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REVIEW ARTICLE

Genetic influences on measures of the environment: a systematic review

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ABSTRACT

Background. Traditional models of psychiatric epidemiology often assume that the relationship between individuals and their environment is unidirectional, from environment to person. Accumulating evidence from developmental and genetic studies has made this perspective increasingly untenable.

Method. Literature search using Medline, PsycINFO, article references and contact with experts to identify all papers examining the heritability of measures of environments of relevance to psychiatry/psychology.

Results. We identified 55 independent studies organized into seven categories: general and specific stressful life events (SLEs), parenting as reported by child, parenting reported by parent, family environment, social support, peer interactions, and marital quality. Thirty-five environmental measures in these categories were examined by at least two studies and produced weighted heritability estimates ranging from 7% to 39%, with most falling between 15% and 35%. The weighted heritability for all environmental measures in all studies was 27%. The weighted heritability for environmental measures by rating method was: self-report 29%, informant report 26%, and direct rater or videotape observation (typically examining 10 min of behavior) 14%.

Conclusion. Genetic influences on measures of the environment are pervasive in extent and modest to moderate in impact. These findings largely reflect 'actual behavior' rather than 'only perceptions'. Etiologic models for psychiatric illness need to account for the non-trivial influences of genetic factors on environmental experiences.

INTRODUCTION

In traditional psychiatric epidemiological models of disease etiology, the causal relationship is conceptualized as moving from the environment to the organism. However, the unidirectionality of this association has been increasingly questioned. In the developmental literature, it is now widely accepted that organisms both impact on and are impacted by their environment (Bell, 1968; Wachs & Plomin, 1991).

Genetic approaches can provide valuable information about the nature of the relationship between individuals and their environment. Many important aspects of human behavior are significantly influenced by genetic factors (Plomin *et al.* 2003; Kendler & Eaves, 2005; Kendler & Prescott, 2006). If the association between individuals and their environment solely takes the form of environment→person,

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then genes ought to have no influence on environmental exposures and the heritability of environment experiences should be zero. However, if an individual's own behavior impacts on the environmental exposures and if the relevant aspect of behavior is itself subject to genetic influences, then these environmental measures ought to be heritable.

The concept that individuals play an active role in selecting, modifying and constructing their environment is widely accepted in evolutionary biology (Dawkins, 1982; Odling-Smee *et al.* 2003). In his book *The Extended Phenotype*, Dawkins (1982) provides many examples of genes that 'extend' their phenotype outside the skin of the organism that possesses them. In what has been termed 'niche construction', animals such as beavers, weaver birds and termites modify their physical environment through building dams or constructing nursery environments for their offspring (Odling-Smee *et al.* 2003). In addition to effects on the physical environment, in social animals, genes can impact on key aspects of the social environment such as parent–offspring, mate and adult–peer relationships. In the evolutionary genetics literature, this is called 'indirect genetic effects' (Agrawal *et al.* 2001). Studies in model organisms have found significant genetic effects on a wide range of behaviors influencing the physical and social environment, including nest construction, selection of micro-environments, maternal and affiliative behaviors, infant behaviors that communicate distress to the mother and intra-species aggression (Kendler & Greenspan, 2006). Plomin *et al.* (1977) distinguished between passive, reactive and active forms of genetic influence on environmental measures, or as they termed it: 'genotype–environment correlation'.

In this paper, we review systematically, for the first time to our knowledge, research that uses human genetically informative samples to examine the heritability of measures of the environment, focusing on those environmental constructs of etiological importance for psychiatric and drug use disorders. (Heritability is defined as the proportion of individual differences for a trait in a particular population that results from inter-individual genetic differences.) Examining the heritability of the environmental exposure, which has been variously called gene–environment covariance, gene–environment correlation or genetic control of exposure to the environment (Kendler & Eaves, 1986), is of interest to the field of mental health because it provides insight into the nature of the causal relationship between humans and their social and physical surroundings.

METHOD

We began with literature searches conducted in Medline and PsycINFO. Search terms included 'twin' or 'genetic' along with: 'stressful life events', 'social support', 'friendship', 'marriage', 'trauma/traumatic', 'parenting', 'peer deviance', 'peer relationship', 'negative life event' and 'parent child relationship'. A blanket search was also conducted with only the terms 'stressful life event' and 'social support' in an effort to ensure that no relevant studies were missed. Next, an extensive review of the references of relevant articles was carried out. Finally, a preliminary list of references was emailed to prominent researchers in their field for review.

