 1991). All four reliabilities were above .90.

Due to occasional equipment failure or failure of the subject to follow instructions, the number of individual subjects with usable electrodermal data varied among measures from a minimum of 607 to a maximum of 623. Of the original 693 participants, valid data on at least one measure were available from 627 twins (90.5%). Of these 627 twins, 542 (86.4%) had complete data on all relevant SCR measures. Subjects with usable data did not differ significantly from subjects with unusable data on any of the demographic variables.

Data Analysis

Phenotypic correlations among the five SCR measures were expressed as product-moment correlations. The assumption of independence among observations was violated for these data because the sample consisted almost entirely of pairs of related individuals. Without correction for this dependency, the significance levels for these correlations would be biased. We therefore used Huber–White corrected tests of significance to account for the paired clustering of observations.

Structural equation modeling was performed with the Rest 1+2, Task, and Trials measures using the maximum-likelihood based program, *Mx* (Neale, Boker, Xie, & Maes, 1999). *Mx* allows for the fitting of models to raw data, rather than to correlation matrices. Thus individuals with partial missing data can still be included, as can individuals whose cotwins have nonvalid data. Models were compared using the likelihood-ratio chi-square (LRC) statistic and the Akaike Information Criterion (Akaike, 1987; Williams & Holahan, 1994). The LRC is obtained by comparing the -2 log-likelihood of a comparison model to the -2 log-likelihood of a nested (reduced) model. The difference in -2 log-likelihood is the LRC statistic. If the LRC between the two models is nonsignificant, the reduced model is generally accepted as the better model. When the LRC is nonsignificant for two or more competing models, the Akaike Information Criterion is used to determine the most parsimonious model. The Akaike Information Criterion indexes both goodness-of-fit and simplicity; the more negative the Akaike Information Criterion, the better the balance between goodness-of-fit and simplicity.

Three primary trivariate genetic factor models were initially tested: the Independent Pathways Model (also called the Biometric Model; Figure 1a), the Common Pathways Model (also called the Psychometric Model; Figure 1b), and the Measurement Model (Figure 1c). Details on differences between these models can be found elsewhere (Kendler, Heath, Martin, & Eaves, 1987; McArdle & Goldsmith, 1990; Neale & Cardon, 1992). Briefly, all three models assume (a) phenotypic correlations among the measured variables and (b) that genetic (A), shared environmental (C), and nonshared environmental (E) factors may each contribute to the phenotypic covariation among measures. The models differ primarily in terms of their stringency concerning the ways in which genetic and environmental factors contribute to variation and covariation.

As seen in Figure 1a, the Independent Pathways Model assumes that the genes and environments influencing covariation among measures operate directly on each variable through

