Rapid Communication

Title: Parental Education Accounts for Variability in the IQs of Probands with Down Syndrome: A Longitudinal Study¹

Parental Education and Proband IQ Variability

David W. Evans 0000-0002-3990-5494 0000-0002-3990-5494, PhD, Bucknell University, Department of Psychology, Lewisburg, PA USA

Mirko Uljarevic, PhD, MD, Olga Tennison Autism Research Centre, School of Psychology and Public Health, La Trobe University, Bundoora, Victoria 3086, Australia.

Corresponding Author: David W. Evans, Bucknell University, Department of Psychology, Program in Neuroscience, 315 O'Leary Center, Lewisburg, PA 17837. Email: dwevans@bucknell.edu. Phone: 570.577.3478

¹ This is the author manuscript accepted for publication and has undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi:[10.1002/ajmga.38519](https://doi.org/10.1002/ajmga.38519)

ABSTRACT

Recent work has demonstrated that variability in proband phenotypes including physical features, cognitive ability, social functioning, and other developmental domains, is influenced by parental traits. Here we examine the role of parental education as a factor contributing to the variability of intelligence quotient (IQ) of offspring with trisomy 21. Participants were forty-three probands with trisomy 21, aged 4 to 21 years of age, and their parents. Data were collected on parental education, and a bi-parental mean education score (BMES) was calculated. Probands' cognitive abilities were assessed by the Stanford-Binet 4th at baseline (T1), and again 24 months later (T2). Probands were placed into one of two age groups: 4 to 12 years and 13 to 21 years. Results indicated higher parent-proband correlations in Age Group 2 (mean $r = .47$) relative to Age Group 1 (mean $r = .33$) and increasing parent-proband correlations across time, with mean correlations of Age Group 1, T1: $r = .26$, T2: $r = .39$; Age Group 2 T1: $r = .49$, T2: $r = .46$. Despite the expected IQ deficits observed in trisomy 21 probands, parental education may still contribute to the variability of probands' cognitive abilities. These findings are consistent with the literature noting increasing heritability of IQ with development.

Key Words: Down Syndrome, IQ, Parental Education, Heritability

INTRODUCTION

Understanding the factors accounting for phenotypic variability in genetic syndromes is a primary objective in psychiatric genetics. Mounting evidence suggests that parental traits play a crucial role in accounting for phenotypic variability on traits with known heritability [Hanson, Bernier, Porche, et al., 2015; Hyman et al., 2008; Klaassen et al., 2016; Malich et al., 2000; Moreno De Luca, Myers, Challman, Moreno-De-Luca, Evans, & Ledbetter, 2013; Moreno De Luca, Evans, Boomer et al., 2015]. Even in cases of *de novo* genetic mutations associated with severe neurodevelopmental disorders, parental traits and family background appear to influence the degree of impairment displayed in their offspring on a variety of morphological, cognitive, and behavioral factors [Hanson, Bernier, Porche, et al., 2015; Hyman et al., 2008; Klaassen et al., 2016; Moreno-De-Luca, Evans, Boomer, et al., 2015].

Parental traits have been shown to influence proband phenotype even in disorders that impact heritable physical characteristics. Turner syndrome (45X0), a neurodevelopmental disorder affecting females caused by a whole sex-chromosomal deletion, is associated with short stature that is three to four standard deviations less than the height of their non-carrier first-degree relatives [Brook et al., 1974; Brook et al., 1977; Holl et al., 1994; Lemli & Smith, 1963; Massa & Vanderschueren-Lodeweyckx, 1991; Rochiccioli et al., 1994]. Remarkably however, despite the impact of the 45X0 aneuploidy on physical stature, the correlation between proband height and bi-parental mean height is similar to what is found in the general population, with r s ranging from .49 to .84 [Brook et al., 1974; 1977]. Similarly, in Klinefelter syndrome [47XXY; Brook et al., 1977] and Prader-Willi syndrome [15q11-q13DEL; Malich et al., 2000], probands

exhibit the expected (increased) deviations in height relative to bi-parental mean, while the parent-proband correlations are similar to those reported in the general population.

