A META-ANALYSIS OF THE GENE-CRIME RELATIONSHIP*

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This investigation used the statistical technique of meta-analysis to probe the putative association between heredity and crime. The data for this study were 54 effect sizes obtained from 38 family, twin, and adoption studies on crime. In addition to the overall gene-crime relationship, the potential moderating effects of gender, sample nationality, date of publication, and quality of the research design were also investigated. It was predicted that heredity and crime would not coincide, although subsequent analyses disclosed a low-moderate correlation between these two variables (mean unweighted phi coefficient = .25; mean weighted phi coefficient = .09). Further analysis of these data revealed that better designed and more recently published studies provided less support for the gene-crime hypothesis than more poorly designed and earlier published investigations. The individual strengths and weaknesses of the meta-analytic technique relative to this effort to achieve insight into the gene-crime relationship are discussed.

Research addressing the putative relationship between heredity and human behavior has generated controversy, debate, and strong opinion from both sides. Nowhere is this more evident than in studies exploring the possibility of a gene-crime nexus. While the majority of studies on this topic intimate that certain forms of criminal activity may be influenced by genetic factors, Walters and White (1989) conclude that many of those studies are so methodologically flawed that they defy singular interpretation. Responding to this summation, Brennan and Mednick (1990) question the objectivity and specificity of many of Walters and White's criticisms, which Walters (1990) counters by reasserting that the problem lies with genetic research studies on crime, not the Walters and White review. Since this controversy shows no signs of abating, it would appear that the time has come for a more objective

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analysis of the relationship presumed to exist between genetic inheritance and criminal conduct.

One way by which such an appraisal might be accomplished is through application of the meta-analytic research technique. Meta-analysis allows for the collection of aggregate data from a series of research reports, which are then converted to a common scale and reanalyzed using various statistical techniques. In contrast to narrative reviews of the literature, which consider the qualitative side of a particular research question, meta-analyses conceptualize outcomes derived from individual studies as data points amenable to quantitative analysis. A common statistic, the effect size, is calculated for each study and then averaged, summarized, and analyzed using standard statistical procedures. (The phi coefficient (Φ), calculated from 2×2 tables of criminal outcomes (present, absent), serves as the effect size measure in this investigation.) The individual strengths and weaknesses of the meta-analytic technique have been discussed at length by other investigators (Glass et al., 1981; Hedges and Olkin, 1985; R. Rosenthal, 1984; Wilson and Rachman, 1983), although there is sufficient evidence to suggest that the meta-analytic technique provides yet another avenue through which the gene-crime question might be addressed.

Three primary methodologies have been employed in research on the heritability of criminal conduct: family studies, twin studies, and adoption studies. Family studies are grounded in the knowledge that family members are more genetically similar than nonfamily members. Initially, an index group comprising proband (persons who display the trait or behavior under study) and control (persons who do not display the trait or behavior under study) subjects is identified. Next, the prevalence of a target trait or behavior (e.g., criminality, delinquency) is measured in the relatives, often first-degree relatives (parents, siblings, children), of the proband and control subjects. A possible genetic effect is indicated when the trait or behavior is found more frequently in the relatives of proband subjects than in the relatives of control subjects. However, because family members share many of the same environmental experiences, family studies cannot distinguish between the individual contributions of heredity and common environment.

Twin studies address the question of heritability by comparing monozygotic (single-egg) and dizygotic (dual-egg) twins, armed with the knowledge that monozygotic (MZ), or identical, twins possess greater genetic similarity than do dizygotic (DZ), or fraternal, twins. This is because MZ twins share the same genetic inheritance, whereas DZ twins share only half their genes. A genetic effect is suggested when the concordance rate (presence of the trait or behavior in both twins) is higher in MZ twins than in DZ twins. Like family studies, however, the twin method suffers from a potential

confounding of genetic and environmental influences because research suggests that MZ twins tend to be treated more similarly by others, spend significantly more time together (Kidd and Matthysee, 1978), and share a greater sense of mutual identity (Dalgard and Kringlen, 1976) than do DZ twins.