Articles were included in this review if they included a methodological design that estimated the genetic influences on an environmental variable. We identified studies using twin, adoption and step-family designs. We only included published articles where the primary language was English and the environment under investigation had been related to risk for psychiatric disorders and/or psychological health. We found at least two studies that examined five broad categories of the environment: social support, parenting behavior, family environment, peer interactions, and stressful life events (SLEs). In general, for a specific variable to be included in this review, we had to identify two articles that examined its heritability. However, exceptions to this rule were made if the article was of particular interest or there were a limited number of studies available in a specific category. To implement this rule it was necessary to make judgments as to whether variables from separate studies were measuring comparable constructs. For example, within the category of SLEs, we concluded that controllable and dependent events (where dependent is defined as probably resulting from the respondent's own behavior), uncontrollable

and independent events (where dependent is defined as probably unrelated to the respondent's own behavior), negative and undesirable events and positive and desirable events were sufficiently similar to be combined together. For parenting behavior, warmth, positivity and acceptance, authoritarianism and control, and anger and negativity were treated as equivalent. Finally, within peer interactions, general peer delinquency and peer substance use were sufficiently similar to be examined together. We use the term 'general' SLEs to refer to those events that were typically assessed in a general life event inventory while 'specific' SLEs (such as divorce or combat exposure) were assessed in studies focusing on that single variable.

Although it would have been ideal to conduct formal meta-analyses of this literature, this was not feasible. Very few primary reports provided confidence intervals (or standard errors) of the estimates or primary data (i.e. contingency tables or correlations).

When multiple reports were found from the same data set, we used the one judged most relevant. The exception to this general rule comes within the category of social support, where three different studies from the Virginia Twin Registry (VTR) are reported. Each of these three studies uses a different interview wave for analyses but on overlapping twin subjects. Because of this, a summary statistic was calculated for the overlapping variables between these three VTR studies. Once all relevant articles were obtained, a weighted mean heritability, with the weights based on the sample size, was calculated for those variables that were assessed in at least two studies and the results are summarized in Table 1. We also prepared for this review nine Appendix tables, termed A1 to A9, that provide a study by study summary. These tables are available in the online version of this paper and from our website (www.vipbg.vcu.edu/~jbaker).

RESULTS

General SLEs

Ten twin studies were identified that examined the heritability of general SLEs (Wierzbicki, 1989; Plomin *et al.* 1990; Kendler *et al.* 1993, 1999; Billig *et al.* 1996; Foley *et al.* 1996; Thapar & McGuffin, 1996; Saudino *et al.* 1997; Bolinskey *et al.* 2004; Wang *et al.* 2005). Six of these studies reported on the heritability of total SLEs (Wierzbicki, 1989; Plomin *et al.* 1990; Kendler *et al.* 1993; Thapar & McGuffin, 1996; Bolinskey *et al.* 2004; Wang *et al.* 2005). In five of these studies (Wierzbicki, 1989; Plomin *et al.* 1990; Kendler *et al.* 1993; Bolinskey *et al.* 2004; Wang *et al.* 2005), the heritabilities ranged from 24% to 47%. The weighted mean across all six studies was 28% (Table 1).

We identified four studies that reported heritabilities of negative and positive SLEs, with the latter being slightly more heritable (Wierzbicki, 1989; Plomin *et al.* 1990; Thapar & McGuffin, 1996; Saudino *et al.* 1997). Seven and five studies respectively examined independent (Plomin *et al.* 1990; Billig *et al.* 1996; Foley *et al.* 1996; Thapar & McGuffin, 1996; Saudino *et al.* 1997; Kendler *et al.* 1999; Bolinskey *et al.* 2004) and dependent SLEs (Plomin *et al.* 1990; Billig *et al.* 1996; Foley *et al.* 1996; Kendler *et al.* 1999; Bolinskey *et al.* 2004). Weighted mean heritabilities were substantially lower for the independent (17%) than for the dependent stressful events (31%).

