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The Heritability of the Skin Conductance Orienting Response: A Longitudinal Twin Study

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Abstract

The orienting response is a widely used experimental paradigm that reflects the association between electrodermal activity and psychological processes. The present study examined the genetic and environmental etiology of skin conductance orienting response (SCOR) magnitude in a sample of twins assessed at ages 9-10, 11-13 and 14-16 years. Structural equation modeling at each visit showed that genetic influences explained 56%, 83%, and 48% of the total variance in SCOR at visit 1, 2, and 3 respectively, with the remaining variance explained by non-shared environmental factors. SCOR was moderately stable across ages, with phenotypic correlations between time points ranging from .35 to .45. A common genetic factor explained 36%, 45% and 49% of the variance in SCOR magnitude across development. Additional age-specific genetic effects were found at ages 9-10 and 11-13 years, explaining 18% and 35% of the variance, respectively. The genetic correlations among the three time points were high, ranging from .55 to .73, indicating a substantial continuity in genetic influences from ages 9 to 16. These findings suggest that genetic factors are important influences in SCOR magnitude during late childhood and adolescence.

Keywords

Electrodermal; Orienting; Heritability; Stability; Longitudinal

The skin conductance orienting response (SCOR) is an autonomic response to novel stimuli and indirectly reflects how much a person attends to and processes novel stimuli in the environment (Dawson, Schell & Filion, 2007). According to information processing theory, novel stimuli are initially stored in short-term memory, where they form neural templates (Öhman, 1979). When subsequent stimuli are presented, they are compared to the template currently stored in short-term memory. If the stimulus is different from the template, additional, controlled processing mechanisms are required and the orienting response is

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elicited. In contrast, if the incoming stimulus matches the template, no additional resource is needed and the orienting response does not occur. Orienting reflexes are generated by the central nervous system, and are conveyed to peripheral parts of the body via sympathetic fibers. The skin conductance (SC, e.g., a part of electrodermal activity) component of the orienting response is triggered by postganglionic sympathetic fibers known as sudomotor nerves (Boucsein, 1992). These nerve fibers activate the eccrine sweat glands, which in turn cause an increase in the hydration of the outer layer of the skin (stratum corneum). Skin conductance is measured by passing a constant voltage between two points (electrode sites) on the corneum. The purpose of the present study was to examine the genetic and environmental etiology of the size of SC response across development using a sample of young twins.

Reliable relationships between SCOR and arousal, attention, information processing, and emotion have been observed in numerous studies (for a review, see Hugdahl, 1995). SCOR impairments have been associated with a variety of psychological disorders, both when examined cross-sectionally and longitudinally. For example, in a study including a cross-sectional sample of boys between the ages 8 and 13 years, researchers found that those with comorbid conditions of Attention-Deficit/Hyperactivity-Disorder (ADHD) and conduct disorder showed reduced SCOR magnitudes compared to age-matched boys with ADHD only (Herpertz, 2001). SCOR deficits have additionally been linked to antisocial behavior, especially in psychopathic, antisocial, and criminal subjects who also exhibit schizotypal features, such as paranoia, reduced emotionality, and inability to make close friends (Raine, 1996). Specifically, one recent study suggested that reduced SCOR magnitude is associated with psychopathic personality traits in children and adolescents, although the association was more salient in boys than in girls (Isen et al., 2010). In contrast, another study reported that *larger* SCOR at age 3 years was associated with elevated psychopathic personality traits at age 28 years in both males and females in a community sample (Glenn et al., 2007). Thus, the relationship of SCOR to psychopathology may vary across both gender and age.

Dysfunction of the prefrontal cortex may contribute to both atypical SCORs and psychological disorders and behavior problems. While the neuroanatomical basis of SC is complex, imaging studies of SCOR have specifically implicated the prefrontal cortex, hippocampus, and anterior cingulate cortex (Raine, Reynolds & Sheard, 1991; Williams et al., 2000). Prefrontal deficits have also been found in antisocial and schizophrenic individuals as uncovered by brain imaging and neuropsychological research (Gao et al., 2010; Raine & Yang, 2006). Taken together, the prefrontal abnormality may give rise to both SCOR impairments and antisocial behavior (Raine, 2002).

