Economics and Human Biology 8 (2010) 320–330

Contents lists available at ScienceDirect

Economics and Human Biology

journal homepage: http://www.elsevier.com/locate/ehb

Nature, nurture and socioeconomic policy—What can we learn from molecular genetics?

Petter Lundborg [a], Anders Stenberg [b,*]

[a] Department of Economics, Lund University and affiliated with VU University Amsterdam, Tinbergen Institute, IZA, Centre for Economic Demography (CED), and the Health Economic Program (HEP Lund), Sweden
[b] Swedish Institute for Social Research, SOFI, Stockholm University, Universitetsv 10 F, SE-106 91 Stockholm, Sweden

ARTICLE INFO

Article history:
Received 27 November 2009
Received in revised form 17 August 2010
Accepted 17 August 2010

JEL classification:
J62
H50

Keywords:
Genes
Environment
Inequality

ABSTRACT

Many countries use public resources to compensate individuals with genetic disorders, identified by behaviors/symptoms such as chronic diseases and disabilities. This paper draws attention to molecular genetic research which may provide a new dimension to our understanding of how socioeconomic outcomes are generated. We provide an overview of the recently emerging evidence of gene–environment interaction effects. This literature points out specific areas where policies may compensate groups of individuals carrying genetic risks, without the need to identify anyone's genetic endowments. Moreover, epigenetics studies, which concern heritable changes in gene functions that occur independently of the DNA sequence, have shown that environments may affect heritable traits across generations. It means that policies which neutralize adverse environments may also increase intergenerational mobility, given that genetic and/or environmental risk factors are more common in socially disadvantaged groups.

1. Introduction

Economists have shown an increased interest in quantitative genetics and how genetic endowments co-vary with earnings, education or human behavior. The hereditary (or nature) components reported for education have been between .45 and .65 and for earnings between .25 and .40 (Behrman and Taubman, 1976, 1989; Taubman, 1976; Plug and Vijverberg, 2003; Björklund et al., 2005, 2006, 2007; Sacerdote, 2002, 2007; Liu and Zeng, 2009) while about .20 of the variation in personal traits such as risk-taking and overconfidence has been explained by heritage (Cesarini et al., 2009a,b, forthcoming). These studies have contributed greatly to the acceptance and understanding that genes play an important role in explaining various outcomes. Methodologically, they adhere to a tradition which spans over a number of academic fields, where genetic variation is indirectly identified by analyzing intergenerational transmissions in families with adopted children, or by comparing correlations of monozygotic (MZ) twins with those of dizygotic (DZ) twins.

The estimated nature components encompass direct genetic effects (G), interactions between different gene types (G × G) and effects of gene–environment *correlations* (rGE). The latter means that the nature component includes the effects of genetically induced environments. To see this more clearly, Plomin et al. (1977) proposed three major processes. First, if the genes of parents are correlated with those of the child, there is *passive* rGE—i.e. the genes of the parents influence the parents' behavior and thereby the environment of the child (e.g. intelligent parents raise their children in an intellectual environment). Second, *active* rGE—the genes of individuals influence how they generate their own environments. Third, *evocative* rGE—the genes of individuals influence

* Corresponding author.
E-mail address: anders.stenberg@sofi.su.se (A. Stenberg).

1570-677X/$ – see front matter © 2010 Elsevier B.V. All rights reserved.
doi:10.1016/j.ehb.2010.08.002

how they are treated by others. Thus, even if one allows for random placement of children, active and evocative rGE generate "endogenous environments", whereas exogenous environments are those which are not genetically induced (Jencks, 1980).[1]

Further, gene–environment *interaction* effects (G × E) are also included in the nature component. A G × E occurs as the genes of the individuals influence the reaction when exposed to a certain environmental factor (whether endogenous or exogenous).[2] The interpretation of heritability estimates in quantitative genetics is thus a subject of debate as it is unclear to what extent they reflect environmental rather than genetic factors.[3] Consequently, in terms of specific policy implications, quantitative genetics is generally silent. However, many governments redistribute resources on the basis of genetic endowments. Medications are subsidized for individuals with chronic diseases, disabled are entitled to extra public resources, dyslexic pupils are offered extra teaching assistance, etc. Individuals could in these cases be said to be identified as carriers of genetic risks through their behaviors/symptoms, and are compensated by environmental improvements in the form of medication, equipment and/or teaching hours.[4] Molecular genetics research may provide additional motivation for such redistributions as it sheds new light on how genes operate by *directly* observing individuals' genetic variants and their associations with outcomes. Starting with two papers by Caspi et al. (2002, 2003), this literature has uncovered an increasing amount of evidence of G × E where the impact of genetic risk has been found related to environments in childhood (pre- or post-natal) or as young adults. Although still tentative, these studies challenge the traditional view of separating between effects of genes and environments. Moreover, epigenetic studies, which concern heritable changes in gene functions that occur independently of the DNA sequence, have shown that environments may affect heritable traits across generations. It means that the influence of even temporary environmental conditions may extend to future generations as environments activate some genes and inhibit others (Mill and Petronis, 2007, 2008; Foley et al., 2009). An early example of epigenetic evidence was provided by Waterland and Jirtle (2003) who showed that by altering the diet of the mother before conception, innate genetic risks for cancer and diabetes disappeared in offspring of mice.

The purpose of this article is to provide an overview of the emerging G × E evidence in molecular genetic research, assess identification issues and discuss possible policy implications. For economists and other researchers concerned with how socioeconomic outcomes are generated, the overview and assessment of this branch of literature may serve to increase the understanding of the nature–nurture interplay.[5] Taken at face value, the G × E evidence points out environmental risk factors which policy may subdue in order to enhance outcomes of those carrying a genetic risk. These groups may thus be compensated without the need to identify anyone's genetic endowments. In addition, epigenetic evidence emphasizes the intergenerational effect of environments and under the plausible assumption that individuals in low socioeconomic groups are more often exposed to environmental and/or genetic risk factors, policies implied by the G × E evidence would also be likely to raise the intergenerational mobility in societies, or at least upward mobility, by increasing the probability that individuals do better than their parents.[6] Thus, if a detrimental environment has unequal effects, which systematically depend on the genetic heritage of individuals, there is increasing impetus for an egalitarian policy maker to offset the environmental risk.[7]

We fully acknowledge that it is crucial for policy that the G × E evidence identifies causal effects and that failed replications may be less likely to be published. Indeed, as our exposition will show, the interpretation of the recent evidence of G × E has not reached a consensus view. However, already at this stage, one may argue that genetic and environmental influences are largely intertwined, and that the evidence of G × E will continue to grow as knowledge about relevant environmental factors improve (Caspi et al., 2004; Lehn et al., 2007; Asbury et al., 2008; Oliver et al., 2008) and/or as data sources of sufficient detail accumulate (Reiss, 2008; Moffitt et al., 2006, including references p. 8). As molecular genetic research points out areas where active policies have the potential to enhance socioeconomic equality and intergenerational mobility, we believe it should be of interest to social scientists.

In the following section, we present traditional quantitative genetic studies suggesting G × E. The main part of the paper is Section 3 which consists of three subsections. We first describe molecular genetic research and the G × E evidence presented in Caspi et al. (2002, 2003). The second subsection gives a detailed account of

[1] Passive rGE is not included in Jenck's definition of endogenous environments, since only the portion of the environment caused by the child's genotype is considered in his definition.

[2] G × E has been addressed within the traditional framework of quantitative genetics (Turkheimer et al., 2003; Björklund et al., 2006), we briefly discuss these studies in Section 2.

[3] From a number of different perspectives, authors have made critical assessments of heritability estimates based on traditional models (e.g. Goldberger, 1979; Jencks, 1980; Scarr and Weinberg, 1994; Stoolmiller, 1999; Smith et al., 2000; Case et al., 2000, 2001; Dickens and Flynn, 2001; Joseph, 2002; Rutter and Silberg, 2002; Horwitz et al., 2003; Richardson and Norgate, 2006; Heckman, 2007, 2008; Cunha and Heckman, 2009).

