

A Critical Analysis of IQ Studies of Adopted Children

Ken Richardson^a Sarah H. Norgate^b

^aHexham, ^bUniversity of Salford, Salford, UK

Key Words

Adoption · Development · Intelligence · IQ

Abstract

The pattern of parent-child correlations in adoption studies has long been interpreted to suggest substantial additive genetic variance underlying variance in IQ. The studies have frequently been criticized on methodological grounds, but those criticisms have not reflected recent perspectives in genetics and developmental theory. Here we apply those perspectives to recent IQ adoption studies and show how they further question two sets of problems: first, the assumption of additive gene and environmental effects; second, the assumption that the adoption situation approximates a randomized-effects design. We show how a number of possible factors having systematic effects in breach of those assumptions can produce the received pattern of correlations without appealing to unusual amounts of additive gene variance. Copyright © 2006 S. Karger AG, Basel

Studying adopted children has long been viewed as a well-controlled, quasi-experimental method for estimating genetic and environmental components of variation in human intelligence which can then be used in models of human development. Because biological mothers and their adopted away children share genes but not environments, covariance (correlation) in traits like IQ is considered to be a direct estimate of genetic effects on trait variance. Because adoptive caregivers and their adopted children share environments but not genes, IQ correlations between them can indicate the magnitude of environmental sources of variance. Correlations between unrelated children reared in the same home are taken to indicate what proportion of the environmental variance is 'shared' (varies between families) or 'non-shared' (varies within families). For a review of all these aspects of behaviour genetic methods see Plomin, DeFries, McClearn and Rutter [1997].

KARGER

Fax +41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

© 2006 S. Karger AG, Basel
0018-716X/06/0496-0319\$23.50/0

Accessible online at:
www.karger.com/hde

Sarah H. Norgate, Psychology Directorate
Faculty of Health and Social Care, University of Salford
Allerton Building, Frederick Rd. Campus
Salford, M6 6PU (UK), Tel. +44 161 295 2324
Fax +44 161 295 2427, E-Mail S.H.Norgate@salford.ac.uk

Table 1. Adoptive child's IQ correlations with parents

	Biological father	Biological mother	Adoptive father	Adoptive mother
TAP1 ^a	–	0.31	0.14	0.12
MAS1 ^b	0.43 ^c	0.33 ^c	0.27	0.21

Dash indicates that no correlation was obtained.

^a Mean age 8 years (range 3–16+) in the TAP; ^b mean age 7 years (range 4–18 years) in the MAS; ^c biological parents not tested, IQs were estimated from education levels.

Table 2. Adopted child-adoptive parent correlations in the TAP2 and MAS2 studies

	Adoptive father	Adoptive mother
TAP2 ^a	0.10	0.02
MAS2 ^b	0.16	0.09

^a Mean age 18 years (means of various test combinations, see table 4.3 in Loehlin et al., 1997). ^b Age range 16–22 years.

Since the 1920s a now-familiar pattern of results has emerged from such studies: IQs of adopted children appear to correlate more with those of biological mothers than with adoptive mothers or adoptive fathers. Table 1 shows typical data from two of the more recent studies: the first sweep of the Texas Adoption Project (TAP1) as reported by Horn, Loehlin and Willerman [1979], and the first Minnesota Adoption Study (MAS1, a transracial study) as reported by Scarr and Carter-Saltzman [1989], involving children covering a wide age range, from 3 to 18 years. The seemingly diminishing correlations between adopted children and their adoptive parents in later years are shown in table 2 (results from TAP follow-up after about 10 years and second MAS study of a new sample of youths, aged 16–22 years).

The more recent Colorado Adoption Project (CAP) replicated this general picture, as shown in table 3 [Plomin, Fulker, Corley, & DeFries, 1997]. This study included a control group of normal families matched to the adoptive families for a number of demographic factors. As can be seen, the correlation between the adopted children and their biological parents almost doubled, from less than 0.2 to nearly 0.4 in the period when the children were 14–16 years old, while the adoptive parents-adopted children correlation remained around 0. Finally, all of these studies report IQ correlations between adopted children and other children in the same family significantly lower than those between natural siblings, even though (it is assumed) they are sharing the same environment.

Table 3. Average correlations between parents and children (at ages shown) in the CAP [data from Plomin, Fulker et al., 1997]

	Adopted child/ adoptive parent	Adopted child/ biological parent	Control child/ control parent
3–4 years	0.09/0.00 ^a	0.12	0.19
7–10 years	0.01	0.18	0.24
12–14 years	–0.06	0.20	0.28
16 years	0.03	0.38	0.31

^aCorrelations for 3- and 4-year-olds, respectively.

Though cautions are often expressed [e.g., Plomin, DeFries, McClearn, & Rutter, 1997], the fact that reported biological mothers-adopted children correlations are higher than adoptive mothers-adopted children correlations has had a big impact in psychology and on theories of development. Most usually, the correlations have been computed into heritability (h^2) estimates, referring to the proportion of the total variation in IQ scores in the sample population that can be attributed to genetic variation. With respect to the TAP data, Loehlin, Horn, and Willerman [1997] ‘estimate the heritability ... to be about 0.78’ (p. 123). Similarly, Scarr [1997] says that the data from adoption studies ‘yield heritability estimates of 0.70 or above’ (p. 27). Data from the CAP are said to show a heritability of ‘general cognitive ability’ of 0.56 at 16 years with ‘an increasing role for nature and a decreasing role for nurture’ over the childhood period [Plomin, Fulker, Corley, & DeFries, 1997, p. 445]. Together with the sibling correlations, these data have encouraged the conclusion that environmental differences between families account for none or little of the variation in IQs of adolescents and adults [Jensen, 1997].

Over several decades, these correlations and interpretations have undoubtedly been widely accepted and influential. The task force set up by the American Psychological Association to report on ‘Intelligence: knowns and unknowns’ [Neisser et al., 1995] cited the degree of genetic variance for IQ estimated from adoption (as well as from twin) studies as one of the definite ‘knowns’. In Britain the Nuffield Council on Bioethics, reporting evidence from several contributors, says that ‘Adoption studies provide a powerful means of examining genetic and environmental influences’ and that ‘adoption studies have added to evidence from studies of twins in demonstrating a genetic contribution to variation’ in IQ [Nuffield Council on Bioethics, 2002]. In a recent critical review of behaviour genetic interpretations of IQ data, Maccoby [2001] nevertheless concludes that adoption studies have made a high heritability for IQ ‘undeniable’. Though referring to ‘problems’ in quantitative genetic designs, Rutter [2002] declares that ‘any dispassionate reading of the evidence leads to the inescapable conclusion that genetic factors play a substantial role in the origins of individual differences in all psychological traits’ (p. 2). In a more recent review Turkheimer [2005] says that the results of adoption and other family studies ‘are no longer in serious dispute’ (p. 161). Accordingly, the substantial additive genetic variance usually inferred has provided grounds for conducting molecular-level searches for single gene components of that variance [e.g., Plomin & Spinath, 2004].