Specific life events

We identified six studies, reviewed in Table A2, that examined the heritability of specific life events (McGue & Lykken, 1992; Lyons *et al.* 1993; Jang *et al.* 2001; Stein *et al.* 2002; Johnson *et al.* 2004; Middeldorp *et al.* 2005). Four of these studies examined the propensity of individuals to select themselves into traumatic situations: three in civilian life (Jang *et al.* 2001; Stein *et al.* 2002; Middeldorp *et al.* 2005) and one in the Vietnam War (Lyons *et al.* 1993). Reported heritabilities ranged from 20% to 63%, with a weighted mean of 36%. Two studies examined the heritability of divorce, reporting heritabilities of 29% (Middeldorp *et al.* 2005) and 53% (McGue & Lykken, 1992) with a weighted mean of 35%. Two studies reported heritabilities of various non-assaultive traumas (Jang *et al.* 2001; Stein *et al.* 2002) that were low, estimated at 7%.

Parenting behavior

We located a total of 19 individual studies that examined the heritability of various aspects of the parent–child relationship (Rowe, 1981, 1983; Rende *et al.* 1992; Perusse *et al.* 1994;

Table 1. *The weighted mean heritability across studies of various aspects of the environment*

Constructs	No. studies	Studies included	Total *N*	Weighted mean
Stressful life events				
Total life events	6	Wierzbicki, 1989; Plomin *et al.* 1990; Kendler *et al.* 1993; Thapar & McGuffin, 1996; Bolinskey *et al.* 2004; Wang *et al.* 2005	6197	0·28
Negative life events	3	Plomin *et al.* 1990; Wierzbicki, 1989; Thapar & McGuffin, 1996	731	0·39
Positive life events	3	Plomin *et al.* 1990; Wierzbicki, 1989; Thapar & McGuffin, 1996	731	0·34
Independent life events	6	Plomin *et al.* 1990; Billig *et al.* 1996; Thapar & McGuffin, 1996; Foley *et al.* 1996; Kendler *et al.* 1999; Bolinskey *et al.* 2004	5056	0·17
Dependent life events	5	Plomin *et al.* 1990; Billig *et al.* 1996; Foley *et al.* 1996; Kendler *et al.* 1999; Bolinskey *et al.* 2004	4459	0·31
Selection into trauma	4	Lyons *et al.* 1993; Jang *et al.* 2001; Stein *et al.* 2002; Middeldorp *et al.* 2005	6558	0·36
Selection into non-assaultive trauma	2	Jang *et al.* 2001; Stein *et al.* 2002	569	0·07
Divorce	2	McGue & Lykken, 1992; Middeldorp *et al.* 2005	5692	0·35
Child-based reports of parenting behavior				
Maternal warmth	7	Rowe, 1981; Rende *et al.* 1992; Plomin *et al.* 1994; O'Connor *et al.* 1995; Kendler, 1996; Lichtenstein *et al.* 2003; Neiderhiser *et al.* 2004	3446	0·37
Paternal warmth	5	Rowe, 1981; Plomin *et al.* 1994; O'Connor *et al.* 1995; Kendler, 1996; Lichtenstein *et al.* 2003	2664	0·34
Maternal control	5	Rende *et al.* 1992; O'Connor *et al.* 1995; Kendler, 1996; Lichtenstein *et al.* 2003; Neiderhiser *et al.* 2004	2330	0·15
Paternal control	3	O'Connor *et al.* 1995; Kendler, 1996; Lichtenstein *et al.* 2003	1448	0·17
Paternal negativity	2	Plomin *et al.* 1994; O'Connor *et al.* 1995	377	0·12
Paternal protectiveness	2	Kendler, 1996; Lichtenstein *et al.* 2003	2198	0·20
Maternal protectiveness	2	Kendler, 1996; Lichtenstein *et al.* 2003	2198	0·26
Parent-based reports of parenting behavior				
Parental warmth	4	Perusse *et al.* 1994; Kendler, 1996; Deater-Deckard *et al.* 1999; Deater-Deckard, 2000	1690	0·35
Parental control	3	Kendler, 1996; Losoya *et al.* 1997; Spinath & O'Connor, 2003	433	0·20
Parental protectiveness	3	Perusse *et al.* 1994; Kendler, 1996; Spinath & O'Connor, 2003	1477	0·23
Parental negativity	7	O'Connor *et al.* 1995; Losoya *et al.* 1997; Deater-Deckard *et al.* 1999, 2001; Deater-Deckard, 2000; Neiderhiser *et al.* 2004; Boivin *et al.* 2005	4766	0·19
Family environment				
Cohesion/connectedness	4	Plomin *et al.* 1988, 1989; Jacobson & Rowe, 1999; Jang *et al.* 2001	1911	0·24
Conflict	3	Plomin *et al.* 1988, 1989; Jacobson & Rowe, 1999	1428	0·30
Organization	3	Plomin *et al.* 1988, 1989; Jacobson & Rowe, 1999	1428	0·25
Expressiveness	3	Plomin *et al.* 1988, 1989; Jacobson & Rowe, 1999	1428	0·24
Active	3	Plomin *et al.* 1988, 1989; Jacobson & Rowe, 1999	1428	0·26
Control	3	Plomin *et al.* 1988, 1989; Jacobson & Rowe, 1999	1428	0·18
Social support				
Friend problem	2	Kendler *et al.* 1997; Agrawal *et al.* 2002	2860	0·23
Relative problem	2	Kendler *et al.* 1997; Agrawal *et al.* 2002	2860	0·38
Friend support	3	Kessler *et al.* 1992; Kendler *et al.* 1997; Agrawal *et al.* 2002	4502	0·17
Relative support	3	Kessler *et al.* 1992; Kendler *et al.* 1997; Agrawal *et al.* 2002	4502	0·31
Confidants	3	Kessler *et al.* 1992; Kendler *et al.* 1997; Agrawal *et al.* 2002	4502	0·31
Social integration	2	Kendler *et al.* 1997; Agrawal *et al.* 2002	2860	0·31

