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The epidemiology of cognitive development☆

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ABSTRACT

The epidemiology of cognitive development is an approach essentially based on large observational studies, which examines individual differences in cognitive abilities throughout childhood and their determinants. Although different in terms of methodology and main interests from developmental psychology, cognitive epidemiology offers complementary viewpoints on cognitive development and addresses fundamental research questions of interest to developmental psychologists. The present paper depicts the contributions of the epidemiological approach to the field of cognitive development and highlights the methodological advances that have made such contributions possible. We discuss the stability and developmental trajectories of cognitive functions, their main predictors, the complex interplay between environmental and genetic predictors, and the relationships between the different domains of cognition from birth to adulthood.

1. Introduction

1.1. What makes each of us a human being?

Jacques Mehler was a pioneer in developmental psychology, a discipline attempting to address some of the most fundamental questions in cognitive science:

- What is the initial state of cognition?
- What are the different stages from the initial to the final state (adulthood)?
- What are the mechanisms leading from one stage to the next?
- How rich must the initial state be for typical cognitive development to proceed, given typical environmental input?

Developmental psychology attempts to identify, describe and dissect basic cognitive processes, which are assumed to be universal (unless pathological). As such, it belongs to the tradition of cognitive psychology, and aims at addressing the fundamental question: "What makes each of us a human being"? The focus is on what is common to all members of the species, with a special interest in what differentiates us from other species.

This intellectual tradition developed a suite of methods appropriate for its purpose. Developmental psychology has traditionally relied on the experimental manipulation of factors that are hypothesised to have an effect on certain aspects of development, and on the comparison of relatively small groups of children using custom-made tasks (McCall, 1977). The main interest is on the effect of experimental conditions and their interactions on group means. Inter-individual variance is treated as noise.

1.2. What makes each of us a unique human being?

As important as the fundamental question of developmental psychology may be, it is not the only question that can be asked about development. Another one is: "What makes each of us a *unique* human being" (different from all the others)? Indeed, a species is defined not only by a set of traits that all members are presumed to share, but also by the variability of those traits. This variability is not an artefact or an imperfection, it is the very material on which natural selection operates. Every individual is a variation on the theme that characterises the species.

In the present paper, we highlight this alternative approach to cognitive development, which focuses on the second fundamental question, that of variability: the epidemiology of cognitive development. Although it is not new, it is seldom presented as a unified, coherent

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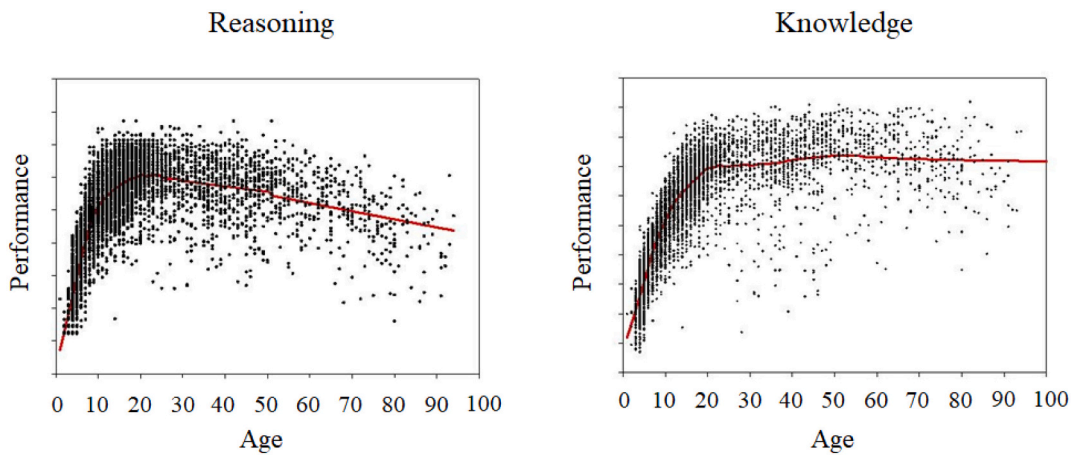

Fig. 1. The evolution of cognitive abilities with age. Cross-sectional data on fluid reasoning ($N = 5712$) and crystallised knowledge ($N = 5315$) measured with the Woodcock-Johson tests of cognitive abilities, 3rd edition, from Tucker-Drob (2019). Caveat: because the data shown do not come from a longitudinal follow-up of the same individuals, the apparent age trends actually exaggerate cognitive decline because of the Flynn effect (i.e., newer generations tend to have higher cognitive abilities). With permission from Elliot Tucker-Drob.

stream of research. We hope to show that it allows one to address questions on development that Jacques Mehler would have found as interesting as those addressed by the traditional developmental psychology approach.

1.3. The epidemiological approach to cognitive development

Epidemiology is the study of the distribution and determinants of health outcomes. Originally focused on mortality and infectious diseases, it now has a much broader scope, including chronic diseases, various health events, but also all sorts of traits. Cognitive epidemiology is a relatively new branch of epidemiology which focuses on cognitive outcomes.

The epidemiology of cognitive development differs from developmental psychology in a number of ways. Asking what makes each of us different from the others leads one to slightly different methodological approaches, where naturally occurring, rather than experimentally-induced variability, is the focus. This approach can therefore be seen as an extension of differential psychology. Because the factors underlying variability are not experimentally designed, the first step is to uncover them and establish their role on the observed variability. To do so, cognitive epidemiology typically relies on observational studies and standardised testing of large populations, often using cohorts constituted for broader purposes (i.e., health-related outcomes).

Although the developmental approach and the epidemiological approach may seem to have diverging interests, it is now becoming apparent that they offer complementary viewpoints on cognitive development. Recent advances suggest that the epidemiological approach can also address questions that are of theoretical interest to developmental psychologists. Beyond identifying simple statistical associations between early factors and cognitive outcomes, the epidemiological approach is able to investigate more complex questions, such as:

- To what extent are individual differences in a cognitive trait stable, and to what extent do they vary throughout development?
- What are the factors that underlie individual developmental trajectories?
- To what extent do different factors interact during development (rather than having additive effects)?
- To what extent is a given cognitive function necessary for the development of another one?
- When two cognitive functions seem interrelated, what is the direction of causation?
- When a factor seems to have an influence on a cognitive trait, what are the mediating mechanisms?

In the present paper, we illustrate how the epidemiology of cognitive development can help address these fundamental questions, and we highlight the methodological advances that have made such contributions possible.

2. Developmental trajectories and stability of individual differences

Developmental change can be studied either in absolute terms (raw scores) or relatively to the same-age population (standardised scores). In absolute terms, all cognitive abilities improve with age, until they reach peak performance, after which they slowly decline with age. The specific pattern of growth, timing of maturity, and speed of decline depends on each specific cognitive ability (Baxendale, 2011). For instance, on the one hand, cross-sectional data show that abstract reasoning abilities (fluid intelligence) grow rapidly until age 20, after which they slowly

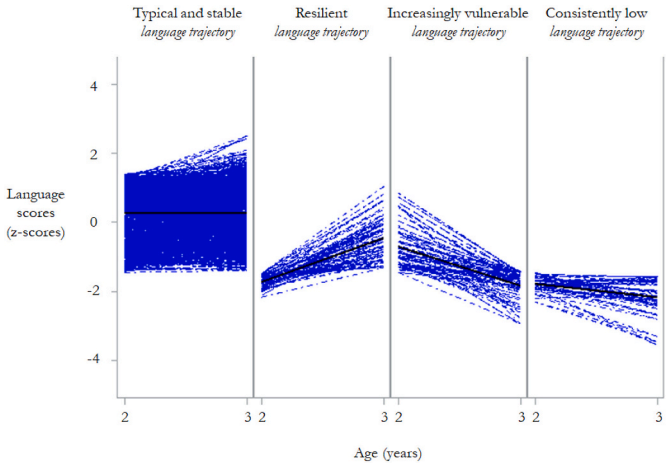

Fig. 2. Trajectories of language development. Illustration of how a group of 1002 children followed from 2 to 3 years old has been divided into four groups, from left to right: children with typical (above the 10th centile), stable language skills ($N = 843$), children with resilient language skills ($N = 59$), children with increasingly vulnerable language skills (N = 59) and children with consistently low language skills ($N = 41$). Scores are age-standardised, so the developmental trajectories shown are relative. Data from Peyre et al. (2014).