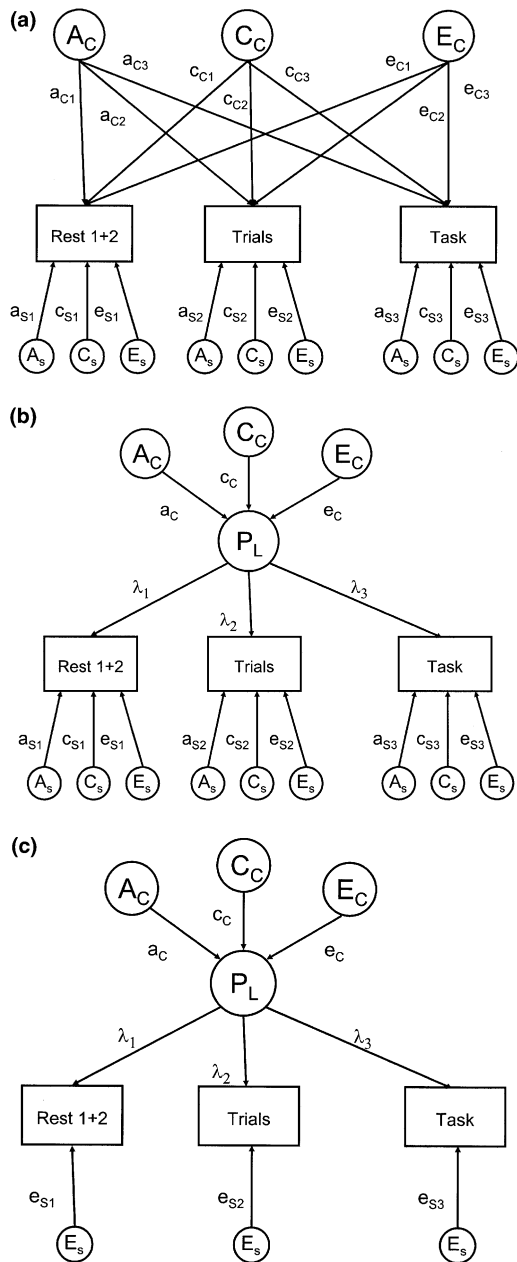


Figure 1. a: The Independent Pathways Model. b: The Common Pathways Model. c: The Measurement Model. For ease of display only one twin is represented in the diagrams in a–c. A_C : Common additive genetic influence, C_C : Common shared environmental influence, E_C : Common nonshared environmental influence. A_s : Specific additive genetic influence, C_s : Specific shared environmental influence, E_s : Specific nonshared environmental influence. P_L : Underlying latent phenotype. The lambda (λ) paths represent factor loadings for the three measured variables on the latent phenotype.

independent genetic and environmental pathways. Thus, the covariation between different pairs of variables can be due, in differing parts, to genetic and environmental influences. For example, the covariation between the Rest 1+2 and Trials measures may be due primarily to genetic factors, whereas the covariation between Rest 1+2 and Task may be due primarily to nonshared environmental factors.

In comparison, the Common Pathways Model (Figure 1b) assumes that a single underlying latent phenotype (P_L) is solely

responsible for the covariation among the three measures. Genetic and environmental influences on covariation are isomorphic and operate through the latent phenotype. That is, genes and environments influence correlation among variables via a common pathway. A key element of the Common Pathways Model is its prediction that the phenotypic covariance is equally apportioned into genetic and environmental components for all combinations of variables. It has been shown that the Common Pathways Model is a submodel of the Independent Pathways Model (McArdle & Goldsmith, 1990). These models can therefore be compared using the LRC statistic. The lambda (λ) paths in the Common Pathways Model correspond to factor loadings of each measure on the latent phenotype and account for the proportion of variance in each measure shared with the latent phenotype.

The final model, the Measurement Model (Figure 1c) is a submodel of the Common Pathways Model. The critical difference between these two models is that the Common Pathways Model allows for unique genetic (A_s) and shared environmental (C_s) factors specific to each measure that do not influence the covariation among measures. In contrast, the Measurement Model assumes that all genetic and environmental influences are operating through the latent phenotype. The only residual variance on each measure not explained by the latent phenotype is assumed to be measurement error and is therefore modeled as nonshared environment (E_s).

In all three models, genetic factors correlate 1.0 for MZ twins and 0.5 for DZ twins. Shared environmental factors are correlated 1.0 across twins regardless of zygosity. Nonshared environmental factors are uncorrelated across twins. The variance of the underlying genetic and environmental factors is fixed at 1.0. Likewise, in the Common Pathways and Measurement models a nonlinear constraint is imposed so that the variance of the underlying latent phenotype also equals unity.

Results

Distribution Statistics

Table 1 presents distribution statistics for the four measures of nonspecific SCR lability and for the number of trials to specific SCR habituation for MZ and DZ twins separately and combined. Nonspecific SCR rate/minute during the digit transformation task was approximately normally distributed and markedly higher than nonspecific SCR rates/minute under resting conditions. In accord with previous reports (e.g., Vossel & Zimmer, 1990), nonspecific SCR rates/minute during rest were positively skewed, as was also the case for trials to specific SCR habituation. In subsequent analyses, scores for the nonspecific SCR resting and specific SCR habituation measures were modified by use of normalizing logarithmic transformations ($\log[x+1]$). There were no significant differences at the alpha = .05 level for mean differences between MZ and DZ twins on any of the measures in Table 1.