Here we suggest that the data do not support such interpretations. We say this because, broadly, they appear to be based on narrow models of both genetic and environmental effects, and of the nature of human development. First, the expectation of meaningful correlations presupposes an additive/independent-effects model of gene and environment influence. Such a model is now known to be invalid, in ways we discuss further below. Second, the interpretations assume that adoption studies approximate a kind of randomized-effects design in which nature provides the genetic 'treatment' while other environmental influences are controlled or randomized. Below we examine a number of uncontrolled factors that may act systematically in breach of that assumption.

Fundamental doubts about the methodology and empirical adequacy of IQ adoption studies have been expressed before [e.g., Lewontin, Rose, & Kamin, 1984; Munsinger, 1975]. But these do not cover more recent studies and the further interpretations of them, including the seemingly negligible effects of parents, or between-family environments [Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; see also Sacerdote, 2004]. Moreover, they do not include more recent developmental perspectives [for reviews see Gottlieb, 2000; Horowitz, 2000; Lerner, 2002]. Focusing on the overly simple causal models employed, rather than the magnitude of heritability estimates as such, our discussion extends earlier critiques to suggest a radical re-appraisal of the interpretations and conclusions mentioned above, including the impact they have had within developmental psychology and elsewhere.

The Additive/Independent-Effects Assumption

IQ adoption studies have traditionally assumed that natural variation in human intelligence is that of a simple quantitative character like height or weight. That is, trait variation consists of the sums of effects of variable 'genes' and variable 'environments', combining additively/independently, as randomly segregating units of equal effect. This assumption is what constitutes the basic model of effects: the seeming separation of them in the adoption process suggests the expected correlations. Degrees of approximation to those expectations offer separate estimates of magnitudes of genetic and environmental sources of variance that could not be predicted under non-additive (especially non-linear) effects.

The first problem with the approach is that the assumption that genes and environments act in this additive/independent (mendelian) fashion is now known to be quite invalid. Gene-gene, gene-environment, or even environment-environment interactions are now known to be the rule rather than the exception in creating variation in complex traits [see e.g., Balaban, 2001; Nijhout & Davidowitz, 2003]. As Merila and Sheldon [1999] point out in their review of animal studies, the 'genetic architectures' of highly evolved traits tend to be more complex, with many more loci utilized under interactive regulatory hierarchies. In molecular studies, the ubiquity with which gene utilization in development is dependent upon genetic background as well as environmental interactions suggests that there are very few truly mendelian traits [Glazier, Nadeau, & Aitman, 2002]. The predominance of regulatory genes sensitive to environmental change has been revealed as 'cascades' of interactive, self-organizing processes in development [Gottlieb, 1998, 2000; Lickliter & Honeycut, 2003; Moore, 2002;

Oyama, 2001]. Accordingly, there is now much experimental evidence of gene-environment interactions in behavioural traits in animals [see, for example, Newman et al., 2005; Suomi, 2003]. In such systems many allelic variants can be rendered functionally equivalent; many deviant environmental effects can be compensated for, and additive sources of variance are much reduced [Peccoud, Venden, Podlich, Winkler, Arthur, & Cooper, 2004]. In addition there is strong evidence of other kinds of gene-environment and environment-environment interactions arising within the special dynamics of the adopted state (discussed in some detail below). In all of these ways, predictability from allelic to phenotypic variation, and therefore to 'expected' trait variances and covariances, becomes very weak.

Some of these negations of the foundation assumptions of the adoption method have been well known for many years. They have been acknowledged to some extent by adoption researchers who have used multivariate methods and 'path' models to assess whether such interactions could influence adoption study data, albeit on the assumption that they will have minor effects against an essentially additive background [e.g., Cherny, Fulker, & Hewitt, 1997]. However, Wahlsten [1990] and Turkheimer and Waldron [2000] have made the point that adoption studies simply do not have the statistical power to detect such interactions, leading Wahlsten [2000] to suggest that 'the whole edifice' of additive models can now only 'be accepted on faith, if it is to be accepted at all' (p. 50). We suggest, however, that the evidence of interactive sources of variance from molecular genetic and other research is now sufficiently strong as to doubt whether adoption studies can *in principle* answer the kinds of questions at issue.

The corollary to the additive/independent effects model is that all other effects (on covariance patterns) have been either controlled or randomized in the adoption design, obviating other explanations for the data. The rest of this paper examines a number of uncontrolled factors with systematic effects that can readily explain the biological mothers-adopted children/adoptive mothers-adopted children pattern of correlations in adoption studies without resort to unlikely assumptions of independent effects.

'Intelligence' Testing in Adoption Studies

'Intelligence' testing in adoption studies has perforce been opportunistic, with much use of modified and/or shortened tests in less than ideal circumstances. This is illustrated in the CAP where Plomin [1986] says, 'the goal was to collect as many valid and reliable data as possible during relatively brief testing periods, sampling extensively and broadly rather than intensively and narrowly' (p. 49). A large variety of tests was administered at different times under widely different circumstances: in late pregnancy in the nursing home; in the family home; in the laboratory, and 'over the telephone' [Plomin, Fulker, Corley, & DeFries, 1997]. Early reports from CAP emphasized 'special cognitive abilities', whereas the most recent stresses 'general mental ability' as reflected in the First Principal Component of the pooled test scores. However, that general factor seemed to account for only 37.2% of the total score variance in the adult tests [DeFries, Plomin, Vandenburg, & Kuse, 1981], and, somewhat curiously, the matrices test, often considered to be virtually a pure measure of general mental ability [Carpenter, Just, & Shell, 1990; Jensen, 1998], had a factor loading of only 0.65

on the First Principal Component. In other words, much more than 'general mental ability' is being tapped in test scores, variances and correlations.