Box 1
The special status of IQ in cognitive development

IQ is generally of little interest to developmental psychologists, who see it at best as a global cognitive measure that ignores the specificity of each cognitive function, and that fails to illuminate important theoretical issues about cognitive development. Yet, it is often the most frequently (if not only) cognitive measure reported in epidemiological studies. One obvious reason for this is the high reliability and stability of the measure. But its global aspect is as much an asset as a disadvantage. If one uses only one cognitive measure, IQ is the most obvious choice, as it provides the best possible summary of all cognitive functions (it is generally calculated as the first unrotated component of a principal component analysis of diverse tests, making it a weighted average of all cognitive scores; Spearman, 1904). As a summary measure of cognitive performance, it is also sometimes regarded as an indicator of "brain health" and of cognitive decline. Thus, it is no surprise that IQ is the cognitive measure with the greatest amount of data available, and this is reflected in the present paper.

When IQ is properly measured (as a composite measure of several tests), each of the tests remains available for analysis of more specific abilities; e.g., of verbal or spatial ability, abstract reasoning or working memory. Furthermore, studies that can spare the time and resources to include additional and more specific cognitive tests have the potential to address more precise and sophisticated cognitive questions. Yet, even in those cases, having a global cognitive measure will allow them to at best disentangle to what extent the effects they report are specific to one cognitive function, and to what extent they are general.

decline. On the other hand, cultural knowledge (crystallised intelligence) only reaches a plateau around 20 and then continues to grow very slowly, declining only at very old ages (Fig. 1).

While it is interesting to track the absolute developmental time-course of each cognitive function, it is equally interesting to track individual trajectories and compare them to same-aged individuals (population norms, symbolised by the red line of Fig. 1). Indeed, one way to understand the developmental trajectory of a given function is to study the factors that make it vary between individuals. This leads to investigating relative developmental trajectories; i.e., how the position of a person compared to their age group varies over time. Some individuals will reach developmental milestones early (individual dots above the red line in Fig. 1), and may remain permanently above the norm, while others will eventually be outperformed by their peers. Some will develop more slowly (individual dots below the red line in Fig. 1) and catch up later; while others will never catch up with their peers. Nevertheless, most individuals have a relatively flat trajectory, meaning that their standing relative to their peers is quite stable (illustrated in Fig. 2: more than 80% of the children sampled have a typical and stable trajectory).

The best example of the stability of individual differences is provided by IQ (intelligence quotient) scores. IQ scores are the standardised version of raw measures of general intelligence, positioning individuals on age-related norms, with standardised scores having a population mean conventionally set at 100 and a standard deviation set at 15 (see Box 1). Correlations of IQ scores between age 11 and age 70-79 ranged from 0.73 to 0.78 in cohorts of Scottish individuals (Deary, 2014) and correlations between ages 18 and 55-60 were as high as 0.90 in a cohort of Swedish subjects (Rönnlund, Sundström, & Nilsson, 2015). Together, these findings illustrate the high stability of relative general intelligence.

Interestingly, the correlation between childhood and adulthood IQ decreases, not just as the time interval between the two measures increases, but primarily as the childhood measure is made earlier. For example, Bayley (1949) found that IQ at 18 years old was correlated 0.81 with IQ at 7, but only at 0.25 with developmental milestones at age 1. Similarly, Breeman, Jaekel, Baumann, Bartmann, and Wolke (2015) found that IQ at 26 years old correlated at 0.62 with IQ at age 8, but at 0.57 with IQ at age 6, 0.47 at age 4 and 0.25 at 20 months, thus showing that early measures of general intelligence are much more unstable (and probably also noisier) than later measures. In our own study of predictors of children's IQ at 5-6 years old, we found that developmental milestones observed at 4, 8 and 12 months of age had little predictive power. Only at 24 months old did those milestones show a moderate correlation (0.42) with later IQ. Interestingly, while the milestones measured in infancy covered fine and gross motor skills and social and language skills, only language skills predicted unique variance in later IQ (Peyre, Charkaluk, Forhan, Heude, & Ramus, 2017). These studies thus support that projections based on very young children's performance are risky.

However, when a child experiences certain risk factors, such as a chromosomal abnormality, or the consequences of very preterm birth and very low birth weight, predictive correlations drastically increase, with 20-month old cognitive abilities already significantly predicting adult IQ (correlation of 0.74 for very preterm and very low birth rate individuals, versus 0.25 for term-born children) (Breeman et al., 2015). This suggests that risk factors that affect brain development tend to canalise cognitive development, making it much more predictable than in the general population. Obviously, brain plasticity has a role to play, but it also has its limits (Ramus, 2006).

Beyond general intelligence, language abilities also show substantial stability during childhood: correlations range from 0.64 between 15 and 25 months old to 0.62 from 25 months to 5 years old, and 0.90 from 5 to 11 years old (Bornstein, Hahn, & Putnick, 2016). Again, later performance seems to be more stable than earlier performance. Curiously, language stability appears to be similar in typically and atypically developing children (Bornstein, Hahn, Putnick, & Pearson, 2018).

In this background of general stability, there are children whose trajectories deviate from the norm, upwards or downwards (resilient and increasingly vulnerable children in Fig. 2), due to the influence of specific factors. Studies that investigate this question may investigate the predictors of growth by analysing the predictors of cognitive skills at one age and adjusting on the same skills at an earlier age. Another possible strategy is to model and classify developmental trajectories, either using simple thresholds such as in Fig. 2 (Law, Rush, Anandan, Cox, & Wood, 2012; Peyre et al., 2014), or by using latent class analysis or growth mixture models to uncover more complex trajectories (see Herle et al., 2020, for the methods, and Ukoumunne et al., 2012, for an application to language development), especially across more than 2 time points.

Many factors are known to influence cognitive skills at various ages. Some factors may influence both absolute cognitive skills and their growth, while others may be associated with either one or the other. Understanding which factors influence cognitive skills at what age, and which factors promote growth or prevent decline, may be important to design prevention and intervention programs that use the right levers at the right time. For instance, such knowledge may aid to counter delays in linguistic skills that may induce lower academic attainments (Johnson, Beitchman, & Brownlie, 2010), lower social-emotional and behavioural adjustment (Schoon, Parsons, Rush, & Law, 2010) as well as periods of unemployment in adulthood (Law, Rush, Schoon, & Parsons, 2009). The following section provides an overview of the main predictors of cognitive development.

Table 1
Main predictors of general cognitive ability.

