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Genetic and Environmental Architecture of Human Aggression

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A meta-analysis was performed on data from 24 genetically informative studies by using various personality measures of aggression. There was a strong overall genetic effect that may account for up to 50% of the variance in aggression. This effect was not attributed to methodological inadequacies in the twin or adoption designs. Age differences were important. Self-report and parental ratings showed genes and the family environment to be important in youth; the influence of genes increased but that of family environment decreased at later ages. Observational ratings of laboratory behavior found no evidence for heritability and a very strong family environment effect. Given that almost all substantive conclusions about the genetics of personality have been drawn from self or parental reports, this last finding has obvious and important implications for both aggression research in particular and personality research.

It has been fairly well established that aggression and antisocial behavior run in families. Researchers have either seen them as delinquency (Rowe, Rodgers, & Meseck-Bushey, 1992), criminality (Mednick, Moffit, Gabrielli, & Hutchings, 1986), conduct disorder (Jary & Stewart, 1985), or antisocial personality (Cadoret, 1978). However, although similarity among family members for aggressive or antisocial behavior has been evident, the study of intact nuclear families has not been able to trace this similarity to shared genetic influences, shared familial environmental factors, or some combination of both genes and environment.

The purpose of this article is to provide an overview of twin and adoption studies on the personality construct of aggression so that genetic and environmental influences may be unconfounded and thus provide more insight into why relatives are similar. Most behavioral genetic studies have used the twin design, estimating heritability and environmentality through the comparison of monozygotic (MZ) and dizygotic (DZ) twin intraclass correlations. However, results have varied from one study to another, even when the same instrument of measure has been used. We illustrate this in the present article with the Psychopathic-deviate (Pd) scale of the Minnesota Multiphasic Personality Inventory (MMPI). Gottesman (1963), reporting on 68 pairs of adolescent twins, gave correlations of .57 for MZ twins and .18 for DZ twins, which suggests a substantial genetic effect. Correlations of similar magnitude were also found by Rose on a larger study of 410 adolescent twin pairs (Rose, personal communication, July 18, 1986). However, although Pogue-Geile and Rose (1985) found significant genetic effects at Age 20, no significant genetic variance was detected at Age 25. Reznikoff and Honeyman (1967) also failed to find significant heritability for the Pd scale in their sample of 34 adult twin pairs.

Studies of children also have had variable findings. Some studies have reported significant heritability (Lytton, Watts, & Dunn, 1988; O'Connor, Foch, Sherry, & Plomin, 1980; Scarr, 1966). Others have reported little genetic influence (Owen & Sines, 1970), whereas yet another suggested heritability was important for males but not for females (Stevenson & Graham, 1988).

Adoption studies on aggression again have shown variable results. The Texas Adoption Project found modest correlations between biological mother and adoptee, but correlations of nearly zero for adopted relationships (Loehlin, Willerman, & Horn, 1985, 1987). However, adoptive siblings in the Colorado Adoption Project correlated .85 (Rende, Slomkowski, Stocker, Fulker, & Plomin, 1992).

Recent reviews of this literature suggest an overall consensus that there is some genetic influence on aggression and antisocial behavior (Carey, 1994; Gottesman & Goldsmith, 1994). However, as discussed above, there is striking variability among these studies. Hence, we think that it is justified to examine in a rigorous quantitative fashion the degree to which this variability can be attributed to factors such as age, sex, measuring instrument, and, of course, statistical sampling error. Meta-analysis allows one to use differential scaling for variable methods of measurement and to account for sample size differences by weighing results from samples accordingly. Through this procedure, researchers can explore how factors such as age and sex may moderate the genetic and environmental architecture of aggressive and antisocial behavior.

Studies

The samples selected for this review include the twin and adoption studies tabulated by Carey (1994) in the National Research Council report on violence, plus additional studies

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published since that report was originally written in 1989. We included a study if it used any measure of aggression, hostility, or antisocial behavior or if the scale was specifically constructed to predict juvenile delinquency. Results for male participants and female participants were treated separately unless the original study reported only results pooled for gender. Table 1 and Table 2 present the samples and data used in this review.

We coded the following variables for the meta-analysis: zygosity (for twins), biological versus adoptive relationship (for adoption studies), sex, age, and type of measurement. Age was coded as youth (mean sample age of 18 or younger) versus adult (over age 18). (Other methods of coding age, such as children, adolescent, and adult, were also tried, but results did not substantively differ and hence are not presented.) Type of measurement was coded into three categories: self report (SR), parental report (PR), and observational (OB). Table 3 presents the number of studies that fit into each of the categories.

We would have liked to code the actual measurement instrument according to its construct or its predictive validity with respect to aggression per se as opposed to general antisocial behavior, which would include aggression among a much wider range of behavior. However, we could not think of an objective way of coding these data along this line for two reasons. First, we examined items from those studies that included a scale with the word *aggression* in the title. Despite the same title, there was a significant amount of heterogeneity of item content, ranging from projective techniques (Owen & Sines, 1970), to an intrapunitive sense of guilt and self-blame (Partanen, Bruun, & Markkanen, 1966), to an excellent psychometric scale with item content ranging from relatively minor feelings of anger and retribution to overt acts of assault (Tellegen et al., 1988). Second, it is well established that the extreme aggression that would be called violence (sexual assault, robbery, murder, etc.) is strongly correlated with other aspects of antisocial behavior (e.g., vandalism, theft). As a consequence, some well-validated scales that predict general antisocial behavior (e.g., the MMPI Pd scale) may actually be more predictive of interpersonal, physical aggression than other scales that are specifically called aggression scales. Lacking intercorrelation matrices among these scales and lacking correlations between the scales and a common criterion variable of interpersonal aggression, we decided on a different tactic, outlined below in the *Method* section, to arrive at an index of measuring a common construct.