In the TAP, most birth mothers were given the Revised Beta Test which, as Loehlin, Horn and Willerman [1997] say, 'has some special problems' (p. 107) (for example, there was a strong ceiling effect). Others were tested on the WISC or the WAIS or the Stanford-Binet. Adoptive parents were tested with both the WAIS and the Beta. All tests had old norms, so scores required various adjustments. This mixed testing regime may explain why correlations between parents and children varied from 0.26 to 0.78, depending on the combination of tests (see further below). In the MAS, birth mothers' IQs were simply estimated from levels of education [Scarr & Carter-Saltzman, 1989].

The point is, of course, that lack of precision in measurement can permit expression of unknown sources of variance/covariance, some of which may create systematic (non-random) effects, and the reported pattern of correlations. It is worth remembering, in this respect, that there is continuing uncertainty about what cognitive tests actually test. IQ scores may not be so much an index of mental efficiency or capacity as one of acquisition of particular cultural tools (e.g., numeracy and literacy skills [Cole, 1999]), and all the issues of inclination, motivation and test preparedness associated with that [Richardson, 2001]. There are reasonable grounds for suspecting that score variance, especially that captured as the First Principal Component in factor analysis (and usually labelled 'general mental ability' or g), could be partly or even entirely non-cognitive in origin [Richardson, 2005].

One non-random source of variance emerging through lack of measurement precision may be the testing of children and adults of widely different ages: 'unlike measures of height, tests for mental ability cannot be presumed to measure the same trait' at different ages [Plomin & DeFries, 1985]. In consequence, larger or smaller correlations in scores between different kinds of pairs of relatives may simply reflect degree of resemblance in age. Biological parents who give up children for adoption are known to be typically younger than adoptive parents (e.g., respective averages of 20 and 33 years in the CAP), so adoptive children are always closer in age to their biological parents than to their adoptive parents (or control children to *their* parents) at age of testing. In the CAP, this is *especially* so at the critical age of 16, when the adopted children were only 3 years younger than their natural parents were, on average, when they were tested on exactly the same test battery. This may explain why, in the CAP, there is a near doubling in IQ correlation between adopted children and their biological parents to 0.38 between 14 and 16 years (before which all correlations are less than 0.2; see table 3). This crucial correlation exceeds even that between control parents and their own children growing up in the same home from birth ($r = 0.31$). A similar puzzling differential occurs in the TAP as reported in Loehlin et al. [1997].

Note that Plomin, Fulker et al. [1997] refer readers to DeFries et al. [1981] for a method of age-adjustment of scores, but that paper is only about 'the adult tests', the children at that time being only 1 year old. The significance of the 'Flynn effect' – or rising average test performances over time [e.g., Flynn, 1998] – which can be non-linear and vary with different kinds of tests, further stresses the ideal of either testing all subjects on the same test at more or less the same time or making properly assessed adjustments.

Epigenetic Inheritance and Transgenerational Effects

As mentioned above, the primary assumption of the adoption design is that adopted children's environments of development will be uncontaminated by that of biological parents. It has long been recognized that this can never be strictly true because adopted away children are dependent on the biological mother's environment during early development in utero. This environment includes not only nutritional, hormonal, immune and other influences, but also others detectable in terms of social and linguistic experiences [e.g., DeCasper & Spence, 1986].

However, other important effects of biological parents' environments on their children's, and even their children's, development have been discovered in much recent research. New insights are now available into what Harper [2005] calls 'this all but ignored' pathway of influence from parents to children and successive generations. Such influence occurs, not by genetic transmission as such, but by experience-dependent epigenetic reprogramming of gene expression in parents: gene identity is unaltered, but regulated gene utilization may be transformed [for review, see Harper, 2005]. Parental experiences now known to have such transgenerational effects include under-nutrition during pregnancy, affecting birth weight and child height across generations [Stein & Lumey, 2000]; transient exposure to environmental toxins [Anway, Cupp, Uzumcu, & Skinner, 2005], and various kinds of stress and trauma [Francis, Diorio, Liu, & Meaney, 1999; Seckl & Holmes, 2001; Yehuda, Engel, Brand, Seckl, Marcus, & Berkowitz, 2005]. In some cases biological details of transmission pathways have now been described [Lin, Di, & Zhou, 2003; Turner, 2000].

Many of these effects of parental experiences could be reflected in cognitive performance measures in the child, and thus in covariances between the biological mothers and their children, including those adopted away. There is a long-standing recognition of a link between affective states (e.g., stress reactivity) and cognitive performance [Zeidner, 1995; and contributions in Maddux, 1995]. The link is acknowledged by some authors of test manuals who warn how 'fatigue, ill health and stress' affect speed and accuracy on tests [Raven, Raven, & Court, 1993, p. 14]. Insofar as these effects impinge on IQ test covariance they can seriously distort parent-child correlations and interpretations of them. Harper [2005] considers a complex design for testing for transgenerational effects in behaviour genetic studies, although it is only feasible in animals. This involves taking eggs that have undergone primary development from a mother that has been subjected to some stressful treatment over that period. The harvested eggs are then fertilized and the zygotes implanted in same-strain females not subjected to the treatment. If transgenerational effects are present, then grandmothers' treatment should account for more of the variance in the offspring than the maternal environment.

Other non-random effects distorting biological mothers-adopted children IQ correlations may be described as 'indirect genetic'. It is well known that attributions about intelligence are made on the basis of height and facial appearance, and that parents, teachers and peers treat children differently accordingly [Langlois, 1988]. Adopted children will tend to be physically more similar to natural parents than to their adoptive parents, and less similar to their adoptive parents than are the latter's natural children. In consequence, reported associations between physical attractiveness and both self-esteem and anxiety levels [Rowe, Clapp, & Wallis, 1987] may continue to affect biological parents and their adopted children similarly, even though

they are apart, so bringing about covariance in IQ test performance. Even though such effects may be small, they could be sufficient to create what are, after all, relatively small differences between small covariations.

Another possible distorting effect mentioned in the literature is called genotype-environment covariance [Jensen, 1997; Scarr & McCartney, 1983]. According to this view, individuals select, seek out and/or create environments best suited to, or even driven by, their personal genotypes. This applies also to the environments different caregivers, with different genotypes, will provide for their children. Though superficially plausible, the mechanisms through which such environmental selection and provision can be 'genetically driven' [Jensen, 1997, p. 42] are unknown, and the extent to which the concept can explain data from adoption (and twin) studies depends on the empirical sufficiency of those studies. Since it is the latter that we are scrutinizing here, we do not discuss gene-environment covariance any further in this paper except to note that it would constitute another breach of the assumption of independent effects.