It is also possible that a genetic predisposition to a psychopathological phenotype may be expressed in part through a psychophysiological characteristic (Raine, 2002). It is therefore important to establish if this psychophysiological characteristic, i.e., the SCOR, is heritable and the extent to which its stability across development is explained by genetic and/or environmental influences. The main aim of the present study is to examine the genetic and environmental etiology of SCOR magnitude in children and adolescents in a large sample of male and female twins.

To the authors' knowledge, very few studies have examined the genetic and environmental influences on SC in general, and these have all been cross-sectional in nature (e.g., Lykken et al., 1988). Twin studies of adults have shown that genetic factors are important for various indices of SC responses (Crider et al., 2004; Lykken et al., 1988). Crider et al. (2004) found that the covariation between rates of non-specific SC responses and habituation (i.e., the number of trials before a subject fails to respond to repetitive stimuli) of SCOR in a sample of 345 twin pairs was explained by a common latent phenotype that was

53% heritable, with the remaining variance explained by non-shared environmental influences (i.e., experiences unique to each twin in a pair). There was no evidence of shared environmental effects (i.e., non-genetic influences that contribute to similarity within pairs of twins). Similarly, Lykken et al. (1988) studied SC responses to loud tones in 121 twin pairs and found that genetic factors accounted for about 40% of the variance in the number of trials necessary for habituation of the orienting response, as well as 59% of individual differences in magnitude of the first four SC responses, with shared environmental influences between twins being of minimal importance. One limitation common to these studies is that they have either not tested for sex differences or failed to include females.

More importantly, no behavioral genetic study has examined how genetic and environmental influences contribute to SCOR from late childhood to adolescence. As mentioned above, Crider et al. (2004) examined the genetic influences on SC habituation, which is a measure of temporal stability of SC responses. When reporting the response size of SCOR when stimulus is presented repeatedly, magnitude or amplitude is usually calculated. Magnitude refers to the mean values computed across all stimulus presentations including those without a measurable response, whereas amplitude is the mean values computed across only those trials on which a measurable (nonzero) response occurred (Dawson et al., 2007). Although it is still arguable as to which assessment is better (Dawson et al., 2007; Venables & Christie, 1980), SCOR magnitude rather than amplitude or habituation was examined in the current study for three reasons: 1) we are interested in the genetic influences on the response size of the SCOR, 2) the full set of observations can be exploited, and 3) SCOR magnitude has been frequently reported in studies involving autonomic system activities in relation to behavioral measures (Scarpa et al., 1997).

In summary, we attempt to fill these research gaps by investigating the genetic and environmental etiology of SCOR magnitude in a longitudinal sample of male and female twins measured on three occasions, i.e., at ages 9-10, 11-13, and 14-16 years. Specifically, our aims are twofold: (1) to examine the genetic and environmental influences on SCOR magnitude during each time point and (2) to explore how genetic and/or environmental factors contribute to the stability of SCOR magnitude from late childhood to adolescence.

Method

Participants

The subjects were participants in the University of Southern California (USC) Twin Study, which is a prospective study of the interplay of genetic, environmental, social, and biological factors on the development of antisocial behavior from childhood to emerging adulthood. Twins were recruited from local schools and advertisements in the Los Angeles community. Both the ethnic and socio-economic status composition of the sample is representative of Southern California: 37% Hispanic, 27% Caucasian, 14% Black, 4% Asian, and the remaining 17% of mixed/other. Socio-economic status (SES) is based on the Hollingshead Four-Factor index of Social Status (Hollingshead, 1979), upper 1.4%, upper-middle 20.0%, middle 41.4%, lower-middle 34.4%, and lower 2.8%. Zygosity was determined based on DNA microsatellite analysis of the same-sex twin pairs. Complete details on the procedures and measures can be found elsewhere (Baker et al., 2006).