Predictors	Parameters	References
Genetic factors		
Genes from twin studies	$R^2 = 50\%$	Polderman et al. (2015)
SNP heritability (from GCTA studies)	$R^2 = 20\%$	Sniekers et al. (2017)
Polygenic Risk Score	$R^2 = 10\%$	Lee et al. (2018)
Genetic syndrome (here: Fragile X)	$\beta^* \sim -30$ pts	Garber, Visootsak, and Warren (2008)
Prenatal exposures		
Maternal tobacco consumption during pregnancy (>1 pack/d)	$\beta^* = -2$ pts. in univariate analysis but $\beta = 0$ pts. in multivariate analysis	Batty, Der, and Deary (2006)
Maternal alcohol consumption during pregnancy (heavy drinking)	$\beta = -8$ pts	Testa (2003)
Drug exposure during pregnancy (e.g. high-dose valproate (>800 mg /d)	$\beta = -10$ pts	Meador et al. (2009)
Birth factors		
Preterm birth (25-37 GA)	$\beta = -10$ pts	Bhutta, Cleves, Casey, Cradock, and Anand (2002)
Small for gestational age	$\beta = -4$ pts	Sommerfelt (2000)
Apgar scores <7 at 1 and 5 min	$\beta = -1.2$ pts	Odd, Rasmussen, Gunnell, Lewis, and Whitelaw (2007)
Parental and social factors		
Parental education	$\beta = +0.7$ pts./year of parental education	Eriksen et al. (2013)
Birth rank (first vs. second born)	$\beta^* = +3$ pts	Kristensen and Bjerkedal (2007)
Breastfeeding (yes vs. no)	$\beta^* = +3.4$ pts. in univariate analysis and $\beta = +2.62$ when controlling for maternal IQ	(Horta et al., 2015)
Parent-child interaction	$\beta = +0.8$ pts. / HOME score points	Espy, Molfese, and DiLalla (2001)
Malnutrition	$\beta = -3.53$ pts. for early-onset persistent stunting in univariate analyses and $\beta = -2.10$ pts. when controlling for other risks factors	Alam et al. (2020)
Screen exposure	$\beta = -0.5/-0.7$ pts. / daily hour of exposure for the within-subject association	Madigan, Browne, Racine, Mori, and Tough (2019)

Note: Proportion of variance explained, or standardised parameters of multivariate regression analysis (or univariate analysis*). Points refer to IQ scores, which are age-standardised with a population mean of 100 and a standard deviation of 15. Thus, having a genetic syndrome decreases general cognitive ability of about 2 standard deviations on average. The HOME (Home Observation Measurement of the Environment) Inventory (Bradley & Caldwell, 1984) assesses the quality of the cognitive stimulation and emotional support provided by parents to their child, rated on 55 items. This list is not exhaustive.

3. What shapes early cognitive development

Cognition is a vast array of abilities and traits – such as language, motor skills, reasoning, working memory or attention, but also social, emotional and behavioural skills. Providing a detailed picture of the multitude of factors that influence these various areas is no easy task. We do not intend to be exhaustive, but rather to report the main factors, for which solid evidence – from meta-analyses when possible, and large cohort studies otherwise – has been provided. In doing so, we chose not to restrict our scope to some specific domains of cognition to illustrate the diverse influence that predictors have on different domains of cognitive development. The influence of these main factors on IQ is summarized in Table 1.

3.1. Sex

Sex differences in cognitive development have been the focus of a multitude of studies in psychology. Although male and female children are largely similar, they show some differences in the developmental trajectories of certain cognitive functions. For instance, while there is no sex difference in general intelligence (Deary, Strand, Smith, & Fernandes, 2007), robust small differences are found in specific cognitive abilities. Meta-analytic evidence shows that girls have better verbal skills than boys (Hyde & Linn, 1988), at least in early childhood (Peyre et al., 2019), while boys perform better on mental rotation tasks (Maeda & Yoon, 2013; Voyer, Voyer, & Bryden, 1995). In terms of mental health, boys tend to be more at risk of having neurodevelopmental disorders (May, Adesina, McGillivray, & Rinehart, 2019) such as autism spectrum disorder (ASD) (male-to-female ratio equal to 3:1, Loomes, Hull, & Mandy, 2017) and attention deficit-hyperactivity disorder (ADHD) (3:1, Willcutt, 2012). Boys are additionally more susceptible to externalising disorders (Mayes, Castagna, & Waschbusch, 2020), while girls are more susceptible to certain internalising disorders such as depression (Salk, Hyde, & Abramson, 2017).

These observations leave open the mechanisms of sex effects: they may be intrinsic to each sex (i.e., mediated by sex chromosomes or hormones), or may be due to differences in terms of exposure to environmental factors. Satisfactorily disentangling these possibilities is difficult in humans and has not been fully achieved in most cases. There is evidence for both intrinsic and extrinsic effects, such that neither can readily be excluded for most cognitive outcomes (Halpern, 2004, 2013).

3.2. Prenatal exposures

Exposure to certain molecules or micro-organisms during pregnancy may have detrimental effects on the child's cognitive development. For instance, children of epileptic mothers who have been exposed in utero to valproic acid, an antiepileptic drug, have on average lower general cognitive abilities (Banach, Boskovic, Einarson, & Koren, 2010) and are at higher risks of developing ASD (Christensen et al., 2013) and various neurodevelopmental disorders (Blotière et al., 2020). Prenatal alcohol exposure also has negative consequences for cognitive development: meta-analytic studies suggest that moderate alcohol intake during pregnancy (3-6 drinks per week) is negatively associated with child behaviour outcomes ($d = -0.15$; Flak et al., 2014), while binge drinking (more than 4 drinks per occasion) and heavy drinking (more than 2 drinks per day) are negatively associated with general cognitive development ($d = -0.13$ for binge drinking, Flak et al., 2014; and $d = -0.53$ for heavy drinking, Testa, 2003). Various infections (e.g. toxoplasmosis, cytomegalovirus, zika, etc.) of the pregnant mother additionally have adverse effects on the foetus' cognitive development (Brecht et al., 2015; Sever et al., 1988; Valdes et al., 2019). Note that these are all raw associations, which may not necessarily reflect causal influences, as discussed in the next section on confounding factors.

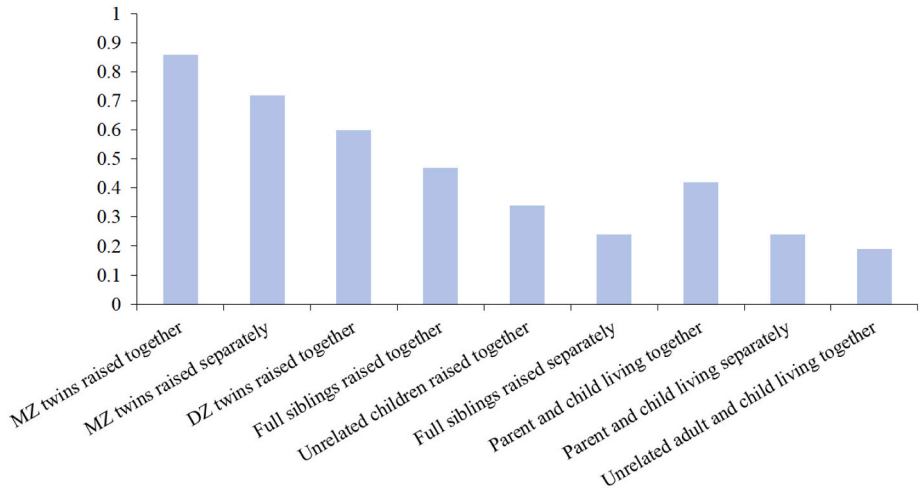

Fig. 3. Intrapair correlations of IQ scores between individuals with various degrees of genetic and environmental relatedness. Fully unrelated individuals have an intrapair correlation of 0. MZ: monozygotic. DZ: dizygotic. Adapted from Loehlin (1989), based on data from Bouchard and McGue (1981).