Selective Placement and Range Restriction

Another departure from a randomized-effects design occurs through selective placement of children for adoption. From their knowledge of the biological parents, agencies tend to have preconceived ideas about the personality and intelligence that the child is going to have, and place him or her in what they think will be a compatible family environment [Rutter, Pickles, Murray, & Eaves, 2001]. There is strong evidence of selective placement in the TAP and MAS studies [e.g., from sizeable correlations between biological mothers of adopted children and unrelated children in the same adoptive family; Scarr & Carter-Saltzman, 1989]. In the CAP, the adoption agencies aimed to place an infant in the 'best family for that particular child' [DeFries et al., 1981, p. 254]. Plomin, Fulker et al. [1997] suggest that selective placement is absent in the CAP on the grounds of similar means and variances of socio-economic scores (SES) in all three groups of fathers. However, this interpretation is based on only those 20% of biological fathers who volunteered to join the study and who may well not be representative. Selective placement could inflate the biological mothers-adopted children correlation in IQ, although it could also affect adoptive parents-adopted children correlations.

More importantly, adoptive parents in all studies, by virtue of the rigorous selection processes they are subjected to, tend to be of higher than average SES, and, as a sample, restricted in range [Rutter et al., 2001]. In the TAP, as Loehlin et al. [1997] explain, 'the clientele of this adoption agency are a selected group and were probably further selected by participation in our study' (p. 109). In the MAS1 the variance of IQ scores in adoptive parents 'was considerably restricted' [Scarr & Carter-Saltzman, 1989, p. 854], while the biological mothers' variance for education levels (used to estimate their IQs) was not restricted [Scarr & Weinberg, 1978]. In the MAS2, adoptive parents' scores were also restricted in range for IQ and other variables [Scarr & Weinberg, 1978]. Stoolmiller [1998] found that adoptive families in the CAP represented only the top third of the American population in terms of socio-economic status. Adoptive and control parents in the CAP all show restricted standard deviations, as well as well-above average means, on test scores on the Wechsler Adult Intelligence

Test [Plomin, Fulker, 1997]. The effect of restricted socio-demographic factors in adoptive families, and their reflection in test score variances, is to reduce adoptive parents-adopted children correlations but not biological mothers-adopted children correlations.

Finally, there is evidence of considerable sample attrition over time in all family studies of this sort, and the evidence tends to be disproportionately from lower SES groups [Rutter et al., 2001]. This factor may further restrict the range of adoptive families of older adopted children, but mostly affects the generalizability of results to the greater population, rather than the results themselves.

Family Attributions and Sociodynamics

The social interactions experienced by adoptive children in families and social networks also produce systematic departures from randomized effects. As Jackson [1993] notes, the adopted-child method

is engaging because of its simplicity. However, it is critically incomplete as a model of what actually happens in family life when an adoption is involved ... All adoption studies that follow the human behavioural genetics model fail to account for adoptive status as a highly important, culturally imposed social factor in the psychological environment of development. (p. 1323)

Among other things, it is a status prone to affective stress. Howe [1998] notes that, 'being an adopter and being adopted are not emotionally neutral states ... Both have developmental tasks to negotiate that are peculiar to adoption' (p. 69). Adopted children may suffer from 'geneological bewilderment' and 'confused sense of identity' [see Feigelman, 1997]. Brodzinsky and colleagues [e.g., Brodzinsky, Smith, & Brodzinsky, 1998] note how adoptees are conscious of the loss of birthparents and associated extended family, which can produce adjustment difficulties [Smith & Brodzinsky, 2002]. Adopted children may also bear residues of 'attachment doubts', such that 'the levels of confidence, self-esteem and self-efficacy are not quite as robust as those achieved by children who enjoy unbroken secure attachments' [Howe 1998, p. 156]. It is also reported that children who are aware of their physical differences from other family members, and that these reflect their adopted status, can feel at a disadvantage, and have lower self-esteem because of it [Rosenberg & Horner, 1991; c.f. Jackson, 1993].

These factors may explain why adoptees tend to be more vulnerable to emotional, behavioural and academic problems than normal children [Ferguson, Lynskey, & Horwood, 1995]. For example, the CAP reports a higher frequency of conduct disorders in adoptees, increasing with age [Coon, Carey, Corley, & Fulker, 1992]. It is well known how self-efficacy beliefs and levels of confidence and anxiety strongly affect test performance [Maddux, 1995]. As mentioned above, levels of reactivity to this enhanced stress may reflect transgenerational effects from the birthmother (who would likewise have experienced unique stressful events), which could increase biological mother-adopted children correlation in test performance while reducing adoptive mother-adopted children correlation. The effects of such affective factors on test performance will be especially important where, as suggested elsewhere [Richardson, 2005], a first principal component largely reflects affective rather than cognitive covariance.

Other dynamics may operate in a more direct sense, as conscious or unconscious aspects of family treatment policy, to 'make adoptees different' from other adoptive family members. Clinicians who work with adoptive families report how adoptive parents, from the moment of adoption, worry about the personalities, blood-lines, and social histories of the natural parents of their adopted child, and how this affects the parent/adopted-child relationship [Hajal & Rosenberg, 1991; cf. Jackson, 1993]. The fact that adoptive parents 'hold stronger beliefs in the influence of heredity' compared with ordinary parents [Terwoegt, Hoeksma, & Koops, 1993] may reduce expectations about the course and targets of adoptees' development. As Scarr, Scarf, and Weinberg [1980] put it, 'Adoptive parents, knowing that there is no genetic link between them and their children, may expect less similarity and thus not pressure their children to become like their parents' (p. 446). Indeed, Larson [1999] reports from one study that adoptive couples 'strove to enhance the differences between their children and themselves in an effort to develop each child's unique talents and capabilities'. Later, adolescent adoptees can become highly conscious of their special identity and react to adoptive parents' standards and values [Brodzinsky, Singer, & Braff, 1984; cf. Baumrind, 1993].

It is especially worth stressing such attributions and respective treatments because information exchange and contact between natural and adoptive families seems to have occurred in the CAP.

Most of these adoptive families had access to information concerning the birthmother, and in some cases concerning the birthfather as well. Some of the adoptive families participating in the CAP have had direct contact with birthparents ... To varying degrees the rearing parents attribute some of their children's characteristics to these known aspects of the birth parents. [Rhea & Corley, 1994, p. 309]

Such attributions (in addition to those based on physical resemblances, mentioned above) may result in conscious or unconscious treatment of the adopted child in a way that increases biological mother-adopted children correlations and reduces adoptive mother-adopted children correlations (see further on such treatment effects below).