[4] Market solutions may of course also compensate genetic differences, e.g. we buy glasses to improve poor eyesight (Goldberger, 1979).

[5] None of the economic papers on quantitative genetics mentioned in the beginning refer to the recent molecular genetic research. In their analysis of the origins of inequality, Cunha and Heckman (2009, p. 330) acknowledge the G × E literature but they do not discuss the evidence or its implications.

[6] See discussions in Bhattacharaya and Mazumder (2009), Bratsberg et al. (2007) and Jäntti et al. (2006). It does not hold in general that a reduced variation in offspring income variation also reduces the parent–offspring income correlation. However, based on evidence from the Nordic countries, the UK and the US, Jäntti et al. (2006) conclude that "increases in overall mobility would most likely occur from interventions designed to increase the mobility of the very poorest".

[7] To be precise, the G × E evidence implies that inequality is reduced across "genetic groups" if individuals carrying a genetic risk benefit more from an improved E than individuals without genetic risk.

identification issues regarding these two studies while the third goes through other published evidence of G × E in order to demonstrate the range of topics covered by this literature. Section 4 presents evidence from epigenetic research and Section 5 broadly summarizes the potential implications of the research presented.

2. Gene–environment interactions in traditional models

Quantitative genetic studies based on twins or adoptees have found heritability to explain a substantial part of the variation in e.g. income, education, IQ and most behavioral traits. Since the ambition of quantitative genetics has been to establish the importance of the genetic variation, the articles explicitly addressing the interplay between genes and environments are in comparison very few and they generally do not make a distinction between exogenous and endogenous environments, i.e. between rGE and G × E.[8]

Turkheimer et al. (2003) suggested that the genetic component of the variation in IQ was higher in groups with high socioeconomic status (SES). Using a twin design, and splitting their sample in two halves, estimates of heritability were .10 if SES was low and .72 if SES was high. Similar findings had already been reported by Rowe et al. (1999), when controlling for parents' level of education, Guo and Stearns (2002) when controlling for parental unemployment and, more recently, Harden et al. (2007) when considering parental income. On adoption data, Sacerdote (2007) divided foster families into three types and found environmental influences to be strongest in large families where parents had low educational achievements. Thus, all these results indicate that poor socioeconomic background is associated with a stronger environmental impact, but it is not entirely clear what the findings imply. A common interpretation has been that genes associated with ability may be suppressed by poor learning environments. A key question, and a potentially important contribution of molecular genetics, would then be to more exactly identify the environmental risk factors.

Björklund et al. (2006) used a different approach to detect G × E. As part of their analysis of intergenerational correlation in schooling between parents and their children, they included interaction variables between adoptive parents' and biological parents' schooling to reflect the children's environmental and genetic factors. In alternative specifications, they used earnings, income or university attendance in similar fashion. They found that the interaction variables were associated with significant and positive coefficients except for fathers' schooling or fathers' university attendance, suggesting that the importance of environmental factors may vary across some distribution of genetic endowments.

More specific evidence of G × E was reported in Cadoret et al. (1996) and Kendler et al. (1995, 1999). These partly contributed to the formulation of the hypotheses tested in the seminal work by Caspi et al. (2002, 2003). Cadoret et al. (1996) analyzed conduct disturbances among adoptees in Iowa. The measure of genetic risk was based on information about antisocial personality disorder in biological parents, and the environmental risk was measured by indicators on antisocial behavior of the rearing parents. The negative environment had no effect on the child's conduct disturbance if there was no biological risk factor involved, but the effect was significant if both conditions applied. Kendler et al. (1995) studied the incidence of major depressions within the framework of the twin model. Stressful life events among MZ and DZ twins were considered as the environmental risk factor, whereas the genetic risk was measured by the incidence of depressive behavior in the co-twin. The findings indicated that given a stressful life event had occurred, the genetic risk altered the probability for depression. Kendler et al. (1999) went one step further by making a distinction between exogenous stressful life events and "personal and network related events", potentially influenced by genetic factors (rGE). They found support for a causal relationship between stressful life events and the onset of depression, but also reported that individuals predisposed for depressive states self-select into high-risk environments.

3. Molecular genetic research

In this section, we discuss molecular genetics research, where genetic variants are directly observable. Section 3.1 contains a background and a description of the studies by Caspi et al. (2002, 2003). In Section 3.2, we provide an account of the literature devoted to scrutinize identification issues in these two studies, encompassing re-analyses with different data sets and several meta-analyses. In Section 3.3, we summarize other potentially policy relevant G × E evidence, primarily related to home environments and adolescent behavior, but abstain from a discussion on identification.

3.1. Molecular genetics and the initial G × E evidence

Molecular genetics is dominated by research where genetic variants are singled out, often derived from experimental evidence on animals, in order to test hypotheses of a statistical association with some personality trait or disease.[9] Since 2005, so-called genome-wide association studies (GWAS) have made it possible to examine hundreds of thousands of genetic variants and their associations with individual traits (Pearson and Manolio, 2008 provide an introduction to GWAS, see also the webpage of the National Human Genome Research Institute). A number of different diseases have been linked to genetic variants but, as pointed out by Hirschhorn (2009) and Goldstein (2009), the estimated associations have in general been tiny.[10] Given that traditional studies

[8] For a more critical and detailed discussion on this topic, see Benjamin et al. (2007). For further references on G × E evidence within the traditional framework, see e.g. Rutter and Silberg (2002), Moffitt et al. (2006) and McGue (2008).

[9] Economists are also active in this area, Knafo et al. (2007), Dreber et al. (2009), Israel et al. (2009), Kuhnen and Chiao (2009), McDermott et al. (2009) and Roe et al. (2009).

[10] For a GWAS on educational achievement, see Beauchamp et al. (2009).

frequently have indicated heritability estimates in excess of .60 for height, IQ and hyperactivity disorders (ADHD), geneticists have been surprised that identified gene associations, including accumulated effects of multiple genes, only explain about 3% of the variation in a trait such as height (Weedon et al., 2008; McEvoy and Visscher, 2009), and even less of the variation in IQ scores (Butcher et al., 2008; Meaburn et al., 2008) and in ADHD (Franke et al., 2009; Wallis et al., 2008; Thapar and Stergiakouli, 2008). Several hypotheses have been put forward to explain the weakness of the findings. It may be that outcomes are affected by a large number of gene types in combination, and that these combinations need to be identified, or that the important genetic variants for a particular behavior or disease are rare, making it necessary with very large samples for statistical power.

Meanwhile, researchers with access to detailed data on genes *and* environments have explored hypotheses based on the idea that outcomes are influenced by genes, but only conditional on some environmental factor. The reported results have had a profound impact on the field.[11] Caspi et al. (2002) showed that antisocial behavior in adulthood, such as criminal activity, was associated with the interaction of childhood maltreatment and a low prevalence of a specific gene variant, but not separately by the gene, whereas Caspi et al. (2003) found depressions to be associated with interactions between a specific gene type and earlier stressful life events.

Caspi et al. (2002, 2003) were the first molecular genetic studies to establish gene–environment interaction effects on human behavior. This team of researchers followed (and still follows) 1037 individuals from the city of Dunedin, New Zealand, born between April 1972 and March 1973. Child–parent data have been collected every second year until aged 15 and then at age 18, 21, 26 and at age 32, when the sample still displayed 96% retention. The longitudinal data contains details on *both* environmental and genetic factors. The point of departure in Caspi et al. (2002) was the hypothesis of a connection between child abuse and later antisocial behavior. Focusing on males, followed from age 3 to 26, they explored variation in maltreatment experiences between age 3 and 11. No maltreatment was reported for 64%, "probable" maltreatment for 28% and "severe" maltreatment for 8%. Their outcomes included five different measures of antisocial behavior; adolescent conduct disorders, convictions of violent crimes, disposition towards violence assessed at age 26, symptoms of antisocial personality disorder at age 26 and a composite index measure of the four. Using an additive model, the composite index of antisocial behavior was not significantly associated with the candidate gene variant ($n = 442$, p-value .89). [12] In the G × E literature, this is a common way to rudimentarily check if the environmental risk factor is exogenous, rather than caused by the genes of the individuals (rGE), which also could imply that the detected G × E in fact is a gene–gene interaction (G × G). When including an interaction variable between genetic risk and maltreatment, a significant association with each measure of antisocial behavior was detected, including the composite index.