The influence of parental traits on proband phenotypes is not limited to physical features, but extends to cognitive ability, social behavior, and other developmental domains [Malich et al., 2000, Moreno De Luca et al., 2015]. Recent work [Moreno De Luca, Evans, Boomer, et al., 2015], demonstrated that variability in the cognitive phenotype of *de novo* 16p11.2 deletion probands may be attributed to parental cognitive ability. As expected, the Full Scale Intelligence Quotients (FSIQ) of the 16p11.2 deletion probands were shifted 1.7 SD from the non-carrier, first-degree relatives. However, despite the shift toward increasing impairment in the probands, parent-child correlations for IQ approached .50 [Moreno De Luca, Evans, Boomer et al., 2015] – similar to the parent-child IQ correlations observed in the general population [Deary, Spinath, & Bates, 2006]. Similar IQ patterns have been observed in Prader-Willi syndrome (15q11.2-q13 deletion/uniparental disomy; [Malich et al., 2000]) where probands' IQs were shifted 2.6 standard deviations relative to maternal mean IQ, with significant maternal-offspring IQ correlations for female ($r=.58$) but not for male ($r=.12$) probands. The differential gender associations reported by Malich and colleagues are difficult to interpret as paternal IQs were not considered, and separate analyses for children with deletions and uniparental disomy (UPD) were not provided -- a significant issue considering that cases due to UPD are less impaired in terms of their cognitive ability [Dykens, Cassidy, & King, 1999; Roof, Stone, MacLean, Feurer, Thompson, & Butler, 2000; Torrado, Araoz, Baialardo, et al., 2007]. Still gender-specific effects may possibly

result from a preponderance of X-linked, IQ-related genomic variations, although this merits further exploration.

Cognitive phenotype of Down syndrome probands and their parents

Over forty years ago, several research groups explored the link between parental cognitive ability and the IQs of their offspring with Down syndrome (DS). In addition to the expected deficits in the IQs of offspring with Down syndrome relative to their parents, probands' IQs remain significantly correlated with parental IQ ($r = .50$ with fathers; $r = .40$ with mothers; [Fraser & Sadovnik, 1976; Gibson, 1967]). Using the highest level of parental education as a proxy for IQ, Golden and Pashayan [1976] reported that the mean IQ for the children with DS whose parents had college or post-graduate education was 50, whereas the DS offspring of parents who had not attained a high school education had mean IQs of 35. Similar results were reported elsewhere [Libb, Meyers, Graham & Bell, 1983; for an overview please see Hodapp & Zigler, 1990]. Further evidence that probands' IQ scores are modulated by parental level of education was provided in a recent study of the 22q11.2 deletion syndrome [Klaassen et al., 2016]: the FSIQ differential between the probands whose parents were at the highest, compared to those at the lowest levels of education, was 9 points (.6 SD), which in this case, spanned from a mean IQ of 76 to 67 – crossing the traditional IQ diagnostic threshold for intellectual disability.

The extant literature is limited in that all previous studies have been cross-sectional designs. This presents limitations that are specific to the study of Down syndrome. First, IQ scores in DS cohorts tend to decrease across development [Carr,

2005; Couzens, Cuskelly, & Haynes, 2011; See Patterson et al., 2013, for a review], making the link between parental IQ/education and proband IQ particularly dynamic and complex. Second, the heritability of IQ is not uniform across development. Large population-based studies reveal increasing parent-child IQ correlations from early school age through adolescence [Bergen et al., 2007; Bouchard, 2013; Plomin et al., 2013; Plomin & Deary, 2015]. Haworth et al. [2010] report that the heritability of general cognitive ability increases linearly across ages 9, 12, and 17 years of age. Such factors highlight the critical importance of longitudinal analysis to understand the dynamic nature of parental characteristics on the cognitive ability of probands with DS.

Here, we present the first longitudinal evidence demonstrating the impact of parental education on the cognitive phenotype of children and adolescents with Down syndrome -- the most common genetic form of intellectual disability. We used parental education status for several reasons. First, we aimed to replicate recent cross-sectional findings by Klaassen et al. [2016]; Second, education level has a moderate to strong relationship with IQ [Deary et al., 2007; Deary, 2012; Mackintosh, 2011; Plomin & Deary, 2015]; and third, the association between spouses' levels of education is about .6, which indicates that, like IQ, education is part of an assortative mating process [Jensen, 1998; for a discussion on relevance of education level for assortative mating for intelligence see Plomin & Deary, 2015].