3.3. Birth factors

Cognitive development is also associated with several birth characteristics, such as gestational age, birth weight, and the newborn's clinical status (quantified by the Apgar score). Preterm children and those with low birth weights experience a variety of cognitive deficiencies including linguistic, sensory, and motor difficulties, compared to term-born children with normal birth weights (Aarnoudse-Moens, Weisglas-Kuperus, van Goudoever, & Oosterlaan, 2009; Barre, Morgan, Doyle, & Anderson, 2011; Beauregard, Drews-Botsch, Sales, Flanders, & Kramer, 2018; Courchia, Berkovits, & Bauer, 2019; de Kieviet, Piek, Aarnoudse-Moens, & Oosterlaan, 2009; Nguyen et al., 2018; Twilhaar et al., 2018). Cognitive losses range from 0.2 to 0.3 SD for preterm (<37 weeks) and early term children (37–38 weeks; Beauregard et al., 2018) and up to 0.86 SD for very preterm children (<32 weeks) and/or with very low birth weight (<1500 g) (Twilhaar et al., 2018). Lastly, a low Apgar score (<7) is additionally associated with lower cognitive abilities (Ehrenstein, 2009; Razaz et al., 2016) and teacher-rated hyperactivity and inattention (Guhn, Emerson, Mahdaviani, & Gadermann, 2020; Razaz et al., 2016).

3.4. Parental and social factors

A higher parental socio-economic status (SES, aggregating educational attainment and income) is positively associated with a wide range of cognitive outcomes, such as general cognitive ability (+0.7 IQ point per additional year of education, Eriksen et al., 2013), executive functions ($r = 0.22$; Lawson, Hook, & Farah, 2018), behavioural outcomes (Hedge's $g = 0.06$ for externalising problems, and 0.08 for internalising problems; Letourneau, Duffett-Leger, Levac, Watson, & Young-Morris, 2013), and language development (Hedge's $g = 0.35$; Letourneau et al., 2013; $r = 0.31$, Scaff & Cristia, in preparation). Many factors which arise from SES are also associated with cognitive development. For instance, breastfeeding is associated with higher offspring's general cognitive abilities (Horta, Loret de Mola, & Victora, 2015: 3.44 more IQ points) and with lower risks of developing certain behavioural problems such as ADHD symptoms (Tseng et al., 2019: Odds ratio for non-breastfeeding = 3.71). Similarly, malnutrition is negatively associated with the child's development (Alam et al., 2020: −3.53 IQ points for early-onset persistent stunting). Adult language input is also an important predictor of the child's language development ($r = 0.29$; Wang, Williams, Dilley, & Houston, 2020) and both advanced maternal and paternal age (older than 40) are associated with a greater risk of developing ASD (Reichenberg et al., 2006; Sandin et al., 2017). Lastly, schooling is an obvious contributor to children's cognitive abilities, with an increase of the equivalent of 3-4 IQ points on average per year of education (Ritchie & Tucker-Drob, 2018).

3.5. Genes

The collective effect of genes on cognition has been investigated in heritability studies, which determine the share of variance in phenotypes that corresponds to genetic variance. Historically, such studies relied on the comparison between mono- and di-zygotic twins (Bartels, Rietveld, Van Baal, & Boomsma, 2002; Bishop, North, & Donlan, 1995; de Zeeuw, de Geus, & Boomsma, 2015; Polderman et al., 2015), but also exploited other situations such as adoption at birth and more generally, trait correlations between relatives of varying genetic and environmental similarity (Bouchard & McGue, 1981; Loehlin, 1989; Plomin, Fulker, Corley, & DeFries, 1997) (see Fig. 3 for an illustration). Across all cognitive traits, heritability has typically been found to lie between 20 and 80% (Plomin, Owen, & McGuffin, 1994; Polderman et al., 2015), making the genome the single most important factor in predicting cognitive development (although each individual genetic variant only has a minute effect on cognition).

Twin studies are not confined to estimating the heritability of simple measures. They can also estimate the genetic contribution to developmental trajectories and to the covariance between cognitive functions. For instance, up to 90% of the stability of general cognitive ability may be accounted for by genetic factors, while the shared environment is more responsible for fluctuations (Petrill et al., 2004; Plomin et al., 1994; Rimfeld et al., 2018). Furthermore, when two cognitive functions are correlated, it is possible to analyse to what extent this covariance is due to the same genes affecting both functions. For instance, it has been shown that about half of the correlation between reading and mathematics ability is due to shared genetic factors (Davis et al., 2014).

Since the beginning of the 21st century, new molecular genetic methods have complemented twin and family studies. Genome-wide Complex Trait Association (GCTA) studies use whole-genome analysis to estimate the proportion of phenotypic variance that can be explained by genetic variance, which is measured across dozens or hundreds of thousands of Single-Nucleotide Polymorphisms (SNPs) (Yang, Lee, Goddard, & Visscher, 2011). For reasons that are well understood, GCTA studies systematically show lower heritability estimates (20-30%) than twin studies (Trzaskowski et al., 2013), but do confirm the substantial influence of genes on most cognitive traits (Davies et al., 2011; Hill et al., 2018; Sniekers et al., 2017). Such genome-wide association results are now being used to compute polygenic scores, which cumulate the

predictive power of thousands of SNPs that are most strongly associated with the phenotype of interest. Current polygenic scores may account for up to 10% of the variance in cognitive performance (Lee et al., 2018).

Beyond documenting the contribution of genes to cognitive development, another important contribution of such genetic studies is to enrich our knowledge of the effects of environmental factors, by allowing one to consider interactions between genetic and environmental factors, and by providing a way to adjust for the confounding effects of genetic factors on environmental ones, as will be explained in the next sections.

4. The importance of controlling for confounding variables

As one can imagine, many of the various predictors of cognitive development are correlated with each other: a textbook case where correlation does not entail causation. It is therefore often necessary to measure as many factors as possible, and adjust them on one another to identify the specific contribution of each one. For instance, family income, quality of medical care, breastfeeding and parent/child interactions are all positively correlated with each other and with cognitive development. Failing to measure and control for any of these factors may lead to overestimating the influence of other factors or misattributing a causal role to them. We develop and illustrate the two main types of such confounding: confounding due to omitted environmental variables, and due to genetic factors (i.e. gene-environment correlations).

4.1. Environmental confounders

A confounder is a factor that is related to both the exposure and the outcome variables and that is thought not to lie on the causal pathway between them. When certain confounding factors are not properly taken into account, this leads to over-estimating or underestimating true associations (see Table 1 for some illustrations). Additionally, potential measurement unreliability in these confounding factors may largely affect estimates and lead to incorrect conclusions (Westfall & Yarkoni, 2016). Some environmental factors were once thought to have an influence on cognitive development, and their effects have vanished once proper confounding factors were adjusted.

An example of a confounded relationship is that between breastfeeding and maternal intelligence. Breastfeeding has been purported to have a positive influence on cognitive development due to the particular composition of maternal milk. However, when controlling for maternal IQ (mothers with higher IQ more frequently breastfeed their child), the positive influence of breastfeeding on the child's intelligence falls from 3.44 IQ points to 2.62 (Horta et al., 2015). Similarly, after matching breastfed children with non-breastfed children on a range of individual and parental characteristics, the difference in IQ considerably shrinks and becomes non-significant (Bernard et al., 2017; Der, Batty, & Deary, 2006). These results suggest that a large part of the association between breastfeeding and the child's cognitive development may stem from higher parental intelligence and its associated effects, rather than nutritional benefits.

In a similar fashion, maternal smoking during pregnancy has long been believed to be associated with decreased cognitive outcomes and with an increased risk of ADHD. However, large scale studies that controlled for a wide range of factors thought to be correlated both with maternal smoking and with children's cognitive development, such as maternal education, found no evidence for such an association (Batty et al., 2006; Gilman, Gardener, & Buka, 2008). Therefore, maternal smoking in itself does not seem to be detrimental to the infant's cognitive development (although it is to the mother's health).