If one gives the results in Caspi et al. (2002) a causal interpretation, any policy that would diminish child maltreatment would also decrease the overall prevalence of antisocial behavior and/or criminal activities, but it would benefit carriers of the genetic risk more strongly. Thus, a permanent decrease in child maltreatment would also decrease the difference in antisocial behavior across "genetic groups" in both present and future generations. In this perspective, it may well be that efforts devoted to reduce child abuse are undersized.[13] The intergenerational policy implication is strengthened by extensive evidence from epigenetic research which will be discussed separately in Section 4.

Caspi et al. (2003) studied the incidence of stressful life events and its association with depression, showing that a genetic variant had a moderating impact on the relation. Stressful life events (SLEs) between ages 21 and 26 were assessed with life-history calendars, and included 14 types of events such as job loss, financial stress, health stressors, marital problems and marital loss. In the Dunedin sample, 30% experienced no SLE, 25% one event, 20% two events, 11% three events and 15% four events or more. The genetic risk was not significantly associated with SLEs (p-value .59). Depressive symptoms in the past year were assessed at age 26 through structured interviews. Again, there was no significant difference in the occurrence of depressive symptoms across the gene type, but an interaction variable between SLE and the genetic variation was significantly linked with depressive symptoms.

A causal interpretation of this result does not necessarily have a meaningful interpretation for policy as the SLEs encompass a number of different events. Instead, several hypotheses are left for future research to explore, e.g. that structural changes following economic shocks may have costly long-term consequences if the adverse consequences of job loss and/or financial stress partly depend on genetic endowments. Policy issues aside, a major contribution of Caspi et al. (2003) is the large literature it has generated committed to assess the quality of the G × E evidence.

3.2. Assessments of the G × E evidence

The first study to replicate the result in Caspi et al. (2002) was Foley et al. (2004) who analyzed the incidence of conduct disorder for a sample of Virginia twins (514 males). Their definition of maltreatment in childhood was based on detailed information from interviews conducted

[11] Google Scholar indicates that Caspi et al. (2002, 2003) are cited more than 4000 times.

[12] More precisely, the gene type in question is a functional polymorphism in the gene encoding the neurotransmitter-metabolizing enzyme MAOA. However, we will consistently avoid biological definitions as our interest is primarily to discuss overall implications for social policy rather than gene-specific aspects.

[13] Corporal punishment of children is illegal in 23 countries. Durrant and Janson (2005) claim that physical abuse in Swedish families did decrease following the legislation in 1979, and infer increased education and a normative impact of the law as two probable causes.

by trained field workers, and then classified as violence between parents, parental neglect or inconsistent parental discipline. Several studies have followed, but a weakness is that the cells of carriers with both genetic risk and environmental risk are typically very small, below 20 individuals. A meta-analysis by Taylor and Kim-Cohen (2007) reviews the results from published articles and report that the G × E prediction "holds up across eight different studies with samples that range in mean age from 7 to 32". Their analysis included two non-replications. Haberstick et al. (2005) was based on interviews of 774 males but they were "self-nominated" (volunteers) and included only four individuals convicted of any violent offence. Huizinga et al. (2006) was the other non-replication, based on self-report questionnaires from a sample of 277 males. We return to this study and other issues of identification below.[14]

The topic studied in Caspi et al. (2003), the relation between SLEs and depressive states, has a long history in the psychiatric literature. A very large number of studies have attempted to replicate their results. Uher and McGuffin (2008) review 17 published works, including three non-replications. They explore the design of all studies in terms of statistical approaches, type of samples studied, the definitions of environmental risk and outcome measures, and assess whether genes influence the probability of environmental risk. A statistical property when estimating interaction effects in regressions is that the results are sensitive to scaling and susceptible to generate false significant as well as false insignificant estimates. In Caspi et al. (2002, 2003), robustness checks where the E factors were represented by several types of indicators with different scaling properties supported the original hypotheses.

Uher and McGuffin (2008) devote particular attention to the three non-supportive studies of Caspi et al. (2003), which they note also reported weaker direct associations between SLE and the onset of depression. As possible explanations, they infer data collection methods and the age composition of the samples. Structured interviews have been judged as more reliable to accurately assess SLEs (Brown and Harris, 1978; Paykel, 1997; McQuaid et al., 2000) as self-report questionnaires tend to over-report adverse events by including trivial occurrences (Gorman, 1993; Dohrenwend, 2006) and under-report events which are important for the onset of depression (Duggal et al., 2000; Monroe, 2008). The less costly self-reported measures of SLEs may as a consequence lead to systematic measurement errors when analysing G × E evidence (Monroe and Reid, 2008; Brown and Harris, 2008). Five studies based on structured interviews replicated the findings, whereas the non-replications were all based on self-report questionnaires (as was Huizinga et al., 2006, the non-replication of Caspi et al., 2002). Concerning the age of the samples studied, the G × E evidence may not hold for older age groups as SLEs have a naturally weaker association with depression among elderly. Depressive states tend to re-occur and the strongest link with SLE concerns the first depressive episode (Meltzer, 1989).

The G × E evidence could be driven by a gene–gene interaction (G × G) rather than G × E. One argument against this is that the hypothesized genetic risks are derived from experimental like evidence on animals, which do not include genetic interactions (references are provided in Caspi et al., 2002, 2003; Uher and McGuffin, 2008). Also, Caspi et al. (2003) found the G × E interaction to be unrelated to depression reported at age 18 or age 21, i.e. in periods *preceding* the SLE. In other words, if G × G is a confounding factor, it is likely to work through the SLE. The same is implied by the insignificant correlation between gene type and the occurrence of SLE. Caspi et al. (2002) also show that none of the E factors in that study (the measures of child maltreatment) correlate significantly with the candidate G factor. However, to be able to completely exclude the G × G hypothesis, one would need a randomly distributed environmental risk, but the G × E evidence presented so far in molecular genetics does not include any such randomly assigned events.

Two recent meta-analyses conducted by Risch et al. (2009) and Munafò et al. (2009) seriously question the G × E evidence reported in Caspi et al. (2003) since both studies fail to replicate a significant association with depression. Risch et al. (2009) was based on accumulated data sets obtained by collecting data from 14 earlier studies while Munafò et al. (2009) used summary statistics from five studies. Moreover, the authors of both articles point out that many studies making claims of replications are not justified since they are not consistent with Caspi et al. (2003) in how they use statistical interactions of continuous and/or categorical variables and in whether they use additive linear regression models or logistic models. Munafò et al. (2009) suggest that the relatively large number of replications reflects that results are sensitive to such seemingly minor differences between studies, but also that researchers may be prone to data mining and scientific journals disposed towards publication bias (Ioannidis, 2005).

In a comment to the two meta-analyses, Uher and McGuffin (2010) extend their original review to 34 studies, encompassing those included in Risch et al. (2009) and Munafò et al. (2009). The review comprises nine non-replications in total (they do not consider different types of statistical interactions). Again, they note that the studies using objective indicators and/or structured interviews to assess the occurrence of SLEs are strongly associated with replications of the G × E findings. None of these 15 studies failed to replicate the results of Caspi et al. (2003), whereas all non-replications are based on self-report questionnaires (half of these are non-replications). Of the 14 studies included in Risch et al. (2009), 11 are based on self-report questionnaires (seven non-replications).[15] Similarly in Munafò et al. (2009), four of the five studies included are based on self-report questionnaires (one non-replication). Uher and McGuffin (2010) conclude that the two meta-

[14] One study, Young et al. (2006), was excluded from their analysis as it was based on a clinical sample of individuals in treatment for conduct problems.

[15] Nine of the studies were based on structured interviews to assess depressive states, but not the SLE.

analyses represent a biased synthesis of the existing evidence since self-report questionnaires are flawed by systematic measurement errors. Unfortunately, neither Risch et al. (2009) nor Munafò et al. (2009) discuss the literature on the assessment of SLE referred to above. To conclude this section, there is still no unanimously accepted evidence confirming or rejecting the results in Caspi et al. (2002, 2003).