METHOD

Participants were forty-three individuals (18 males; 25 females) with Down syndrome (trisomy 21) aged 4 to 21 years ($M_{age}=11.7$ [11 years; 7 months]; $SD=4.8$ years) and their parents (father $M_{age}= 45.8$; $SD = 10.5$; mother $M_{age}= 43$; $SD=8.6$).

Probands were placed into two age groups based on data indicating progressively high heritability of IQ from middle childhood through adolescence. Age Group 1 (n=21) ranged in age from 4 to 12 years ($M_{age}=7.97$ years; $SD=2.8$). Age Group 2 (n=23) ranged from 12.2 to 21.1 years ($M_{age}=15.3$ years; $SD=2.79$). All of the participants were tested again two years following baseline. Full descriptive statistics are presented in Table 1.

Participants were recruited from several sites in an urban region of the Southeastern United States, including both public and private facilities. Parents completed a demographic form including information on parental education, marital status, ethnicity, religion, child's health status, sex, and information on any siblings. Parents received a written description of the proposed study, along with informed consent. Willing participants were asked to return the signed consent forms and completed inventories to their child's school, or to the investigator. Children's cognitive abilities were assessed with the Stanford-Binet 4th edition, at the site of their school, daycare, or home. For Age Group 1 at Time 1, NVIQ data were available on only 12 children (owing to non-compliance or fatigue effects of the children), and for Age Group 1 at Time 2 NVIQ n=17. Parents were administered a range of other measures as part of a larger study [see Evans & Gray, 2000; Evans et al., 2014].

Parental education was classified along an 8-point scale: 1=less than 7th grade education (2.3% fathers; 0 mothers); 2=junior high school only (n=0); 3=some high school only (4.5% fathers; 0 mothers); 4=high school only: (18.2% fathers; 27.3% mothers); 5=technical training program post high school (6.8% fathers; 13.6% mothers);

6=some college (29.5% fathers; 15.9% mothers); 7=college degree (22.7% fathers; 20.5% mothers); 8=advanced/professional degree: (13.6% fathers; 22.7% mothers). Bi-parental mean for education level was calculated for each parental dyad. In the event that only one parent's education level was available (one father's education was missing) the remaining parent's education level was used.

RESULTS

Zero order correlations were conducted between Bi-parental Mean Education Scores (BMES) and children's Full Scale IQ (FSIQ), Verbal IQ (VIQ) and Non-Verbal IQ (NVIQ) for each Age Group at both time points. The full list of correlations is presented in Table 2. For Age Group 1, Time 1 none of the correlations between BMES and child IQ scores was statistically significant. At Time 2, VIQ approached significance ($r(19)=.41$, $p=0.06$, and BMES and NVIQ were significantly correlated $r(17)=.56$, $p=0.021$. For Age Group 2, Time 1, significant correlations emerged between BMES and FSIQ $r(23)=.45$, $p=.031$; VIQ ($r(23)=.56$, $p=.006$; NVIQ $r(22)=.46$, $p=.03$. For Age Group 2, Time 2, BMES correlated with FSIQ $r(23)=.42$, $p=.045$; VIQ $r(23)=.57$, $p=.005$; NVIQ $r(22)=.38$, $p=.082$.

Considering these cross-sectional and longitudinal time points as four distinct developmental time points, respectively, Age Group 1, T1, T2, Age Group 2, T1, T2, we observed the BMES-IQ correlations changed over time as follows: BMES-FSIQ: $r=.34$, $r=.20$, $r=.45$, $r=.42$; BMES-VIQ: $r=.25$, $r=.41$, $r=.56$, $r=.57$; BMES-NVIQ: $r=.19$, $r=.56$, $r=.46$, $r=.38$. Mean correlations coefficients were as follows: Age Group 1, T1: $r=.26$, T2: $r=.39$; Age Group 2 T1: $r=.49$, T2: $r=.46$.

