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The influence of nonshared environmental factors on number and word recall test performance

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ABSTRACT

A large body of research indicates that variation in intelligence is influenced by genetic and environmental factors. Despite this knowledge, much of the research examining environmental influences on intelligence is not conducted using genetically informative research designs. In order to address this gap in the literature, this study examines the potential association between nonshared environments and measures of intelligence (recall ability) in adulthood using monozygotic (MZ) difference scores analyses. Analysis of MZ twin pairs drawn from the National Longitudinal Study of Adolescent to Adult Health revealed that none of the nonshared environmental variables were consistently related to recall ability. One nonshared environmental variable, maternal disengagement, was found to be a significant predictor of recall ability in two of the four recall tasks. In addition, measures of maternal attachment and delinquent peers were found to be associated with only one test of word recall ability and none of the three other recall tests.

1. Introduction

Intelligence is a relatively stable trait that is characterized by a significant amount of variability in the population (Deary, Whalley, Lemmon, Crawford, & Starr, 2000). Of particular importance, intelligence has been found to be a predictor for an array of life outcomes. For instance, intelligence has been associated with increased educational outcomes, occupational status, and incomes in longitudinal studies (Strenze, 2007; Deary et al., 2005). In addition, intelligence as also been linked with health related outcomes including cardiovascular disease (Hart et al., 2004), obesity (Chandola, Deary, Blane, & Batty, 2006), stroke (Hart et al., 2004), and premature mortality (Batty, Deary, & Gottfredson, 2007). Overall, research indicates that intelligence has a broad influence that affects life factors in almost all domains of life.

Against this backdrop, it is not surprising that there is a significant amount of attention placed on identifying the etiology of intelligence and related cognitive abilities. Evidence from this body of research indicates that intelligence is under significant genetic influence with heritability estimates consistently above .50 (Plomin, 1999). The remaining variance tends to be accounted for by environmental influences that are unique to each child. Research has identified a host of specific environments that might be involved in the creation of variation in intelligence; however, a major limitation of most of this research is that it might be misspecified because of a failure to control for the potentially confounding effects of genetic influences.

2. Etiology of intelligence

There is a wealth of literature exploring the etiology of intelligence. Evidence from this line of research indicates that variation in intelligence is influenced by genetic and environmental factors (Nisbett et al., 2012). Studies on the heritability of intelligence indicate that genetic factors account for between 40% and 80% of the variance in intelligence (Nisbett et al., 2012; Deary, Johnson, & Houlihan, 2009). Moreover, findings from this area of research reveal an age-related pattern with low estimates of heritability during childhood and increasing estimates of heritability with age (Spinath, Ronald, Harlaar, Price, & Plomin, 2003; Haworth et al., 2010). Overall, findings from these studies indicate that genes account for approximately 80% of the variance in intelligence in adulthood (Gottfredson & Saklofske, 2009; Edmonds et al., 2008).[1] The remaining variance in intelligence is explained by environmental factors (and error).

Studies that examine the influence of genetic factors on intelligence frequently employ twin-based research designs. Twin-based research designs operate by comparing phenotypic similarity between monozygotic (MZ) and dizygotic (DZ) twin pairs which allows for phenotypic variance to be partitioned into genetic and environmental components. The logic of this approach is grounded in the fact that MZ twins share approximately 100% of their DNA and DZ twins share approximately

[1] A reviewer pointed out that gene-environment interactions and active/evocative gene-environment correlations may help to explain the high estimates of heritability for intelligence. For an in-depth discussion of this issue see Plomin, DeFries and Loehlin (1977)

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50% of their distinguishing DNA.[2] As a result, if the assumptions of twin research designs are met, greater phenotypic similarity between MZ twins compared to DZ twins is interpreted as being the result of genetic influences, with the remaining variance being the result of environmental influences (and error). The environmental component is divided into a shared and a nonshared component.[3] Shared environmental influences make children who live in the same household similar to one another whereas nonshared environmental influences make children who live in the same household different from one another. In general, findings from twin-based research studies on the etiology of intelligence indicate that nonshared environments account for variation in adult intelligence scores, while shared environmental factors appear to have limited effects (Edmonds et al., 2008; McGue & Bouchard, 1998; Bouchard & McGue, 2003).

Aside from genetically informed studies on intelligence, there is a growing line of research examining specific environments that may account for variation in intelligence. For instance, previous research has identified family-level measures, such as parental involvement (Bradley et al., 1993; Fan & Chen, 2001), parental attachment (Guo & Harris, 2000), and parental encouragement (Koutsoulis & Campbell, 2001) as being related to intelligence and academic performance. In addition, findings from studies that incorporate natural experiments and reviews of the literature on the relationship between education and intelligence indicate that additional years of schooling are associated with intelligence scores in adulthood (Brinch & Galloway, 2012; Ceci, 1991). Most of the existing studies in this area, however, have examined the influence of environmental factors on intelligence using standard social science methodologies that examine only one child per family and do not estimate genetic effects. Furthermore, these studies are unable to distinguish between shared and nonshared environments.