Peer interactions				
Peer deviance	4	Iervolino *et al.* 2002; Rose, 2002; White *et al.* 2003; Walden *et al.* 2004	3012	0·21
Marital quality				
Marital satisfaction	2	Spotts *et al.* 2004*b*, 2005	752	0·28
Marital conflict	2	Spotts *et al.* 2005; Spotts, unpublished data	1659	0·13
Marital warmth	3	Spotts *et al.* 2004*b*, 2005; Spotts, unpublished data	1985	0·17
Observer/informant reports				
Maternal control	3	Rende *et al.* 1992; O'Connor *et al.* 1995; Neiderhiser *et al.* 2004	639	0·12
Maternal affection	4	Rende *et al.* 1992; O'Connor *et al.* 1995; Kendler, 1996; Deater-Deckard, 2000; Neiderhiser *et al.* 2004	1695	0·14
Maternal negativity	3	O'Connor *et al.* 1995; Deater-Deckard, 2000; Neiderhiser *et al.* 2004	635	0·06
Heritability of self-report measures	197 constructs	All studies that gave sample size information	124 464	0·29
Heritability of observer-report measures	42 constructs	All studies that gave sample size information	9032	0·14
Heritability of informant-report measures	18 constructs	All studies that gave sample size information	8856	0·26
Heritability of all non-self-report measures	60 constructs	All studies that gave sample size information	16 210	0·21
Heritability of all environmental measures	265 constructs	All studies that gave sample size information	141 460	0·27

Plomin *et al.* 1994; O'Connor *et al.* 1995; Kendler, 1996; Elkins *et al.* 1997; Losoya *et al.* 1997; Deater-Deckard *et al.* 1999, 2001; Deater-Deckard, 2000; Wade & Kendler, 2000; Lichtenstein *et al.* 2003; Spinath & O'Connor, 2003; Neiderhiser *et al.* 2004; Walden *et al.* 2004; Boivin *et al.* 2005; Herndon *et al.* 2005). We divided them into studies that examined this relationship from the perspective of the child *versus* from the perspective of the parent. While child-based designs assess the role of genetic factors in the *elicitation* of parental behavior, parent-based designs evaluate how genes impact on the *provision* of parental care. The results are shown in Tables A3–A5.