Given previous work reporting differential correlations between mother-proband and father-proband dyads, we conducted correlations separately for each parent-offspring pair. For Age Group 1 at Time 1, only the correlation between father education and proband FSIQ reached significance: $r(19)=.49$, $p=.034$. None of the mother-proband correlations reached statistical significance. At Time 2, the correlation between NVIQ and father education approached significance $r(16)=.48$, $p=.06$, and NVIQ was significantly correlated with mother education $r(17)=.55$, $p=.022$.

For Age Group 2, Time 1, paternal education correlated with FSIQ $r(23)=.59$, $p=.003$; VIQ $r(23)=.67$, $p=.0004$; NVIQ $r(22)=.61$, $p=.003$. At Time 2, paternal education correlated with FSIQ $r(23)=.44$, $p=.035$, VIQ $r(23)=.51$, $p=.012$, and NVIQ $r(22)=.42$, $p=.049$. Maternal education was significantly correlated with Time 2 VIQ only $r(23)=.52$, $p=.012$.

Finally, consistent with the previous literature for parents of typically-developing children, it is noteworthy that maternal and paternal education levels were correlated $r(20)=.68$, $p<.001$ for Age Group 1, and $r(23)=.64$, $p<.0001$ for Age Group 2.

DISCUSSION

Many cognitive, social, behavioral, motor, and physical traits are highly heritable, and as such, parental characteristics continue to play a role in the development and outcomes of their offspring -- even when their offspring have a neurodevelopmental disorder of known genetic etiology [Brook, 1974; 1977; Hansen et al., 2015; Malich et al., 2000; Moreno De Luca et al., 2013; 2015; Klaassen, 2016]. Here, we examined the

role of parental education and its possible influence on the cognitive ability (IQ) in probands with Down syndrome. This study addresses an important limitation in previous work on this topic given a growing body of work revealing increases in heritability of IQ from early childhood into the school-age and adolescent years in typical development [Plomin et al., 2013; Plomin & Deary, 2015; Haworth et al., 2010]. This presents interesting challenges for research on genetic syndromes that are marked by unique developmental trajectories across various domains of functioning. The cognitive development of probands with DS is known to follow a particular trajectory that yields what appears to be decreasing IQ scores over time, as probands' chronological and cognitive developmental levels grow increasingly disparate. Therefore, we employed a longitudinal approach in order to better understand the potential impact of parental education (as a proxy for parental cognitive ability; [Golden & Pashayan, 1976; Klaassen et al., 2016; Libb, et al., 1983] on the variability of IQ in probands with DS at different points in development.

Our findings suggest that parental education does indeed appear to modulate the cognitive level of offspring with Down syndrome, and that this modulation is dynamically related to the age of the offspring. The weakest correlations between parental education and proband IQ emerged in the youngest cohort at Time 1. Indeed none of the correlations between bi-parental mean education levels and proband IQ scores reached significance. While paternal education correlated with proband FSIQ, none of the other parent-child correlations reached significance. At Time 2 in Age Group 1 bi-parental mean education correlated with both VIQ and NVIQ of the probands. For the older cohort – Age Group 2 at Time 1, parental education was most robustly correlated with

proband FSIQ, VIQ, and NVIQ; these were particularly robust for paternal education, where 45% of the variance in proband IQ was accounted for by paternal education level. At Time 2 FSIQ and VIQ remained significantly related to parental education levels, but the education-NVIQ was rendered non-significant in this oldest cohort; still, all three IQ scores remained correlated with paternal education.

Some limitations are noteworthy. First, although our sample size was not small considering the fact that the estimated prevalence of DS is 8.27 in 10,000 [Presson et al., 2013], replication of our findings using larger samples of individuals will be a goal for future research. This is particularly relevant to the Age Group 1, Time 1 cohort for whom only a subset of participants had NVIQ data, which would have reduced power. Second, although previous work has used parental education as a proxy for IQ [Klaassen et al., 2016] and shows moderate to high association with IQ [Mackintosh, 2011; Plomin & Deary, 2015], parental level of education is still an indirect measure that can be influenced by other factors, such as the broader socio-economic context. Despite these limitations, to our knowledge, this is the first longitudinal analysis demonstrating the role of parental education as a contributing factor to offspring cognitive ability in Down syndrome. Consistent with previous research on the general population, we observed a relative increase in shared variance between parental cognitive/educational status and their offspring with DS with development. The correlations between parental education and proband IQ increased over time, both cross-sectionally and longitudinally, with a leveling off or slight decrease in the oldest cohort. The contribution of parental traits and family background factors on proband outcomes merits further exploration in other neurodevelopmental disorders of various etiologies.