Because the family and twin methods were found to be less than ideal for isolating genetic and environmental contributions to criminal outcome, adoption studies were introduced. Adoption studies cross-lag and compare the behavior of biological and adoptive parents with the behavior of index subjects adopted away from their biological homes at a relatively early age. The advantage of the adoption method is that the shared environmental effect of being raised in a particular home and the effect of biological parentage can be studied separately due to the fact that the subject was not raised by his or her biological parents. Although the adoption method of investigating the gene-crime relationship is superior to the family and twin methods, potential problems of interpretation arise when the adoption does not take place shortly after birth or the adoption agency follows a practice of matching the biological and adoptive homes on such potentially important characteristics as family income or socioeconomic status (see Walters and White, 1989). It is possible to combine these individual methods, as exemplified by studies comparing MZ twins reared apart and reared together, although mixed-method studies are rarely found in the gene-crime literature.

The decision to employ a case-to-case statistical model, such as is provided by the phi coefficient, rather than a case-to-base rate model, such as has been suggested by Gottesman and Carey (1983) in their use of the tetrachoric coefficient procedure, is central to the logic of this meta-analysis. A procedure that allows for direct comparisons of subjects from the same sample—whether this involves contrasting subjects with and without a family history of criminality or examining the relative concordance of MZ and DZ twins—seems to capture more clearly the spirit of gene-crime research than a procedure that pits subject groups against some estimated population base rate. After all, the purpose of the twin method is to compare MZ and DZ twins with each other rather than with an estimate of the population base rate, which research suggests, may vary by as much as 20 percentage points from one study to the next.

Researchers in the area of behavior genetics often make use of the heritability coefficient (h), in which the concordance rate is doubled to compensate for the fact that genetic studies do not typically compare genetically identical pairs of subjects with genetically unrelated pairs of subjects. Twin studies, as noted, compare MZ twins, who share 100% of their genetic inheritance, with DZ twins, who share 50% of their genetic inheritance. Adoption studies, on the other hand, contrast child-biological parent pairs, who share 50% of their genes in common, with child-adoptive parent pairs, who are genetically unrelated. Consequently, researchers in the field of behavior genetics estimate the

heritability coefficient by doubling the concordance rate or simple correlations obtained in family, twin, or adoption studies. The heritability coefficient was not used to estimate effect sizes in this investigation because (1) data sufficient to calculate this coefficient were not always available, (2) the case-by-case analysis employed seemed more compatible with other major meta-analyses of criminology-related issues (cf. Andrews et al., 1990; Tittle et al., 1977), and (3) the heritability coefficient, regardless of how scientific it may sound, is no more immune from interpretation problems introduced by a research design that confounds genetic and environmental influences than any other statistic (see Trasler, 1987).

This investigation was undertaken to document the existence, strength, and magnitude of a heredity-crime relationship. The principal null hypothesis was that various indices of genetic inheritance and criminal outcome would not correlate. In addition to assessing the overall relationship between heredity and crime, the effect sizes for family, twin, and adoptive studies were calculated individually. Finally, the influence of several potentially important moderating variables (subject gender, nationality of the research sample, publication date of the referenced article, quality of the research design) on the gene-crime relationship was gauged. The respective null hypotheses predicted that each of the four moderating variables (gender, nationality, publication date, design quality) would fail to modify the gene-crime relationship significantly. The rationale for selecting these variables was based on their facility of operationality, prior inclusion in other meta-analyses, and potential relevance to the criminal justice field.

METHOD

SAMPLE OF STUDIES

Thirty-eight family, twin, and adoption studies yielding data pertinent to the proposed affiliation of heredity and crime indicators were subjected to meta-analysis. The studies were identified through an exhaustive review of the genetic literature on crime, and all major studies addressing the gene-crime relationship were included. Several of the studies, however, used overlapping (or even identical) samples. Data from overlapping studies were only included in the meta-analysis if they shed new light on the gene-crime question by way of variations in procedure, methodology, or the measurement of criminal outcome.

PROCEDURE

The 38 studies included in this meta-analysis produced 54 2 $\times$ 2 contingency tables relevant to the hypothesized link between heredity and crime. In

studies following a family methodology, the gene-crime correlation was estimated by comparing the family criminal backgrounds of proband and control index subjects. This is a relatively weak test of the gene-crime hypothesis in that family studies are incapable of distinguishing between genetic and environmental influences. With regard to twin studies, the gene-crime association was calculated on the basis of a comparison of concordance rates for MZ and DZ twins. The genetic status-criminal outcome connection was quantified in adoption studies by contrasting the rate of biological parent criminality observed in the backgrounds of criminal, adopted-away offspring with the rate of biological parent criminality observed in the backgrounds of noncriminal controls.