3.3. Other topics covered by G × E evidence

We now turn to other examples of G × E evidence associated with factors potentially shaping individuals' socioeconomic outcomes in adult life. The environmental factors mainly concern children's pre- and post-natal environments or behavioral traits of adolescents or young adults. As in the preceding section, one should be cautious about giving the reported results a causal interpretation, especially since most of the evidence has not been subject to the same scrutiny as Caspi et al. (2002, 2003) and since there has been no studies where the environment has been randomly assigned.

An often studied outcome associated with pre-natal environment is the incidence of hyperactivity disorders ADHD. This is a highly policy relevant area of research as ADHD is estimated to affect more than 5% of the world's school aged children (Polanczyk et al., 2007). On a sample of 161 children, aged from 6 months up to 5 years, Kahn et al. (2003) showed that a specific gene type increased the risk of ADHD if the mother had been smoking during pregnancy. Replications of the result include Neuman et al. (2007) who used a larger sample, consisting of 1494 male and female twins aged 7–19. Becker et al. (2008) confirmed the finding for 146 males, where data allowed controlling for post-natal smoke exposure and psychosocial adversity, but not for females ($n = 159$). Brookes et al. (2006) did not replicate the finding but instead found ADHD linked to pre-natal alcohol use which exacerbated the genetic risk. The studies first of all suggest that there may be more to gain from policies affecting home environments than previously thought. Second, efforts towards keeping pregnant women away from drinking and/or smoking could also decrease differences in behaviors if they compensate for genetic risks associated with conduct disorders.

Post-natal environmental factors further stress the importance of home environments. Epidemiologists have recognized that genetically transferable diseases clustered in families most often also depend on environmental risk factors.[16] Asthma is related to specific genes but the risks increase for children exposed to tobacco smoke early in life (Bouzigon et al., 2008) and decrease for children who attend day care during the first year (Hoffjan et al., 2005). Returning to behavioral traits, we already discussed a potential relation between child maltreatment and antisocial behavior. Additional results in Caspi et al. (2003) reported that child maltreatment as E factor (instead of SLE) generated significant G × E for the incidence of depression. Also mentioned above was that molecular genetic researchers have struggled to find a direct genetic link to IQ score. Caspi et al. (2007) used the fact that breastfeeding of children has been known to predict IQ scores later in life (Anderson et al., 1999; Mortensen et al., 2002). Controlling for intrauterine growth, social class and maternal cognitive ability, they found breastfeeding was significantly related to IQ, as was the interaction term between their candidate gene type and breastfeeding. The gene by itself was not significantly associated with IQ or any of the covariates. Besides their Dunedin sample ($n = 858$), they confirmed their findings by studying a British sample of same-sex twins drawn from a 1994 to 1995 birth register ($n = 1772$).[17] However, importantly, breastfeeding enhances IQ of those with a favorable genetic variant. Thus, a policy increasing breastfeeding to improve IQ overall could *increase* inequality across "genetic groups" if breastfeeding increases equally across socioeconomic groups but the gene type is more common in affluent families.

There are relatively scattered examples of detected G × E evidence related to adolescent or young adult behavior. Shanahan et al. (2008) explored a genetic variant which has been associated with dysfunctional schooling. Among individuals with this gene type, they found a lower probability of engaging in tertiary level education, but the genetic risk was compensated by environments where parents had high socioeconomic status, were highly involved in school and the school itself was of high quality. Caspi et al. (2005) examined cannabis use in adolescence, which is considered to be a weak risk factor for developing various psychotic symptoms such as schizophrenia. They found a genetic variant which was related to a significantly higher likelihood of psychotic symptoms following cannabis use in adolescence, whereas another gene type was associated with no relation between cannabis use and psychotic symptoms. Guo et al. (2008) found an association between the number of sexual partners and a specific gene type, which did not hold in school environments where a high proportion started having sex early. Settle et al. (2008) found that for individuals with a specific gene variation, the number of friendships in adolescence was significantly associated with a liberal political ideology. The relation did not hold for individuals who did not have the specified genetic makeup.[18]

Other studies on adolescents or young adults have used definitions of G × E comparable to those used in Caspi et al. (2003), but altered the depressive state outcome to instead focus on criminal behavior, alcohol and drug use (Reif et al., 2007; Covault et al., 2006; Nilsson et al., 2005; Kaufman et al., 2007). The results imply a complex set of effects of G × E for which Uher and McGuffin (2008) suggest three

[16] For example, exposure to smoking increases the risk for lung-cancer but the effect is moderated by gene types (Amos et al., 2008; Hung et al., 2008; Thorgeirsson et al., 2008). Other fields include bacterial infections (malaria, streptococci), cardiovascular disease and type 2 diabetes, for references, including replications, see Moffitt et al. (2006, p. 8).

[17] From the Environmental Risk Longitudinal Twin Study.

[18] The result lends some support to a controversial study by Alford et al. (2005) who, with a traditional twin study, found quite specific political preferences (on e.g. nuclear power, censorship, abortion laws) were determined by genetic heritage. See Charney (2008) for a discussion.

possible interpretations. It may be (1) that the same G × E has several separate effects, or (2) that the same G × E contributes to several outcomes along the same causal pathway (depression leads to substance use, which leads to criminality), or (3) that there is some intermediate outcome which is connected in a causal link between gene type and several different outcomes. These remarks do not question the inherent policy issue of the G × E evidence, i.e. that the identification of environmental risk factors may enable policies to improve these environments, and thereby in particular assist carriers of genetic risk.

4. Epigenetics

Epigenetics concern heritable information outside the DNA sequence (the Greek prefix *epi* means "on" or "over"). Although DNA sequences remain unchanged, it has been shown that environmental factors may cause genes to "behave" differently, by activating some genes and deactivating others. The term used is that they modify gene expression, and that such epigenetic modifications are heritable.[19] Consequently, monozygotic twins differ significantly in their gene expressions already when they are 5 years old (Mill et al., 2006) and the differences increase with age, and/or if they have a history of non-shared environments (Fraga et al., 2005; Oates et al., 2006). The implications of these studies include that heritable traits are not entirely "random" variants of the ancestors, but partly adapted to the environment.

Human longevity makes it difficult to collect historical data on behaviors and/or environments across generations. The epigenetic hypothesis is therefore easier to study on animals with short life-spans. Waterland and Jirtle (2003) studied agouti mice. Its name stems from a particular gene, the agouti gene, a variant of which makes it prone to cancer and diabetes. By altering the diet of the mother just before conception, certain genes were switched off. The offspring was no longer susceptible to cancer or diabetes and lived much longer, and their different physical traits (color and size) and lower disease risks were passed on to the next generation. Similarly, Bertram et al. (2008) found that under-nutrition of pregnant guinea pigs affected the risk for cardiovascular disease across two generations. Evidence on animals outside gestation include Champagne et al. (2006) who reported from experiments on rats that increased grooming behavior of the mother altered gene expression and induced a similar behavior in female offspring. This behavior was transmitted across generations. Recent evidence in Nätt et al. (2009) indicates that trained abilities may also be transmitted to the offspring. They found that chickens who were exposed to unpredictable food access adapted their strategies to better survive, and their offspring showed similar foraging behavior. The epigenetic evidence suggests that events in childhood or in utero may work jointly with genetic factors to affect adult life. However, interestingly, Weaver et al. (2004, 2006) showed on rats that epigenetic changes arising due to childhood events, and which explained stable differences in adults, could be reversed by medications.