Our study replicates previous findings noting the parental contributions to the cognitive abilities of probands with DS (and other genetic syndromes). Here we provide the first evidence of increasing heritability of IQ across development in a DS cohort. Taken in the broader philosophical and historical context, these findings speak to several important theoretical issues in the development of intellectual disabilities. Early efforts to characterize the development of persons with intellectual disabilities resulted in various theoretic models [e.g., Dingman & Tarjan, 1960; Zigler, 1967], most notably, Zigler's "two group" approach [Zigler, 1967]. According to the original two-group approach, the development of persons whose intellectual disability is caused by non-organic factors (cultural-familial) is similar to typically-developing children in both sequence and structure, whereas, for disabilities with organic etiology (e.g., known genetic causes, such as DS), development involves a qualitatively different process. Subsequent work [Burack, 1990; Cicchetti & Pogge-Hesse, 1982; Hodapp & Zigler, 1990], however, expanded the two-group approach, noting the relevance of models of typical development to the development of those intellectual disabilities with known organic etiology. The work reported here demonstrates that despite the obvious biological origins of the intellectual impairments in probands with Down syndrome, familial factors nonetheless play a critical role in modulating the phenotypic outcome of their offspring. Thus, not only do these (and previous) findings offer practical applications in the way of informing expectations for future development of children with DS, they speak to the broader theoretical issues of the continuity that underlies typical and atypical development.

Conflict of Interest: None stated.

References

- Bergen, S. E., Gardner, C. O., & Kendler, K. S. (2007). Age-related changes in heritability of behavioral phenotypes over adolescence and young adulthood: A meta-analysis. *Twin Research and Human Genetics*, 10, 423-433.
- Brook, C. G., Mürset, G., Zachmann, M., & Prader, A. (1974). Growth in children with 45,XO Turner's syndrome. *Archives of Disease in Childhood*, 49(10), 789-795.
- Brook, C. G., Gasser, T., Werder, E. A., Prader, A., Vanderschueren-Lodewykx, M. A. (1977). Height correlations between parents and mature offspring in normal subjects and in subjects with Turner's and Klinefelter's and other syndromes. *Annals of Human Biology*, 4(1), 17-22.
- Bouchard, T. J. (2013). The Wilson Effect: The increase in heritability of IQ with age. *Twin Research and Human Genetics*, 16, 923-930.
- Burack JA. (1990). Differentiating mental retardation: Two-group approach and beyond. In Hodapp RM, Burack JA, & Zigler E, editors, *Issues in the developmental approach to mental retardation* (pp 27-48). NY: Cambridge University Press.
- Carr, J. (2005). Stability and change in cognitive ability over the life span: A comparisons of populations with and without Down's syndrome. *Journal of Intellect Disabilities Research*, 49, 915-928.
- Cicchetti D & Pogge-Hesse P. (1982). Possible contributions of the study of organically retarded persons to developmental theory. In Zigler E, & Balla, D, editors, *The developmental-difference controversy* (pp. 277-318). Hillsdale, NJ: Erlbaum.