Study 1

Method

The purpose of this study was to examine the validity of two crucial assumptions for twin and adoption data: the absence of selective placement for the trait and the equal environments assumption for MZ and DZ twins. Selective placement occurs when there is some correlation in the environments of biological relatives who are raised in separate households. Strong selective placement in environments relevant to aggression and antisocial behavior will compromise interpretation of the studies of twins raised apart and adoptees that are presented in Tables 1 and 2. The second assumption is that identical twins raised together do not experience more similar environments for antisocial behavior and aggression than do fraternal twins raised together. Given evidence of twin imitation with regard to registered criminality (Carey, 1992) and the presence of selective placement in some adoption studies (Plomin, DeFries, & Fulker, 1988), it is crucial to examine both of these assumptions in the data set before proceeding with substantive analysis.

We performed three analyses for the three instruments of measure presented in Tables 1 and 2 that have been gathered on both twins and adoptees—the California Psychological Inventory Socialization (CPI So) scale, the MMPI Pd scale, and the Multidimensional Personality Questionnaire (MPQ) Aggression scale.

The model of analysis assumes that the phenotype (P) is a linear function of genotype (G) and environment (E), giving the structural equation $P = hG + eE$, where h and e are regression weights. We assumed the simple model of additive gene action with random mating. We also assumed that the phenotype of parents (P_m and P_f for mother and father, respectively) impinge on the environments of their offspring (E_o), giving the structural equation $E_o = tP_m + tP_f + U$, where U is a residual and the ts are regression weights. We also assumed that the environments of siblings are correlated by the amount r_s in addition to the correlation in their environments that results from the influence of the parental phenotypes. We used two parameters to measure selective placement. We used the quantity s to denote the correlation between a biological parent and an adoptive parent of an adopted child. We used the quantity w to measure the extent to which the environments of twins raised apart are correlated with respect to siblings raised in the same household; $w = 0$ implies that the environments for twins raised apart are random, and $w = 1$ implies that the environments of twins raised apart are as similar as those of siblings and twins raised together. Finally, we used the parameter a to denote imitative effects with separate as for MZ and DZ twins (see Carey, 1992). The expected correlations are presented in Table 4.

This full model is not identified with the data at hand. However, it is possible to fix certain parameters to examine the effect of the violations of assumptions on other parameter estimates. Typically, models used in behavioral genetics have assumed no selective placement of either singleton adoptees or twins raised apart. This is one extreme model in which $s = w = 0$. At the other extreme, one can assume perfect selective placement so that the correlation between biological parent and adoptive parent is at its upper mathematical limit of 1 and that the correlation for the environments of twins raised apart is equal to that of siblings raised together (i.e., $w = 1$).

We fit five models to each of the three scales. The first allowed perfect selective placement with the possibility of genetic influence. The second also had selective placement but no heritability. The third and fourth models assumed no selective placement with heritability (Model 3) or no heritability (Model 4). The final model fitted only heritability.

Results

The results of fitting selective placement models to the MMPI Pd scale are presented in Table 5. Model 1, in which there is perfect selective placement, gives a satisfactory fit. However, Model 2 demonstrates that heritability cannot be set to 0; even under such extreme circumstances, this model must be rejected on the basis of both goodness of fit and likelihood ratio (LR), $\chi^2(1) = 14.83$, $p < .001$. A model that takes the extreme genetic position of no selective placement also fits well (Model 3), but again, heritability cannot be set to 0 (Model 4). In terms of information, the last model fits best. It uses only one parameter, h^2, and satisfactorily accounts for all the data.

Table 6 gives the results of fitting these data to the CPI So scale. These results parallel those for the MMPI. The two models that assume no heritability, Models 2 and 4, must be rejected. There is no evidence for selective placement, and the simplest

Table 1
Twin Studies Included in Meta-Analysis

Study	Sample	Measure used	Group	Male		Female		Age (years)	Method
				N	*r*	*N*	*r*		
Twins raised together, genders analyzed separately									
Gottesman (1966)	1	CPI Socialization	MZ	34	.32	45	.52	14–25	SR
			DZ	32	.06	36	.26	14–25	SR
Partanen, Bruun, & Markkaren (1966)	2	Aggression items	MZ	157	.25	—	—	28–37	SR
			DZ	189	.16	—	—	28–37	SR
Scarr (1966)	3	ACL n Aggression	MZ	—	—	24	.35	6–10	PR
			DZ	—	—	28	−.08	6–10	PR
Owen & Sines (1970)	4	MCPS Aggression	MZ	10	.09	8	.58	6–14	SR
			DZ	11	−.24	13	.22	6–14	SR
Loehlin & Nichols (1976)	5	CPI Socialization	MZ	202	.52	288	.55	18	SR
			DZ	124	.15	193	.48	18	SR
		ACL n Aggression	MZ	216	.20	293	.24	18	SR
			DZ	135	−.05	195	.06	18	SR
Rowe (1983)	6	No. of delinquent acts	MZ	61	.62	107	.66	13–18	SR
			DZ	38	.52	59	.46	13–18	SR
Rushton, Fulker, Neale, Nias, & Eysenck (1986)	7	23 aggression items from IBS	MZ	90	.33	106	.43	19–60	SR
			DZ	46	.16	133	.00	19–60	SR
			DZ-OS	98	.12	—	—	19–60	SR
Lytton, Watts, & Dunn (1988)	8	Rutter Antisocial subscale	MZ	13	.89	—	—	9	PR
			DZ	20	.67	—	—	9	PR
Stevenson & Graham (1988)	9	Rutter Antisocial subscale	MZ	46	.61	53	.29	13	PR
			DZ	48	.40	58	.49	13	PR
Twins raised together, genders pooled									
Gottesman (1963, 1966) Reznikoff & Honeyman (1967)	1	MMPI Psychopathy	MZ	120	.48	—	—	14–18	SR
			DZ	132	.27	—	—	14–18	SR
Canter (1973)	10	Acting Out Hostility of Foulds Hostility Scale	MZ	39	.14	—	—	16–55	SR
			DZ	44	.30	—	—	16–55	SR
O'Connor, Foch, Sherry, & Plomin (1980)	11	Conners's bullying	MZ	52	.72	—	—	5–11	PR
			DZ	32	.42	—	—	5–11	PR
Plomin, Foch, & Rowe (1981)	11	Median (three objective aggression ratings)	MZ	53	.39	—	—	5–11	OB
			DZ	32	.42	—	—	5–11	OB
Pogue-Geile & Rose (1985)	12	MMPI Psychopathy	MZ	71	.35	—	—	20–25	SR
			DZ	62	.18	—	—	20–25	SR
Rose (personal communication July 18, 1986)	12	MMPI Psychopathy	MZ	228	.47	—	—	14–34	SR
			DZ	182	.23	—	—	14–34	SR
Ghodsian-Carpey & Baker (1987)	13	CBC Aggression	MZ	21	.78	—	—	4–7	PR
			DZ	17	.31	—	—	4–7	PR
		MOCL Aggression	MZ	21	.65	—	—	4–7	PR
			DZ	17	.35	—	—	4–7	PR
Tellegen et al. (1988)	14	MPQ Aggression	MZ	217	.43	—	—	19–41	SR
			DZ	114	.14	—	—	19–41	SR
Twins raised apart, genders pooled									
Gottesman, Carey, & Bouchard (1984)	15	MMPI Psychopathy	MZ	51	.64	—	—	19–68	SR
			DZ	25	.34	—	—	19–68	SR
Tellegen et al. (1988)	15	MPQ Aggression	MZ	44	.46	—	—	19–68	SR
			DZ	27	.06	—	—	19–68	SR
Bouchard & McGue (1990)	15	CPI Socialization	MZ	45	.53	—	—	19–68	SR
			DZ	26	.39	—	—	19–68	SR