In all of these aspects of treatment policy there is 'creation of differences' between adopted children, their adoptive parents and other children in the same family. By influencing cognitive, affective, and 'test preparedness' factors in IQ test performance, they would tend to reduce adoptive mothers-adopted children correlations in IQ, and increasingly so with age. Having a 'control' group matched to adoptive families in superficial demographic features does not, of course, control for any of these special socio-dynamic interactions. Recognizing that a real control would entail, in effect, parents rearing a child from birth in the absence of knowledge on both sides that it is adopted, would at least acknowledge the scale of the problem.

Evidence of these Effects in Adoption Data

The fact that special interactions in the adopted families may actually *create* differences between natural family members and the adopted child may be evident in some aspects of the correlational data. For example, just as adoptive mother-adopted children correlations are smaller than biological mother-adopted children correla-

Table 4. Direct and indirect IQ heritability estimates based on correlations in two adoption studies

Study	Heritability estimates	
	direct	indirect
TAP at 8 years	0.62	0.17
TAP at 17 years	0.70	0.32
CAP at 1 year	0.28	0.00
CAP at 2 years	0.22	0.20
CAP at 3 years	0.30	-0.04
CAP at 4 years	0.50	-0.08

Adapted with permission from Plomin and Loehlin [1989].

tions, so the former may also be smaller than adoptive father-adopted children correlations, because the adoptees will probably have spent more time with their adoptive mothers than their adoptive fathers. In fact, father-child correlations higher than mother-child correlations is a consistent finding in adoption studies. In the TAP, though scarcely different from 0 in any case, 5 out of 6 adoptive father-adopted children correlations (based on different test combinations) are greater than the equivalent AM-AC correlations [Loehlin et al., 1997, their table 4.3]. In the MAS the IQ correlations were also higher for adoptive father-adopted children pairs than for adoptive mother-adopted children (0.27 vs. 0.21 for the younger children; 0.16 vs. 0.09 for adolescent children). In the young CAP children, DeFries et al. [1981] found it 'perplexing' that 'significant correlations occurred between fathers and their offspring for the biological, adoptive and control relationships' (p. 271) – values ranged from 0.18 to 0.45 – but not between mothers and children, which were near 0. A similar effect was found for 'general cognitive ability' when the children were 16 years old [Plomin, Fulker et al., 1997].

In commenting on this 'particularly intriguing feature of the data', in which increasing contact seems to reduce correlations, Loehlin et al. [1997] say that, 'Thus, we may need to entertain the hypothesis that parent's interactions with their children do have an effect on the latter's IQ, but one that can tend to make them less, not more, like the parents' (p. 114). Further evidence of them is probably seen in the way that 'direct' estimates of IQ heritability tend to be so much higher than 'indirect' estimates (see table 4; Plomin & Loehlin, 1989). The former are based on correlations between genetically related pairs reared apart, as in biological mother-adopted children pairs, where the kinds of family interactions just discussed do not exist. Indirect estimates are based on comparisons of correlations between pairs reared together, as between adopted children and their adoptive parents or other children in the same family, where the effects of negative as well as positive interactions cannot be avoided. Such data may be confirming that different kinds of factors and interactions can influence these correlations in systematic ways, and that sharing of home and parents does not necessarily mean shared environment.

Table 5. Effects that can increase biological mother-adopted children (BM-AC) correlations while reducing adoptive mother-adopted children (AM-AC) correlations

Kind of effect	On BM-AC correlation	On AM-AC correlation
Age at testing	+	?
In utero environmental effects	+	-
Inheritance of maternal epigenetics	+	-
Indirect genetic (e.g. physical resemblance)	+	-
Selective placement	+	+
Restriction of range	+	-
Adoptive parents' information about birth family	+	-
Family stress effects	+	-
Family treatment effects	+	-

Discussion and Conclusion

The pattern of IQ correlations reported in a number of adoption studies over the last 50 years or more has had a significant impact on models of human development, and of the nature of intelligence and causes of individual differences in it [Maccoby, 2001; MacDonald & Hershberger, 2005]. Most of that impact derives from studies reporting that biological mother-adopted children correlations in IQ are significantly higher than adopted mother-adoptive children correlations. Interpretation of these correlations, however, presupposes a simple additive/independent-effects model of genes and environments. And it assumes that the methodology approximates a simple randomized effects design in which genetic effects on variance are uncontaminated by environmental effects and environmental effects are uncontaminated by genetic effects.

We have argued that these assumptions are naïve. First, the simple additive-effects model is now widely accepted to be false for both genetic and environmental effects (we also suggest, as an aside, that it is a mistake to treat IQ as a simple quantitative trait). Second, there are numerous other interactive effects that can explain the received pattern of IQ correlations (increasing biological mother-adoptive children correlations, reducing adoptive mother-adopted children correlations) without resort to unlikely additive effects. These are summarized in table 5.

Note that we do not suggest that these factors merely attenuate the conventional interpretations of the correlations of adoption studies. Rather, we suggest that they are *more likely* explanations of them than improbably huge amounts of additive genetic effects. Indeed, given that knowledge of at least some of these factors has been available for a long time, it is surprising to us that results of adoption studies are still widely quoted and duly interpreted as if they did not exist.

One reason for this imperviousness to criticism may be the oft-quoted consistency with the results of twin studies, although twin studies, too, have been criticized for methodological defects, as well as also assuming an additive-effects model [Richardson & Norgate, 2005; Joseph, 2003]. Whatever the reason, the force of traditional argument seems to be strongly adhered to even though its foundations have been

crumbling in the face of molecular genetic evidence. For example, Turkheimer, Haley, Waldron, D'Onofrio, and Gottesman [2003] note that:

In the fractious history of scientific investigations of the heritability of intelligence ... there has been only one contention with which everyone could agree: Additive models of linear and independent contributions of genes and environment to variation in intelligence cannot do justice to the complexity of the development of intelligence in children.

Yet Plomin, Fulker, et al. [1997] categorically state, on the basis of their data, that 'there is little evidence for nonadditive genetic influence' (p. 444). For a similar recent view see MacDonald & Hershberger [2005], who, on the basis of CAP data, argue that $G \times E$ interaction is non-existent, and that the very idea has been used merely 'to satisfy the utopian fantasies of social scientists' (p. 44). We suggest that, far from accepting such conclusions, a 'fit' of additive models to adoption data should lead us to suspect the statistical parameters being used, and thus the causal model of the factors producing the correlations.