For humans, the famine during the winter of 1944–1945 in the Netherlands has been explored to study the effects of variation in nutrition during pregnancy. It has been shown that the famine had detrimental health effects for the children and grandchildren of those mothers who were exposed (Painter et al., 2005; Lumey et al., 2007).[20] Moreover, the hypothesis that a pregnant mother's diet also affected inherited traits is supported by Heijmans et al. (2008) who show evidence of epigenetic effects on those pre-natally exposed to the famine compared with their unexposed same-sex siblings. With a slightly different hypothesis in mind, Kaati et al. (2002) studied the occurrence of food shortage in northern Sweden by going through records of harvests and food prices during the 19th century. They were especially interested in food supplies during children's so-called "slow growth period" (SGP) which occurs at age 8–10 for girls and 9–12 for boys. It turned out that individuals experiencing food shortages in the SGP, had descendants with lower risks of mortality from cardiovascular disease and diabetes (i.e. a period of fasting decreased descendants' risks). Pembrey et al. (2006) analyzed the effect of smoking during the SGP by exploring UK data from the Avon Longitudinal Study of Parents and Children, consisting of interviews with parents of newborn 1991–1992. Of the 5000 fathers who had said they were smokers, 166 of them had stated they were regular smokers already during the SGP. They found that the sons of these fathers had higher body mass index as 9 year olds. Among the hypotheses to explain these findings, epigenetics is a major candidate but, of course, the evidence from studies of animals is more compelling.

The epigenetic findings have also stimulated proposals of hypotheses with extremely long time perspectives. Jasienka (2009) argues that epigenetic effects of slavery in the 18th and the 19th century partly explain the lower average birth weight among African-Americans in the US. Wells (2010) coins the term "metabolic ghetto" as an environment where systematic exploitation of a population takes place, inferring the Dutch winter famine as an example. In a mould similar to that of Jasienka (2009), he uses the term to discuss long-term consequences of imperialism, which forced many non-European populations into a cumulative exposure to chronic malnutrition. He also argues that there are analogous implications of socioeconomic marginalization today as an environmental risk factor may be transmitted to future generations.

5. Concluding discussion

The emerging molecular genetic evidence on gene–environment interactions (G × E) as well as epigenetic findings makes it important to think about its applicability. Until now, public resources support individuals with

[19] The development of a fetus illustrates one example of changing gene expression. A fertilized egg takes new shapes, developing many cell types (venes, muscles, etc.) as it continues to divide through a process where some genes are activated while others are not.

[20] A quasi-experimental set-up is also used by Almond and Mazumder (2009) who study the effects of under nutrition in gestation due to the Ramadan and find adverse effects on children's learning abilities.

genetic disorders which are identified through symptoms/behaviors such as learning disabilities and/or chronic diseases. We argue that if the current G × E evidence stands the test of time, it points out areas of policy where subdued adverse environments may enhance the relative prospects of individuals carrying genetic risks, without the need to identify anyone's genetic endowments. Epigenetic evidence also implies possible long-term effects on future unborn generations as it has shown that genes and environments work jointly to construct our heritable traits. In addition, such policies are also likely to increase social mobility if environmental and/or genetic risk factors are more prevalent in poor socioeconomic groups. The policy examples we have discussed include ways to decrease child maltreatment (leading to antisocial behavior, including criminal activities), to decrease smoking and/or drinking among pregnant women (reducing the incidence of ADHD) and/or to increase mothers' breast-feeding of children (enhancing IQ).[21] Thus, a broadly brushed picture of the overall findings suggests that health policies aimed at improving maternal, foetal and infant health may generate substantial benefits for several successive generations. However, for any policy, an important reservation is that the G × E findings must reflect causal effects, but convincing evidence has so far not been presented and the understanding of the most often replicated result has therefore not yet settled into a consensus view.

For economists concerned with how socioeconomic outcomes are generated, co-operation with geneticists is an interesting path. Economists, with their long tradition of analyzing and exploiting exogenous shifts in the environment, can make important contributions to combine exogenous shifts in the environment with genetic information in order to identify causal G × E effects. Several authors provide guidelines for G × E research (Hunter, 2005; Moffitt et al., 2006; North and Martin, 2008; Uher and McGuffin, 2008). Major caveats include that G × E may develop in several stages, requiring longitudinal data, and that variations in environments are measured quite crudely compared with genetic variations. This suggests that relatively small samples, with more accurate (and expensive) measures of E, may be preferred to larger data sets. Simulations of measurement errors in Wong et al. (2003) indicate that if correlations with some true value of E increase from .4 to .7 it corresponds to a 20-fold increase in sample size needed to detect effects with similar precision. Of course, whether researchers uncover a direct genetic effect, a G × E or no effect depends on the variation in E and G. If a genetic variant influences an outcome only conditional on an E factor, it will emerge as a direct effect if E is very common or no effect if E is very rare, i.e. in order to detect G × E, sufficient variation in E is required. Usually, however, environments vary greatly and even MZ twins who share the same class-room environment have been found to perceive it very differently (Asbury et al., 2008). Also, as traditional studies based on twins or adopted children have found genetic endowments to co-vary with most diseases, mental disorders and behavioral traits, it seems likely that the sensitivity to environmental factors would also partly depend on the genes.

[21] Many of the suggested policies are in line with the literature proffering early interventions to improve child development and promote equity (Doyle et al., 2009 and references therein).

References

Alford, J., Funk, C., Hibbing, J., 2005. Are political orientations genetically transmitted? American Political Science Review 99 (2), 153–167.

Almond, D., Mazumder, B., 2009. Health Capital and the Prenatal Environment: The Effect of Maternal Fasting During Pregnancy. Federal Reserve Bank of Chicago, WP 2007-22, Revised November 2009.

Amos, C.I., Wu, W., Broderick, P., et al., 2008. Genome-wide association scan of Tag SNPs identifies a susceptibility locus for lung cancer at 15q25.1. Nature and Genetics 40 (5), 616–622.

Anderson, J.W., Johnstone, B.M., Remley, D.T., 1999. Breast-feeding and cognitive development: a meta-analysis. American Journal of Clinical Nutrition 70, 525–535.

Asbury, K., Almeida, D., Hibel, J., Harlaar, N., Plomin, R., 2008. Clones in the classroom: a daily diary study of the nonshared environmental relationship between MZ differences in school experience and MZ differences in school achievement. Twin Research and Human Genetics 11 (6), 686–695.

Bhattacharaya, Mazumder, B., 2009. Nonparametric Analysis of Intergenerational Mobility with Application to the United States. Federal Reserve Bank of Chicago, WP 2007-12, Revised June 12, 2009.

Beauchamp, J., Cesarini, D., Rosenquist, N., Fowler, J.H., Christakis, N., 2009. A Genome Wide Association Study of Educational Attainment. Harvard University Working Paper. .

Becker, K., El-Faddagh, M., Schmidt, M., Esser, G., Laucht, M., 2008. Interaction of dopamine transporter genotype with prenatal smoke exposure on ADHD symptoms. The Journal of Pediatrics 152, 263–269.

Behrman, J., Taubman, P., 1976. Intergenerational transmission of income and wealth. American Economic Review 66, 436–440.

Behrman, J., Taubman, P., 1989. Is schooling "mostly in the genes"? Nature–nurture decomposition using data on relatives. Journal of Political Economy 97 (6), 1425–1446.

Benjamin, D.J., Chabris, C.F., Glaeser, E.L., Gudnason, V., Harris, T.B., Laibson, D., Launer, L., Purcell, S., 2007. Genoeconomics. In: Weinstein, M., Vaupel, J., Wachter, K. (Eds.), Biosocial Surveys. National Academies Press, Washington.

Bertram, C., Khan, O., Ohri, S., Phillips, D., Matthews, S., Hanson, M., 2008. Transgenerational effects of prenatal nutrient restriction on cardiovascular and hypothalamic–pituitary–adrenal function. Journal of Physiology 586 (8), 2217–2229.

Björklund, A., Jäntti, M., Solon, G., 2005. Influences of nature and nurture on earnings variation. A report on a study of various sibling types in Sweden. In: Bowles, S., Gintis, H., Osborne, M. (Eds.), Unequal Chances: Family Background and Economic Success. Princeton University Press, Princeton, NJ.

Björklund, A., Jäntti, M., Solon, G., 2007. Nature and nurture in the intergenerational transmission of socioeconomic status: evidence from Swedish children and their biological and rearing parents. The B.E. Journal of Economic Analysis and Policy 7 (2) (Article 4).

Björklund, A., Lindahl, M., Plug, E., 2006. The origins of intergenerational associations: lessons from Swedish adoption data. Quarterly Journal of Economics 121, 999–1028.