The phi coefficient was used to measure the strength and direction of the putative association between heredity and crime. In line with recommendations made by R. Rosenthal (1984), these phi coefficients were transformed into Fisher z_r's for the purpose of combining and comparing the results of individual studies. These z_r values were also used to construct a mean Z estimate by which the statistical significance of the average effect sizes for family, twin, adoption, and combined sample studies was evaluated. The mean z_r values were subsequently converted back to phi coefficients following completion of the data analysis phase of this study. In addition to a standard, unweighted mean estimate of phi, a weighted mean estimate, based on the value of z_r and degrees of freedom ($N = 3$) for the particular study in question, was also calculated.

Four potential moderating variables were also examined: gender, nationality, year of publication, and quality of the research design. Gender of the research sample was coded male, female, or (in studies that failed to provide separate rates for males and females) both. The nationality of the research sample was coded according to whether the study was conducted in the United States or in a foreign (usually European) nation. The year 1975 served as the cutoff point for the publication date measure; studies published prior to 1975 were put into one category and studies published in 1975 or thereafter were put into a second category. The year 1975 was selected as the cutoff because it was the year in which D. Rosenthal published one of the first critical reviews of genetic studies on crime with clear implications and recommendations for future research.

The quality of a study's research design was rated adequate (+) when it satisfied three (four in the case of twin studies) basic criteria: use of a clearly defined outcome measure of criminality, use of a control group, at least 10 subjects per research cell, and for twin studies, a reliable measure of zygosity. Studies failing to satisfy any one of the three (or for twin studies, four) criteria were judged to have had problems with their research design and were given a rating of (−) on this measure. A second rater provided independent ratings of the design quality measure for all 54 gene-crime comparisons, the results of

which indicated a respectable degree of interrater reliability for the two sets of ratings (K = .86). The four gene-crime comparisons on which there was disagreement were discussed and a consensus rating was derived.

RESULTS

The studies included in this meta-analysis of the gene-crime literature are summarized in Tables 1 (family studies), 2 (twin studies), and 3 (adoption studies). The three tables provide information on each study's location, basic subject characteristics (sample size, gender), and the results obtained for proband and control subjects. In each case the criterion behavior is the definition of criminality employed in the study, be it a global definition like delinquency or a more specific delineation such as documented convictions or results from a formal self-report measure of past criminal activity. The rates of familial criminality attained by proband and control index subjects are also provided in each table—familial being defined as biological parents or first-degree relatives in family studies, co-twins in twin studies, and biological parents in adoption studies. The status of the four moderating variables and the phi coefficient effect sizes for each of the 54 comparisons generated by the meta-analysis are also provided in these tables.

Characteristics of the overall relationship between heredity and crime, as well as the characteristics of the effect sizes for family, twin, and adoption studies are provided in Table 4. As is readily discernible from a cursory inspection of this table, the unweighted mean phi coefficient is modest (Φ = .25), and the weighted mean phi coefficient falls in the low-modest range (Φ = .09).* Nevertheless, the mean overall, family, twin, and adoption study effect sizes attained statistical significance using a procedure (Z transformation) that takes into account the size of the subject sample (see Table 4). A nonparametric binomial test of the proportion of positive to negative effect sizes revealed a pattern of results in favor of positively valenced effect sizes ($p < .001$). It should be noted, however, that the mean effect sizes obtained with adoption studies (weighted mean, Φ = .07; unweighted mean, Φ = .11), perhaps the strongest of the three methods commonly used to investigate the heritability of crime, are low.

* That the unweighted mean effect size is nearly three times the size of the weighted mean effect size can be explained by the fact that the adoption studies, by virtue of their large sample sizes, contributed disproportionally to the mean weighted effect size.