4.2. Gene-environment correlations

While it is commonplace in social science and epidemiological research to measure and control for a maximum of confounding variables, this approach is often restricted to environmental factors. For example, a study evaluating the effect of the type of school on cognitive development and educational attainment will control for a child's socioeconomic status, which may differ between schools. However, if the genetic predispositions of children also vary across schools, genes should similarly affect the interpretation of differences in outcomes. Yet, these genetic contributions are often ignored.

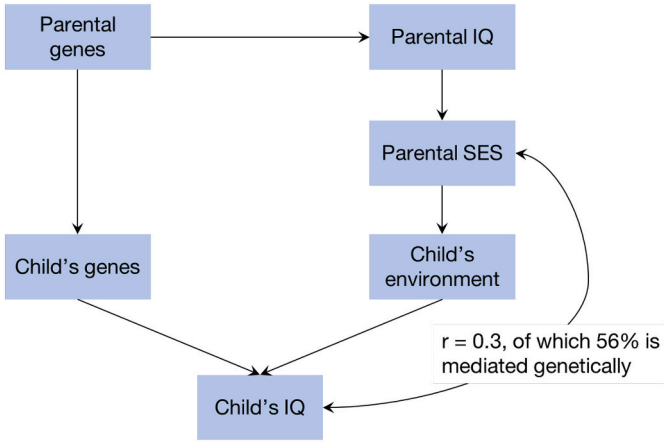

Fig. 4. Illustration of how the effect of parental SES on child IQ may be partly genetically mediated. Phenotypic correlation of 0.3 and proportion of 56% of covariance from Krapohl and Plomin (2016) (IQ measured at age 12).

Across epidemiological studies, genetic factors are often intertwined with environmental factors. This has been known for a long time, with the paradoxical discovery of the heritability of environmental factors, also known as "the nature of nurture" (Plomin & Bergeman, 1991). The very exposure to life events (accidents and trauma) - an unambiguous environmental factor - is more concordant between monozygotic than between dizygotic twins, suggesting that certain environmental factors are to some extent genetically influenced. This can be explained by genetic influences on cognitive traits such as risk-taking or impulse control, or less directly, by genetic influences on intelligence and educational achievement, which in turn affect the likelihood of knowing, understanding and following basic safety recommendations. Gene-environment correlations can take different forms (Pingault et al., 2018; Rutter, 2007). They can be *passive*, such as when parents with good language skills both genetically transmit their predispositions to their children and provide a richer linguistic environment for these children to grow up in. They can be *evocative*, such as when children with good language learning predispositions talk more and better, and therefore elicit richer language input in return. And finally, they can be *active*, such as when children with good language learning predispositions actively seek peers with good verbal skills, books, and challenging linguistic environments that further improve their language skills. This positive correlation between genotype and environment contributes to explaining why cognitive abilities are both highly heritable and largely influenced by the environment, which has long been perceived as a paradox (Dickens & Flynn, 2001). In all cases, studies measuring the association between the linguistic environment and children's language abilities may make incorrect (or inflated) causal inferences if they omit genetic transmission.

Confounding genetic factors even affect the mother of all environmental factors: SES (Trzaskowski et al., 2014). Any study claiming that parental SES influences children's cognitive development and/or educational attainment raises the following questions: To what extent is the correlation between SES and cognitive outcomes due to the environment provided by parents, and to what extent is it due to genes transmitted by parents? Historically, evidence for some degree of genetic transmission of SES came from studies of siblings adopted by

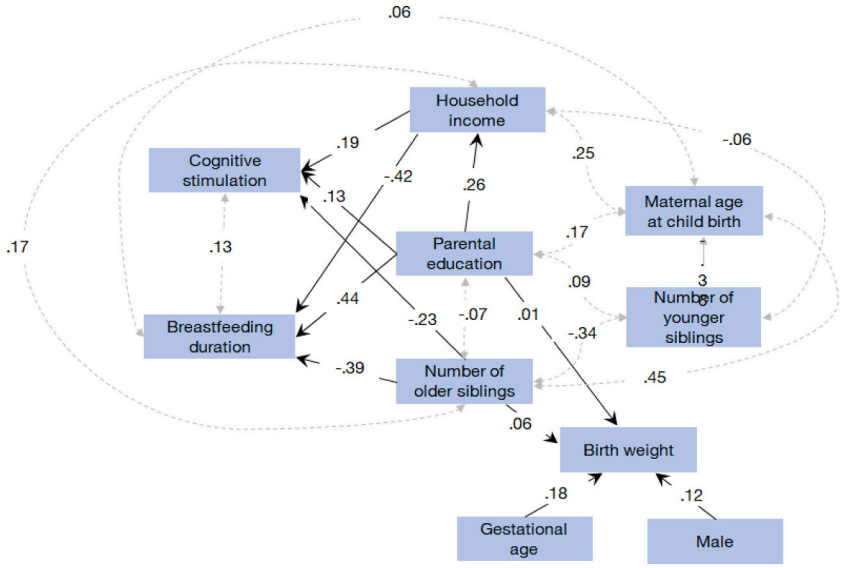

Fig. 5. Illustration of the complexity of relationships between predictors of cognitive development. Here, the relationships between multiple distal and proximal factors of cognitive development has been modelled using Structural Equation Models (SEMs). All of these factors are also simultaneous predictors in an analysis of cognitive scores (not shown), which takes into account the entire network, and allows one to estimate separately the direct effects of each factor and its indirect effects (mediated by other factors). Solid lines and arrows reflect postulated directional relationships. Dashed lines and double-headed arrows reflect correlations, potentially reflecting mutual influence, or unidentified factors. From Peyre et al. (2016).

different families, showing that adult social class was more highly correlated between siblings reared apart, than between genetically unrelated children raised together (Teasdale & Owen, 1981). Nowadays, molecular genetics offers a way to directly measure and control for genetic influences, using previously mentioned GCTA and polygenic score analyses. Some polygenic scores are significantly correlated with environmental factors known to have an effect on cognitive development. These studies suggest that at least 50% of the covariance between parental SES and child IQ or educational achievement may be explained by shared genetic factors (Krapohl & Plomin, 2016; Trzaskowski et al., 2014) (see Fig. 4).

Similarly, genetic factors seem to confound associations between cognitive/educational outcomes and environmental factors such as breastfeeding duration, smoking during pregnancy, and even whether the TV is usually on and spanking or slapping (Krapohl et al., 2017).

It has long-been recognised that studies that do not use "genetically-informed designs" (usually involving relatives with different degrees of genetic relatedness) overestimate or sometimes spuriously attribute the effects of certain environmental factors on cognition (Lemery & Goldsmith, 1999; Liu & Neiderhiser, 2017). Yet genetic confounding, often unrecognised, continues to plague countless social science and cognitive studies on the environmental factors related to cognitive development. It is to be hoped that this will progressively improve, thanks to molecular genetics, which now offers new ways to control for genetic transmission that do not require the inclusion of genetic relatives.

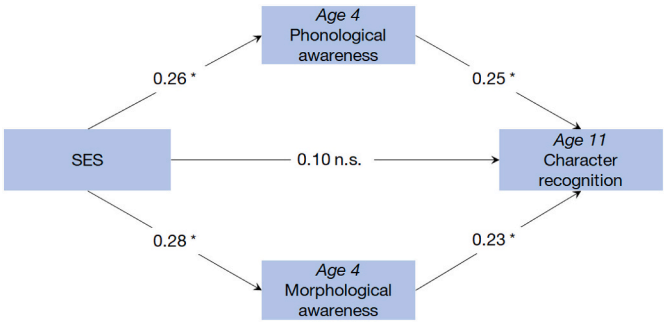

Fig. 6. Mediation of the effect of SES on reading skills through early phonological and morphological awareness in 262 Chinese students. Adapted from Su et al. (2017).