Phenotypic Correlations

Correlations among the five SCR measures are displayed in Table 2. Substantial covariation was observed between nonspecific SCR lability during the initial and final rest periods, a measure of stability, as well as between the combined rest periods and the task period, a measure of consistency. Stable and consistent individual differences in nonspecific SCR lability accounted for approximately 50% of the variance in each case. Effect sizes for

Table 1. Distribution Statistics for Measures of Nonspecific SCR Liability (SCRs/min) and for Trials to Specific SCR Habituation

	<i>N</i>	Mean	<i>SD</i>	Median
Rest 1				
MZ	325	2.54	3.14	1.25
DZ	285	2.26	2.72	1.00
All	610	2.41	2.96	1.25
Rest 2				
MZ	316	3.07	3.05	2.00
DZ	291	2.63	2.55	2.00
All	607	2.86	2.83	2.00
Rest 1+2				
MZ	310	2.76	2.81	1.71
DZ	282	2.42	2.41	1.57
All	592	2.60	2.63	1.57
Task				
MZ	324	7.25	4.20	7.10
DZ	291	6.75	3.89	6.80
All	615	7.01	4.06	6.90
Trials				
MZ	329	7.38	7.03	4.00
DZ	294	6.80	6.65	4.00
All	623	7.10	6.85	4.00

cross-situational consistency were somewhat reduced when considering the two rest periods separately, as would be expected on psychometric grounds. Substantial covariation was also found between nonspecific SCR liability during the combined rest periods and trials to specific SCR habituation. This effect size was unaltered when nonspecific SCR liability during the first rest period alone was considered. The magnitudes of the correlations in Table 2 were not appreciably different when rank order correlations were computed, ruling out the possibility that the Pearson correlations might be inflated by the presence of outliers. Because of the substantial stability of nonspecific liability across the first and second rest periods, the combined rest measure was subsequently used to calculate twin correlations as well as in the structural equation modeling.

Twin Correlations

Prior to testing the three primary structural equation models, we ran a saturated model in *Mx*. This model fits the observed data perfectly and therefore serves as a comparison model (using the LRC statistic) for all the theoretical models tested. In addition, the saturated model yields the observed covariance within and

Table 2. Pearson Correlations (*n*) among Measures of Nonspecific SCR Liability and Trials to Specific SCR Habituation

	1.	2.	3.	4.
1. Rest 1*				
2. Rest 2*	.70 (596)			
3. Rest 1+2*	.93 (596)	.91 (596)		
4. Task	.64 (602)	.67 (603)	.71 (588)	
5. Trials*	.70 (611)	.57 (607)	.69 (592)	.61 (616)

Note: $p < .001$ in all cases.

* $\log(x+1)$.

across twins. Table 3 presents the full 6×6 correlation matrices for MZ and DZ twins generated by this model. Inspection of the correlations in bold and in italics gives some indication of the extent to which genetic and environmental factors contribute to variation in each measure and to covariation across measures. Specifically, the correlations in bold are the cross-twin, within-trait correlations. DZ correlations for resting SCR liability and trials to specific SCR habituation are less than one-half the respective MZ correlations, suggesting that genetic factors influence variation in these measures and that shared environmental influences are negligible. For task SCR liability, the DZ cross-twin, within-trait correlation is slightly greater than one-half the MZ correlation, suggesting a strong genetic influence and a small shared environmental influence. The pattern of cross-twin, cross-trait correlations presented in italics suggests that genetic factors are the primary source of familial covariance across traits. Specifically, for four of the six correlations, DZ correlations are less than or equal to one-half the MZ correlations, suggesting strong genetic influence and no effect of shared environment. For the remaining two correlations, DZ correlations are only slightly greater than one-half the respective MZ correlations, suggesting that shared environmental influences are modest at best. Finally, within MZ twins the cross-twin, cross-trait correlations are lower in magnitude than the cross-twin, within-trait correlations, which suggests some genetic or shared environmental influence on the specific measures independent of the genetic and shared environmental factors that influence covariation across measures.