Table 1. Family Studies Addressing the Gene-Crime Relationship

		Index Subjects			Family Subjects				Outcome(%)			Moderating Var.			
Study	Location	*N*	Sex	Criteria	*N*	Sex	Relation	Criteria	Proband	Control	Φ	Sex	Nat	Pub	Des
Glueck & Glueck (1930)	Concord, Mass.	402	M	Delinquency	2,404	M&F	1st-degree relatives	Arrest of fam. mem.	53.2	26.7	.27	M	USA	Pre	−
Glueck & Glueck (1934)	Framingham, Mass.	426	F	Delinquency	3,165	M&F	1st-degree relatives	Arrest of fam. mem.	45.5	28.6	.25	F	USA	Pre	−
Robins & Lewis (1966)	St. Louis, Mo.	67	M	Delinquency	134	M&F	Parents	Delinquency/ criminality	28.0	13.0	.20	M	USA	Pre	+
Guze et al. (1967)	St. Louis, Mo.	93	M	Felony conviction	519	M&F	1st-degree relatives	Sociopathy	9.0	2.7	.13	M	USA	Pre	−
Cloninger & Guze (1973)	St. Louis, Mo.	66	F	Sociopathy	288	M&F	1st-degree	Arrest	26.0	3.8	.31	F	USA	Pre	−
					106	M&F	relatives	Sociopathy	19.8	2.8	.27	F	USA	Pre	−
Cloninger et al. (1975)	St. Louis, Mo.	58	M	Sociopathy	272	M&F	1st-degree	Sociopathy	10.3	2.2	.17	M	USA	Post	−
		28	F		115	M&F	relatives		27.8	4.3	.32	F	USA	Post	−
Robins et al. (1975)	St. Louis, Mo.	86	M	Delinquency	172	M&F	Parents	Delinquency/ criminality	43.0	0.0	.41	M	USA	Post	+
		59	F		118	M&F			23.0	0.0	.32	F	USA	Post	+
Stewart & Leone (1978)	Iowa City, Iowa	36	M	Unsocialized aggr. reaction	72	M&F	Parents	Antisocial personality	26.5	2.6	.35	M	USA	Post	−
Farrington (1979)	London, England	409	M	Self-reported delinquency	818	M&F	Parents	Criminal conviction	43.8	26.5	.21	M	For	Post	+
Lewis et al. (1983)	St. Louis, Mo.	131	M	Antisocial		M&F	1st-degree	Antisocial	18.2	5.2	.20	M	USA	Post	−
		281	F	personality		M&F	relatives	acts	41.3	14.1	.26	F	USA	Post	−
Farrington et al. (1988)	London, England	63	M	Felony conviction	126	M&F	Parents	Criminal conviction	78.3	41.2	.35	M	For	Post	+

NOTES: Outcome: Proband = percentage of criminal outcome index subjects with one or more criminal outcome relatives. Control = percentage of noncriminal index subjects with one or more criminal outcome relatives. Because all of the index subjects in the Glueck & Glueck (1930, 1934), Guze et al. (1967), Cloninger & Guze (1973), and Cloninger et al. (1975) studies had criminal outcomes, the control concordance for these studies was determined from a base-rate estimate of criminal outcome calculated on the basis of data collected by Cloninger et al. (1975) and organized according to the sex (male, female) and race (white, nonwhite) of family subjects in the proband conditon.

Moderating variables: Sex = gender of index subjects—M = males, F = females, and B = mixed samples of males and females; Nat = nationality of the subject sample—USA = a US-based sample and For = a European- or Asian-based sample; Pub = year of publication—Pre = a publication date earlier than 1975 and Post = a publication date of 1975 or later; Des = whether the design of the study satisfies three criteria, i.e., a clearly defined outcome, use of a control group, and an adequate sample size (+ vs −).

Table 2. Twin Studies Addressing the Gene-Crime Relationship

			Monozygotic Twins			Dizygotic Twins					Moderating Var.		
Study	Location	Criteria	*N*	Sex	Concord.(%)	*N*	Sex	Concord.(%)	Φ	Sex	Nat	Pub	Des
Lange (1930)	Germany	Criminal arrest	13	M	76.9	17	M	11.8	.66	M	For	Pre	−
LeGras (1932)	Holland	Imprisonment	4	M&F	100.0	5	M&F	0.0	.99	B	For	Pre	−
Rosanoff et	United	Criminal	38	M	76.3	23	M	21.7	.53	M	USA	Pre	−
al. (1934)	States	conviction	7	F	85.7	4	F	25.0	.59	F	USA	Pre	−
Kranz (1936)	Germany	Criminal arrest	31	M&F	64.5	43	M&F	53.5	.11	B	For	Pre	+
Stumpfl (1936)	Germany	Registered	15	M	60.0	17	M	41.2	.19	M	For	Pre	−
		criminality	3	F	66.7	2	F	0.0	.67	F	For	Pre	−
Slater (1938)	United Kingdom	Criminal arrest	2	M&F	50.0	10	M&F	30.0	.14	B	For	Pre	−
Borgstrom (1939)	Finland	Registered criminality	4	M&F	75.0	5	M&F	40.0	.36	B	For	Pre	−
Yoshimasu (1961)	Japan	Criminal arrest	28	M	60.7	18	M	11.1	.49	M	For	Pre	−
Hayashi (1967)	Japan	Delinquency	15	M	80.0	4	M	75.0	.06	M	For	Pre	−
Christiansen (1970)	Denmark	Registered criminality	67	M	35.8	114	M	12.3	.28	M	For	Pre	+
Dalgard &	Norway	Legal violat.	49	M	22.4	89	M	18.0	.05	M	For	Post	+
Kringlen (1976)		Felonies	31	M	25.8	54	M	14.8	.14	M	For	Post	+
Shields (1977)	United Kingdom	Delinquency	5	M	80.0	9	M	77.8	.03	M	For	Post	−
Rowe (1983)*	United	Self-Reported	61	M	73.8	38	M	68.4	.06	M	USA	Post	+
	States	antisocial acts	107	F	75.7	59	F	67.8	.09	F	USA	Post	+
Gurling et	United	Criminal	14	M&F	7.1	14	M&F	14.3	−.11	B	For	Post	−
al. (1984)	Kingdom	conviction											