In the first visit during 2000-2004 the twins were 9-10 years old (mean age = 9.6, $SD = .58$). During the second visit 2003-2006, the twins were 11-13 years old (mean age = 11.8, $SD = .90$). During the third visit in 2005-2009, the twins were 14-16 years old (mean age = 14.6, $SD = .69$). The total sample consists of 1,416 subjects, including 310 monozygotic (MZ) male, 324 MZ female, 204 dizygotic (DZ) male, 214 DZ female, and 364 DZ opposite-sex twins. The present study used data from all three visits. Table 1 presents the number of

twins, by zygosity, with complete data on SCOR. There were 1,157 twins with data on SCOR during the first visit, 161 twins during the second visit, and 642 twins during the third visit.

Procedures

Participants were invited to USC for a laboratory assessment. When arriving at USC, the subject was seated in a room adjacent to the interviewer. Before administering the tasks, baseline recordings were obtained for three minutes during which time the subject was instructed to sit quietly and relax.

SCOR Measures

The orienting paradigm consists of four types of stimuli with 12 tones in total. There were three consecutive trials within each of four stimulus types. The four types of stimuli were presented in the following order: 1) 1000-Hz tones (75 dB), 2) consonant-vowel (“da”) sounds (65-95 dB), 3) novel sounds (cuckoo clock, bird chirping, and rooster crowing, 70-105 dB), and 4) baby cries (90-95 dB). The interstimulus intervals ranged from 18 to 28 seconds, with a mean of 22.5 seconds. The stimuli consisted of complex waveforms that never exceeded a sound pressure level of 105 dB. The duration of the baby cries was exceptionally long (8 seconds) in order to be more ecologically valid, whereas the durations of the other stimuli never exceeded one second.

SC was recorded from bipolar leads on the distal phalanges of the index and middle fingers using a constant voltage system (Lykken & Venables, 1971). Silver-silver chloride (Ag-AgCl) electrodes (7 mm in diameter) were placed on the palmar surface of the non-dominant hand. A water soluble lubricant was used, supported by an adhesive electrode collar that maintained full contact with the skin. The electrodes were further fastened in place using waterproof tape.

SC was recorded with equipment and software from the James Long Company (1999; Caroga Lake, New York). A 31 channel Isolated Bioelectric Amplifier was used and physiological data were recorded online directly into a data acquisition computer. The signal from the amplifier had a sampling rate of 512 Hz. Responses elicited by the stimuli were scored through the James Long Company software (SCOR component of the Orientation Response Analysis System). As part of our scoring criteria, a latency window of 1.5 - 4.5 seconds following each stimulus onset was used to determine whether a response occurred. Although latencies are typically in the range of 1 to 4 seconds (Dawson et al., 2007), we started our latency window at 1.5 seconds out of concern that our subjects would need additional time to register some of the more complex (non-tone) stimuli. Additionally, the slope in SC level was required to exceed the baseline slope (at stimulus onset) by a minimum of 0.05 μ S / second. SCOR amplitude was defined as the peak change in SC occurring within seven seconds of response initiation, and mean SCOR magnitude was calculated. If no response was detected, then the magnitude for that particular trial assumed a value of zero rather than being omitted.

The variable of interest was the mean SCOR magnitude to the 12 stimuli. Prior to analyses, the three SCOR magnitudes were ranked (PROC RANK, Blom option) and normalized (PROC STANDARD) to reduce the positive skew in their distributions using the statistical software SAS 9.1.3 (SAS, 2005).

Statistical Analyses

Descriptive Statistics and Correlations—Descriptive statistics, including means and standard deviations, were first computed for SCOR magnitude prior to transformation, as well as phenotypic correlations for SCOR magnitude across the three time points.

In the classical twin design data from monozygotic (MZ) and dizygotic (DZ) twins are used to decompose the variance in measured trait to genetic and environmental components. MZ twins share their common environment and they are assumed to share 100% of their genes. DZ twins also share their common environment and they are assumed to share about 50% of their genes. By comparing the resemblance between MZ and DZ twins the total phenotypic variance of a measured trait can be divided into additive genetic factors (A), shared environmental factors (C), and non-shared environmental factors (E). Shared environmental factors refer to non-genetic influences that contribute to similarity within pairs of twins. Non-shared environmental factors are those experiences that make siblings dissimilar, and this parameter also includes measurement error. Heritability is the proportion of total phenotypic variance due to genetic variation. To get a first indication of the underlying sources of variance in SCOR magnitude, comparisons were made among twin intraclass correlations (Twin-1 - Twin-2 correlations). A DZ intraclass correlation approximately half the value of the MZ intraclass correlation would indicate the presence of additive genetic effects, whereas a DZ intraclass correlation more than half an MZ intraclass correlation indicates the presence of both genetic and shared environmental effects. However, this is a descriptive approach which does not specifically identify latent factors underlying covariance across measures. Thus, formal genetic modeling is necessary to test the accuracy of the inferences made from these observations (Neale & Cardon, 1992).