Bouzigon, E., Corda, M.S., Hugues, A., Dizier, M.-H., Boland, A., Bousquet, J., et al., 2008. Effect of 17q21 variants and smoking exposure in early-onset asthma. The New England Journal of Medicine 359, 1985–1994.

Bratsberg, B., Røed, K., Raaum, O., Naylor, R., Jäntti, M., Eriksson, T., Österbacka, E., 2007. Nonlinearities in intergenerational earnings mobility: consequences of cross-country comparisons. The Economic Journal 117, C72–C92.

Brookes, K.J., Mill, J., Guindalini, C., Curran, S., Xu, X., Knight, J., et al., 2006. A common haplotype of the dopamine transporter gene associated with attention-deficit/hyperactivity disorder and interacting with maternal use of alcohol during pregnancy. Archives of General Psychiatry 63, 74–81.

Brown, G.W., Harris, T.O., 1978. Social Origins of Depression. A Study of Psychiatric Disorder in Women, 5th ed. Routledge, London.

Brown, G.W., Harris, T.O., 2008. Depression and the serotonin transporter 5-HTTLPR polymorphism: a review and a hypothesis concerning gene–environment interaction. Journal of Affective Disorders 111, 1–12.

Butcher, L.M., Davis, O.S.P., Craig, I.W., Plomin, R., 2008. Genome-wide quantitative trait locus association scan of general cognitive ability using pooled DNA and 500K single nucleotide polymorphism microarrays. Genes, Brain, and Behavior 7, 435–446.
Cadoret, R.J., Winokur, D., Langbehn, E., Troughton, E., Yates, W.R., Stewart, M.A., 1996. Depression spectrum disease I: The role of gene environment interaction. American Journal of Psychiatry 153, 892–899.
Case, A., Lin, F.I., McLanahan, S., 2000. How hungry is the selfish gene? Economic Journal 1110, 781–804.
Case, A., Lin, F.I., McLanahan, S., 2001. Educational attainment of siblings in stepfamilies. Evolution and Human Behavior 22, 269–289.
Caspi, A., McClay, J., Moffitt, T., Mill, J., Martin, J., Craig, I.W., 2002. Role of genotype in the cycle of violence in maltreated children. Science 297, 851–854.
Caspi, A., Sugden, K., Moffitt, T., Taylor, A., Craig, I.W., Harrington, H., 2003. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. Science 301, 386–389.
Caspi, A., Moffitt, T., Morgan, J., Rutter, M., Taylor, A., Arsenault, L., et al., 2004. Maternal expressed emotion predicts children's antisocial behavior problems: using monozygotic twin differences to identify environmental effects on behavioral development. Development Psychology 40, 149–161.
Caspi, A., Moffitt, T., Cannon, M., McClay, J., Murray, R., Harrington, H., et al., 2005. Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-O-methyltransferase gene. Biological Psychiatry 57, 1117–1127.
Caspi, A., Williams, B., Kom-Cohen, J., Craig, I., Milne, B., Poulton, R., et al., 2007. Moderation of breastfeeding effects on the IQ by genetic variation in fatty acid metabolism. Proceeding of the National Academy of Sciences of the United States of America 104 (47), 18860–18865.
Cesarini, D., Dawes, C., Johannesson, M., Lichtenstein, P., Wallace, B., 2009a. Genetic variation in preferences for giving and risk-taking. Quarterly Journal of Economics 124 (2), 809–842.
Cesarini, D., Johannesson, M., Lichtenstein, P., Wallace, B., 2009b. Heritability of overconfidence. Journal of the European Economic Association 7 (2–3), 617–627.
Cesarini, D., Johannesson, M., Lichtenstein, P., Sandewall, Ö., Wallace, B., forthcoming. Genetic variation in financial decision making. Journal of Finance.
Champagne, F.A., Weaver, I.C.G., Diorio, J., Dymov, S., Szyf, M., Meaney, M.J., 2006. Maternal care associated with methylation of the estrogen receptor-α1b promoter and estrogen receptor-α expression in the medial preoptic area of female offspring. Endocrinology 147 (6), 2909–2915.
Charney, E., 2008. Genes and ideologies. Perspectives on Politics 6 (2), 299–319.
Covault, J., Tennen, H., Armeli, S., Conner, T.S., Herman, A.I., Cillessen, A.H., et al., 2006. Interactive effects of the serotonin transporter 5-HTTLPR polymorphism and stressful life events on college student drinking and drug use. Biological Psychiatry 61, 609–616.
Cunha, F., Heckman, J.J., 2009. The economics and psychology of inequality and human development. Journal of the European Economic Association 7 (2–3), 320–364.
Dohrenwend, B.P., 2006. Inventorying stressful life events as risk factors for psychopathology: toward resolution of the problem of intracategory variability. Psychological Bulletin 132, 477–495.
Doyle, O., Harmon, C., Heckman, J., Tremblay, R., 2009. Investing in early human development: timing and economic efficiency. Economics and Human Biology 7 (1), 1–6.
Dreber, A., Apicella, C., Eisenberg, D., Garcia, J., Zamore, R., Lum, K., Campbell, B., 2009. The 7R polymorphism in the dopamine receptor D4 gene (DRD4) is associated with financial risk-taking in men. Evolution and Human Behavior 30, 85–92.
Dickens, W., Flynn, J., 2001. Heritability estimates versus large environmental effects: the IQ paradox resolved. Psychological Review 108 (2), 346–369.
Duggal, S., Malkoff-Schwartz, S., Birmaher, B., Anderson, B.P., Matty, M.K., Houck, P.R., et al., 2000. Assessment of life stress in adolescents: self-report versus interview methods. Journal of the American Academy of Child and Adolescent Psychiatry 39, 445–452.
Durrant, J.E., Janson, S., 2005. Law reform, corporal punishment and child abuse: the case of Sweden. International Review of Victimology 12 (2), 139–158.
Foley, D.L., Craig, J., Morley, R., Olsson, C., Dwyer, T., Smith, K., Saffery, R., 2009. Prospects for epigenetic epidemiology. American Journal of Epidemiology 169, 389–400.
Foley, D.L., Eaves, L.J., Wormley, B., Silberg, J.L., Maes, H.H., Kuhn, J., 2004. Childhood adversity, monoamine oxidase A genotype and risk for conduct disorder. Archives of General Psychiatry 61 (7), 738–744.
Fraga, M.F., Ballestar, E., Paz, M.F., Ropero, S., Setien, F., et al., 2005. Epigenetic differences arise during the lifetime of monozygotic twins. Proceeding of the National Academy of Sciences of the United States of America 102, 10604–10609.
Franke, B., Neale, B., Faraone, S., 2009. Genome-wide association studies in ADHD. Human genetics 126 (1), 13–50.
Goldberger, A., 1979. Heritability. Economica 46, 327–347.
Goldstein, D., 2009. Common genetic variation and human traits. New England Journal of Medicine 360 (17), 1696–1698.
Gorman, D.M., 1993. A review of studies comparing checklist and interview methods of data collection in life event research. Behavioral Medicine 19, 66–73.
Guo, G., Yuying, T., Tianji, C., 2008. Gene by social context interactions for number of sexual partners among white male youths: genetics-informed sociology. American Journal of Sociology 114, 36–66.
Guo, G., Stearns, E., 2002. The social influences on the realization of genetic potential for intellectual development. Social Forces 80 (3), 881–910.
Haberstick, B., Lessem, J., Hopfer, C., Smolen, A., Ehringer, M., Tiimberlake, D., Hewitt, J., 2005. Monoamine oxidase A (MAOA) and antisocial behaviors in the presence of childhood and adolescent maltreatment. American Journal of Medical Genetics Part B (Neuropsychiatric Genetics) 135B, 59–64.
Harden, K.P., Turkheimer, E., Loehlin, J.C., 2007. Genotype by environment interactions in adolescents' cognitive aptitude. Behavioral Genetics 37, 273–283.
Heckman, J.J., 2007. The economics, technology, and neuroscience of human capability formation. Proceeding of the National Academy of Sciences of the United States of America 104, 13250–13255.
Heckman, J.J., 2008. Schools, Skills, and Synapses. IZA Working Paper 3515. .
Heijmans, B., Tobi, E., Stein, A., Putter, H., Blauw, G., Susser, E., Slagboom, E., Lumey, L.H., 2008. Persistent epigenetic differences associated with prenatal exposure to famine in humans. Proceeding of the National Academy of Sciences of the United States of America 105, 17046–17049.
Hirschhorn, J., 2009. Genome wide association studies—illuminating biologic pathways. New England Journal of Medicine 360 (17), 1699–1701.
Hoffjan, S., Nicolae, D., Ostrovnaya, I., Roberg, K., Evans, M., Mirel, D., et al., 2005. Gene–environment interaction effects on the development of immune responses in the 1st year of life. American Journal of Human Genetics 76, 696–704.
Horwitz, A., Videon, T., Schmitz, M., Davis, D., 2003. Rethinking twins and environments: possible social sources for assumed genetic influences in twin research. Journal of Health and Social Behavior 44, 111–129.
Huizinga, D., Haberstick, B., Smolen, A., Menard, S., et al., 2006. Childhood maltreatment, subsequent antisocial behavior, and the role of monoamine oxidase A genotype. Biological Psychiatry 60, 677–683.
Hung, R.J., McKay, J.D., Gaborieau, V., et al., 2008. A susceptibility locus for lung cancer maps to nicotinic acetylcholine receptor subunit genes on 15q25. Nature 452 (7187), 633–637.
Hunter, D.J., 2005. Gene–environment interactions in human diseases. Nature Genetics 6, 287–298.
Ioannidis, J.P.A., 2005. Why most published research fi ndings are false. PLoS Med 2 (8), e124.
Israel, S., Lerer, E., Shalev, I., Uzefovky, F., Riebold, M., Laiba, E., et al., 2009. The oxytocin receptor (OXTR) contributes to prosocial fund allocations in the dictator game and the social value orientations task. PLoS One 4 (5), e5535.
Jasienka, G., 2009. Low Birth Weight of Contemporary African Americans: An Intergenerational Effect of Slavery? American Journal of Human Biology 21, 16–24.
Jäntti, M., Bratsberg, B., Røed, K., Raaum, O., Naylor, R., Österbacka, E., Björklund, A., Eriksson, T., 2006. American Exceptionalism in a New Light: A Comparison of Intergenerational Earnings Mobility in the Nordic Countries, the United Kingdom and the United States. IZA Discussion Paper 1938. .
Jencks, C., 1980. Heredity, environment, and public policy reconsidered. American Journal of Sociology 45, 723–736.
Joseph, J., 2002. Twin studies in psychiatry and psychology: science of pseudoscience? Psychiatric Quarterly 73 (1), 71–82.
Kaati, G., Bygren, L.O., Edvinsson, S., 2002. Cardiovascular and diabetes mortality determined by nutrition during parents' and grandparents' slow growth period. European Journal of Human Genetics 10, 682–688.
Kahn, R., Khoury, J., Nichols, W., Lanphear, B., 2003. Role of dopamine transporter genotype and maternal prenatal smoking in childhood hyperactive-impulsive, inattentive, and oppositional behaviors. The Journal of Pediatrics 143, 104–110.