Model Fitting

Results from the structural equation modeling are presented in Table 4. None of the three theoretical models tested fitted the data very well, as indexed by significant p values based on comparisons with the saturated model. Significant p values are often found in twin models with relatively large sample sizes, because small differences in variances across twins or across zygosity contribute to model misfit. In these data, within-person corre-

Table 3. Correlation Matrices (*r*) of SCR Measures for MZ and DZ Twins

	Rest 1+2-A	Trials-A	Task-A	Rest 1+2-B	Trials-B	Task-B
MZ twins						
Rest 1+2-A	1.0					
Trials-A	.68	1.0				
Task-A	.77	.64	1.0			
Rest 1+2-B	.49	.37	.40	1.0		
Trials-B	.41	.51	.35	.68	1.0	
Task-B	.36	.40	.48	.69	.64	1.0
DZ twins						
Rest 1+2-A	1.0					
Trials-A	.66	1.0				
Task-A	.60	.51	1.0			
Rest 1+2-B	.14	.21	.15	1.0		
Trials-B	.15	.24	.10	.75	1.0	
Task-B	.18	.27	.27	.74	.63	1.0

Note: Correlations were obtained from *Mx* by fitting a saturated model to the raw data. Variables with -A are for twin A; those with -B are for twin B. Correlations in bold are cross-twin, within-trait correlations and yield information about genetic and environmental influences on each individual measure. Correlations in italics are cross-twin, cross-trait correlations and yield information regarding genetic and environmental covariation among the three variables.

Table 4. Model Fitting Results for Covariance among SCR Measures

Model		-2LL	df	AIC	p
1	Independent Pathways	5616.60	1809	-15.34	.025
2	Common Pathways	5619.40	1813	-20.53	.039
3	Measurement	5659.31	1819	+7.38	<.001

Note: -2LL: -2 log-likelihood. AIC: Akaike Information Criterion. *p* values are based on comparisons with the saturated model (-2LL = 5565.93, *df* = 1776).

lations among the three SCR measures were relatively similar across A and B twins and across MZ and DZ twins (Table 3). However, there was some variability in estimates of overall variance in each measure. Although there was a tendency towards lower overall variances among DZ compared with MZ twins (Table 1), patterns were not consistent across A and B twins.

Of the three theoretical models tested, the Common Pathways Model did not fit the data significantly more poorly than the Independent Pathways Model (LRC = 2.80, *df* = 4, *p* = .59), indicating that the genetic and environmental factors that influence covariation across the three SCR measures operate through a single underlying latent phenotype. In contrast, the Measurement Model fit significantly more poorly than the Common Pathways Model (LRC = 39.91, *df* = 6, *p* < .001), indicating that there were significant genetic and/or shared environmental influences specific to the three measures that did not overlap with the genetic and shared environmental influences on the underlying latent phenotype. The Common Pathways Model also had the lowest Akaike Information Criterion value, indicating that it was the best-fitting model of the three.

Inspection of the results from the Common Pathways Model revealed that 95% confidence intervals for the specific genetic and shared environmental influences on each measure contained zero (results available from author), suggesting that none of these specific influences were statistically significant by themselves. Nevertheless, the above model fitting results clearly indicate that not all of the specific genetic and shared environmental influences could be dropped simultaneously from the model. Thus, to determine why the Measurement Model did not fit the data, we ran a series of additional submodels that dropped different sets of parameters. In particular, we examined the fit of two different models: (1) a model that dropped all three specific genetic factors and (2) a model that dropped all three specific shared environmental factors.

The model that dropped all three specific genetic factors fit the data significantly more poorly at the trend level than the full Common Pathways Model (-2 log-likelihood = 5626.13, model *df* = 1816, LRC = 6.73, *df* = 3, *p* = .08). In contrast, dropping all three shared environmental influences did not fit the data significantly more poorly than the full Common Pathways Model (-2 log-likelihood = 5620.67, model *df* = 1816, LRC = 1.27, *df* = 3, *p* = .74). Once the specific shared environmental factors had been dropped from the model, the specific genetic influences on trials to specific SCR habituation and task SCR liability became statistically significant (LRC = 12.36, *df* = 1, *p* < .001 for trials to habituation; LRC = 15.54, *df* = 1, *p* < .001 for task liability). However, dropping the specific genetic influence on resting liability did not significantly reduce model fit (LRC = 0.00, *df* = 1, *p* > .99). Finally, shared environmental influences on the latent

trait were estimated at zero, and dropping this parameter did not significantly worsen model fit (LRC = 0.00, *df* = 1, *p* > .99).