Biometric Analyses

Within each time point univariate models were fit to estimate the relative contributions of additive genetic factors (A), shared environmental factors (C), and non-shared environmental factors (E), to SCOR magnitude. To test for sex differences in the variance components, a model in which the genetic and environmental effects were allowed to differ between boys and girls were compared against a model in which the estimates were constrained to be equal. A saturated model, which estimates the variances, covariances, and means of SCOR magnitude, were first fit and used as a baseline model to which subsequent models were compared.

In addition to the univariate genetic analyses within each time point, an Independent Pathway model was fit to the three time points simultaneously. In this model, genetic and environmental effects are of two types: common and specific. The model includes genetic, shared environmental, and non-shared environmental effects that are common to SCOR magnitude across the three time points. The model also specifies genetic, shared environmental and non-shared environmental effects that are specific to each measurement time point. That is, the degree to which SCOR magnitude across development share common genetic and environmental factors will be reflected in the relative loadings of the common versus age-specific factors. We did not constrain the age-specific non-shared environmental loadings to zero as that would imply, unrealistically, that SCOR magnitudes were measured without error (McArdle & Goldsmith, 1990).

Genetic correlations (r_g) among SCOR magnitudes at the three time points were also estimated using a multivariate Cholesky factor decomposition model. A multivariate Cholesky model decomposes the variance at each time point, as well as the co-variances among time points into additive genetic, shared environmental, and non-shared environmental factors. The genetic correlation indicates the extent to which individual

differences in SCOR magnitude at two time points reflect overlapping (i.e., correlated) genetic influences. This statistic varies from -1.0 to +1.0, and is independent of the extent to which each trait is influenced by genetic factors (Posthuma et al., 2003). It should be noted that an Independent Pathway model and a multivariate Cholesky model including three variables estimates the same number of parameters. A saturated model, in which the means and 6×6 matrices of variances and covariances among the three measures in each twin were freely estimated, was used as a baseline model to which subsequent multivariate models were compared.

There are several assumptions related to the classical twin design. Chief among these assumptions is that MZ twin pairs are no more likely than DZ pairs to share the environmental factors that are etiologically relevant to the phenotype under study. If this equal environment assumption is violated, then higher correlations among MZ twins may be due to environmental factors, rather than genetic factors and heritability estimate may be overestimated. A more detailed discussion of these and other assumption in the classical twin design can be found elsewhere (Neale & Cardon, 1992).

Models were fit with the structural equation program Mx (Neale et al., 2003), using a maximum likelihood estimation procedure for raw data. Raw maximum likelihood yields a goodness of fit index called log-likelihood. The adequacy of fit is assessed by computing twice the difference between the log-likelihood of a full model and that of a submodel, in which parameters are fixed to be zero or constrained to be equal. This difference follows a χ^2 distribution with the difference in the number of estimated parameters in the two models as the degrees of freedom. A significant χ^2 indicates that the model with fewer parameters to be estimated fits the data worse. The suitability of the models was also determined by comparing the model's Akaike Information Criterion ($AIC = -2LL - 2 \cdot df$). The AIC represents the balance between model fit and the number of parameters (parsimony), with lower values indicating the most suitable model (Akaike, 1987). The last model-selection statistic was the Bayesian Information Criterion ($BIC = -2LL + df \ln N$), where increasingly negative values correspond to increasingly better fitting models (Raftery, 1995).

Results

Descriptive Statistics

Table 1 presents the number of participants, means and standard deviations for the raw (untransformed) SCOR magnitudes. No significant mean or variance differences were found between twin-1 and twin-2, nor were there any mean or variance differences in SCOR magnitude between zygosity groups during any of the time points. Significant mean differences were found between boys and girls at ages 9-10 years, with boys showing higher mean values ($t = 2.36$, $df = 1,159$, $p = 0.02$). The phenotypic stability (r_p) across the three time points were moderate, between time 1 and time 2 $r_p = .35$, between time 1 and time 3 $r_p = .42$, and between time 2 and time 3 $r_p = .45$.

