Genetics of Mathematical Aptitude

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Normative variation in mathematical aptitude (MA) is relevant to many aspects of societal functioning, in particular for occupations in science, technology, engineering and mathematics (STEM). Though elements of numerosity have been identified in some animals, complex MA is uniquely human. Several studies, using a variety of designs, have investigated the heritability of MA, and the extreme ends of the dimension of this trait – mathematics learning disability (MLD) and mathematical talent (MT). Initial estimates of the heritability of MA ranged from 0.2 to 0.9, while later studies identify more modest heritabilities for MA. Modest heritabilities have also been identified for MLD and MT. Despite this, genome-wide association studies have had limited success. Additionally, MA may have similar genetic architectures to MLD and MT. Finally, while existing studies have tried to understand the genetic architecture of MA as a whole, different domains within MA may have different genetic architectures.

Introduction

Mathematical aptitude (MA) is important for several jobs, particularly in the science, technology, engineering and mathematics (STEM) fields. Between 5% and 8% of children and adolescents have difficulties in mathematics, and poor mathematical skills can have negative consequences for employment and consequently mental well-being (Geary, 2004). While complex mathematics is thought to be uniquely human, there are certain aspects of MA that are not solely related to humans. Indeed, several species of nonhuman animals have been shown to perform simple counting and arithmetic, suggesting evolutionary conservation of brain regions for numerosity (Hubbard *et al*., 2008). Several studies have established numerosity in infants, suggesting that certain aspects of MA may be innate, and these are supported by studies that have identified a heritable component to MA. More recent studies have focused on other aspects of MA, including subdomains of MA, and genetic correlation with other traits such as reading and general intelligence (g). These studies have also investigated changes in heritability of MA with time. A few studies have also investigated the heritability of mathematical talent (MT) and mathematical learning disability (MLD). Over the last decade, molecular genetic investigations have provided novel insights into the genetic architecture of MA, suggesting that MA is polygenic.

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Quantifying Mathematical Aptitude

One of the key challenges in the study of MA is to quantify the high variability in the trait. Several different instruments have been developed, but there is a lack of consensus on the best instrument. Most studies have investigated MA in children or students. Some of these studies have used teacher reports (Oliver *et al*., 2004; Walker *et al*., 2004; Docherty *et al*., 2010a) but these are subjective, and cannot be replicated easily. Indeed, twin studies show that MA has different heritabilities when twins are graded by the same or different teachers (Oliver *et al*., 2004). In addition, these are sensitive to cultural trends in education: one cannot expect different countries to have the same teaching and grading patterns for MA (Huntsinger *et al*., 2010; Chiu and Klassen, 2010). Other studies have used school examination results (Baron-Cohen *et al*., 2014; Rimfeld *et al*., 2015), which can change over time owing to changes in examination standards, and self-report measures (Krinzinger *et al*., 2009), which can be confounded by self-report biases. An alternative is to use standardised tests such as the Weschler Objective Numerical Dimension and the Test of Early Mathematical Ability. More recently, a few studies have used web-based testing either as a stand-alone measure of MA or in addition to school grades and other standardised scores (Docherty *et al*., 2010a). Web-based testing is cheap, easy to administer, less prone to error and can be used to collect data from a large participant cohort. In addition, these tests can be timed, and the time taken to answer each

question can also be calculated. Many of these tests have been designed to test a wide range of abilities in mathematics that do not require more than elementary training in mathematics.

Despite the advances in instrument development, two challenges remain. The first is developing a test that captures most if not all the variation in MA. This includes all the subdomains in MA and the variation across the ability spectrum. This is a challenge because we do not yet know of all the different domains in MA and also because these tests need to be short to be feasible. To work around this, some studies have also focused on specific areas of MA and tested these extensively (Kovas *et al*., 2007). A second area of concern is developing tests for adults. Most existing tests target children or adolescents who are currently in or have only recently left mathematical education. As a result, we know little about the biological correlates of MA in adults and how these change with time. To address this, we have recently developed the Mathematics Quotient (MQ), a short online test that can be completed by adults and adolescents.

Mathematical Aptitude, Disability and Talent

It is important to distinguish between MA, MLD and MT. MA refers to normative variance in mathematical ability. This includes several different components such as number processing, shape processing, mathematical modelling and non-numerical processes. MA is thought to be normally distributed in the population with MLD and MT on the two extremes of this distribution. MLD is a neurodevelopmental condition where individuals have specific learning difficulties in mathematics skills that are markedly below what is expected at their age or general IQ (American Psychiatry Association, 2013). It is estimated that about 7% of children and adolescents have MLD (Geary *et al*., 2007). It is thought that MLD with deficits in intelligence (or general developmental delay) is different from MLD without deficits in intelligence. Different studies use different criteria for identifying individuals with MLD, typically using a predefined threshold on one or a battery of testing instruments (Oliver *et al*., 2004; Alarcón *et al*., 1997; Kovas *et al*., 2007; Docherty *et al*., 2010a). More recent studies categorise children who score below the 10th percentile on standardized maths tests but having intelligence above the 15th percentile as having MLD (Geary, 2011). Owing to the different criteria adopted by different studies referenced in this article, we refer to low mathematical ability as MLD even if they may not necessarily meet the diagnostic criteria for MLD.