Figure 2 presents the parameter estimates from the final reduced model: the Common Pathways Model with significant specific genetic influence on trials to specific SCR habituation and on task SCR liability only, and with no shared environmental influences. Estimates were very similar to results from the full Common Pathways Model (not shown). In this reduced model, the latent phenotype was influenced approximately equally by genetic (53%; 95% CI = 41–64%) and nonshared environmental factors (48%; 95% CI = 37–61%). The latent phenotype accounted for 83% of the variance in resting SCR liability (95% CI = 77–86%), 59% of the variance in trials to specific SCR habituation (95% CI = 52–64%), and 61% of the variance in task SCR liability (95% CI = 55–66%).

Table 5 presents the total estimates of heritability and non-shared environmental influences for each of the three measures. The total heritabilities were approximately equal across measures, ranging from .43 to .48. However, for trials to specific SCR habituation and task SCR liability, approximately one-third of this total genetic influence was due to specific genetic factors that did not overlap across measures. Nonshared environmental influences accounted for just over one-half of the variance in each measure, with 30–50% of the total nonshared environmental influence due to measure-specific nonshared environmental influences (including measurement error).

Discussion

The major findings of this study are as follows: (1) individual variation in nonspecific EDR liability showed substantial retest stability and cross-task consistency, (2) resting and task measures of nonspecific EDR liability were inversely related to the speed of habituation of the specific EDR to an iterated stimulus, and (3) nonspecific EDR liability and specific EDR habituation speed loaded on a single latent phenotype with substantial genetic and unique environmental influences but absent influence from the common environment.

The retest stability of nonspecific EDR liability under resting conditions has been confirmed in numerous investigations (Crider, 1993; Freixa i Baqué, 1982). Our intrasession correlation of *r* = .70 suggests that stable individual differences account for

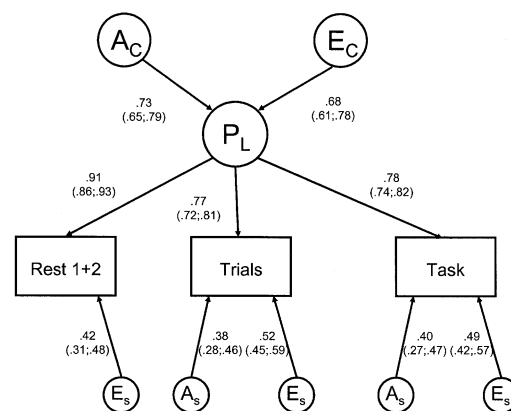


Figure 2. Standardized parameter estimates from the reduced Common Pathways Model. For ease of display only one twin is represented in the diagram. Numbers in parentheses are 95% confidence intervals.

Table 5. Calculations of Genetic (h^2) and Environmental (e^2) Effects Using the Reduced Common Pathways Model

	Rest 1+2	Trials	Task
h^2 due to latent phenotype	.43	.31	.32
h^2 due to specific genetic influence	—	.15	.16
Total h^2	.43	.46	.48
Proportion h^2 due to specific factors	.00	.32	.33
e^2 due to latent phenotype	.39	.27	.29
e^2 due to specific environmental influence	.18	.27	.24
Total e^2	.57	.54	.53
Proportion e^2 due to specific factors	.31	.50	.45