Twin Correlations

The MZ intraclass correlations were larger than the DZ intraclass correlations at all three time points, see Table 2, suggesting genetic influences for SCOR magnitude. At the third visit, the DZ intraclass correlations were slightly more than half the MZ intraclass correlations, indicating both genetic and shared environmental influences. All MZ intraclass correlations were less than one, which suggests the presence of non-shared environmental influences.

Biometric Analyses

Model-fitting results for each of the three time points are displayed in Table 3. A full ACE model described the data better than the baseline saturated model at all three time points (e.g., visit 1 Model # 2, $\chi^2 = 12.03$, $df = 9$, $p = .21$). A model constraining genetic and environmental components to be equal in boys and girls provided a better fit than the full ACE model (e.g., visit 1 Model # 3, $\chi^2 = .45$, $df = 3$, $p = .93$). This model could be further reduced by dropping the shared environmental component (e.g., visit 1 Model # 4, $\chi^2 = .00$, $df = 1$, $p = 1.00$), and this AE model with parameter estimates constrained to be equal across boys and girls was deemed the best fitting and most parsimonious model for SCOR magnitude at each time point (e.g., visit 1 Model # 4, AIC = 842.13, BIC = -2128.69). Genetic influences accounted for 56% ($p < .05$), 83% ($p < .05$), and 48% ($p < .05$) of the phenotypic variance at time points 1, 2 and 3, respectively, and non-shared environmental factors accounted for the remaining variance in SCOR magnitude: 44% ($p < .05$), 17% ($p < .05$), and 52% ($p < .05$), at time points 1, 2, and 3, respectively.

Next, a series of longitudinal multivariate models were fit to the data across the three time points simultaneously. As there were no significant sex differences in the univariate analyses, the longitudinal analyses were conducted combining MZ twins into one group and DZ twins into another group. The Independent Pathway model provided a better fit to the data than the saturated model based on the AIC and BIC criteria (Table 4, Model # 2, AIC = 1320.14, BIC = -3788.52), and did not significantly differ from the saturated model ($\chi^2 = 45.16$; $df = 33$; $p = .08$). This model could be reduced by dropping common shared environmental influences (Model # 2a), age-specific shared environmental influences (Model # 2b), common non-shared environmental influences (Model # 2c) and age-specific genetic effects at time point 3 (Model # 2d).

Figure 1 displays standardized estimates for the common (A) and age-specific (As) genetic effects as well as for the age-specific (Es) non-shared environmental effects from the reduced Independent Pathway model (Model 2d).

Three results are noteworthy. First, squaring and summing the genetic paths that load on SCOR magnitude at each time point gives the heritability estimates (Time 1: $.60^2 + .42^2 = .54$, or 54%; Time 2: 80%; and Time 3: 49%) These results are consistent with the heritability estimates derived from the univariate models, apart from slight variation in the parameter estimates resulting from additional information available in cross-twin cross-age covariance. Second, a common genetic factor explained the majority of the covariance in SCOR magnitude across the three time points. Specifically, this common genetic factor explained 36% (i.e., $.60^2$), 45% and 49% of the total variance in SCOR across development. Third, age-specific genetic effects were found at ages 9-10 years and ages 11-13 years, explaining 18 % and 35% of total variance at each age, respectively. Age-specific non-shared environmental effects (including measurement error) were found at all three time points; explaining 46%, 20% and 51% of the phenotypic variance.

Genetic correlations were estimated using a multivariate Cholesky decomposition model. This multivariate Cholesky decomposition model also provided a better fit to the data than the saturated model based on the AIC and BIC criteria (Table 4, Model # 3, AIC = 1320.04, BIC = -3788.57), and did not significantly differ from the saturated model ($\chi^2 = 45.06$; $df = 33$; $p = .08$). This model could be further reduced by dropping the shared environmental effects (Model # 3a, $\chi^2 = 7.42$; $df = 6$; $p = .28$). The genetic correlations (r_g) for SCOR magnitude are: $r_g = .55$ (95% CI, .32-.75) between time point 1 and 2, $r_g = .71$ (95% CI, .55-.87) between time point 1 and 3, and $r_g = .73$ (95% CI, .42-.98) between time point 2 and 3. This indicates a very strong genetic stability for SCOR from late childhood through adolescence.