Note. Dashes indicate data were not obtained. SR = self-report; PR = parental report; OB = observational data; CPI = California Psychological Inventory; ACL = Gough's Adjective Checklist; n = number of adjectives checked; MCPS = Missouri Children's Picture Series; IBS = Interpersonal Behavior Survey; MMPI = Minnesota Multiphasic Personality Inventory; CBC = Child Behavior Checklist; MOCL = Mothers' Observations Checklist; MPQ = Multidimensional Personality Questionnaire; DZ = dizygotic; MZ = monozygotic; DZ-OS = dizygotic opposite sex.

model of only heritability (Model 5) gives a satisfactory fit by using only one parameter.

The fits for the MPQ Aggression scale are given in Table 7. The results are very similar but are not identical to those for the MMPI and CPI. Once again, the best fitting model is Model 5, which explains the observed correlations by using only h^2, and heritability cannot be set to 0 when there is no selective placement. The difference between this MPQ Aggression and

Table 2
Adoption Studies Included in Meta-Analysis

Study	Sample	Measure used	Relationship	*N*	*r*	Age (years)	Method
Loehlin, Willerman, & Horn (1985)	16	CPI Socialization	Adoptive father–child	241	.00	parents	SR
			Adoptive mother–child	253	−.02	39–76	SR
			Biological father–child	52	.16	39–76	SR
			Biological mother–child	53	.06	39–76	SR
			Adoptive–adoptive siblings	76	.03	14–36	SR
			Adoptive–biological siblings	47	.10	14–36	SR
			Biological–biological siblings	15	−.01	14–45	SR
Loehlin, Willerman, & Horn (1987)	16	MMPI Psychopathy	Adoptive father–child	180	.07	parents	SR
			Adoptive mother–child	177	.01	39–76	SR
			Biological father–child	81	.12	39–76	SR
			Biological mother–child	81	.07	39–76	SR
			Birth mother–adopted child	133	.27	39–76	SR
			Adoptive–adoptive siblings	44	.02	14–36	SR
			Adoptive–biological siblings	69	.06	14–36	SR
			Biological–biological siblings	20	−.06	14–45	SR
Parker (1989)	17	CBC Aggression	Adoptive siblings	45	.47	4–6	PR
			Biological siblings	66	.44	4–6	PR
Rende, Slomkawski, Stacker, Fulker, & Plomin (1992)	17	Conflict scale	Adoptive siblings	57	.85	3–11	OB
			Biological siblings	67	.91	3–11	OB

Note. SR = self-report; PR = parental report; OB = observational data; CPI = California Psychological Inventory; MMPI = Minnesota Multiphasic Personality Inventory; CBC = Child Behavior Checklist.

the MMPI and CPI occurs in comparing Model 2 with Model 1. With the MPQ scale, heritability can be set to 0 under perfect selective placement; it cannot be set to 0 under perfect selective placement with the MMPI or CPI scales.

The importance of this analysis lies less in its providing a strong argument for the presence of heritability than in its demonstrating that even if selective placement were occurring in the extreme, genetic effects would still be present. The appropriate data points here are the estimates of heritability under Model 5 in contrast with the estimates under Model 1 in Tables 5, 6, and 7. Despite the unreasonable assumption of perfect selective placement, heritability estimates change from .48 to .44 (MMPI Pd), from .53 to .40 (CPI So), and from .43 to .42 (MPQ Aggression). Selective placement and correlations in the environments of twins raised apart reduce heritability, as they should. They do not make the influence of heritability go away, and they do not dramatically alter the magnitude of the genetic influence.

Perfect selective placement implies that the personnel at an adoption agency would be able to ascertain perfectly all aspects of psychopathy in a biological mother and in an adoptive mother and father and would then be able to match them. Even if MMPI Pd scores were available on all three parents, the limited number of adoptees available at any one time would prevent perfect matching on observed scores.