5. From simple associations to complex relationships

More often than not, causal factors and cognitive outcomes may be related in more complex ways than are currently portrayed. In addition, cognitive skills are correlated with each other, due to the dynamic nature of cognitive development, also known as the idea that "skills beget skills" (Cunha & Heckman, 2007; Van Der Maas et al., 2006). To better understand the complex mechanisms by which a predictor influences an outcome, more sophisticated statistical models can be used, as they provide answers to the following inquiries: Is the effect mediated by a third factor? Does the effect depend on particular circumstances? To what extent do two factors exert reciprocal influences on each other?

5.1. Mediation effects

When considering multiple, potentially confounded factors that influence an outcome, it may be important to consider the distinction between distal and proximal factors. Take for instance the effect of parental education on children's language development (r = 0.2-0.3; Letourneau et al., 2013; Scaff, C., & Cristia, A. (in preparation)). If you enter another related factor as a covariate in the model, such as how often parents talk to their children, it will show an association with both child language and parental education and will therefore diminish the estimate of the effect of parental education on child language (which may even become non-significant (see Newman, Rowe, & Bernstein Ratner, 2016). But does this mean that parental education has no genuine causal effect on child language? No, it doesn't. This example instead illustrates that correlated predictors are not necessarily confounded: they may themselves show a causal relationship. Parental education may correspond to a distal factor, in the sense that it is an abstract factor very far upstream of the outcome, and its effect on the outcome would be indirect, as it is mediated by proximal factors. In the present case, parental education has an effect on child language that is partly confounded by genetic transmission (see previous section) and by environmental factors, mediated by more proximal factors such as parental speech, parental practices, child health care, investment into child education, etc. (see Fig. 5). Environmental factors can therefore form a complex network of causal influences, mutual relationships, and correlations of unknown or complex origin, which can be modelled using SEMs. Directed acyclic graphs (DAGs) are a useful tool to visualise causal relationships between variables and distinguish the confounding variables that need to be controlled for (those which impact both the predictor and the outcome of interest) from those which should not

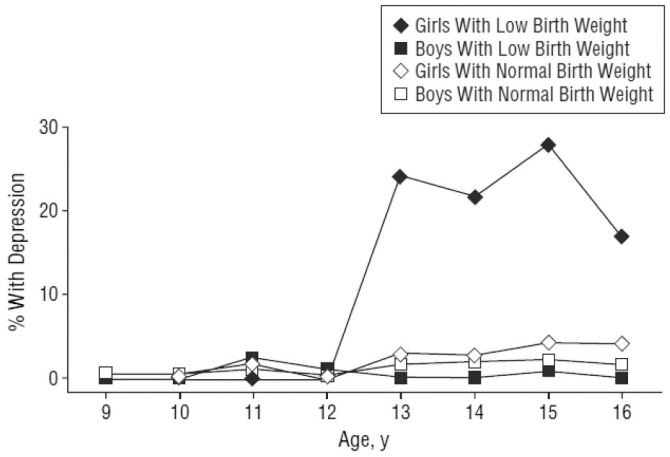

Fig. 7. Moderation of the effect of low birth weight on 3-month depression by sex. From Costello et al. (2007).

(mediators and colliders). A correctly specified DAG can thus enable the identification of causal effects of interest (Rohrer, 2018).

The binary distinction between proximal and distal factors nonetheless remains artificial and insufficient. There could be a cascade of progressively more proximal factors influencing each other between distal factors and the outcome. Beyond specific direct environmental factors, another common category of proximal factors to consider consists of early measures of cognitive skills. For instance, when one considers the relationship between familial factors (home literacy environment) and reading acquisition, they may be partly mediated by early cognitive skills, such as language or visuo-spatial ability. In a study of 262 Chinese children followed from age 3 to 11, we found that the distal effect of parental SES on Grade 5 word reading skill (raw association: beta = 0.22, R^2 = 0.047) was entirely mediated by children's phonological and morphological awareness at 4 years old (Su et al., 2017) (Fig. 6).

Similarly, in another study, we found that the influence of parental education on French children's arithmetic problem-solving skills at 11 years old (raw association: 0.2) was entirely mediated by their language and visuo-spatial skills at 5.5 years old. However, the effect of sex on problem solving skills was not mediated by such early cognitive skills (direct effect: beta = 0.43) (Guez, Peyre, & Ramus, submitted). This suggests that sex differences in arithmetic do not emerge from early cognitive skills, but arise only once formal schooling begins, consistent with previous observations (Robinson & Lubienski, 2011).

Statistical models allow to estimate the extent to which the distal factor is mediated by the proximal factor. Initial statistical approaches that relied on a three-step regression process (Baron & Kenny, 1986) are now being superseded by more complex SEMs, which outperform them (Iacobucci, Saldanha, & Deng, 2007) and can include multiple mediators in the same model (Preacher & Hayes, 2008). Lastly, causal mediation analysis, a method based on counterfactual reasoning, provides a rigorous framework for estimating such relationships (VanderWeele, 2016).

5.2. Moderation/interaction effects

When exposed to similar environmental factors, distinct individuals may react differently. This may be due to differences in terms of genes, sex, or developmental history conferring different vulnerability or potential. This phenomenon is known as the moderation of the effect of one factor by another, or as an interaction between two factors; i.e., when the effect of one factor depends on the presence or the value of another (see Fig. 7 for an illustration). For instance, the negative influence of prenatal alcohol exposure on a child's executive functions is greater when the mother is older (Burden, Jacobson, Sokol, & Jacobson, 2005; Chiodo et al., 2010). One possible interpretation of this effect is that among mothers who consumed alcohol during pregnancy, older women, who have been drinking for longer, may have a greater tolerance for alcohol and suffer more from liver dysfunction, which can increase alcohol levels in the foetus. Another suggestion is that the ratio of body fat to water increases with maternal age, such that older mothers are more likely to have higher concentrations of alcohol in their blood when consuming alcohol over a longer period of time.

Beyond environmental factors interacting with each other, the child's sex seems to moderate the effects of certain environmental factors on cognitive development. For instance, low birth weight is a long-term risk factor for depression in adolescent girls (see Fig. 7), but not in boys, and only in conjunction with other childhood risk factors (Costello, Worthman, Erkanli, & Angold, 2007). According to the authors, this result suggests that low birth weight is not simply another risk factor for depression, but a marker for poor intrauterine conditions for growth and development. The foetus would be forced to adapt to the deficient environment in order to maximize its chances for survival during gestation, but would do so at a cost: Its response to future stressors would be less adapted. The effects of low birth weight thus appear to be latent until the individual faces adversity. As another example, the well-known male advantage in spatial skills has been found to emerge only at middle/high SES, but not at low SES, constraining the potential explanations for this sex difference (Levine, Vasilyeva, Lourenco, Newcombe, & Huttenlocher, 2005). A possible interpretation for this finding is that boys have more access to toys and games that promote spatial skills in higher- than in low-SES households.

Genetic makeup also interacts with environmental factors. Understanding such interactions may shed additional light on well-established environmental effects. For instance, it has long been known that childhood maltreatment is associated with conduct disorder and with later antisocial personality behaviour. This may be interpreted as a form of learning by imitation. However, not all maltreated children become maltreating parents. In a landmark study, Caspi et al. (2002) showed that a particular polymorphism of the gene coding for monoamine oxydase A (MAO-A) interacted with childhood maltreatment. Carriers of the low protein expression variant were more at risk of developing conduct disorder only if they were maltreated; while the effect of maltreatment on the likelihood of developing conduct disorder was limited in carriers of the high expression variant. This result suggests that the learning-by-imitation interpretation is at best incomplete. It supports the idea that genetics may contribute to why some individuals are very vulnerable to maltreatment, while others are more resilient.