NOTES: For monozygotic and dyzogotic twins: Concord. = percentage of criminal outcome index twins with criminal outcome co-twin. Because all of the index subjects in the Lange (1930), LeGras (1932), Kranz (1936), Stumpfl (1936), Slater (1938), Borgstrom (1939), Rosanoff et al. (1934), Yoshimasu (1961), and Shields (1977) studies had criminal outcomes, the control concordance for these studies was determined by the base rate of criminality calculated according to sex (male, female) using rates provided by Christiansen (1970, 1974).

Moderator Variables: Sex = gender of index subjects M = males, F = females, and B = mixed samples of males and females; Nat = nationality of the subject sample—USA = a US-based sample and For = a European- or Asian-based sample; Pub = year of publication—Pre = a publication date earlier than 1975 and Post = a publication date of 1975 or later; Des = whether the design of the study satisfies four criteria, i.e., a clearly defined outcome, a reliable procedure for determining zygosity, use of a control group, and an adequate sample size (+ vs −).

* Cutting scores were derived separately for males and females based on the median number of antisocial acts reported by each group (males = 18, females = 6).

Table 3. Adoption Studies Addressing the Gene-Crime Relationship

		Criterion Behavior		Proband Subjects			Control Subjects			Moderating Var.				
Study	Location	Adoptee	Bio. Parent	*N*	Sex	Outcome(%)	*N*	Sex	Outcome(%)	Φ	Sex	Nat	Pub	Des
Bohman (1972)	Sweden	School adj. prbl.	Registered criminality (father)	118	M	26.3	117	M	35.9	−.10	M	For	Pre	−
				59	F	47.4	140	F	31.4	.15	F	For	Pre	−
Crowe (1972)	United States	Arrest	Incarceration (mother)	52	M&F	15.4	52	M&F	3.8	.20	B	USA	Pre	−
Schulsinger (1972)	Denmark	Psychopathy	Psychopathy	54	M&F	9.3	56	M&F	1.8	.15	B	For	Pre	−
Crowe (1974)	United States	Antisocial personality	Incarceration (mother)	46	M&F	28.2	46	M&F	4.3	.32	B	USA	Pre	−
Hutchings & Mednick (1975)	Denmark	Criminal conviction	Criminal conviction	164	M	48.8	807	M	33.8	.17	M	For	Post	+
Bohman (1978)	Sweden	Registered criminality	Registered criminality	169	M	12.5	812	M	13.0	−.01	M	For	Post	+
				32	F	3.1	1,167	F	2.8	.01	F	For	Post	+
Cadoret (1978)	United States	Antisocial personality	Antisocial personality	9	M	11.1	38	M	7.9	.04	M	USA	Post	−
				3	F	33.3	34	F	2.9	.32	F	USA	Post	−
Cloninger et al. (1982)	Sweden	Registered criminality	Registered criminality	76	M	15.8	786	M	3.4	.17	M	For	Post	+
Mednick et al. (1984)	Denmark	Criminal conviction;	Criminal conviction;	1,226	M	20.2	2,492	M	13.0	.09	M	For	Post	+
		3 or more convictions	3 or more convictions	147	M	25.2	3,571	M	10.7	.09	M	For	Post	+
Cadoret et al. (1985)	United States	Antisocial personality	Antisocial personality	21	M	57.1	106	M	26.4	.25	M	USA	Post	+
				9	F	33.3	78	F	20.5	.09	F	USA	Post	−
Cloninger & Gottesman (1987)	Sweden	Criminality w/o Alcohol	Registered criminality	39	M	20.5	823	M	8.3	.09	M	For	Post	−
				16	F	50.0	897	F	14.5	.13	F	For	Post	−
Moffitt (1987)	Denmark	Nonviolent conviction;	Criminal conviction;	1,252	M	16.0	2,555	M	10.6	.08	M	For	Post	+
		Violent conviction	Criminal conviction	164	M	3.5	3,643	M	3.4	.00	M	For	Post	+
Baker et al. (1989)	Denmark	Property offense	Property offense	408	M	35.5	3,222	M	23.8	.08	M	For	Post	+
				88	F	42.0	3,834	F	25.8	.05	F	For	Post	+