Table 3
Studies Separated by Method of Report

Method (age in years)	Twins raised: Together	Twins raised: Apart	Adoption	*N*	Sex analyzed separately[a]
Self-report					
Young (6–25)	5	0	0	5	4
Old (14–76)	6	3	2	11	2
Parent report (4–11)	5	0	1	6	3
Observational (3–11)	1	0	1	2	0

[a] Only twins raised together were analyzed separately by sex.

Study 2

Method

In this section, we outline the methods that we used for the actual meta-analysis. The first and most general model expresses heritability

Table 4
Expected Correlations for Different Kinship Correlations

Kinship correlation	Expected correlation
Adoptive parent–offspring	te
Adoptive siblings	$(2t^2 + r_s)e^2$
Genetic parent–adoptive child	$\frac{1}{2}h^2 + 2st^2e^2$
Siblings	$\frac{1}{2}h^2 + (2t^2 + r_s)e^2$
DZ twins together	$[(1 + a_{DZ}^2)y_{DZ} + 2a_{DZ}]/(1 + a_{DZ}^2 + 2a_{DZ}y_{DZ})$
MZ twins together	$[(1 + a_{MZ}^2)y_{MZ} + 2a_{MZ}]/(1 + a_{MZ}^2 + 2a_{MZ}y_{MZ})$
DZ twins apart	$\frac{1}{2}h^2 + w(2t^2 + r_s)e^2$
MZ twins apart	$h^2 + w(2t^2 + r_s)e^2$

Note. $y_{DZ} = \frac{1}{2}h^2 + (2t^2 + r_s)e^2$; $y_{MZ} = h^2 + (2t^2 + r_s)e^2$. DZ = dizygotic; MZ = monozygotic; a_{DZ} = imitative effect for DZ twins; a_{MZ} = imitative effect for MZ twins; e = environmental effect; h^2 = heritability; r_s = environmental correlation for siblings; s = selective placement coefficient; t = regression weight; w = environmental correlation for twins raised apart.

Table 5
Testing Assumptions on the MMPI Pd Scale

Model	Goodness of fit			Parameter estimates				
	df	χ^2	*p*	h^2	*t*	r_s	a_{DZ}	a_{MZ}
1: Perfect selective placement[a]	8	8.16	.42	.44	.00	.16	−.03	−.04
2: Perfect selective placement,[a] no heritability	9	22.99	<.01	—	.10	.22	.00	.13
3: No selective placement[b]	8	9.12	.33	.51	−.04	−.02	.00	−.02
4: No selective placement,[b] no heritability	9	41.41	<.0001	—	.06	.02	.11	.23
5: Only heritability[c]	13	9.55	.73	.48	—	—	—	—

Note. Dashes indicate parameter was set to zero. MMPI = Minnesota Multiphasic Personality Inventory; Pd = Psychopathic-deviate; DZ = dizygotic; MZ = monozygotic; a_{DZ} = imitative effect for DZ twins; a_{MZ} = imitative effect for MZ twins; h^2 = heritability; r_s = environmental correlation for siblings; s = selective placement coefficient; t = regression weight; w = environmental correlation for twins raised apart.
[a] $s = 0.5$, $w = 1$. [b] $s = w = 0$. [c] $s = w = t = r_s = a_{DZ} = a_{MZ} = 0$.

and common environment by using contrast codes for sex, age category, rating type (self vs. parental), and measurement mode (observational vs. psychometric). The specific formula for heritability was

$$\text{heritability} = h^2 + \beta_1\text{sex} + \beta_2\text{age} + \beta_3\text{report} + \beta_4\text{measure}, \quad (1)$$

where h^2 is a constant and β_1, β_2, β_3, and β_4 are parameters for sex, age, rating type, and measurement mode, respectively, in accordance with the observed correlation in Tables 1 and 2. Sex was coded numerically as -1 = *female sample*, 0 = *mixed female–male sample*, and 1 = *male sample*. Age was coded as 0 = *adult* and 1 = *youth*. Rating type was coded as 0 = *self-report* and 1 = *parental rating*. Measurement mode was coded as 1 = *observational* and 0 = *psychometric*. A similar equation was written for common environment:

$$\text{common environment} = c^2 + \delta_1\text{sex} + \delta_2\text{age} + \delta_3\text{report} + \delta_4\text{measure}. \quad (2)$$

Naturally, it would have been desirable also to code for interactions, but the presence of empty cells in the data made this impossible.

In family data, the observed correlation between relatives is the product of the reliability of the measure and the true correlation between the relatives. Thus, differences in reliability between measures will contribute to differences in observed correlations among samples. To control for this, we used the parameter α_i, where α is the reliability coefficient for the measure and i denotes the ith measure.

The predicted correlation for a pair of relatives from the jth sample using the ith measure becomes

$$r_{ij} = \alpha_i[\gamma_j(h^2 + \beta_1\text{sex}_j + \beta_2\text{age}_j + \beta_3\text{report}_j + \beta_4\text{measure}_j) + \eta_j(c^2 + \delta_1\text{sex}_j + \delta_2\text{age}_j + \delta_3\text{report}_j + \delta_4\text{measure}_j)]. \quad (3)$$

Here γ_j denotes the coefficient of genetic relatedness for the kinship in the jth sample. If the sample involves adoptive relatives, $\gamma_j = 0$; for biological parent and offspring, biological siblings, and DZ twins; $\gamma_j = .5$; and for MZ twins, $\gamma_j = 1.0$. The quantity η_j denotes the coefficient of environmental relationship of the jth sample; $\eta_j = 1.0$ when the relatives are raised together, and $\eta_j = 0$ when the relatives are raised apart.