It should be noted that, while this particular result seems to have stood the test of time so far (Byrd & Manuck, 2014), most gene x environment interaction studies based on a single candidate gene have proved to be false positives (e.g., Border et al., 2019; Caspi et al., 2003). It is now understood that the effect of single genes are generally too small to yield interactions of meaningful size. The future thus lies with studies harnessing the collective effects of genetic variations with polygenic scores. The greater predictive power of polygenic scores provides more accurate estimations of the extent to which the effects of an environmental factor on a phenotype depend on genetic predispositions (e.g., Domingue, Trejo, Armstrong-Carter, & Tucker-Drob, 2020; Harden, 2021). For instance, the polygenic score for educational achievement has been found to interact with family SES in predicting the child's educational achievement (Papageorge & Thom, 2020). Polygenic scores can also be used to investigate to what extent an educational/psychological intervention or a policy change differentially affects people with different genetic predispositions (e.g., Barcellos, Carvalho, & Turley, 2018; Kuo et al., 2019).

Thus, given individual differences in the response to environmental factors or to experimental interventions, it is important to consider genetic factors as one possible source of variability.

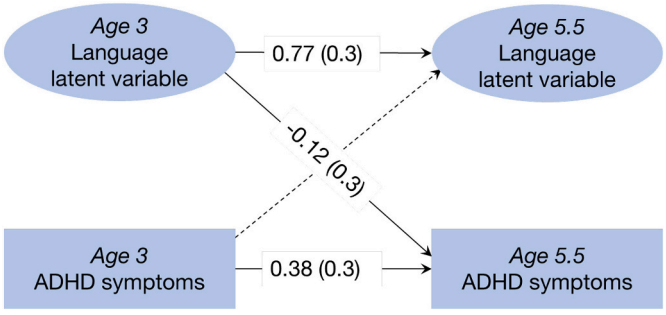

Fig. 8. Bidirectional relationships between language skills and inattention/hyperactivity symptoms. Numbers are standardised regression coefficients, with standard deviations in parentheses. Interpretation: An increase of language ability at 3 by 1 standard deviation decreases ADHD symptoms at 5.5 by 0.12 standard deviations. Adapted from Peyre, Galera, et al. (2016).

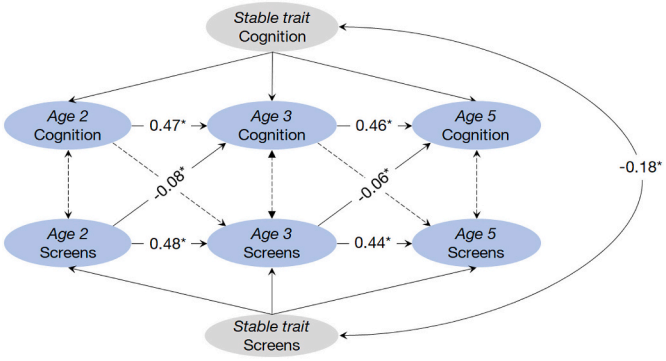

Fig. 9. Bidirectional relationships between cognitive abilities and exposure to screens. The model used disentangles within-individual variance from stable between-individual variance, thus estimating the influence of within-individual changes in screen exposure on cognition. Single-headed arrows reflect regression coefficients, double-headed arrows reflect correlations. Numbers are standardised regression coefficients or correlation coefficients. Interpretation: An increase of 1 standard deviation in daily screen exposure at age 2 is associated with a decrease of 0.08 standard deviations in general cognitive ability at age 3, after adjusting for the stable covariance between screen exposure and cognitive ability (whose correlation is $r = -0.18$). Dashed lines reflect non-significant coefficients and correlations. Adapted from Madigan et al. (2019).

5.3. Reciprocal relationships

When two variables that evolve in time are correlated, it is often hard to determine which one causes the other, or, if they both influence each other, which one has the larger effect (a phenomenon illustrated in Figs. 8-9). For example, language abilities and behavioural problems are two cognitive outcomes that are correlated and for which the direction of the relationship is not obvious. It is possible that early behavioural problems impair language development, but also that early language difficulties prevent children from properly regulating their behaviour. It is also possible that there is no causal link between the two outcomes, but that both are caused by a third, potentially unobserved factor, which creates a correlation between them. Cross-lagged panel models are a type of SEM that can help disentangle such reciprocal relationships. In these models, the two variables are measured at different time points and are simultaneously regressed on past values of themselves and on past values of each other. When measures are available at more than two time points, more sophisticated models can be used and can distinguish between-person from within-person variance by, for instance, including random intercepts (Hamaker, Kuiper, & Grasman, 2015). These models are able to estimate to what extent a variable A affects the within-person change in variable B, and vice-versa. Cross-lagged panel models examining the relationships between language abilities and ADHD symptoms have thus shown that better early language skills prevent the development of ADHD symptoms, but that early ADHD symptoms do not impair language acquisition (Petersen et al., 2013; Peyre et al., 2016) (see Fig. 8).

Similar methods can be applied when exposure to a risk factor varies with time and its relationships with cognitive outcomes are unclear. For example, exposure to screens is negatively correlated with children's cognitive abilities (Madigan, McArthur, Anhorn, Eirich, & Christakis, 2020; Walsh et al., 2018). However, we do not know a priori if this correlation conceals a causal relationship from screen time to cognitive abilities (e.g., if watching TV or playing a video game deters children from doing activities that are more beneficial to cognitive development), a causal relationship from cognitive abilities to screen time (e.g., if children with lower cognitive abilities are more attracted to screens), or if it is simply due to external factors (e.g., children from lower socio-economic backgrounds could both have lower cognitive abilities and be more exposed to screens). Going beyond simple associations by using a random intercept cross-lagged panel model showed that there is a small negative link from screen time to general cognitive development, but not the reverse (Madigan et al., 2019) (see Fig. 9). However, this presumed causal effect is much smaller (−0.5 to −0.7 IQ points per daily hour of exposure) than the raw association.

6. Discussion

What makes each of us a unique human being? This is the broad question that the epidemiological approach to cognitive development tackles, enabling us to understand to what extent cognitive abilities are variable, and which factors explain this variability. Through the statistical analysis of large samples gathering rich sets of cognitive, environmental, and increasingly, genetic data, epidemiological studies inform cognitive science on fundamental questions related to the variability of cognitive development across humans. Throughout this review, we aimed to provide an overview of such contributions, including:

- Modelling how cognitive abilities change throughout life, and how these trajectories may differ across individuals;
- Uncovering the main associations between genetic and environmental factors and cognitive abilities;
- Controlling for a multitude of confounding factors, including genetic ones;
- Disentangling the complex relationships between cognitive, environmental, and genetic factors, such as: the mediation of certain factors by others, the interactions between these factors, and their dynamic and reciprocal relationships.

These contributions illustrate that cognitive development is a dynamic and complex process, shaped by the influences and synergies of a large array of environmental, biological, and genetic factors.

While these advances are substantial, epidemiological studies are subject to some limitations. First of all, the vast majority of studies are based on individuals from a particular subset of social, political, demographic and cultural contexts, namely WEIRD populations (Western, Educated, and from Industrialized, Rich, and Democratic countries; Henrich, Heine, & Norenzayan, 2010). On top of this, most findings are from English-speaking countries. Therefore, while these studies can illuminate our understanding of the mechanisms underlying cognitive development in the particular populations that are sampled, it would be far-fetched to assume similar results would be found in other populations. More studies from non-WEIRD populations are needed in order to assess the generalisability of findings.