NOTES: Proband subjects = adoptees who satisfied the criminal outcome criterion; control subjects = adoptees who failed to satisfy the criminal outcome criterion. Outcome = percentage of index subjects with at least one criminal outcome biological parent.

Moderating variables are coded as in Table 1.

Table 4. Effect Sizes for Studies Addressing the Gene-Crime Relationship

	Overall Effect	Family Studies	Twin Studies	Adoption Studies
Number of Φ Estimates	54	15	18	21
Maximum Φ	.99	.41	.99	.32
Median Φ	.17	.27	.16	.09
Minimum Φ	−.11	.13	−.11	−.10
Weighted Mean (Φ)	.09	.26	.21	.07
Unweighted Mean (Φ)	.25	.27	.30	.11
T-Test of Mean *Z*	9.42*	7.87*	4.18*	5.54*

* $p < .001$ (one-tailed test).

Because there were statistically significant between-group differences in the standard error variance estimates for three of the four moderating variables included in the meta-analysis, separate error variance estimates were used in calculating the *t*-score values for moderating variable effects. Subsequent analyses revealed statistically significant differences for year of publication, $t(52) = 2.26, p < .05$, and quality of the research design, $t(52) = 2.11, p < .05$, but not for gender, $t(52) = 1.19, p > .10$, or nationality, $t(52) = 0.22, p > .10$. Overall moderating variable effects, as well as moderating variable effects for each of the three types of studies, are displayed in Table 5.

A multiple regression analysis was then performed with the four moderating variables serving as predictor measures and the phi coefficient serving as the criterion measure. This analysis produced a multiple correlation of .46 ($R^2 = .21$) and individual beta weights of -.37 for year of publication, -.16 for nationality, -.07 for design quality, and .02 for gender. Since the year of publication and design quality measures were significantly correlated ($\Phi = .48, p < .001$), a separate series of regression analyses were calculated, the results of which indicated that the multiple correlation fell from .46 to .33 when the year of publication was dropped from the regression equation but remained reasonably stable at .45 when design quality was removed from the equation.

DISCUSSION

The results of this meta-analysis of the proposed gene-crime nexus reveal a consistent and statistically significant association between various indices of heredity and crime. With a mean unweighted effect size of .25, median effect size of .17, and mean weighted effect size of .09, there would appear to be guarded optimism for a genetic interpretation of certain facets of criminal behavior. However, several factors limit the significance of the overall relationship observed in this investigation. Due, in part, to the large samples

Table 5. Effects of Moderating Variables on the Gene-Crime Relationship

	Subject Gender		Nationality		Year of Publication		Design Quality	
	Male	Female	USA	Non-USA	Pre-75	75-on	+	−
Overall Effect								
Number of Φ Estimates	31	15	23	31	23	31	22	32
Maximum Φ	.66	.67	.59	.99	.99	.41	.41	.99
Median Φ	.17	.26	.26	.13	.27	.09	.10	.22
Minimum Φ	−.10	.01	.04	−.11	−.10	−.11	−.01	−.11
Mean of Φ Distribution	.19	.27	.26	.24	.38	.15	.15	.33
SD of Φ Distribution	.19	.21	.15	.45	.49	.13	.12	.48
Family Studies								
Number of Φ Estimates	9	6	13	2	6	9	5	10
Maximum Φ	.41	.32	.41	.35	.31	.41	.41	.35
Median Φ	.21	.29	.27	.28	.26	.32	.32	.26
Minimum Φ	.13	.25	.13	.21	.13	.17	.20	.13
Mean of Φ Distribution	.26	.29	.27	.28	.24	.29	.30	.25
SD of Φ Distribution	.10	.03	.08	.11	.07	.09	.08	.07
Twin Studies								
Number of Φ Estimates	10	3	4	14	12	6	6	12
Maximum Φ	.66	.67	.59	.99	.99	.14	.28	.99
Median Φ	.16	.59	.31	.16	.42	.06	.10	.42
Minimum Φ	.03	.09	.06	−.11	.06	−.11	.05	−.11
Mean of Φ Distribution	.27	.48	.34	.43	.54	.04	.12	.49
SD of Φ Distribution	.26	.36	.32	.60	.60	.08	.09	.63
Adoption Studies								
Number of Φ Estimates	12	6	6	15	5	16	11	10
Maximum Φ	.25	.32	.32	.17	.32	.32	.25	.32
Median Φ	.08	.11	.22	.09	.15	.09	.08	.14
Minimum Φ	−.10	.01	.04	−.10	−.10	−.01	−.01	−.10
Mean of Φ Distribution	.08	.12	.20	.08	.14	.10	.09	.14
SD of Φ Distribution	.09	.11	.12	.08	.16	.09	.08	.13