Similar considerations apply to causal models of the nature and role of the environment in producing the correlations. For example, the fact that the environment is rich in different kinds of interactions impinging in different ways on different relationships, may also explain why simple additive models have revealed little effect of shared environments on IQ variance [e.g., Plomin, Asbury, & Dunn, 2001]: what is described as 'genetic' variance is really interactive environmental and/or gene-environmental in origin. This possibility has been well discussed. For example, Turkheimer and Waldron [2000] implicate the role of 'complex reciprocal' effects in phenotype \times environment interactions in which the environment 'is all interaction and little main effect' (p. 92). Again, this necessitates looking at IQ correlations as products of acting, representing, and cognizing individuals, operating in a cultural milieu with differentiated expectations, instead of passive products of genes and environments.

Another reason for the apparent neglect of interactive gene and environmental effects may be that they demand a paradigmatically different view of development and variation in it. On the one hand, it may mean dropping the idea of intelligence and its development as a simple quantitative trait varying as the sums of additive factors. The alternative view is that intelligence is itself a cognitive system that evolved for abstracting the statistical structure from highly changeable circumstances. In humans this means the acquisition of the cultural tools that mediate such adaptability [Richardson, 1998; Rogoff, 2003]. While it will always be possible to devise indices of trait values that portray trait variation 'as if' that of a simple, quantitative trait, it is meaningless to attempt to describe such variation and its development independently from the ecological conditions in which it is embedded [Coen, 1999].

On the other hand, an appreciation of interactive factors will demand modification of simple path diagrams of linear causation from independent factors to additively predictable trait values. Recent dynamic system perspectives [e.g., Lickliter & Honeycut, 2003] urge a move from elemental, deterministic models of development to a focus on interactive, self-organizing processes among numerous non-linear factors. Causes of normal, adaptive variation in such systems need to be distinguished from those creating pathological variation. 'Pathological' elements in traffic flows, such as one or several defective vehicles or drivers, may have cataclysmic effects on specific movements at specific times. But a focus on such independent elements would be hopeless in describing normal ('non-pathological') variation in traffic flows. This cat-

egory error may explain why ‘main effects’ models have been disappointing in the study of human development [see Harper, 2005]. The point is that non-linear, self-organizing systems are adaptive in a creative sense, often constructing very unexpected results, rather than passive outputs of linear, deterministic systems. In describing such systems, the aims and priorities of science move from the prediction of specific outcomes in a system (such as predicting specific values of IQ in specific children), to encouraging the researcher to understand the overall organization and true nature of adaptable systems, and thus to promoting the development of them in a more general sense [Mainzer, 1997].

This does not make genetic variation immaterial in development at any levels. However, in view of the role of these robust, interactive, developmental systems at various levels, we suggest that standard behaviour genetic assumptions may only be valid in specific cases (and at specific levels) where ‘main effects’ do indeed arise. These include the effects of well-known deleterious genetic mutations, and teratogenic environmental effects, which produce categorical disease states (and, thus, entailing a causal pattern quite different from that involved in ‘normal’ variation). In such cases adoption studies may (with care) become informative [Strachan & Read, 1999], though always bearing in mind that many pathological effects are ‘complex’ (in the sense mentioned earlier) and that genetic effects are always environmentally conditioned, and vice versa. Otherwise ignoring the interactive systems has, we suggest, been most responsible for the erroneous interpretations of adoption studies we have discussed here, and the deep controversies the studies have produced within psychology and elsewhere.

References

- Anway, M.D., Cupp, A.S., Uzumcu, M., & Skinner, M.K. (2005). Epigenetic transgenerational actions of endocrine disruptors and male fertility. *Science*, 308, 1466–1469.
- Balaban, E. (2001). Behaviour genetics: Galen's prophecy or Malpighi's legacy. In R.S. Singh, C.B. Krimbas, D.B. Paul, & J. Beatty (Eds.) *Thinking about evolution*. (Vol. 2, pp. 429–466). Cambridge, UK: Cambridge University Press.
- Baumrind, D. (1983). The average expectable environment is not good enough: A response to Scarr. *Child Development*, 64, 1299–1317.
- Brodzinsky, D.M., Singer, L.M., & Braff, A.M. (1984). Children's understanding of adoption. *Child Development*, 55, 869–878.
- Brodzinsky, D.M., Smith, D.W., & Brodzinsky, A.B. (1998). *Children's adjustment to adoption: Developmental and clinical issues*. Thousand Oaks, CA: Sage.
- Carpenter, P.A., Just, M.A., & Shell, P. (1990). What one intelligence test measures: A theoretical account of the processing in the Raven Progressive Matrices Test. *Psychological Review*, 97, 404–431.
- Cherny, S.S., Fulker, D.W., & Hewitt, J.K. (1997). Cognitive development from infancy to middle childhood. In R.J. Sternberg & E. Grigorenko (Eds.), *Intelligence, heredity and environment*. (pp. 463–482). Cambridge, UK: Cambridge University Press.
- Coen, E. (1999). *The art of the genes: How organisms make themselves*. Oxford: Oxford University Press.
- Cole, M. (1999). Culture-free versus culture-based measures of cognition. In R.J. Sternberg (Ed.), *The nature of cognition*. (pp. 645–664). Cambridge, MA: MIT Press.
- Collins, W.A., Maccoby, E.E., Steinberg, L., Hetherington, E.M., & Bornstein, M.H. (2000). Contemporary research on parenting. *American Psychologist*, 55, 218–232.
- Coon, H., Carey, G., Corley, R., & Fulker, D. (1992). Identifying children in the Colorado Adoption Project at risk for conduct disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 31, 503–511.
- DeFries, J.C., Plomin, R., Vandenberg, S.G., & Kuse, A.R. (1981). Parent-offspring resemblance for cognitive abilities in the Colorado Adoption Project: Biological, adoptive and control parents and one-year-old children. *Intelligence*, 5, 245–277.