MT, on the other hand, has been less investigated than both MLD and MA. Identifying cognitively talented individuals is usually achieved using 'talent searches'. Individuals are identified as being cognitively talented if they score in the top percentile on standardized subtests such as the Scholastic Aptitude Test-Mathematics (SAT-M). One of the oldest studies of mathematically proficient children classified children as mathematically talented if they scored above 500 or 700 out of 800 on the SAT-M before the age of 13 (Lubinski and Benbow, 2006). Another study, which looked at mathematical profiles in individuals with autism, defined MT (the term used in the study was hypercalculia) as children who scored average on calculation skills, but scored below average on domains of reading and applied problems (Wei *et al*., 2015). Other studies used self-report measures of talent (Vinkhuyzen *et al*., 2009) and have used participants in the top 85 percentile (Kovas *et al*., 2007) based on scores on tests of MA to define MT.

Individual and Group Differences in MA

A few studies have investigated the heritability of MA. Heritability is the proportion of variance in a trait that can be explained by genes in a given population at a given time. Most studies that have investigated the heritability of MA have used a twin design. These studies use genetic information shared between different twin pairs to calculate heritability. One of the earliest studies to calculate heritability of MA used report card grades in 1020 13-year old twins (Husen, 1959). The study identified a heritability of 0.66 for arithmetic, higher than the heritabilities of grades in history, reading and writing in the same cohort. Two subsequent studies reported different heritabilities for MA, one reporting heritability as low as 0.19 (Thompson *et al*., 1991) and the other as high as 0.90 (Alarcon *et al*., 2000). Estimates of shared and unique environment also varied considerably between these studies. These wide estimates can be attributed to a few factors such as differences in age of the participants and the different instruments used to measure MA.

In response to the discrepancies in heritabilities, subsequent studies have tried to address these issues by investigating MA in two well-defined cohorts – the Twins Early Development Study (TEDS) in the United Kingdom and the Western Reserve Reading and Math Project (WRRMP) in the United States. Heritability of MA has been investigated at several time points in the TEDS sample, and the genetic and environmental components have remained largely the same, even though there have been some differences in how MA has been assessed at various stages. Heritability has remained largely constant between 7 years of age and 12 years of age and explains about two-thirds of the variance in MA (Kovas *et al*., 2013). Similarly, unique environment accounts for a quarter of the total variance, while shared environment has minimal contribution. At approximately 16 years of age, the heritability of MA as measured using the scores on the General Certificate of Secondary Education (GCSE) examinations ranges from 0.55 (Shakeshaft *et al*., 2013) to 0.65 (Rimfeld *et al*., 2015).

Further evidence for high heritability of MA in the TEDS cohort comes from a genetic study that used genome-wide single nucleotide polymorphism (SNP) data to calculate heritability (Davis *et al*., 2014). All SNPs, additively, resulted in a heritability of 0.51 in the sample of 12-year-old twins. However, all SNPs, additively, resulted in a heritability of only 0.21 (Rimfeld *et al*., 2015) for maths GCSE scores, which declined to 0.14 after removing the effect of intelligence. The difference in SNP-based heritability is interesting, and may point to different genetic architecture for MA measured using GCSE scores and web-based testing.

The TEDS sample has also been used to investigate the heritability of subdomains of MA at ages 7, 9 and 10 years. At ages 7 and 9, subdomains had similar heritabilities to the composite maths score (Haworth *et al*., 2007), while the heritabilities were lower for the subdomains at age 10 than that of composite MA (Kovas *et al*., 2007). Notably, the heritabilities for each individual subdomain remained constant at ages 7 and 9 years. Different subdomains were measured at age 10, which does not allow for longitudinal analyses in this age group. The TEDS samples have also been used to identify the heritability of MA after removing known correlates. One study used GCSE scores of mathematics to calculate the heritability of MA independent of intelligence. In this cohort, the independent component of MA had a heritability of 0.45 (Rimfeld *et al*., 2015), suggesting that intelligence is an important contributor of MA. Another study identified that 'pure maths' (MA after the common reading and intelligence components have been removed) has a heritability of 0.44 (Tosto *et al*., 2013) in 12-year-old twins. Mathematical literacy also has a higher heritability than general cognitive ability (g), which increases linearly from childhood to early adulthood (Haworth *et al*., 2010).