approximately half of the retest variance in this measure. This effect size is virtually identical with intrasession values over 40 and 60 min retest periods reported by Schuler and Papousek (1992). Other studies examining the retest stability of resting EDR lability over intervals of 1 day to 1 year report correlations both above and below the intrasession value found here. We identified 10 such studies employing normal subjects. The data from two of these studies were excluded because the obtained reliabilities for both resting EDR lability and a second electrodermal measure were speciously lower than their reported intercorrelation (Strube, 2000). The remaining eight studies reported retest reliabilities (r or ρ) ranging from .50 to .76, with an unweighted mean of .66 (Baugher, 1975; Crider & Lunn, 1971; Dykman, Ackerman, Galbrecht, & Reese, 1963; Hustmyer & Burdick, 1965; Johnson, 1963; Lacey & Lacey, 1958; Schuler & Papousek, 1992; Vossel & Zimmer, 1990). In general, higher reliabilities were associated with shorter retest intervals, longer rest periods, and rank order score transformations, but the data were insufficient to confirm the significance of these trends. Although the sources of the variation in effect sizes among these studies are unclear, the aggregate results nevertheless converge on the effect size reported here.

Our results also indicate that consistent individual differences account for about 50% of the cross-situational variance in EDR lability, in this case between resting nonspecific EDR lability and nonspecific EDR lability during an effortful processing task. This finding agrees with the original Lacey and Lacey (1958) finding, as well as with the conclusions of O'Gorman and Horneman (1979). That retest stability and cross-task consistency each account for similar proportions of total nonspecific EDR lability variance is quite plausible and consistent with a trait formulation of EDR lability.

The validity of our results is strengthened by the replication of the well-known covariation of resting nonspecific EDR lability and resistance to specific EDR habituation. The effect size ($r = .70$) was slightly larger than consensual estimates (Crider, 1993). However, the reliability of trials to habituation appears to be greater for more intense stimuli (Siddle & Heron, 1976), and the moderately intense stimulus employed here may have optimized its covariation with resting nonspecific EDR lability. As would be expected on the grounds of cross-task consistency, we also found a similar relationship between resistance to specific EDR habituation and nonspecific EDR lability assessed under task conditions.

The present study revealed moderate heritabilities between .40 and .50 for the three phasic EDR activity measures. The finding for resistance to specific EDR habituation replicates the

result of Lykken et al. (1988), despite procedural differences between the two studies in habituation procedures, genetic models, and sample gender composition. In addition, our results revealed heritabilities of similar magnitude for resting and task measures of nonspecific EDR lability.

Structural equation modeling showed that the phenotypic covariation among the three EDR measures operated through a single latent phenotype, which was influenced in approximately equal measure by genetic and nonshared environmental factors. This latent phenotype represents a dimension of phasic EDR activation manifested by nonspecific EDR lability as well as by resistance to specific EDR habituation. We suggest that the psychological significance of this dimension is related to the putative role of phasic EDR activity in information processing. Specifically, phasic EDR activity can be considered a peripheral manifestation of a central arousal system that functions to increase cognitive capacity in the service of efficient information processing (Kahneman, 1973; Öhman, 1979; Öhman, Hamm, & Hugdahl, 2000). In the case of the specific EDR, the arousal system is triggered by motivationally relevant external events that mobilize attentional resources and prompt effortful processing of the stimulus. In Öhman's (1979) account, the specific EDR represents a call for cognitive resources in the service of effortful processing in a capacity-limited central channel. The specific EDR does not reflect effortful processing per se; rather it signals an increase in resource availability in anticipation of processing. Because of capacity limitations, the call for resources indexed by the specific EDR may not be answered if the central channel is occupied with other tasks.

The information processing account of specific EDR activity can be extended to the case of nonspecific EDR activity occurring in the absence of an eliciting stimulus. Recent investigations suggest that nonspecific EDRs observed under resting conditions are associated with conscious rumination on motivationally relevant but unrealized goals and intentions (Nikula, Klinger, & Larson-Gutman, 1993). Because such rumination can be considered an effortful process, nonspecific EDRs may reflect arousal in the service of increased cognitive capacity during rumination. Therefore, stable and consistent individual differences in resting nonspecific EDR lability may index differential propensities toward effortful ideation. Indeed, nonspecific EDR lability is correlated with self-reports of emotional ideation under resting conditions (Baugher, 1975) and with observer ratings of active suppression of negative thoughts while anticipating an aversive stimulus (Hare, 1966). In sum, both specific and nonspecific EDRs may reflect a common information processing mechanism, although different classes of events trigger the two types of response.