Kaufman, J., Yank, B.-Z., Douglas-Palumberi, H., et al., 2007. Genetic and environmental predictors of early alcohol use. Biological Psychiatry 61, 1228–1234.
Kendler, K.S., Kessler, R.C., Walters, E.E., MacLean, C., Neale, M.C., et al., 1995. Stressful life events, genetic liability, and onset of an episode of major depression in women. American Journal of Psychiatry 152 (6), 833–842.
Kendler, K.S., Karkowski, L.M., Prescott, C.A., 1999. Causal relationship between stressful life events and the onset of major depression. American Journal of Psychiatry 156 (6), 837–841.
Knafo, A., Israel, S., Darvasi, A., Bachner-Melman, R., Uzefovsky, F., et al., 2007. Individual differences in allocation of funds in the dictator game associated with length of the arginine vasopressin 1a receptor RS3 promoter region and correlation between RS3 length and hippocampal mRNA. Genes, Brains, and Behavior 7, 266–275.
Kuhnen, C., Chiao, J., 2009. Genetic determinants of financial risk taking. PLoS One 4 (2), e4362.
Lehn, H., Derks, E.M., Hudziak, J.J., Heutink, P., van Beijsterveldt, T.C.E.M., Boomsma, D.I., 2007. Attention problems and attention-deficit/hyperactivity disorder in discordant and concordant monozygotic twins: evidence of environmental mediators. Journal of the American Academy of Child and Adolescent Psychiatry 46, 83–91.
Liu, H., Zeng, J., 2009. Genetic ability and intergenerational earnings mobility. Journal of Population Economics 22, 75–95.
Lumey, L.H., Stein, A., Kahn, H., van der Pal-de Bruin, K., Blauw, G.J., Zybert, P., Susser, E., 2007. Cohort profile: the Dutch hunger winter families study. International Journal of Epidemiology 36, 1196–1204.
McDermott, R., Tingley, D., Cowden, J., Frazzetto, G., Johnson, D.D.P., 2009. Monoamine oxidase A gene (MAOA) predicts behavioural aggression following provocation. Proceeding of the National Academy of Sciences of the United States of America 106, 2118–2123.
McEvoy, B.P., Visscher, P.M., 2009. Genetics of human height. Economics and Human Biology 7, 294–306.
McGue, M., 2008. The end of behavioral genetics? Acta Psychologica Sinica 40 (10), 1073–1087.
McQuaid, J.R., Monroe, S.M., Roberts, J.E., Kupfer, D.J., Frank, E., 2000. A comparison of two life stress assessment approaches. Prospective prediction of treatment outcome in recurrent depression. Journal of Abnormal Psyhchology 109 (4), 787–791.
Meaburn, E.L., Harlaar, N., Craig, I.W., Schalkwyk, L.C., Plomin, R., 2008. Quantitative trait locus association scan of early reading disability and ability using pooled DNA and 100K SNP microarrays in a sample of 5760 children. Molecular Psychiatry 13, 729–740.
Meltzer, H., 1989. Serotonergic dysfunction in depression. British Journal of Psychiatry Suppl. 155, 25–31.
Mill, J., Dempster, E., Caspi, A., Williams, B., Moffitt, T., Craig, I., 2006. Evidence for monozygotic twin discordance in methylation level at two CPG sites in the promoter region of the catechol-O-methyltransferase gene. American Journal of Medical Genetics 141, 421–425.
Mill, J., Petronis, A., 2007. Molecular studies of major depressive disorder: the epigenetic perspective. Molecular Psychiatry 12, 799–814.
Mill, J., Petronis, A., 2008. Pre- and peri-natal environmental risks for attention-deficit hyperactivity disorder (ADHD): the potential role of epigenetic processes in mediating susceptibility. Journal of Child Psychology and Psychiatry 49 (10), 1020–1030.
Moffitt, T., Caspi, A., Rutter, M., 2006. Measured gene–environment interactions in psychopathology. Perspectives on Psychological Science 1 (1), 5–27.
Mortensen, E.L., Michaelsen, K.F., Sanders, S.A., Remich, J.M., 2002. Association between duration of breastfeeding and adult intelligence. Journal of the American Medical Association 287 (19), 2365–2371.
Monroe, S.M., 2008. Modern approaches to conceptulizing and measuring human life stress. Annual Review of Clinical Psychology 4, 33–52.
Monroe, S.M., Reid, M.W., 2008. Gene–environment interactions in depression research: genetic polymorphisms and life-stress polyprocedures. Psychological Science 19, 947–956.
Munafò, M., Durrant, C., Lewis, G., Flint, J., 2009. Gene × environment interactions at the serotonin transporter locus. Biological Psychiatry 65, 211–219.
Nätt, D., Lindqvist, N., Stranneheim, H., Lundeberg, J., Torjesen, P., Jensen, P., 2009. Inheritance of acquired behavior adaptations and brain gene expression in chickens. PLoS One 4 (7), e6405.
Neuman, R., Lobos, E., Reich, W., Henderson, C., Sun, L.-W., Todd, R., 2007. Prenatal smoking exposure and dopaminergic genotypes interact to cause a severe ADHD subtype. Biological Psychiatry 61, 1320–1328.
Nilsson, K.W., Sjöberg, R.L., Damberg, M., Alm, P.O., Ohrvik, J., Leppert, J., et al., 2005. Role of the serotonin transporter gene and family function in adolescent alcohol consumption. Alcoholism: Clinical and Experimental Research 29, 564–570.
North, K.E., Martin, L.J., 2008. The importance of gene environment interaction: implications for social scientists. Sociological Methods & Research 37 (2), 164–200.
Oates, N.A., Van Vliet, J., Duffy, D.L., Kroes, H.Y., Martin, N.G., Boomsma, D.I., 2006. Increased DNA methylation at the AXIN1 gene in a monozygotic twin from a pari discordant for a caudal duplication anomaly. American Journal of Human Genetics 79, 155–162.
Oliver, B., Pike, A., Plomin, R., 2008. Nonshared environmental influences on teacher-reported behavior problems: monozygotic twin differences in perceptions of the classroom. Journal of Child Psychology and Psychiatry 49 (6), 646–653.
Painter, R., Roseboom, T., Bleker, O., 2005. Prenatal exposure to the Dutch famine and disease in later life: an overview. Reproductive Toxicology 20, 345–352.
Paykel, E.S., 1997. The interview for recent life events. Psychological Medicine 27, 301–310.
Pearson, T., Manolio, T., 2008. How to interpret genome-wide association study. Journal of the American Medical Association 299 (11), 1335–1344.
Pembrey, M., Bygren, L.O., Kaati, G., Edvinsson, S., Northstone, K., Sjöström, M., Golding, J., 2006. Sex-specific, male-line transgenerational responses in humans. European Journal of Human Genetics 14, 159–166.
Plomin, R., DeFries, J.C., Loehlin, J.C., 1977. Genotype–environment interaction and correlation in the analysis of human behavior. Psychological Bulletin 84, 309–322.
Plug, E., Vijverberg, W., 2003. Schooling, family background and adoption: is it nature of is it nurture? Journal of Political Economy 111, 611–641.
Polanczyk, G., de Lima, M.S., Horta, B.L., Biederman, J., Rhode, L.A., 2007. The worldwide prevalence of ADHD: a systematic review and metaregression analysis. American Journal of Psychiatry 164, 942–948.
Reif, A., Rosler, M., Freitag, C.M., Schneider, M., Eujen, A., Kissling, C., 2007. Nature and nurture predispose to violent behavior: serotonergic genes and adverse childhood environment. Neuropsychopharmacology 32, 2375–2383.
Reiss, D., 2008. Social processes and genetic influences in child development: novel uses of twin and adoption designs. Acta Psychologica Sinica 40 (10), 1099–1105.
Richardson, K., Norgate, S., 2006. A critical analysis of IQ studies of adopted children. Human Development 49, 319–335.
Risch, N., Herrell, R., Lehner, T., Liang, K.-Y., Eaves, L., Hoh, J., et al., 2009. Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: a meta-analysis. Journal of the American Medical Association 301, 2462–2471.
Roe, B., Tilley, M., Gu, H., Beversdorf, D., Sadee, W., Haab, T., Papp, A., 2009. Financial and psychological risk attitudes associated with two single nucleotide polymorphisms in the nicotine receptor (CHRNA4) gene. PLoS One 4 (8), e6704.
Rowe, D.C., Jacobson, K.C., van den Oord, E., 1999. Genetic and environmental influences on vocabulary IQ: parental education level as moderator. Child Development 70 (5), 1151–1162.
Rutter, M., Silberg, J., 2002. Gene–environment interplay in relation to emotional and behavioral disturbance. Annual Review of Psychology 53, 463–490.
Sacerdote, B., 2002. The nature and nurture of economic outcomes. American Economic Review 92, 344–348.
Sacerdote, B., 2007. How large are the effects from changes in family environment? A study of Korean American Adoptees. Quarterly Journal of Economics 122, 119–157.
Scarr, S., Weinberg, R., 1994. Educational and occupational achievements of brothers and sisters in adoptive and biologically related families. Behavior Genetics 24, 301–325.
Settle, J., Dawes, C., Hatemi, P., Christakis, N., Fowler, J., 2008. Friendships moderate an association between a dopamine gene variant and political ideology. Paper presented at the Annual Meeting of the APSA 2008, Boston.
Shanahan, M., Vaisey, S., Erickson, L., Smolen, A., 2008. Environmental contingencies and genetic propensities: social capital, educational continuation and dopamine receptor gene DRD2. American Journal of Sociology 114, 260–286.
Smith, S.L., Howard, J.A., Monroe, A.D., 2000. Issues underlying behavior problems in at-risk adopted children. Children and Youth Services Review 22, 539–562.
Stoolmiller, M., 1999. implications of the restricted range of family environments for estimates of heritability and non-shared environment in behavior-genetic adoption studies. Psychological Bulletin 125, 392–409.