Results from the US cohort have been substantially lower than those from the UK-based cohort. Data on various facets of MA from two different waves of testing at approximately 9–11 had much lower heritabilities (Petrill *et al*., 2012; Hart *et al*., 2009). For example, in 9 year olds, genetics, shared environment and unique environment explained about a third of the variance each in MA as measured using the mathematics subset of the Wide Range Aptitude Test (Hart *et al*., 2009). Additionally, subdomains of MA had low heritabilities at age 9 (0–0.63), though this increased at age 11 (0.29–0.54) (Hart *et al*., 2009). The difference in heritabilities between the two cohorts is intriguing, but it is difficult to directly compare them as the measures used were different between the cohorts. Additionally, the WRRMP cohort is smaller than the TEDS cohort and the age range of the participant during any one wave of testing was larger in the WRRMP cohort than in the TEDS cohort.

Two studies have also investigated the heritability of self-rated MA. In the TEDS sample, self-rated MA had a heritability of approximately 40% at ages 9 and 12 (Luo *et al*., 2011). The bulk of this was explained by dominant genetic effects (0.38). In a second study, carried out using participants from the Netherlands Twin Registry, self-rated MA had a higher heritability in adolescent and adult twins. Heritability from additive genetic factors was 0.11 (Vinkhuyzen *et al*., 2009), while heritability from dominant genetic factors was 0.56. Unique environmental contributions were similar in both the studies, accounting for approximately a third of the variance. Additionally, modest heritability has also been identified for number sense (Tosto *et al*., 2014), spatial ability (Vuoksimaa *et al*., 2010) and maths anxiety (Wang *et al*., 2014).

Fewer studies have investigated the heritability of MLD and MT. Family members of individuals with MLD are 10 times more likely to be diagnosed with MLD, indicating a heritable component (Geary, 2004). The first study in twins identified a modest heritability of 0.38 for MLD (Alarcón *et al*., 1997). Studies using the TEDS sample have identified higher heritability of MLD, though they used the 15% cut-off criteria to define MLD. At both 7 and 9 years of age, heritability of MLD was similar to heritability of MA (approximately two-thirds of the total variance) in the TEDS sample (Haworth *et al*., 2007). The genetic correlation between the two ages was quite high, indicating that the same set of genes contribute to variation at both the ages. However, by age 10, the heritability declines to 0.45, while both shared and unique environments have greater contribution to the variation in MLD when compared with MA (Kovas *et al*., 2007). Interestingly, subdomains of mathematics are more heritable in the lowest 5% of the cohort when compared to the lowest 15% of the cohort, based on performance on the various maths measures (Kovas *et al*., 2007). In the TEDS cohort, the heritability of MT was investigated using the top 85 percentile 10-year-old twins. Heritability was modest for the composite math score (0.53) and the heritabilities for the three mathematical subdomains ranged from 0.09 (non-numerical processes) to 0.52 (understanding numbers) (Petrill *et al*., 2009). There was some variation between heritabilities in the high ability group and the whole cohort, indicating that there may be a different genetic architecture between the two groups. However, another study identified much higher heritability for MT in adult and adolescent Dutch twins. Using self-report data, the study identified a heritability of 0.87 for additive genetic components (Vinkhuyzen *et al*., 2009). Unique environment accounted for the remainder of the variance. The discrepancy in the heritabilities can be attributed, in part, to the type of measures used (self-report vs performance based). In addition, other factors such as sample size, cultural differences, and model fitting all contribute to differences in heritability. Neither of the studies, however, have investigated individuals with extreme mathematical ability, individuals in the top 1–5% (such as winners of the Fields Medal or the Wolf Prize), and indeed it will be difficult to study this using a traditional twin design.

It is unclear to what extent MA, MLD and MT are related genetically. The generalist genes hypothesis suggests that the same genes are associated with MA and MD. Alternatively, different genes can contribute to different ability levels on the MA spectrum. Some forms of MD may be a result of brain injuries (Benavides-Varela *et al*., 2014), functional deficits in specific brain regions (Dehaene *et al*., 2004) and changes in grey matter (Dahaene et al. Dehaene *et al*., 2004). Difficulties in mathematics are also associated with Turner syndrome, Williams Syndrome, and Prader–Willi Syndrome, conditions that have distinct genetic profiles (Mazzocco, 2006; Bertella *et al*., 2005; O'Hearn and Luna, 2009). On the other hand, a subset of children with autism have hypercalculia (Wei *et al*., 2015), and children with autism use complicated number decomposition strategies during arithmetic problem solving (Iuculano *et al*., 2014). These suggest that at least for some individuals, MD and MT might be caused by distinct genetic and nongenetic factors.