An important implication of the foregoing is that individuals with characteristically higher levels of nonspecific EDR activity (EDR labiles) will have less spare capacity available for meeting the information processing requirements of imposed tasks than those with lower levels of activity (EDR stabiles). That is to say, capacity-demanding ideation will compete with the efficient processing of subsidiary tasks because of limitations on spare capacity. Several studies confirm that EDR labiles perform more poorly on capacity-demanding tasks than their more stabile counterparts, presumably because of reduced spare capacity secondary to ideational preoccupation (O'Gorman & Lloyd, 1988; Schuler & Papousek, 1992; Zimmer, Vossel, & Frohlich, 1990). In contrast, EDR labiles appear to be superior to EDR stabiles on less complex tasks, like simple and choice reaction time, that

require alertness and/or sustained attention rather than capacity (Crider, 1993).

Limitations on capacity may also explain the phenotypic covariation between nonspecific EDR lability and resistance to specific EDR habituation. Because specific EDR habituation requires central processing capacity, habituation will be slowed to the extent that ideational preoccupation competes for limited processing resources. In other words, individuals with higher levels of nonspecific EDR lability will have less spare capacity available for meeting the processing demands of the habituation task. In this perspective, individual variation in specific EDR habituation speed would appear to be a secondary consequence of individual differences in ideational preoccupation as indexed by nonspecific EDR activity.

A salient finding from the present study is the apparent lack of shared environmental influences on nonspecific EDR lability and on resistance to specific EDR habituation. Although the 95% confidence intervals on the specific genetic and shared environmental factors suggest that we were somewhat underpowered to discriminate between specific genetic and shared environmental influences, results from the full Common Pathways Model (available from author) suggest that shared environmental influences were negligible, accounting for less than 1% of the overall variation in each of the three EDR measures. This finding is quite consistent with the genetic analysis of psychological traits and various forms of psychopathology (Plomin & Caspi, 1999). The absence of shared environmental influences does not indicate that other sources of environmental influence are unimportant. Rather, such findings indicate that only those environmental influences unique to the individual exert a significant influence on phasic EDR activation.

Although our results suggest that a single latent phenotype best captures the relationship among the three measures of phasic EDR activity, we also found evidence of specific genetic influences on nonspecific EDR lability during the task and on resistance to specific EDR habituation. Unique genetic factors accounted for approximately one-third of the overall total ge-

netic variance in these two measures. In contrast, resting EDR lability showed negligible specific genetic influence while loading strongly on the latent phenotype. The present study does not clarify the psychological concomitants of the two unique genetic influences. Nevertheless, these findings indicate that genetic influences are not necessarily uniform across different measures of phasic EDR activity. Genetic influences specific to particular EDR measures suggest the operation of psychophysiological factors over and above the information processing function we have attributed to the latent phenotype.

A limitation of this study is that the generalizability of our results may be constrained by the composition of the sample, which consisted primarily of Caucasian males. However, our results are generally consistent with a number of previous reports based on subject samples of varying demographic composition. In addition, there may be limits to the cross-situational consistency of nonspecific EDR lability, and this study would have benefited from the inclusion of more than two assessment situations. Strengths of the study include the large sample size, the use of a general population sample, and the examination of middle-aged subjects, which are rare in studies of electrodermal phenomena.

In conclusion, this twin study has identified a latent phenotype represented by various forms of phasic EDR activation, which may operate in the service of effortful information processing in a limited capacity central channel. The latent phenotype is characterized by substantial genetic and negligible common environmental influence, with the remaining variance accounted for by unique environmental influences. An intriguing future direction will be to determine the extent to which the genes that influence phasic EDR activation are related to genes that influence psychopathology. Behavioral genetic research combining measures of phasic EDR activation with measures of psychopathology and related traits should help clarify the extent to which individual variation in phasic EDR activation acts as an endophenotype associated with specific forms of psychopathology.

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