- DeCasper, A.J., & Spence, M.J. (1986). Prenatal maternal speech influences newborns' perception of speech sounds. *Infant Behaviour and Development*, 9, 133–150.
- Feigelman, W. (1997). Adopted adults: Comparisons with persons raised in conventional families. In H.E. Gross & M.B. Sussman (Eds.), *Families and adoption*. (pp. 199–223). New York: Haworth Press.
- Ferguson, D.M., Lynskey, M., & Horwood, L.J. (1995). The adolescent outcomes of adoption: A 16-year longitudinal study. *Journal of Child Psychology and Psychiatry*, 36, 597–615.
- Flynn, J.R. (1998). IQ gains over time: Towards finding the causes. In U. Neisser (Ed.), *The rising curve*. (pp. 25–66). Washington, American Psychological Association.
- Francis, D., Diorio, J., Liu, D., & Meaney, M.J. (1999). Nongenetic transmission across generations of maternal behaviour and stress response in the rat. *Science*, 286, 1155–1157.
- Glazier, A.M., Nadeau, J.H., & Aitman, T.J. (2002). Finding genes that underlie complex traits. *Science*, 298, 2345.
- Gottlieb, G. (1998). Normally occurring environmental and behavioral influences on gene activity: From central dogma to probabilistic epigenesis. *Psychological Review*, 105, 792–802.
- Gottlieb, G. (2000). Environmental and behavioral influences on gene activity. *Current Directions in Psychological Science*, 9, 93–97.
- Hajal, F., & Rosenberg, E.G. (1991). The family life cycle in adopted families. *American Journal of Orthopsychiatry*, 61, 78–85.
- Harper, L.V. (2005). Epigenetic inheritance and the intergenerational transfer of experience. *Psychological Bulletin*, 131, 340–360.
- Horn, J.M., Loehlin, J.C., & Willerman, L. (1979). Intellectual resemblance among adoptive and biological relatives: The Texas Adoption Project. *Behavior Genetics*, 9, 177–207.
- Horowitz, F.D. (2000). Child development and the PITS: Simple questions, complex answers, and developmental theory. *Child Development*, 71, 1–10.
- Howe, D. (1998). *Patterns of adoption*. Oxford: Blackwell.
- Jackson, J.F. (1993). Human behavioral genetics, Scarr's theory, and her views on interventions: A critical review and commentary on their implications for African American children. *Child Development*, 6, 1318–1332.
- Jensen, A.R. (1997). The puzzle of nongenetic variance. In R.J. Sternberg & E. Grigorenko (Eds.), *Intelligence, heredity and environment*. (pp. 42–88). Cambridge, UK: Cambridge University Press.
- Jensen, A.R. (1998). *The g-factor*. Westport, CA: Praeger.
- Joseph, J. (2003). *The gene illusion*. Ross-on-Wye, UK: PCCS Books.
- Langlois, J.H. (1988). From the eye of the beholder to behavioural reality: Development of social behaviors and social relations as a function of physical attractiveness. In C.P. Herman, M. Zanna, & E.T. Higgins (Eds.), *Physical appearances, stigma & social behaviour*. (pp. 23–51). Hillsdale, NJ: Erlbaum.
- Larson, G. (1999). *The parenting experiences of infertile couples who adopt*. Unpublished Ph.D. dissertation, Chicago School of Professional Psychology.
- Lerner, R.M. (2002). *Concepts and theories of human development*. Mahwah, NJ: Erlbaum.
- Lewontin, R., Rose, S., & Kamin, L. (1984). *Not in our genes*. Harmondsworth: Penguin.
- Lickliter, R., & Honeycutt, H. (2003). Developmental dynamics: Toward a biologically plausible evolutionary psychology. *Psychological Bulletin*, 129, 819–835.
- Lin, Q., Di W., & Zhou, J. (2003). The PTS element facilitates and restricts a distant enhancer to a single promoter in the transgenic *Drosophila* embryos. *Development*, 130, 519–526.
- Loehlin, J.C., Horn, J.M., & Willerman, L. (1997). Heredity, environment and IQ in the Texas Adoption Project. In R.J. Sternberg & E. Grigorenko (Eds.), *Intelligence, heredity and environment*. (pp. 105–125). Cambridge, UK: Cambridge University Press.
- Maccoby, E. (2001). Parenting and its effects on children: Misreading behavior genetic evidence. *Annual Review of Psychology*, 51, 1–28.
- MacDonald, K., & Hershberger, S. (2005). Theoretical issues in the study of evolution and development. In R. Burgess & K. MacDonald (Eds.), *Evolutionary Perspectives on Human Development*. (2nd ed.). Thousand Oaks, CA: Sage.
- Maddux, J.E. (Ed.). (1995). *Self-efficacy, adaptation and adjustment: Theory, research and application*. New York: Plenum.
- Mainzer, K. (1997). *Thinking in complexity: The complex dynamics of matter, mind and mankind*. Berlin: Springer.
- Merila, J., & Sheldon, B.C. (1999). Genetic architecture of fitness and nonfitness traits. *Heredity*, 83, 103–109.
- Moore, D.S. (2002). *The dependent gene: The fallacy of nature vs. nurture*. New York: Freeman.
- Munsinger, H. (1975). The adopted child's IQ: A critical review. *Psychological Bulletin*, 82, 623–659.
- Neisser, U., Boodoo, G., Bouchard, T.J. Jr., Boykin, A.W., Brody, N., Ceci, S.J., et al. (1995). Intelligence: Knowns and unknowns. *American Psychologist*, 51, 77–101.