NOTE: The mean of Φ distribution is the unweighted mean.

employed by several of the investigators in this area of research, the gene-crime connection was shown to be highly statistically significant. However, the actual magnitude of the relationship was modest and the practical significance of the findings uncertain. Further, higher quality studies and those published in 1975 and later provided less support for the gene-crime hypothesis than lower quality studies and those published prior to 1975. Finally, the mean effect size produced by studies using an adoptive methodology, possibly the strongest of the three primary strategies used to investigate gene-behavior relationships, was less favorable to the gene-crime hypothesis than those produced by family and twin studies.

In considering the three primary methodologies used to examine the gene-crime relationship, a few comments are in order. First, since the family method is incapable of distinguishing between the individual contributions of heredity and common environment, the effect sizes observed in the family studies should be viewed as an "upper limit" estimate of genetic influence. Consequently, the actual heritability of criminal conduct is somewhere between zero and this upper limit, and, in all likelihood, resides at a level substantially lower than that displayed in Table 1 and Table 4. Second, twin studies were introduced in an effort to resolve the heredity-environment confound problem that hinders interpretation of family study data on the strength of the assumption that environmental similarity is largely equivalent across sets of identical and fraternal twins. However, several investigators have uncovered the presence of significant MZ-DZ differences in twin influence (Dalgard and Kringlen, 1976; Rowe, 1985), which when controlled, place the heritability of criminal conduct at a level commensurate with that obtained using the more conservative adoption method and considerably lower than that traditionally associated with the outcome of twin research (Carey, 1992). It is worth noting that adoption studies not only provide the most accurate test of the gene-crime hypothesis, but also yield the lowest heritability estimates.

Although the heritability coefficient was not used as the effect size measure in this investigation, it is still possible to estimate this coefficient by taking the weighted and unweighted mean phi correlations, partialling out the variance attributable to the four moderating variables (i.e., 21%), and then multiplying that figure by two (because index group differences in genetic similarity for the studies included in this meta-analysis vary by 50% rather than 100%). This procedure yields heritability estimates of 41 to 43% for family studies, 33 to 47% for twin studies, and 11 to 17% for adoption studies. These heritability estimates can then be used to partition variation in criminal outcomes into the three component sources (genetic, common environmental, specific environmental) of the behavior genetic model. The genetic component would

seem to be best captured by the heritability estimate from the adoption studies (namely, 11 to 17%). Since family studies contain both genetic and common environmental sources of variance, the common environmental component (experiences shared by family members) might be estimated by subtracting the heritability estimate obtained in adoption studies from the heritability estimate obtained in family studies. This results in a common environmental component estimate of 24 to 32%. The remaining 51 to 65% of variance in criminal outcome would appear to comprise specific environmental influences (experiences unique to the individual) and measurement error.

Besides differences in outcome occurring as a result of variations in the research method employed (family studies vs. twin studies vs. adoption studies), this investigation suggests that at least two features of the study itself (year of publication and quality of the research design) modify the observed relationship between heredity and crime. Studies published prior to 1975, many of which were of poorer methodological quality than studies published in 1975 and later, tend to yield results more favorable to the genetic hypothesis than more recently conducted and better designed investigations. However, the multiple regression results obtained when year of publication and design quality were systematically removed from the moderating variable equation suggest that year of publication, not design quality, was the most salient moderating condition in this meta-analysis. Although merely speculative at this point, this finding may indicate an important shift in the etiological foundations of crime over time, with heredity playing a more significant role in the criminal behavior of subjects raised during the early part of this century than is the case with more contemporary samples of subjects. In a related vein, a shift in the economic-crime relationship was observed between the nineteenth and twentieth centuries whereby crime became less closely tied to adverse economic conditions and began to take on the appearance of being more clearly motivated by opportunity and self-centered goals (Wilson and Herrnstein, 1985). Further study is obviously required for a fuller understanding of how year of publication impacts on the gene-crime relationship. However, this investigation suggests that there is something about the recentness of publication, independent of the quality of the research design, that is important in defining support for the gene-crime hypothesis.