Discussion

The key findings in the current study were that SCOR magnitude is heritable across development from ages 9 to 16 years. Genetic influences accounted for 56%, 83%, and 48% of the variance in SCOR magnitude at ages 9-10, 11-14 and 16-17 years, respectively, with the remaining variance due to non-shared environmental influences. SCOR magnitude was moderately stable across development, with phenotypic correlations ranging from .35 to .45. Longitudinal model fitting results showed that the majority of the variance in SCOR was explained by a common genetic factor. This common genetic factor explained 36%, 45% and 49% of the variance in SCOR magnitude across development. Age-specific genetic effects were found at ages 9-10 years and ages 11-13 years, explaining 18% and 35% of the variance, respectively. Further, the genetic correlations between the three time points were high (time 1 to time 2, .55; time 1 to time 3, .73; and time 2 to time 3, .71), indicating a large continuity of the same genetic influences across time. To the authors' knowledge, this is the first behavioral genetic study examining the genetic and environmental etiology of SCOR magnitude from late childhood to adolescence.

We found that within each assessment SCOR magnitude was primarily explained by genetic and non-shared environmental influences. This finding is well in line with findings from previous research on SC using adult samples (Crider et al., 2004; Lykken et al., 1988). These studies have reported that genetic and non-shared environmental factors each explain about half of the variance in various indices of SC responses. In our study, genetic influences accounted for 55%, 82%, and 48% of the variance in SCOR magnitude at ages 9-10, 11-14 and 16-17 years, respectively. This finding that genetic effects vary across development has been found for other phenotypes as well, for example, heritability estimates of cognitive functioning increase from around 30% in preschool children to 80% in early adolescence (Polderman et al., 2006). Further, our longitudinal analyses showed that age-specific genetic effects were important at ages 9-10 years and ages 11-13 years. In other words, not all genetic influences were accounted for by the common genetic factor across development. This indicates that there may be genetic effects that uniquely impact SCOR magnitude at these ages, over and above the genetic influence from the common genetic factor. It has been suggested that genes that are activated at puberty may be an important influence on traits or behaviors (Jacobson, Prescott & Kendler, 2002). It is possible that the age-specific genetic effect found at ages 11-13 years in the current study are related to the onset of puberty.

Similar to previous research, we found no evidence of significant shared environmental influences in SCOR magnitude. The absence of shared environmental influences does not imply that environmental influences are of no importance. Rather, this finding indicates that those environmental experiences unique to each twin are especially important in explaining individual differences in SCOR magnitude. These non-shared environmental experiences may for example include pre- and postnatal experiences, differential treatment by parents, and head trauma.

We found no evidence for sex specific effects at any of the visits, and genetic and environmental components could be equated across sexes. This indicates that the relative magnitude of genetic and non-shared environmental factors in SCOR magnitude is the same in boys and girls.

Our longitudinal analyses showed that a common genetic factor explained the variance in SCOR magnitude across development. Non-shared environmental influences (including measurement error) were age-specific. The longitudinal analyses further demonstrated that genetic influences were stable across development. The genetic correlations between each

pair of time points were rather high, indicating a large continuity of the same genetic influences on SCOR magnitude across the three time points. The importance of stable genetic influences has previously been reported in studies on child psychopathology (Eley, Lichtenstein & Moffitt, 2003; Haberstick et al., 2005; O'Connor et al., 1998).

The finding that genetic factors were the most important influence explaining the variance in SCOR magnitude, cross-sectionally as well as longitudinally, has important implications. It is possible that a genetic predisposition for a psychopathological phenotype is partly expressed through biological correlates, e.g., a psychophysiological characteristic, such as abnormal autonomic orienting to various stimuli in one's environment (Raine, 2002). An important next step would therefore be to examine the genetic covariation between SCOR magnitude and those psychopathological behaviors that have been found to be correlated with SCOR magnitude.