This method permits all the predicted correlations for the samples in Tables 1 and 2 to be made functions of 25 parameters: h^2, the 4 betas, c^2, the 4 deltas, and the 15 alphas. The quantities γ and η will, of course, be fixed for the individual sample. For example, the predicted correlation for the CPI from an adult, mixed-sex sample of identical twins raised apart is

Table 6
Testing Assumptions on the CPI So Scale

Model	Goodness of fit			Parameter estimates				
	df	χ^2	*p*	h^2	*t*	r_s	a_{DZ}	a_{MZ}
1: Perfect selective placement[a]	12	18.11	.11	.40	−.03	.13	.03	.03
2: Perfect selective placement,[a] no heritability	13	27.43	<.01	—	.01	.20	.06	.18
3: No selective placement[b]	12	19.01	.07	.46	−.04	.05	.04	.02
4: No selective placement,[b] no heritability	13	35.66	<.0007	—	.01	.05	.15	.26
5: Only heritability[c]	16	22.25	.14	.53	—	—	—	—

Note. Dashes indicate parameter was set to zero. CPI = California Personality Inventory; So = Socialization; DZ = dizygotic; MZ = monozygotic; a_{DZ} = imitative effect for DZ twins; a_{MZ} = imitative effect for MZ twins; h^2 = heritability; r_s = environmental correlation for siblings; s = selective placement coefficient; t = regression weight; w = environmental correlation for twins raised apart.
[a] $s = 0.5$, $w = 1$. [b] $s = w = 0$. [c] $s = w = t = r_s = a_{DZ} = a_{MZ} = 0$.

Table 7
Model-Fitting Results on the MPQ Aggression Scale

Model	Goodness of fit			Parameter estimates				
	df	χ^2	*p*	h^2	*t*	r_s	a_{DZ}	a_{MZ}
1: Perfect selective placement[a]	2	1.01	.60	.42	−.04	.07	−.02	−.06
2: Perfect selective placement,[a] no heritability	3	3.01	.39	—	.18	.23	.08	−.08
3: No selective placement[b]	2	0.67	.71	.43	−.05	.11	−.04	−.07
4: No selective placement,[b] no heritability	3	10.24	<.02	—	.18	.22	.09	−.07
5: Only heritability[c]	6	2.30	.89	.43	—	—	—	—

Note. Dashes indicate parameter was set to zero. MPQ = Multidimensional Personality Questionnaire; DZ = dizygotic; MZ = monozygotic; a_{DZ} = imitative effect for DZ twins; a_{MZ} = imitative effect for MZ twins; h^2 = heritability; r_s = environmental correlation for siblings; t = regression weight; w = environmental correlation for twins raised apart.
[a] $w = 1$. [b] $w = 0$. [c] $w = t = r_s = a_{DZ} = a_{MZ} = 0$.

$$\alpha_{CPI}(h^2). \tag{4}$$

Similarly, the correlation for the young, mixed-sex, DZ twins on the Child Behavior Checklist (CBC) in Ghodsian-Carpey and Baker (1987) is

$$\alpha_{CBC}[1/2(h^2 + \beta_2 + \beta_3) + (c^2 + \delta_2 + \delta_3)]. \tag{5}$$

An exact method for fitting the model to the data and for assessing the fit of the model is not possible with the information available. The obstacle is the dependence among several observed correlations that are presented in Tables 1 and 2. For example, the correlations for the MMPI, CPI, and MPQ on the Minnesota series of twins raised apart (Bouchard & McGue, 1990; Gottesman, Carey, & Bouchard, 1984; Tellegen et al., 1988) came from three different reports from the same series of twins. To control for this statistical dependency, we would need the raw data from this sample and would need to use quantitative pedigree analysis.

We chose to treat observed correlations from such overlapping samples as independent. This approach leads to very minimal bias in the parameter estimates but may lead to conservative hypothesis testing (McGue, Wette, & Rao, 1984). That is, there is more power to reject a false hypothesis, but this is gained at the expense of increased Type I errors. Simulations, however, suggest that this bias is not large (McGue et al., 1984). Hence, the fitting function we used was

$$\chi^2 = \sum_{i=1}^{k} (N_i - 3)(\hat{z}_i - z_i)^2, \tag{6}$$

where i denotes the ith data point in Tables 1 and 2, N denotes the sample size for that correlation, $\hat{z}_i$ denotes the zeta transformation of the predicted correlation, and z_i denotes the zeta transformation of the observed correlation.

Also, at least one of the αs must be fixed to permit identification. To avoid estimates of reliability considerably greater than 1.0, we fixed α for the sample with the largest correlations (Rende et al., 1992) to be 1.0 and the αs for the MMPI, MPQ, and CPI to be .80, close to their test–retest correlations over a short interval.

Results

Table 8 presents the results of the fitting of models to all studies included in Tables 1 and 2. The general model fits all the parameters. The numbered models set one or more effects to zero for both heritability and common environment. For example, in Model 1, sex = 0 sets both β_1 and δ_1 to zero.

Because the actual parameter estimates depend on arbitrary decisions for fixing the values of the αs, we scaled our estimates so that the sum of the absolute values of each row equals zero. In this way, the absolute values of the estimates for each row give the percentage of the familiality for aggression attributable to a variable. For example, in the general model, $\beta_1 = .05$ and $\delta_1 = -.05$, so that 10% of familiality may be attributable to gender differences, with heritability being slightly higher in males and common environment being slightly higher in females. Similarly, the largest source of variance in the general model (and in all other models where the parameters are free) is attributable to mode of measurement. Observational studies account for 45% (.16 + .29) of the familial variability, greatly decreasing heritability and increasing common environment.

The fits of the models in Table 8 may be judged by the two columns labeled GOF p and LR p. The first is the p value for the goodness-of-fit chi-square. The second is the p value for the likelihood ratio chi-square, comparing one of the numbered models in Table 8 against the general model. Because of the lack of independence among the correlations, the p values should not be interpreted literally as rejecting or not rejecting models. Instead, they should be viewed as nonparametric estimates of fit, with larger p values suggesting satisfactory fits and smaller p values implying poor fits.