Child-based designs

As outlined in Tables A3 and A4, we identified 12 studies examining the heritability of parental behavior through child reports. Two or more studies were found that reported on maternal warmth (Rowe, 1981; Rende *et al.* 1992; Plomin *et al.* 1994; O'Connor *et al.* 1995; Kendler, 1996; Lichtenstein *et al.* 2003; Neiderhiser *et al.* 2004), paternal warmth (Rowe, 1981; Plomin *et al.* 1994; O'Connor *et al.* 1995; Kendler, 1996; Lichtenstein *et al.* 2003), maternal control (Rende *et al.* 1992; Kendler, 1996; O'Connor *et al.* 1998; Lichtenstein *et al.* 2003; Neiderhiser *et al.* 2004), paternal control (O'Connor *et al.* 1995; Kendler, 1996; Lichtenstein *et al.* 2003), paternal negativity (Plomin *et al.* 1994; O'Connor *et al.* 1995), and paternal and maternal protectiveness (Kendler, 1996; Lichtenstein *et al.* 2003). The weighted heritabilities were highest for measures of parental warmth (34–37%), intermediate for measures of protectiveness (20–26%) and lowest for measures of control and negativity (12–17%).

Parent-based designs

As outlined in Table A4, 10 studies were identified that examined the heritability of parental behavior from parent reports (Perusse *et al.* 1994; Plomin *et al.* 1994; Kendler, 1996; Losoya *et al.* 1997; Deater-Deckard *et al.* 1999, 2001; Deater-Deckard, 2000; Spinath & O'Connor, 2003; Neiderhiser *et al.* 2004; Boivin *et al.* 2005). Two or more studies were found that reported on parental warmth (Perusse *et al.* 1994; Kendler, 1996; Deater-Deckard *et al.* 1999,

2001), control (Kendler, 1996; Losoya *et al.* 1997; Spinath & O'Connor, 2003), protectiveness (Perusse *et al.* 1994; Kendler, 1996; Spinath & O'Connor, 2003) and negativity (O'Connor *et al.* 1995; Losoya *et al.* 1997; Deater-Deckard *et al.* 1999, 2001; Deater-Deckard, 2000; Neiderhiser *et al.* 2004; Boivin *et al.* 2005). As shown in Table 1, heritability was substantially higher for the dimension of parental warmth (35%) than for the remaining dimensions of parental behavior (19–23%).

Family environment

We located seven studies that examined various aspects of the environment either in the family of origin or in the current family (Plomin *et al.* 1988, 1989; Hur & Bouchard, 1995; Deater-Deckard *et al.* 1999; Jacobson & Rowe, 1999; Jang *et al.* 2001; Herndon *et al.* 2005) (Table A6). From these reports, we were able to extract weighted estimates for the heritability of six dimensions of family functioning: cohesion (Plomin *et al.* 1988, 1989; Jacobson & Rowe, 1999; Jang *et al.* 2001), conflict (Plomin *et al.* 1988, 1989; Jang *et al.* 2001), organization (Plomin *et al.* 1988, 1989; Jang *et al.* 2001), expressiveness (Plomin *et al.* 1988, 1989; Jang *et al.* 2001), activity (Plomin *et al.* 1988, 1989; Jang *et al.* 2001) and control (Plomin *et al.* 1988, 1989; Jang *et al.* 2001). These estimates were relatively similar across these constructs, varying from 18% to 30%.

Social support

We identified five studies that examined genetic influences on social support (Table A7) (Bergeman *et al.* 1990; Kessler *et al.* 1992; Kendler *et al.* 1997; Agrawal *et al.* 2002; Raynor *et al.* 2002). Three studies come from different interviews with the VTR, from which we could construct six overlapping measures (Kessler *et al.* 1992; Kendler *et al.* 1997; Agrawal *et al.* 2002): friend problems, relative problems, relative support, friend support, confidants, and social integration. Weighted means for these measures ranged from 17% to 38%. Two other studies report heritabilities for constructs closely related to social support that were broadly consistent with those estimated from the VTR samples (Bergeman *et al.* 1990; Raynor *et al.* 2002).

Peer interactions

We found six studies examining the heritability of various aspects of peer relationships (Table A8) (Manke *et al.* 1995; Iervolino *et al.* 2002; Rose, 2002; White *et al.* 2003; Walden *et al.* 2004; Rushton & Bons, 2005). Four studies assessed peer deviancy (Iervolino *et al.* 2002; Rose, 2002; White *et al.* 2003; Walden *et al.* 2004), with a weighted heritability estimate of 21%. Two other studies examined other aspects of friend relationships, suggesting that genetic factors had a significant impact on the chances of having negative interactions with friends (Manke *et al.* 1995) and choosing friends similar to oneself (Rushton & Bons, 2005).