The results of this study are subject to several potential limitations. First, the sample size at the second visit was relatively small; this could have affected the outcome. For example, with a larger sample we might have been able to detect significant shared environmental influences. Therefore it will be important to replicate our findings in larger samples with more power.

Second, our method to assess SCOR does not allow the differentiation of the influences of central and peripheral nervous system. Peripheral factors such as number of sweat glands are associated with SC, especially the SC levels, a measure of arousal (Freedman et al., 1994), but firing up those glands in the orienting paradigm is in a way over and above those peripheral influences (and over and above tonic SC levels). Specifically, SCOR has been found to be sensitive to a variety of stimuli, including stimulus novelty, intensity, arousal content, and significance, and it has been widely used as a psychophysiological index of information processing capability (Dawson et al., 2007). Among all forms of autonomic nervous system activity, individual differences in SC responses appear to be most reliably associated with psychopathological states, again suggesting that it is not all noise. The psychological implications of SCOR are further manifested by the reported significant relationship between psychopathic personality and SCOR magnitude in 9-10-year olds (Isen et al., 2010), and the fact that this personality trait continues to be linked to SCOR magnitude in our male participants during adolescence. Despite these findings, we cannot tease out the possibility that the heritability of SCOR magnitude found in our study mainly reflects the characteristics of underlying physiological rather than psychological factors.

In conclusion, our results showed that SCOR magnitude was roughly equally explained by genetic and non-shared environmental factors within each measurement time point. A common genetic factor explained the majority of the variance in SCOR magnitude across development. These findings increase our understanding of SCOR by demonstrating the importance of genetic influences for individual differences in SCOR magnitude.

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Research Highlights

- Skin conductance orienting response (SCOR) was primarily explained by genetic and non-shared environmental influence.
- SCOR was moderately stable across development.
- The genetic correlations between waves were high, indicating a substantial continuity in genetic influences for SCOR from ages 9 to 16.

Figure 1.

Best-fitting Independent pathway model, showing one twin in a pair: circles indicate variance components, and rectangles indicate observed variables. SCOR= skin conductance orienting response. Standardized variance components: Ac=common additive genetic component; Au= age-specific additive genetic component; Eu= age-specific non-shared environmental component.

Means, Standard Deviations, and Number of Participants (n) for Skin Conductance Orienting Response (SCOR) across Visits (Ages 9-10, 11-13 and 14-16 Years), by Sex and Zygosity

Table 1

	Boys		Girls		DZ opposite sex	
	MZ	DZ	MZ	DZ	Boys	Girls
Means (Standard Deviations)						
Visit 1 (ages 9-10) N=1,157	.43 (.42) n=261	.41 (.37) n=164	.36 (.36) n=277	.38 (.36) n=173	.38 (.36) n=143	.33 (.31) n=139
Visit 2 (ages 11-13) N=161	.33 (.36) n=40	.30 (.26) n=21	.22 (.37) n=36	.24 (.32) n=20	.16 (.29) n=27	.35 (.44) n=17
Visit 3 (ages 14-16) N=642	.52 (.47) n=131	.43 (.42) n=98	.46 (.44) n=141	.43 (.38) n=117	.47 (.43) n=77	.38 (.35) n=78

Notes.

MZ=monozygotic, DZ=dizygotic, N and n=numbers of individual participants; results presented for untransformed SCOR values

Table 2
Intraclass Correlations for Skin Conductance Orienting Response (SCOR) across Visits (Ages 9-10, 11-13 and 14-16 Years)

	MZ male	DZ male	MZ female	DZ female	DZ - OS
Visit 1 (ages 9-10)	.58*	.24*	.55*	.31*	.25*
Visit 2 (ages 11-13)	.81*	.31	.82*	.47*	.61*
Visit 3 (ages 14-16)	.54*	.34*	.46*	.36*	.16

Notes.
MZ=monozygotic, DZ=dizygotic, DZ-OS=dizygotic opposite sex,
* $p < .05$; results presented for transformed SCOR values

Table 3
Univariate Model Fitting Analyses for Skin Conductance Orienting Response at Ages 9-10, 11-13, and 14-16 Years