- Newman, T.K., Sygailo, Y.V., Barr, C.S., Jens, R., Wendland, J.R., Champoux, M., et al. (2005). Monoamine oxidase: A gene promoter variation and rearing experience influences aggressive behavior in rhesus monkeys. *Biological Psychiatry*, *57*, 167–172.
- Nijhout, H.F., & Davidowitz, G. (2003). Developmental perspectives on phenotypic plasticity, canalization, and fluctuating asymmetry. In M. Polak (Ed.), *Developmental instability: causes and consequences*. (pp. 3–13). Cambridge, MA: MIT Press.
- Nuffield Council on Bioethics (2002). *Genetics and human behavior: The ethical context*. London: Nuffield Council on Bioethics.
- Oyama, S. (2001). *Evolution's eye*. Durham, NC: Duke University Press.
- Peccoud, J., Venden, K.V., Podlich, D., Winkler, C., Arthur, L., & Cooper, M. (2004). The selective values of alleles in a molecular network model are context dependent. *Genetics*, *166*, 1715–1725.
- Plomin, R. (1986). *Development, genetics and psychology*. Hillsdale, NJ: Erlbaum.
- Plomin, R., Asbury, K., & Dunn, J. (2001). Why are children in the same family so different? Nonshared environment a decade later. *Canadian Journal of Psychiatry*, *46*, 225–243.
- Plomin, R., & DeFries, J.C. (1985). *Origins of individual differences in infancy: The Colorado Adoption Project*. New York: Academic Press.
- Plomin, R., DeFries, J.C., McClearn, G.E., & Rutter, M. (1997). *Behavioral genetics*. (3rd ed.). New York: Freeman.
- Plomin, R., Fulker, D.W., Corley, R., & DeFries, J.C. (1997). Nature, nurture and cognitive development from 1 to 16 years: A parent-offspring adoption study. *Psychological Science*, *6*, 442–447.
- Plomin, R., & Loehlin, J.C. (1989). Direct and indirect heritability estimates: A puzzle. *Behavior Genetics*, *19*, 331–342.
- Plomin, R., & Spinath, F.M. (2004). Intelligence: Genetics, genes, and genomics. *Journal of Personality and Social Psychology*, *86*, 112–129.
- Raven, J., Raven, J.C., & Court, J.H. (1993). *Manual for Raven's Progressive Matrices and Vocabulary Scales: Section 1*. Oxford: Oxford Psychologists Press.
- Rhea, S.A., & Corley, R. (1994). Applied issues. In J.C. DeFries, R. Plomin, & D.W. Fulker (Eds.) *Nature and nurture in middle childhood*. (pp. 295–309). Oxford: Blackwell.
- Richardson, K. (1998). *Origins of human potential: Evolution, development & psychology*. London: Routledge.
- Richardson, K. (2001). What IQ tests test. *Theory & Psychology*, *12*, 283–314.
- Richardson, K. (2005). G is about variance not contents. *Cortex*, *41*, 1–3.
- Richardson, K., & Norgate, S.H. (2005). The equal environments assumption of classical twin studies may not hold. *British Journal of Educational Psychology*, *75*, 339–350.
- Rogoff, B. (2003). *The cultural nature of human development*. Oxford: Oxford University Press.
- Rosenberg, E.G., & Horner, T.M. (1991). Birthparent romances and identity formation in adopted children. *American Journal of Orthopsychiatry*, *61*, 70–77.
- Rowe, D.C., Clapp, M., & Wallis, J. (1987). Physical attractiveness and the personality resemblance of identical twins. *Behaviour Genetics*, *17*, 191–201.
- Rutter, M. (2002). Nature, nurture and development: From evangelism through science toward policy and practice. *Child Development*, *73*, 1–21.
- Rutter, M., Pickles, A., Murray, R., & Eaves, L. (2001). Testing hypotheses on specific environmental causal effects on behavior. *Psychological Bulletin*, *127*, 291–324.
- Sacerdote, B. (2004). What happens when we randomly assign children to families? *National Bureau for Economic Research*, Working Paper No. 10894.
- Scarr, S. (1997). Behavior genetic and socialization theories of intelligence: Truce and reconciliation. In R.J. Sternberg & E. Grigorenko (Eds.), *Intelligence, heredity and environment*. (pp. 3–41). Cambridge: Cambridge University Press.
- Scarr, S., & Carter-Saltzman, L. (1989). Genetics and intelligence. In R.J. Sternberg (Ed.), *Handbook of human intelligence*. (pp. 792–896). Cambridge: Cambridge University Press.
- Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype-environment effects. *Child Development*, *54*, 424–435.
- Scarr, S., Scarf, E., & Weinberg, R.A. (1980). Perceived and actual similarities in biological and adoptive families: Does perceived similarity bias genetic inferences? *Behavior Genetics*, *10*, 445–458.
- Scarr, S., & Weinberg, R.A. (1978). The influence of 'family background' on intellectual attainment. *American Sociological Review*, *43*, 674–692.
- Seckl, J.R., & Holmes, M.C. (2001). Glucocorticoid programming of brain corticosteroid receptors and corticotrophin-releasing hormone: Possible implications for behaviour. *Neuroscience*, *104*, 1–23.
- Smith, D.W., & Brodzinsky, D. (2002). Coping with birthparent loss in adopted children. *Journal of Child Psychology & Psychiatry*, *43*, 213–223.
- Stein, A.D., & Lumey, L.H. (2000). The relationship between maternal and offspring birth weights after maternal prenatal famine exposure: The Dutch Famine Birth Cohort Study. *Human Biology*, *72*, 1–10.

- Stoolmiller, M. (1998). Implications of the restricted range of family environments for estimates of heritability and nonshared environment in behavior-genetic adoption studies. *Psychological Bulletin*, *125*, 392–409.
- Strachan, T., & Read, A.P. (1999). *Human molecular genetics*. Oxford, UK: Bios Scientific Publishers.
- Suomi, S.J. (2003). Gene-environment interactions and the neurobiology of social conflict. *Annals of the New York Academy of Sciences*, *1008*, 132–139.
- Teerwong, M.M., Hoeksma, J.B., & Koops, W. (1993). Common beliefs about the heredity of human characteristics. *British Journal of Psychology*, *84*, 499–503.
- Turkheimer, E. (2005). Spinach and ice cream: Why social science is so difficult. In Di Lalla, L.F. (Ed.), *Behavior genetics principles: Perspectives in development, personality and psychopathology*. (pp. 161–189). Washington, American Psychological Association.
- Turkheimer, E., & Waldron, M. (2000). Nonshared environment: A theoretical, methodological and quantitative critique. *Psychological Bulletin*, *126*, 78–108.
- Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., & Gottesman, I.I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*, *14*, 623–628.
- Turner, B.M. (2000). Histone acetylation and an epigenetic code. *Bioessays*, *9*, 836–845.
- Wahlsten, D. (2000). Analysis of variance in the service of interactionism. *Human Development*, *43*, 46–50.
- Yehuda, R., Engel, S.M., Brand, S.R., Seckl, J., Marcus, S.M., & Berkowitz, G.S. (2005). Transgenerational effects of posttraumatic stress disorder in babies of mothers exposed to the World Trade Center attacks during pregnancy. *The Journal of Clinical Endocrinology & Metabolism*, *90*, 4115–4118.
- Zeidner, M. (1995). Personality trait correlates of intelligence. In D.H. Saklofske & M. Zeidner (Eds.), *International handbook of personality and intelligence*. (pp. 299–319). New York: Plenum.

Copyright: S. Karger AG, Basel 2006. Reproduced with the permission of S. Karger AG, Basel.
Further reproduction or distribution (electronic or otherwise) is prohibited without permission
from the copyright holder.