- Couzens, D., Cuskelly, M., & Haynes, M. (2011). Cognitive development and Down syndrome: Age-related change on the Stanford-Binet Test (Fourth Edition). *American Journal of Intellectual and Developmental Disabilities*, 116, 181-204.
- Deary, I. J., Spinath, F. M & Bates, T.C. (2006). Genetics of intelligence. *European Journal of Human Genetics*, 14, 690-700.
- Dingman, H. F., & Tarjan, G. (1960). Mental retardation and the normal distribution curve. *American Journal of Mental Deficiency*, 64, 991-994.
- Dykens, E. M., Cassidy, S. B., & King, B. H. (1999). Maladaptive behavior differences in Prader-Willi syndrome due to paternal deletion versus maternal uniparental disomy. *American Journal of Mental Retardation*, 104, 67–77.
- Evans, D. W. & Gray, F. L. (2000). Compulsive-like behavior in individuals with Down syndrome: It's relation to mental age level, adaptive, and maladaptive behavior. *Child Development*, 71, 288-300
- Evans, D. W., Kleinpeter, F. L., Slane, M. M., & Boomer, K. B. (2014). Adaptive and maladaptive correlates of repetitive behavior and restricted interests in persons with Down syndrome and developmentally-matched typical children: A two-year longitudinal sequential design. *PLoS One*, 9:e93951.
- Hanson, E., Bernier, R., Porche, K., Goin-Kochel, R. P., Snyder, L. G., Snow-Gallagher, A., Wallace, A. S., Campe, K., Zhang, Y., Chen, Q., Moreno De Luca, A., Orr, P. T., Boomer, K. B., Evans, D. W., Martin, C. L., Ledbetter, D. H., Spiro, J., Chung, W. (2015). The cognitive and behavioral phenotype of the 16p11.2 deletion. *Biological Psychiatry*, 77(9), 785-93.

Haworth, C. M. A., Wright, M. J., Luciano, M., de Geus, E. J. C., van Beijsterveldt, C. E. M., Bartels, M., Posthuma, D., Boomsma, D. I., David, O. S. P., Kovas, Y., Iacono, M. G., McGue, M., Thompson, L. A., Hart, S. A., Lubinski, D., & Plomin, R. (2010). The heritability of general cognitive ability increases linearly from childhood to young adulthood. *Molecular Psychiatry*, 15, 1112-1120.

Hodapp R. M. Burack J. A. & Zigler E. (1990). Issues in the developmental approach to mental retardation. NY: Cambridge University Press.

Hodapp RM & Zigler E. (1990). Applying the developmental perspective to individuals with Down syndrome. In Cicchetti D, & Beeghly M, editors, *Children with Down syndrome: A developmental perspective*. NY Cambridge University Press.

Holl, R. W., Kunze, D., Etzrodt, H., Teller, W., & Heinze, E. (1994). Turner syndrome: final height, glucose tolerance, bone density and psychosocial status in 25 adult patients. *European Journal of Pediatrics*, 153(1), 11-16.

Fraser, F. C., & Sadovnick, A. D. (1976). Correlation of IQ in subjects with Down syndrome and their parents and sibs. *Journal of Mental Deficiency Research*, 20(3), 179-182.

Golden, W., & Pashayan, H. M. (1976). The effect of parental education on the eventual mental development of noninstitutionalized children with Down syndrome. *Journal of Pediatrics*, 89, 603-605.

Klaassen, P., Duijff, A., Swanenburg, de V, Beemer, F., Sinnema, G., Breetvelt, E., Schappin, R., & Vorstman. J. (2016). Explaining the variable penetrance of

CNVs: Parental intelligence modulates expression of intellectual impairment caused by the 22q11.2 deletion. *American Journal of Medical Genetics*, 171, 790-796.

Lemli, L., & Smith, D. W. (1963). The XO syndrome: a study of the differentiated phenotype in 25 patients. *Journal of Pediatrics*, 63(4), 577-588.

Libb, J. W., Meyers, G. J., Graham, E., & Bell, B. (1983). Correlates of intelligence and adaptive behavior in Down's syndrome. *Journal of Intellect Disabilities Research*, 27, 205-201.

Malich, S., Largo, R. H., Schinzel, A., Molinari, L., & Eiholzer, U. (2000). Phenotypic heterogeneity of growth and psychometric intelligence in Prader-Willi syndrome: variable expression of a contiguous gene syndrome or parent-child resemblance? *American Journal of Medical Genetics*, 91(4), 298-304.

Massa, G. G., & Vanderschueren-Lodeweyckx, M. (1991). Age and height at diagnosis in Turner syndrome: Influence of parental height. *Pediatrics*, 88(6), 1148-1152.