Quantitative genetics has also been used to investigate sex differences in MA. Sex difference in MA, if any, is very small (Lindberg *et al*., 2010). However, at the extreme end of MA, there are greater number of males who have won prestigious awards in mathematics such as the Fields Medal and the Abel Prize. Data from more than 1.5 million school students identify an overrepresentation of males in the top percentiles (5 and above) for mathematics (Stoet and Geary, 2013). Twin-based genetic studies

have consistently identified similar heritabilities for MA at various ages, and across different cohorts. Similarly, twin analyses have not identified any sex differences for subdomains of MA, MLD and self-reported MT (Haworth *et al*., 2007; Markowitz *et al*., 2005; Vinkhuyzen *et al*., 2009). Additionally, molecular genetic studies of systemizing, which is correlated with MA, have not identified any significant difference in heritabilities between males and females (results available from authors). All these studies point to a similar genetic architecture for various aspects of MA, MT and MLD in males and females. However, other factors such as hormones, gene–environment interactions or sociological factors such as the lack of female role models may all contribute to the small average differences in MA between males and females.

Molecular Genetic Studies

A few studies have investigated the molecular genetic architecture of MA. To date, there have been four genome-wide association studies (GWAS) of MA in the general population, though none of them have identified a replicable significant SNP at a threshold of 5×10^{-8}. Two GWAS for MA have been conducted in the TEDS sample. The first followed a two-stage screening protocol in a sample of 1200 10-year olds from a larger cohort of 5019 participants and used a pooled DNA (deoxyribonucleic acid) analysis to identify differences in allele frequencies (Docherty *et al*., 2010a). They used a case–control design, including the 600 lowest scoring and the 600 highest scoring participants from the cohort in the study, but did not identify any SNP significant at a genome-wide threshold. They subsequently investigated the effects of the top 46 SNPs on normative variation in MA in 2356 individuals, and identified 10 nominally significant SNPs ($P < 0.05$). Together, these 10 SNPs accounted for 2.9% of the phenotypic variance. The 10-SNP set was also associated with teacher ratings of MA at ages 7 and 9 and web-based measure of MA at age 12 (Docherty *et al*., 2010b). However, the 10-SNP set was also associated with reading and general cognitive ability at age 10, suggesting a role in other cognitive processes for these variants (Docherty *et al*., 2010b). The 10-SNP set also interacted with environmental variables such as parental negativity, home chaos and teacher negativity to predict MA (Docherty *et al*., 2011). The second study used individual genotyping to investigate MA in 12-year olds using a linear regression model. The study did not identify any significant variant associated with MA (Davis *et al*., 2014).

The third GWAS also used a pooled-DNA approach in 602 school and college students to identify SNPs associated with MA (Baron-Cohen *et al*., 2014). Participants were categorized into low or high maths group based on GCSE grades in mathematics, while controlling for verbal ability. Using a relaxed threshold of 1.5×10^{-5}, the authors reported one SNP (rs789859, OR = 1.6) that was significant for MA. rs789859 lies in the promoter/regulatory region of *FAM43A*, a gene that is expressed in the brain and spinal cord.

Ludwig and colleagues used the correlation between dyslexia and dyscalculia to investigate the genetics of mathematics (Ludwig *et al*., 2013). They performed a GWAS of MA in an initial cohort of 200 children and replicated the top SNP (rs133885 in *MYO18B*) in two independent samples of children with and without dyslexia. However, a subsequent study failed to replicate this (Pettigrew *et al*., 2015).

In addition, one study has also investigated the role of copy number variation in MA. Davis and colleagues investigated the association of DUF1220 copy numbers with IQ and MA (Davis *et al*., 2015). Copy number variation in DUF1220 has previously been associated with both brain size and brain evolution. Copy numbers in DUF1220 was associated with MA in a subsample of 75 individuals who were selected for extremes of brain circumference.

GWAS have currently not identified any variants associated with MA. This may be due to the high polygenicity of the trait and the fact that variants are likely to have small effects. The current sample sizes are not large enough to capture such small effects. However, common variants account for a substantial fraction of the variance in MA, providing further evidence for the heritability of MA.

Conclusions

Variations in MA, including MD and MT are difficult to accurately capture. Several different methods have emerged to try and capture this. Yet, despite the differences in study methodologies, a few themes have emerged, which have been largely substantiated across different studies. First, the entire spectrum of MA is modestly heritable. It is polygenic though syndromic conditions can cause MD in some individuals. Heritabilities and genetic architecture are largely similar between males and females, and this provides further evidence for the small differences in MA between males and females. It is not entirely clear if MA, MLD and MT share the same genetic architecture, although they are genetically correlated. Finally, while molecular genetic studies have not been successful yet in identifying genetic variants associated with MA, they have provided further evidence for the heritability of the trait.

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