Taubman, P., 1976. The determinants of earnings: genetics, family and other environments; a study of white male twins. American Economic Review 66 (5), 858–870.
Taylor, A., Kim-Cohen, 2007. Meta-analysis of gene–environment interactions in developmental psychopathology. Development and Psychopathology 19 (4), 1029–1037.
Thapar, A., Stergiakouli, E., 2008. An overview of the genetics of ADHD. Acta Psychologica Sinica 40 (10), 1088–1098.
Thorgeirsson, T.E., Geller, F., Sulem, P., et al., 2008. A variant associated with nicotine dependence, lung cancer and peripheral arterial disease. Nature 452 (7187), 638–642.
Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., Gottesman, I., 2003. Socioeconomic status modifies heritability of IQ in young children. Psychological Science 14 (6), 623–628.
Uher, R., McGuffin, P., 2008. The moderation by the serotonin transporter gene of environmental adversity in the etiology of mental illness: review and methodological analysis. Molecular Psychiatry 13 (2), 131–146.
Uher, R., McGuffin, P., 2010. The moderation by the serotonin transporter gene of environmental adversity in the etiology of depression: 2009 update. Molecular Psychiatry 15, 18–22.
Wallis, D., Russell, H., Muenke, M., 2008. Review: genetics of attention deficit/hyperactivity disorder. Journal of Pediatric Psychology 33 (10), 1085–1099.
Waterland, R., Jirtle, R., 2003. Transposable elements: targets for early nutritional effects on epigenetic gene regulation. Molecular and Cellular Biology 23 (15), 5293–5300.
Weaver, I.C., Cervoni, N., Champagne, F.A., D'Alessio, A.C., Sharma, S., et al., 2004. Epigenetic programming by maternal behavior. Nature Neuroscience 7, 847–854.
Weaver, I.C., Meaney, M.J., Szyf, M., 2006. Maternal care effects on the hippocampal transcriptome and anxiety-mediated behaviors in the offspring that are reversible in adulthood. Proceeding of the National Academy of Sciences of the United States of America 103, 3480–3485.
Weedon, M.N., Lango, H., Lindgren, C.M., et al., 2008. Genome-wide association analysis identifies 20 loci that influence adult height. Nature Genetics 40, 575–583.
Wells, J.C.K., 2010. Maternal capital and the metabolic ghetto: an evolutionary perspective on the transgenerational basis of health inequalities. American Journal of Human Biology 22, 1–17.
Wong, M.Y., Day, N.D., Luan, J.A., Wareham, N.J., 2003. The detection of gene–environment interaction for continuous traits: should we deal with measurement error by bigger studies or better measurement? International Journal of Epidemiology 32 (1), 51–57.
Young, S.E., Smolen, A., Hewitt, J.K., Haberstick, B.C., Stallings, M.C., Corley, R.P., et al., 2006. Interaction between MAO-A genotype and maltreatment in the risk for conduct disorder: failure to confirm in adolescent patients. American Journal of Psychiatry 163, 1019–1025.