3. Limitations of environmental research on intelligence

Despite the large body of research indicating that intelligence is genetically influenced, the majority of studies examining environmental effects on intelligence do not take into account genetic influences. As a result, the models used to examine the etiology of intelligence might be misspecified and thus produce incorrect or biased coefficients for environmental measures. Further complicating the matter is that a large review of the studies examining genetic influences on variation in environmental measures revealed that variability in parenting measures and other environments predicted to influence the development of intelligence is due to genetic influences (Kendler & Baker, 2007).[4] Findings from studies that have revealed that specific environmental measures are significantly related to variation in intelligence, therefore, may be confounded by unmeasured genetic influences as these studies usually do not attempt to control for genetic confounding. Importantly, studies that have examined the development of intelligence using genetically informative designs have revealed relatively small or even nonsignificant effects for specific environmental variables, including family- and parent-based measures (Beaver et al., 2014; Haworth et al., 2010; Bouchard & McGue, 2003).

[2] Genetic differences may arise between MZ twins due to point mutations and epigenetic markers (Li et al., 2013; Fraga et al., 2005).

[3] Some researchers in this area further decompose environmental factors into effectively and objectively shared environments. For a thorough discussion of the difference between effectively and objectively shared/nonshared environments see Turkheimer and Waldron (2000).

[4] This phenomenon is referred to as a gene-environment correlation. Gene-environment correlations refer to the process through which genetic propensity can influence and structure environments. For a more thorough discussion on gene-environment correlations see Plomin, DeFries, and Loehlin (1977).

4. Current study

There is a significant amount of research examining environmental influences on intelligence, but most of this research does not take into account the influence of genetics. As a consequence, it is not possible to determine the precise role that specific environmental factors have on measures of intelligence. In order to address this gap, the current study estimated the association between nonshared environmental factors and intelligence (as measured by number and word recall tasks). We use MZ difference scores analyses to examine these associations and, at the same time, take into account genetic confounding.

5. Methods

5.1. Data

This study uses data drawn from waves 1 and 4 of the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is a multi-wave longitudinal and nationally representative sample of adolescents in the United States (Udry, 2003). Data collection for the first wave of the Add Health survey was conducted during the 1994–1995 school year and included information from more than 90,000 students who were between the ages of 12 and 18. Questions in the first wave of the Add Health survey covered topics such as daily activities, relationships with parents and peers, and involvement with delinquency. The fourth wave of the survey was administered between 2007 and 2008 and was completed by more than 15,000 of the original participants from wave 1 (Harris et al., 2003). At wave 4, participants were young adults between the ages of 24 and 32 and were asked questions related to educational histories, employment status, and marital status.

The Add Health survey contains a subsample of siblings, twins, and cousins that can be used to conduct genetically informed analyses. During the first wave of the survey, kinship pairs were deliberately oversampled by asking respondents if they lived with a twin, sibling, cousin, or unrelated sibling. If the participants answered affirmatively, and the sibling, was between the ages of 11 and 20, then the sibling was added into the sample. The full sibling sample contains more than 3000 kinship pairs (Beaver, 2008). This study uses data from 289 pairs of monozygotic (MZ) twins with sample sizes ranging from 161 to 163 MZ twin pairs for the analyses of this study.

5.2. Measures

5.2.1. Outcome measures

5.2.1.1. Number recall. At wave 4 participants were asked to complete seven tasks that involved repeating a sequence of numbers backwards to the survey administrator. The length of the number sequences ranged from two to eight numbers. A total-number recall score was created by summing together how participants performed on each of these number recall tasks. Participants were awarded one point for correctly completing each of the number recall sequence tasks. For this analysis we use the total-number recall score and a mid-range (4 number) recall score to measure number recall ability. The total-number recall test measure has been used previously as a measure of short-term memory (Lundberg, 2015) and as a measure of neuropsychological deficits with Add Health data (Beaver, Vaughn, DeLisi, Barnes, & Boutwell, 2012).

5.2.1.2. Word recall. At wave 4 participants were asked to perform two word recall tasks where they were asked to repeat as many words off a list that they could remember in a set time period. In the first task, respondents were shown a word list and then immediately asked to repeat as many words off the list that they could remember in a 90-second time period. In the second task, administered later in the survey,