The most striking conclusion one can draw from Table 8 concerns the magnitude of the mode of measurement effect—an observational rating of videotaped or real-life performance versus a parental or self-report. In every case in which mode of measurement was set to zero, models had poor fits on the basis of either the GOF or the LR chi-square. Similarly, for every model where measurement was fitted, this effect was the largest predictor of familiality, accounting for roughly 50% of the general differences among the correlations in Tables 1 and 2. The effect was also consistent: Observational ratings greatly decreased heritability and increased common environment.

In interpreting all of those models in which the measurement effect was fitted (Models 1, 2, 3, 5, 6, 8, and 11, and the general

Table 8
Model Fits and Proportions of Familial Resemblance for All Data

Model	χ^2	*df*	GOF *p*	LR *p*	h^2	Sex β_1	Age β_2	Report β_3	Measurement β_4	c^2	Sex δ_1	Age δ_2	Report δ_3	Measurement δ_4
General	66.44	57	.18		.22	.05	−.01	−.04	−.16	.00	−.05	.05	.13	.29
1: Sex = 0	69.54	59	.16	.21	.25	.00	−.03	−.09	−.17	.00	.00	.07	.07	.32
2: Age = 0	72.39	59	.11	.05	.21	.05	.00	−.06	−.17	.01	−.06	.00	.15	.30
3: Report = 0	69.01	59	.18	.28	.27	.05	−.07	.00	−.14	.00	−.05	.11	.00	.30
4: Measurement = 0	76.37	59	.06	<.01	.22	.00	−.23	.24	.00	.00	−.01	.26	−.03	.00
5: Sex, age = 0	76.61	61	.09	.04	.24	.00	.00	−.13	−.19	.01	.00	.00	.10	.33
6: Sex, report = 0	71.38	61	.17	.29	.30	.00	−.08	.00	−.15	.00	.00	.13	.00	.33
7: Sex, measurement = 0	79.94	61	.05	<.01	.22	.00	−.24	.17	.00	.00	.00	.27	−.11	.00
8: Age, report = 0	82.67	61	.03	<.01	.26	.07	.00	.00	−.20	.02	−.07	.00	.00	.37
9: Age, measurement = 0	84.86	61	.02	<.001	.39	.10	.00	−.06	.00	.09	−.12	.00	.25	.00
10: Report, measurement = 0	161.50	61	<.001	<.001	.32	.02	−.29	.00	.00	.00	−.03	.35	.00	.00
11: Sex, age, report = 0	86.42	63	.03	<.01	.30	.00	.00	.00	−.24	.03	.00	.00	.00	.43
12: Sex, age, measurement = 0	9.33	63	<.01	<.001	.49	.00	.00	−.22	.00	.11	.00	.00	.17	.00
13: Age, report, measurement = 0	241.41	63	<.001	<.001	.54	.15	.00	.00	.00	.15	−.16	.00	.00	.00
14: Sex, age, report, measurement = 0	244.95	65	<.001	<.001	.78	.00	.00	.00	.00	.22	.00	.00	.00	.00
15: No heritability	239.58	61	<.001	<.001	.00	.00	.00	.00	.00	.18	−.03	.28	.22	.29
16: No common environment	17.77	61	<.001	<.001	.24	−.01	.05	.18	.51	.00	.00	.00	.00	.00

Note. Coded such that for sex (β_1, δ_1), male = 1, mixed male–female = 0, female = −1; for age (β_2, δ_2), adult = 0, child or adolescent = 1; for report (β_3, δ_3), self = 0, parental = 1; for measurement (β_4, δ_4), psychometric = 0, observational = 1. GOF = goodness of fit; LR = likelihood ratio; h^2 = heritability; c^2 = common environment.

model), it appears that the influence of sex, age, and type of report was consistent but not very strong. Heritability was slightly more influential in male participants than in female participants, whereas common environment was more important in female participants than in male participants. The effect of heritability was lesser and that of common environment greater among younger samples compared with adult samples. Finally, parental reports resulted in lower heritability but greater common environment than did psychometric self-reports. The worst model fits occurred when age was set to zero (Models 2, 5, 8, and 11).

Observational data were available for only two studies: Plomin, Foch, and Rowe (1981) and Rende et al. (1992). Because of the large observational effect, we redid the analysis eliminating these two studies as a post hoc exploration of the effects of sex, age, and type of report. Results from this analysis are presented in Table 9. The fundamental patterning of results remained unchanged. Models that assume no heritability or no common environment (Models 9 and 10) gave the worst fits. Models with no age effect had less satisfactory fits than did those that permit no sex and report effect. The most parsimonious model was Model 6, which permits only age differences to moderate the genetic and environmental architecture of aggression.

General Discussion

The meta-analysis gives four major conclusions. The first major conclusion is that heritability and common environment are definitely responsible for individual differences in aggression. It is highly unlikely that heritability is a methodological artifact. Even the unrealistically high estimates of an artifact effect in Study 1 do not dramatically alter heritability. Unless there is some as yet unidentified methodological flaw in the twin or adoption strategies or both, the results from Study 2 suggest an important contribution of heritability for aggression, perhaps accounting for as much as 50% of the variance.

The second important conclusion concerns the extent to which observational methods of measuring aggression gave different results for either self-report or parental report. The two observational studies suggested a very strong influence of common environment with little evidence of heritability, a result that cannot be explained by the age of the two samples (both were young). The reason for this is unclear. Perhaps this is a chance finding—there were, after all, only two studies. Perhaps different aspects of aggression are tabulated by observers than are reported by individuals. Or perhaps one or both of the studies capitalized on state-specific, reciprocal influences of twin or adoptive dyads when they are tested at the same time. The very large sibling correlations of Rende et al. (1992)—.85 for adoptive siblings and .91 for biological siblings—are, to our knowledge, much larger than any other ever reported for any behavioral measure. They are also consistent with the possibility of capturing an episode of dyadic interaction where both siblings are either aggressive or nonaggressive.