Marital quality

We located four studies that assessed genetic influences on various aspects of marital relationships (Table A9) (Spotts *et al.* 2004*a*, *b*, 2005; Spotts *et al.* in press). Two or more reports were found that examined three marital dimensions: satisfaction, conflict and warmth. Weighted heritabilities for these constructs ranged from 13% to 28%.

Summary results

The articles we reviewed in Tables A1 to A9 included a total of 265 variables and over 100 000 assessments (although multiple assessments were often performed on the same subject). The total weighted mean heritability for all these environmental measures was 27%.

DISCUSSION

The literature we have reviewed suggests that genetic influences on measures of the environment are pervasive in extent and modest to moderate in impact. Every aspect of the environment that we were able to examine was significantly influenced by genetic factors. However, the role of genetic influences on these behaviors was far from overwhelming. The weighted heritability estimates for the 35 constructs that were assessed in at least two studies ranged from 7% to 39%, with most falling between 15% and 35%. These results are consistent with extensive evidence, from non-genetically informative studies, of 'person–environment covariance' (see, for example, Wachs, 1992, ch. 7).

Three trends in these summary results are noteworthy. First, SLEs that are largely dependent on an individual's own behavior are more heritable than 'fateful' events independent of the individual's actions. Second, whether reported by the parent or the child, parenting behavior reflecting the positive emotional quality of the parent–child relationship is more heritable that parenting behavior related to disciplinary styles (e.g. control or protectiveness). This pattern might arise because positive emotionality in parent–child relationships is strongly impacted by the genetically influenced temperament of both parties. By contrast, disciplinary style may be more like a social attitude – an approach towards parenting learned by the parent during their own life experiences and which they attempt to apply equally to all their children.

Third, consistent with expectation, genes from each party in a relationship appear to contribute to its quality. This can be best seen in the parent–child relationship, where the quality of that relationship appears to be impacted in similar ways by the genotype of the parent and the genotype of the child. These results suggest one obvious reason why the heritabilities of interpersonal environments are modest. That is, the quality of an interpersonal relationship is impacted on by at least two genotypes – that of the informant and that of the other individual. Our assessments only measure the former, while the latter typically comes out in the analyses as 'environment'.

Methodological concerns

These results should be interpreted in the context of four major methodological concerns. First, in nearly all of the studies included in this review, measures of the environment were obtained at a single point in time. If we instead examined the stable aspects of experiences, would the heritability of the environment increase? We know of two studies that address this question. Foley *et al.* (1996) examined SLEs reported in the previous 12 months at two interviews separated by at least a year. Using a twin 'measurement model', they were able to separate the contribution of random or occasion-specific events on SLEs from those that are stable over time. The model can then partition the influences on the stable liability to SLEs into its genetic and environmental components. Their best-fitting model indicated that stable individual differences were most important for personal SLEs and only make a small contribution to network events. Of particular interest, they found that about 55% of the variation in personal SLEs results from occasion-specific effects or error. However, the heritability of the stable liability to personal SLEs was 65%. This was approximately twice as high as the standard heritability for SLEs calculated from the same data. That is, correcting for transient environmental and measurement effects, genetic factors were quite important in discriminating those individuals with a stable tendency to have few *versus* large numbers of SLEs.

Using similar methods, Kendler (1997) examined social support assessed twice 5 years apart. Levels of social support were moderately stable over time. The heritability of the temporally stable aspects of social support (which ranged from about 45% to 75%) was more than twice as great as that obtained by measurements on one occasion. These two studies suggest that studies examining one 'snapshot' of the environment might underestimate genetic contributions to environmental experiences. Genetic factors are likely to strongly influence the temporally stable patterns of our environmental interactions.

The second major methodological concern is that a substantial majority of the studies reviewed relied on self-report. Could these studies have examined the heritability of the *perception* of the environment rather than heritability of the actual environmental experiences themselves? We have the ability to address this question because a substantial number of studies that we reviewed assessed the environment by direct observation of behavior (either 'live' or by videotape) or by informant reports, most commonly from relatives. All of these studies are individually summarized in Table 2. As is clear from Table 2, heritability estimates derived from direct behavioral observation are on average substantially lower than those obtained using other assessment methods. By contrast, estimates of heritability obtain from informants appear to be broadly similar in magnitude to that outlined from all sources in Tables 1 and A1–A9.