Model #	Overall fit						Chi-square difference test					Parameter estimates (95% CI)	
	-2LL	df	AIC	BIC	χ^2	df	p	Compared to #	χ^2	df	p	A	E
<i>Visit 1 (age 9-10)</i>													
1 Saturated	3153.66	1149	855.66	-2093.40									
2 ACE (boys ≠ girls)	3165.69	1158	849.69	-2116.14	12.03	9	.21						
3 ACE (boys = girls)	3166.13	1161	844.13	-2125.50	12.47	12	.41	2	.45	3	.93		
4 AE (boys = girls)	3166.13	1162	842.13	-2128.69	12.47	13	.49	3	.00	1	1.00	.56 (.48-.62)	.44 (.38-.52)
5 CE (boys = girls)	3185.28	1162	861.28	-2119.12	31.62	13	.002	3	19.15	1	<.001		
6 E (boys = girls)	3295.36	1163	969.36	-2067.27	141.70	15	<.001	3	129.23	2	<.001		
<i>Visit 2 (age 11-13)</i>													
1 Saturated	363.49	134	95.49	-109.29									
2 ACE (boys ≠ girls)	369.63	149	83.63	-125.77	6.14	9	.73						
3 ACE (boys = girls)	371.93	146	79.93	-131.13	8.44	12	.75	2	2.30	3	.51		
4 AE (boys = girls)	372.95	147	78.95	-132.79	9.46	13	.74	3	1.02	1	.31	.83 (.71-.89)	.17 (.11-.29)
5 CE (boys = girls)	380.15	147	86.15	-129.20	16.66	13	.22	3	8.22	1	.004		
6 E (boys = girls)	424.68	148	128.68	-109.10	61.19	15	<.001	3	52.75	2	<.001		
<i>Visit 3 (age 14-16)</i>													
1 Saturated	1775.78	630	515.78	-965.36									
2 ACE (boys ≠ girls)	1784.11	639	506.11	-987.67	8.33	9	.50						
3 ACE (boys = girls)	1786.50	642	502.50	-995.30	10.72	12	.55	2	2.39	3	.50		
4 AE (boys = girls)	1786.98	643	500.98	-997.99	11.20	13	.59	3	.48	1	.49	.48 (.36-.58)	.52 (.42-.64)
5 CE (boys = girls)	1789.87	643	503.87	-996.55	14.09	13	.37	3	3.37	1	.07		
6 E (boys = girls)	1833.20	644	545.20	-977.83	57.42	15	<.001	3	46.7	2	<.001		

Notes.

-2LL=-2(log-likelihood), AIC=Akaike's Information Criterion, BIC=Bayesian Information Criterion, χ^2 =difference in log-likelihoods between models, df=change in degrees of freedom

Table 4
Longitudinal Model Fit Indices for Skin Conductance Orienting Response for Three Visits

	Models #	Overall fit					Chi-square difference test					
		-2LL	df	AIC	BIC	χ^2	df	P	Compared to #	χ^2	df	P
1	Saturated	5154.98	1907	1340.98	-3702.42							
2	Independent Pathway	5200.14	1940	1320.14	-3788.52	45.16	33	.08	1			
2a	Drop common shared environmental effects	5203.87	1943	1317.87	-3796.53	48.89	36	.07	3	3.73	3	.29
2b	Drop unique shared environmental effects	5204.54	1946	1312.54	-3806.08	49.56	39	.12	3	4.4	6	.62
2c	Drop common non-shared environmental effects	5207.30	1949	1309.30	-3814.57	52.32	42	.13	3	7.16	9	.62
2d	Drop unique genetic effects	5208.37	1950	1308.37	-3817.33	53.39	43	.13	3	8.23	10	.61
3	Multivariate Cholesky	5200.04	1940	1320.04	-3788.57	45.06	33	.08	1			
3a	Drop shared environmental effects	5207.46	1946	1315.46	-3804.62	52.48	39	.07	2	7.42	6	.28

Notes.

-2LL=-2(log-likelihood), AIC=Akaike's Information Criterion, BIC=Bayesian Information Criterion, χ^2 =difference in log-likelihoods between models, df=change in degrees of freedom