Whatever the cause, this result suggests the need for returning to multitrait–multimethod strategies (Campbell & Fiske, 1959) for measuring aggression in families. Indeed, in the behavioral genetics literature, there have been very few attempts to assess a similar personality construct in multiple modes of measurement. Virtually all results on adult personality have been based on self-reports (for recent reviews, see Eaves, Eysenck, & Martin, 1989; Loehlin, 1992). The very possibility of obtaining different types of results from a different mode of measurement has strong implications for research on the genetics of all personality traits, not simply aggression.

The third conclusion, made in a more tentative fashion than

Table 9
Model Fits and Proportions of Familial Resemblance for Aggression, Deleting Two Observational Studies

Model	χ^2	*df*	GOF *p*	LR *p*	h^2	Sex β_1	Age β_2	Report β_3	c^2	Sex δ_1	Age δ_2	Report δ_3
1: General	66.36	54	.12		.40	.08	−.03	−.07	.00	−.10	.09	.23
2: Sex = 0	69.46	56	.11	.21	.49	.00	−.05	−.18	−.01	.00	.13	.14
3: Age = 0	72.31	56	.07	.05	.40	.10	.00	−.11	.01	−.11	.00	.27
4: Report = 0	68.93	56	.11	.28	.49	.09	−.13	.00	.00	−.10	.20	.00
5: Sex, age = 0	76.53	58	.05	.04	.49	.00	.00	−.27	.02	.00	.00	.22
6: Sex, report = 0	71.30	58	.11	.29	.58	.00	−.16	.00	−.01	.00	.25	.00
7: Age, report = 0	82.59	58	.02	<.01	.61	.16	.00	.00	.06	−.17	.00	.00
8: Sex, age, report = 0	86.35	60	.01	<.01	.91	.00	.00	.00	.09	.00	.00	.00
9: No heritability	237.40	57	<.001	<.001	.00	.00	.00	.00	.25	−.04	.40	.31
10: No common environment	83.97	57	<.01	<.001	.50	−.03	.10	.38	.00	.00	.00	.00

Note. Coded such that for sex (β_1, δ_1), male = 1, mixed male–female = 0, female = −1; for age (β_2, δ_2), adult = 0, youth = 1; for report (β_3, δ_3), self = 0, parental = 1. GOF = goodness of fit; LR = likelihood ratio; h^2 = heritability; c^2 = common environment.

the prior two, is that the genetic and family environmental architecture of aggression may change over time. Longitudinal studies of individuals highlight both the consistency of aggression (Farrington, 1986, 1989) and the changes in developmental patterns over time (Moffitt, 1993; Patterson, 1992). However, there are no longitudinal genetic data researchers could use to test how much consistency and change may be due to genes and environment. The results of our cross-sectional meta-analysis suggest that in youth, genes and common environment equally promote similarity among relatives. For adults, however, the influence of common environment is negligible but that of heritability increases. These results are completely consistent with the literature on juvenile versus adult criminality in twins (Carey, 1994; Gottesman & Goldsmith, 1994; Rowe, 1990; Rowe & Rodgers, 1989). Common environment has been very important in juvenile delinquency, whereas genes have been relatively more important for adult criminality. This phenomenon suggests that aspects of the family (imitating or reacting to the same parents, living in the same neighborhood, having overlapping groups of friends, etc.) may be very important in the initiation and early maintenance of aggression but may fade over time.

We hypothesized that this age effect is due to what has been informally termed the "smorgasbord" model[1] of gene–environment interplay. This model analogizes the various tidbits on the smorgasbord to different types of environments and the taste preference of the diner to a genotype. Faced with a large number of choices, the diner samples a little of each but then returns to the most enjoyable dishes. Thus, the adult genotype ends up choosing the environments most compatible with the genotype. With aggression in youth, the initial choices on the table may be limited by the family environment. Departing from the family household as an adult, one experiences a broader range of environments that either positively or negatively reinforce aggression. The adult then prefers those environments compatible with his or her genotype, creating a gene–environment correlation that is indistinguishable from heritability. The smorgasbord model is consistent with longitudinal data that suggest that familial factors such as parental inconsistency and failure to set limits predict juvenile and adolescent antisocial behavior (Patterson, Capaldi, & Bank, 1991; Patterson, DeBarsyshe, & Ramsey, 1989). However, testing this hypothesis requires genetically informative, longitudinal data that also include appropriate measures of the environment.

The fourth, and indeed most tentative, conclusion is that of a relative lack of common environment, at least in adults, for aggressive behavior. Although this finding is consistent with Rowe's (1994) review and conclusions about a minor role for family environment, we hesitate to endorse his position in the cases of aggression and antisocial behavior. There are two reasons for our view.

First, we assumed in creating our model that all gene action is additive. This means that there is virtually no genetic dominance or gene–gene interaction for every single locus predicting individual differences in aggression. When this assumption is violated, twin data will overestimate heritability and underestimate common environment.

Second, the adoption data that might resolve the issue of additivity in the twin design have very serious problems of their own when they are applied to severe antisocial behavior, including aggression. Those families with exceptionally high risk environments for violence are seldom allowed to adopt. Examples would include a single-parent household, extreme poverty, and parental alcohol abuse, substance abuse, and criminality. If this tail of the distribution is missing among adoptive families, the influence of family environment may be underestimated, particularly when the effect is nonlinear. That is, those families screened out of the adoption pool may have a very strong influence, whereas the regression line between family environment and offspring aggression is flat among those intact and psychologically healthy families permitted to adopt. For these reasons, we feel confident that heritability is important for aggression and that the causes of familial resemblance change over time, but we await the results of future research before quantifying the effects of the family environment.

Much of personality psychology has yet to face the implications of important heritability for aggression. One dominant

[1] We attribute the "smorgasbord" analogy to Lindon Eaves (personal communication, August 4, 1984). The ideas of gene–environment covariance behind it are discussed in Eaves, Last, Martin, and Jinks (1977); Plomin, DeFries, and Loehlin (1977); and Scarr and McCartney (1983).

perspective that is used to explain the familial resemblance for aggression is social learning theory (Bandura & Walters, 1959). According to this theory, a child learns to behave aggressively by observing aggressive behavior, encoding these acts to memory, and incorporating aggressiveness into his or her code of conduct (Bandura, 1986). Although peers and the media are important contributors to a child's behavior, it is the family environment that may have the greatest influence on the similarity found among family members.