Table 2. *Non-self-report measures of the environment*

Study/Construct	Construct assessment	*N*	h^2
Rende *et al.* (1992)		124	
Maternal control	Observer		0·31
Maternal affection	Observer		0·00
Maternal attention	Observer		0·61
Responsiveness	Observer		0·00
Manke *et al.* (1995)		190	
Delinquency orientation	Maternal report		0·71
Delinquency orientation	Paternal report		0·49
O'Connor *et al.* (1995)			
Adolescent's behavior towards mother		186	
Warmth	Observer		0·25
Assertive	Observer		0·27
Positive mood	Observer		0·11
Control	Observer		0·33
Anger	Observer		0·27
Adolescent's behavior towards father		186	
Warmth	Observer		0·33
Assertive	Observer		0·34
Positive control	Observer		0·35
Control	Observer		0·10
Anger	Observer		0·29
Mother's behavior towards adolescent		186	
Warmth	Observer		0·07
Assertive	Observer		0·00
Positive mood	Observer		0·28
Control	Observer		0·13
Monitoring	Observer		0·08
Anger	Observer		0·00
Father's behavior towards adolescent		186	
Warmth	Observer		0·15
Assertive	Observer		0·00
Positive mood	Observer		0·02
Control	Observer		0·15
Monitoring	Observer		0·18
Anger	Observer		0·00
Kendler (1996)		937	
Paternal warmth	Co-twin report		0·20
Maternal warmth	Co-twin report		0·33
Paternal protectiveness	Co-twin report		0·17
Maternal protectiveness	Co-twin report		0·29
Paternal authoritarianism	Co-twin report		0·46
Maternal authoritarianism	Co-twin report		0·26
Thapar & McGuffin (1996)			
SLE (females)	Parent report	109	0·00
SLE (males)	Parent report	89	0·00
Independent SLE (females)	Parent report	109	0·15
Independent SLE (males)	Parent report	89	0·00
Negative impact events (females)	Parent report	109	0·54
Negative impact events (males)	Parent report	89	0·16
Positive impact events (females)	Parent report	109	0·74
Positive impact events (males)	Parent report	89	0·47
Deater-Deckard (2000)		120	
Maternal negative affect	Observer		0·06
Maternal positive affect	Observer		0·00
Negative control	Observer		0·00
Positive control	Observer		0·00
Responsiveness	Observer		0·49
Child difficult behavior	Observer		0·00
Child conduct problems	Parent report		0·59
Neiderhiser *et al.* (2004)			
Maternal positivity (TM)	Observer	326	0·00
Maternal negativity (TM)	Observer	326	0·09
Maternal control (TM)	Observer	326	0·00

Table 2 (*cont.*)

Study/Construct	Construct assessment	*N*	h^2
Maternal positivity (NEAD)	Observer	138	0·23
Maternal negativity (NEAD)	Observer	138	0·00
Maternal control (NEAD)	Observer	138	0·12
Walden *et al.* (2004)		690	
Peer deviance	Teacher report		0·00
Number of substances used	Maternal report		0·10
Number of substances used	Co-twin report		0·25
Spotts *et al.* (2005)		326	
Wives' marital conflict	Observer		0·02
Wives' marital warmth	Observer		0·21

SLE, Stressful life event; TM, Twin Moms Project; NEAD, Non-shared Environment and Adolescent Development.

To obtain a crude quantitative assessment of the impact of different measurement methods, we calculated a weighted heritability for all of our environmental measures obtained by these three assessment methods: self-report (197 variables) 29%, observer report (42 variables) 14%, and informant report (18 variables) 26%. Only five of the studies we reviewed (Plomin *et al.* 1994; Kendler, 1996; Thapar & McGuffin, 1996; Lichtenstein *et al.* 2003; Neiderhiser *et al.* 2004) included heritability estimates of similar environmental measures by two of these three methods. Similar to what we found when aggregating across a wide variety of environmental measures, in these studies, which permit a more controlled comparison, self-report measures had the highest heritabilities, observer report the least, and informant reports had intermediate results typically only modestly less than those obtained by self-report.