Moreno De Luca, A^{*},² Evans, D. W^{*}, Boomer, K., Hanson, E., Bernier, R., Goin-Kochel, R., Myers, S. M., Challman, T. D., Moreno, De Luca, D., Spiro, J., Chung, W., Martin, C. L., & Ledbetter, D. H. (2015). Parental cognitive, behavioral and motor profiles impact the neurodevelopmental profile of individuals with de novo mutations. *JAMA Psychiatry*, 72(2), 119-26.

Moreno-De-Luca, A., Myers, S. M., Challman, T. D., Moreno-De-Luca, D., Evans, D. W. & Ledbetter, D. H. (2013). Developmental brain dysfunction (DBD): Revival and

² * denotes joint first authorship

expansion of an old concept based on new genetic evidence. *Lancet Neurology*, 12(4), 406-414.

Patterson, T., Rapsey, C., & Glue, P. (2013). Systematic review of cognitive development across childhood in Down syndrome: Implications for treatment interventions. *Journal of Intellectual Disabilities Research*, 57(4), 306-318.

Plomin, R., & Spinath, F. M. (2004). Intelligence: genetics, genes, and genomics. *Journal of Personality and Social Psychology*, 86 (1), 112-129.

Presson, R., & Deary, I. J. (2015). Genetics and intelligence differences: Five special findings. *Molecular Psychiatry*, 20, 98-108.

Roof, E., Stone, W., MacLean, W., Feurer, I. D., Thompson, T., Butler, M. G. (2000). Intellectual characteristics of Prader-Willi syndrome: comparison of genetic subtypes. *Journal of Intellectual Disabilities Research*, 44(1), 25–30.

Torrado, M., Araoz, V., Baialardo, E., Abraldes, K., Mazza, C., Krochik, G., Ozuna, B., Leske, V., Caino, S., Fano, V., & Chertkoff, L. (2007). Clinical-etiological correlation in children with Prader-Willi syndrome (PWS): an interdisciplinary study. *American Journal of Medical Genetics: Part A*, 143, 460–468.

Zigler, E. (1967). Familial mental retardation: A continuing dilemma. *Science*, 155, 292-298.

Table 1. Descriptive statistics

	Age (months)		Full Scale IQ		Verbal IQ		Non-Verbal IQ	
	M	SD	M	SD	M	SD	M	SD
Age Group 1*								
Time 1	95.70	29.61	46.10	8.26	58.21 ^a	10.61	53.23 ^b	8.06
Time 2	118.9	30.22	46.19	10.07	60.29 ^c	10.96	53.65 ^d	7.59
Age Group 2**								
Time 1	184	33.46	38.48	4.32	49.09	6.58	47.27 ^e	9.57
Time 2	208.78	33.05	39.26	5.43	50.21	6.58	45.64 ^e	9.57

Note: *Total N=20; ** Total N=23; ^aN=19; ^bN=12; ^cN=20; ^dN=17; ^eN=22

Table 2. Parental education-proband IQ associations

	Full Scale IQ				Verbal IQ				Non-Verbal IQ			
	Age Group 1		Age Group 2		Age Group 1		Age Group 2		Age Group 1		Age Group 2	
	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
BMES	.34	.20	.45*	.42*	.25	.41*	.56**	.57**	.19	.55*	.46*	.38
FES	.49*	.25	.59**	.44*	.38	.52*	.67**	.51*	.27	.48	.61**	.42*
MES	.17	.12	.22	.32	.10	.47*	.33	.52*	.09	.56*	.22	.26

Note: * $p < 0.05$; ** $p < 0.01$

Minerva Access is the Institutional Repository of The University of Melbourne

Author/s:

Evans, DW;Uljarevi#, M

Title:

Parental education accounts for variability in the IQs of probands with Down syndrome: A longitudinal study.

Date:

2018-01

Citation:

Evans, D. W. & Uljarevi#, M. (2018). Parental education accounts for variability in the IQs of probands with Down syndrome: A longitudinal study.. Am J Med Genet A, 176 (1), pp.29-33.
<https://doi.org/10.1002/ajmg.a.38519>.

Persistent Link:

<http://hdl.handle.net/11343/293893>