While the meta-analytic technique provides many advantages over the traditional literature review, it is not without certain problems and limitations. A major concern voiced by critics of this technique is that studies of differing quality are mixed and given equal weight. One might answer this admonition by pointing out that the quality of a study's research design was one of the moderating variables considered in this investigation and that higher quality designs tended to produce results that were less supportive of the crime-gene hypothesis than lower quality designs. Interpretation of the

overall effect size, however, is hindered by the fact that the procedure for twin studies on the one hand, and for family and adoption studies on the other, differed slightly. Whereas the phi coefficients for family and adoption studies were based on the rate of familial/parental criminality among subjects possessing and failing to possess criminal records, the phi coefficients obtained from twin studies were based on a comparison of MZ and DZ twins. For this reason, moderating variable effects for family, twin, and adoption studies were presented separately in Table 5.

Perhaps one of the reasons why genetic research on crime has not contributed more to an understanding of criminal behavior is that researchers have become preoccupied with the either-or thinking that sometimes dominates discussions of the nature-nurture question. Rather than presupposing a genetic or environmental explanation for a particular human behavior like crime, the science of criminology might be better served by investigations that search for a workable integration of genetic and environmental concerns. In so doing, researchers will have to determine exactly what is being inherited by persons who are at increased biological risk for later criminality. A careful reading of several of the articles published on the topic of heredity and crime reveals that some authors appear to propose a single and/or direct link between heredity and crime that may even be specific for certain categories of criminal offense (see Cloninger et al., 1982). A more realistic and scientifically defensible interpretation of gene-crime data, however, might be found in Rowe and Osgood's (1984) approach wherein genetic factors are viewed as contributing to certain individual differences that in turn interact with specific sociological and environmental conditions to bring about criminal and delinquent outcomes. Dividing the variance obtained from their analyses of twins into its genetic, common environmental, and specific environmental components, Rowe and Osgood were able to show how individual variations in genetic background may contribute to delinquency outcomes, but also how genetic findings can be effectively integrated with conventional sociological theory on peer associations. Rowe and Osgood's study also points out the importance of considering the gene-environment interaction in addition to the individual contributions of nature and nurture in understanding criminal behavior.

Since it seems unlikely that a direct genetic link for crime exists anywhere but in the minds of a handful of investigators, future research in this area should probably be directed at exploring the personality/behavioral characteristics that likely bridge the modest gene-crime association observed in this study. Variables potentially capable of explaining the observed relationship between heredity and criminal behavior include intelligence (Hirschi and Hindelang, 1977), temperament (Olweus, 1980), and physiological reactivity (Venables, 1987), all of which are significantly affected by heredity and have been shown to correlate meaningfully with criminal outcome. Investigators

in this area of research endeavor could enhance the relevance of their studies further by developing behaviorally oriented definitions of criminality, rather than relying exclusively on legal criteria, and including designs that highlight the gene-environment interaction (see Cadoret et al., 1983) rather than focusing on heredity and environment as if they were independent and mutually exclusive entities.

The task, then, is to formulate a coherent theory of gene-crime interrelationships that effectively integrates person-oriented considerations like heredity with the more popular environmental interpretations of crime and delinquency. Given the paucity of meaningful outcomes generated by research on the gene-crime hypothesis, one might wonder why heredity has been selected from the large audience of potential person-based correlates of crime to receive so much attention from certain investigators and scholars (see Rushton, 1987; Wilson and Herrnstein, 1985). Perhaps this reveals a reluctance to reject the biological positivistic roots of our criminologic forebearers, or maybe it simply denudes the natural human tendency to look for easy answers to intricate and complex questions. Many criminologists, on the other hand, seem inclined to reject genetic explanations of criminal involvement before they have even had a chance to examine the pertinent data. As students of crime we must come to realize that to develop a comprehensive understanding of crime and criminal behavior we will have to consider many variables, including genetic factors, common environmental influences, and a host of specific environmental correlates, in our explanatory equation. But most important, we must be willing to examine how these individual components of the behavior genetic model interact to bring about criminal and delinquent outcomes.

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