These results provide strong evidence that our estimates of the heritability of the environment are not solely the result of subjective perceptions but reflect 'real' environmental experiences. When the environment of an individual is judged by an external informant, the estimated heritability is only slightly less than when assessed by the individual her/himself. This would suggest that reporting bias contributes only modestly to the estimated heritability of the environment. However, when assessed by direct observation, the heritability of the environment is substantially less than when obtained from self- or informant-report. There are two plausible interpretations of this finding. Self- and informant-reports could share consistent biases that inflate heritability estimates. Alternatively, the heritabilities obtained from direct observation could be biased downwards because they sample such a small 'slice' of time compared to the two other assessment methods. With the exception of a single report (Rende *et al.* 1992), all the studies reviewed in Table 2 had a duration of observation of only 10 min. Given the two studies outlined above that showed the substantial gain in heritability when correcting for measurement error from self-report measures, it is likely that these very short sampling frames substantially increase the impact of 'error' and other short-term fluctuations. This in turn should result in a downward bias on heritability estimates, which are always limited by unreliability of measurement. We would suggest that the second of these two explanations is the more plausible but this question can only be resolved definitively by further research. We conclude that while reporting bias almost certainly contributes to the evidence for heritability of measures of the environment, most of the findings in the literature probably reflect actual behaviors and not only perceptions.

The third major methodological concern was that we found very few studies that examined the heritability of the environment in a developmental context. One such report, by Elkins *et al.* (1997), shows the possible richness of this approach. In male twins aged 11 and 17, significant genetic influences were found on measures of parent–son conflict, regard, involvement, and overall support. Of particular interest, heritabilities were significantly higher in older twins, demonstrating increased genetic influence with

age. These results, consistent with the prior proposal of Scarr and McCartney (1983), suggest that the heritability of the environment might increase during adolescence as individuals become more able to control and influence their environment. In accord with this prediction, we see, in male twins from the VTR, increasing heritability of peer group deviance from childhood through early adulthood (Kendler *et al.* unpublished observations).

The fourth methodological concern is that we relied solely on published reports. If there is a publication bias such that studies reporting higher heritability for environmental measures are more likely to be published than those reporting low or absent heritability, our aggregate heritability estimates will be biased upward.

Possible mediators of the genetic effects on the environment

Clearly, genetic factors do not, in any direct way, 'code' for specific environments. Of the possible mediators between genotype and environmental measures, by far the most studied has been personality, especially neuroticism and extraversion (e.g. Horwood & Fergusson, 1986; Fergusson & Horwood, 1987; Headey & Wearing, 1989; Poulton & Andrews, 1992; Magnus *et al.* 1993; Billig *et al.* 1996; Saudino *et al.* 1997; Krueger *et al.* 2003; Spinath & O'Connor, 2003). Shared genetic influences have been implicated between neuroticism and parenting (Spinath & O'Connor, 2003), while neuroticism scores have also been shown to significantly predict SLEs, the quality of interpersonal relationships (Kendler *et al.* 2003) and to predispose individuals to experiencing more negative life events (Magnus *et al.* 1993). By contrast, extraversion has been shown to significantly predict the occurrence of positive life events (Magnus *et al.* 1993) as well as to share common genetic variance with controllable and desirable life events (Saudino *et al.* 1997).

Implications

The results of this review have three major implications. First, these findings strongly support bidirectional models of person environment inter-relationships (Scarr & McCartney, 1983). Human beings actively create important aspects of their social environment and interpersonal relationships. Second, the results have crucial implications for how we understand gene action in psychiatry. With startling advances in molecular genetics, our field has turned increasingly towards reductionist models of 'inside the skin' gene effects. While such research approaches are likely to be very fruitful, they will not result in a complete understanding of the pathway from genes to disorders. To achieve that goal, it will be necessary to also consider 'outside the skin' pathways, where the impact of genes on disease risk is mediated through self-selection into pathogenic environments (Kendler, 2001). Third, standard heritability estimates cannot discriminate between inside and outside the skin pathways. Our results suggest that a non-trivial proportion of genetic effects assessed by twin and adoption studies for psychiatric and substance use disorders may involve selection into environmental adversity that then feeds back to increase disease risk.

NOTE

Supplementary material accompanies this paper on the Journal's website (http://journals.cambridge.org).

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DECLARATION OF INTEREST

None.

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