The family environment may also foster the development of aggression through such means as interactions among family members and parental disciplinary techniques. Through detailed observation of aggressive and nonaggressive families, Patterson (1982) has identified a particular coercive pattern that serves to elicit, maintain, and increase aggression among family members. According to this pattern, a child often acts aggressively in reaction to the aversive behavior of another family member. If the other family member withdraws from the interchange and the aversive behavior is stopped, the child's aggressive behavior is reinforced, encouraging similar behavior in the future. This coercion process tends to be found in families that are having problems with an aggressive child. Other factors found among aggressive families are lack of parental monitoring, parental aggression, permissiveness or inconsistency in discipline, and parental rejection (Perry, Perry, & Boldizar, 1990). These environmental factors provide models and reinforcement such that a child learns to act aggressively, thereby increasing a child's level of aggressive behavior.

The results of our meta-analysis suggest that genetics must be seriously considered when explaining the similarity found in the levels of aggression among family members. Adult samples and data on self-report measures and parental ratings on aggressive behavior have suggested moderate heritability with some small influence from common environment. These results do not support the social learning theory, which emphasizes the role of common environmental effects. The observed pattern of correlations for parental ratings and self-report data cannot be explained by methodological biases. Individual analyses on the MMPI Pd, CPI So, and the MPQ Aggression scales all lead to similar results. Each of these measures suggests moderate levels of heritability, supporting the importance of genetic influences.

It has only been fairly recently that researchers have considered biological–genetic factors important in the development of human aggression. The main areas in which researchers have focused include the relationship between aggression and hormones, temperament, arousal, and the central nervous system. Hormone research has concentrated mainly on the correlation between elevated levels of testosterone and aggressive behavior. However, the variable results of several studies have led to difficulty in making any conclusions. Other studies have found relationships between aggression and neurochemicals related to adrenaline. For example, delinquents have been reported to show lower than average levels of epinephrine (adrenaline) and norepinephrine (Zuckerman, 1989). Zuckerman also reported that undersocialized conduct disorder is associated with low levels of dopamine-beta-hydroxylase, the enzyme that breaks down dopamine. Antisocial individuals have also been reported to show lowered levels of monoamine oxidase, an enzyme that breaks down the neurotransmitters serotonin, dopamine, epinephrine, and norepinephrine (Ellis, 1991).

For temperament, infant difficultness has been found to be related to parental ratings of hostility in preschool-age children (Parke & Slaby, 1983). Parke and Slaby also suggested that the way an infant's temperament influences parent–child interactions may account for the coercive patterns of aggression seen by Patterson (1982) that were mentioned earlier. Personality characteristics, which may play a role in the expression of aggression, have also been found to be heritable. A recent twin study found significant heritabilities for empathy, behavioral inhibition, and expressions of negative affect in infants (Emde et al., 1992). Personality research on adults has found antisocial behavior to be highly correlated with high scores on the three personality dimensions of the Eysenck Personality Questionnaire: extraversion, neuroticism, and psychoticism, which have all shown significant heritability (Eysenck & Gudjonsson, 1989).

A strong connection between physiological arousal and aggression has also been demonstrated by several researchers (Berkowitz, 1988; Brancombe, 1985; Zillmann, 1988). However, researchers have emphasized that the relationship is interactive and that aggression occurs only in very specific circumstances. On the other hand, Eysenck and Gudjonsson (1989) proposed the general arousal theory of criminality, which suggests that the inheritance of a nervous system insensitive to low levels of stimulation may make an individual inclined to participate in high-risk activities associated with antisocial behavior, such as crime, substance abuse, and sexual promiscuity, in order to increase their arousal. Studies on the central nervous system have suggested that defects in the limbic system and cortical functioning may also play a role in aggressive behavior (Eichelman, 1983; Gorenstein, 1990). Research in each of these areas has just begun to suggest possible biological influences on aggression.

Aggression is also a common behavior among many primate species, and agonistic behavior is often adaptive. For example, in many groups, agonistic behavior seems to be about maintaining dominance relationships (Lauer, 1992). In turn, a dominance hierarchy helps to limit aggression through the development of stable, cooperative, and nonaggressive relations among members of the group. Such relationships promote individual fitness by diminishing the likelihood of injury or death that might result from continued aggression. Perhaps similar adaptive mechanisms exist for humans. Observational data available on preschool children suggest that agonistic exchanges ending with submission revealed dominance hierarchies (Strayer, 1992). Strayer speculated that differences in dominance ranking during the preschool years may contribute to individual differences in later acquisition of social skills. However, few ethological studies focus on the development of agonistic behavior, and most studies are performed on young children.

An evolutionary theory of sociopathy proposed by Mealey (1995) suggests that sociopathy develops through two pathways. *Primary* sociopaths participate in antisocial behavior because of a lack of normal moral development and an inability to feel social responsibility, which is a product of their genotype, physioptype, and personality. On the other hand, *secondary* sociopathy is contingent on the environment and develops in re-

sponse to disadvantages in social competition. These individuals participate in antisocial behavior because they are unable to succeed through socially acceptable means at a particular time. Secondary sociopathy is also associated with other risk factors, such as low socioeconomic status, urban residency, low intelligence, and poor social skills, whereas primary sociopathy is not associated with a particular background. Although secondary sociopathy appears to be more responsive to the environment, both types of sociopathy may be heritable and may account for some of the genetic evidence that has been found. This brief overview suggests only some of the possible mechanisms that may lead to aggression and the development of antisocial behavior, and the search for other possible adaptive mechanisms merits further research.

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