Similarly, estimates of the proportion of variance explained by a given factor is specific to each population, as it depends on the set of environmental factors encountered and their variance. For instance, the relative (although not the absolute) importance of genetic factors in

Box 2
Inferring causality

How can we determine whether the relationship between a factor and a cognitive trait is causal? Our conception of causality relies on counterfactual reasoning: "If a given individual had not been exposed to factor A, what would his/her outcome have been?". Current research has limited predictive power at the individual level, since all experimental and observational study results represent group probabilities (e.g. IQ difference between a group of children born preterm vs at term) rather than individual causal links (e.g. an individual's low IQ is caused by the fact that he/she is born preterm).

Randomized controlled trials (RCTs) provide a way to make causal inferences between a factor and a cognitive trait. However, most factors influencing cognitive development cannot (practically or ethically) be experimentally manipulated (see Table 1). Among the few exceptions are studies that have randomized maternity hospitals either to receive an intervention to promote breastfeeding or not (Kramer et al., 2008; Yang et al., 2018).

In observational studies, if the main confounding variables are properly controlled for, results from multivariate regression analyses may reflect a causal relationship between a factor and a cognitive trait. However, the validity of this inference largely depends on how confounding variables have been selected. In a meta-analysis of the studies examining the association between breastfeeding and IQ, Der et al. (2006) showed that the results of multivariate regression analyses were largely discordant because of differences in the number and nature of confounding variables that were considered.

Advanced design and analysis of observational studies have been developed to better take into account known and unknown factors that may influence cognitive development, such as:

(i) Family-based design, which is an approach that enables researchers to account for genetic confounding and unknown familial factors. For example, several epidemiological studies reported an association between maternal tobacco consumption during pregnancy and attention deficit and hyperactivity disorder (ADHD). Yet, comparisons of siblings - only one of whom was prenatally exposed to tobacco - recently demonstrated that the associations between maternal tobacco consumption during pregnancy and ADHD were largely due to familial confounding (D'Onofrio et al., 2010; Knopik et al., 2016).

(ii) Regression discontinuity design (RDD) are another type of natural experiment, in which participants are assigned to a treatment based on a continuous assignment variable with an arbitrary cutoff. Identification is possible by comparing participants barely above and below this cutoff. Gormley, Gayer, Phillips, and Dawson (2005) used this method to assess the causal effect of a pre-kindergarten program on children's cognitive abilities, taking advantage of the strict birthday eligibility criterion to be enrolled in a given year. By comparing cognitive outcomes of children born right before and after the cutoff date, the authors found significant positive effects of the program on prereading and reading skills, prewriting and spelling skills, and math reasoning and problem-solving abilities.

(iii) SEMs with longitudinal data, which allow for the inclusion of confounding factors and are particularly well suited for bi-directional relationships. For instance, the random-intercept cross-lagged panel model (Hamaker et al., 2015) simultaneously estimates the within-person associations between two variables measured at different time points, controlling for individuals' stable deviations from the group means. The model thus assesses the effect of a within-person change in a variable A on the change in variable B, and vice-versa. Using this method, Madigan et al. (2019) reported a small negative effect of screen exposure on cognitive development, but not the reverse.

(iv) Mendelian randomization, which is another technique which uses genetic variations (i.e. SNPs) as instrumental variables to investigate the causal relationships between a predictor and an outcome of interest (Davies, Holmes, & Davey Smith, 2018). In this framework, genetic variants are assumed to be related to the cognitive outcome only through the predictor. It is thus possible to assess the effect of the predictor on the outcome free of confounds, through the estimation of the effect of genetic variations on the outcome. With this approach, Bonilla et al. (2012) have reported that maternal vitamin B-12 intake may have a small effect on offspring cognitive skills.

cognitive development is expected to be much lower in countries where schooling is not universal and is of heterogeneous quality and duration (large educational variance). The same should be true for countries where a proportion of children are malnourished (large nutritional variance), considering that these sources of environmental variance in WEIRD populations are nearly null (see https://unstats.un.org/sdgs/indicators/database). We hope that future research will focus on understudied populations, thus ensuring the generalisability of current results, or improving our understanding of how these results vary according to circumstances.

A second limitation is the well-known caveat that correlation is not causation. Many epidemiological studies rely on longitudinal, observational designs, with sometimes sophisticated methods that bring researchers closer to establishing causality - but without definitive proof. Although experimental interventions, and in particular randomised controlled trials, remain the gold standard for proving causation, correlational studies are an indispensable tool in the quest for causality. Indeed, they are often the first piece of evidence in favour of a causal relationship, that will inspire and motivate future experimental/intervention studies. Furthermore, experiments or interventions are often costly and sometimes unethical or unfeasible, making observational studies the only resort. Fortunately, there are methods that allow causal interpretations from observational data, when certain assumptions are met (some of which are detailed in Box 2). These methods enable one to identify the causal effect of a predictor by assessing its effect 'everything else being equal', i.e., by establishing comparability between individuals, and ensuring that differences in the outcome of interest are not due to other observed or unobserved characteristics. This is done through the inclusion of the necessary confounders in the model when they have been measured, or through quasi-experimental 'as good as random' methods such as regression-in-discontinuity designs or instrumental variables, when available. Yet, in spite of these possibilities, researchers working with observational data are often reluctant to explicitly formulate their research questions in causal terms and conduct causal analyses – although they implicitly interpret their findings as causal. As Grosz, Rohrer, and Thoemmes (2020) point out, such a taboo hinders progress in observational psychology research and limits the relevance of the field to policymaking. It is therefore desirable that more researchers clearly articulate causal research questions, hypotheses, identification strategy and their underlying assumptions.

Another limitation of the epidemiological approach, from the perspective of developmental psychology, lies in the nature of the investigated measures. Available cohort databases often include general cognitive measures (standardised tests and scales) that may seem

Table 2
Large cohort studies in the general population containing data on cognitive development.

Cohort name	Country	N	Age range	Cognitive phenotypes
The Longitudinal Study of Australian Children (LSAC)	Australia	10,000	1-20 years	IQ, language, psychomotor development, socio-emotional and behavioural outcomes, academic skills, temperament
Etiology, Risk Factors and Interactions of Enteric Infections and Malnutrition and the Consequences for Child Health and Development (MAL-ED)	Bangladesh, Brazil, India, Nepal, Peru, Pakistan, South Africa, Tanzania	2100	0-5 years	IQ battery, psychomotor development, temperament, language
National Longitudinal Survey of Children and Youth (NLSCY)	Canada	26,000	0-23 years	socio-emotional and behavioural outcomes, temperament, psychomotor development, academic skills, language
Québec Longitudinal Study of Child Development (QLSCD)	Canada	3000	5 months-25 years	IQ, psychomotor development, socio-emotional and behavioural outcomes, language, academic skills
Young Lives Study	Ethiopia, India, Peru, Vietnam	12,000	1-15 years	socio-emotional and behavioural outcomes, language, fluid intelligence, academic skills
Eden	France	2000	0-11.5 years	IQ, language, psychomotor development, academic skills, socio-emotional and behavioural outcomes
ELFE	France	18,000	0-9 years	Language, psychomotor development, socio-emotional and behavioural outcomes
National Educational Panel Study	Germany	60,000	Several cohorts including newborns to adults	IQ, academic skills, language, executive functions
Growing up in Ireland	Ireland	20,000	9 months-10 years	IQ, motor development, socio-emotional and behavioural outcomes, temperament, academic skills
Norwegian Mother, Father and Child Cohort Study (MoBa)	Norway	100,000	0-14 years	Language, socio-emotional and behavioural outcomes, psychomotor development, temperament
Avon Longitudinal Study of Parents and Children (ALSPAC)	UK	14,000	0-13 years	IQ, social cognition, language, psychomotor development, socio-emotional and behavioural outcomes
Millenium Cohort Study	UK	19,000	9 months-17 years	IQ, language, motor skills, visuo-spatial skills, academic skills, socio-emotional and behavioural outcomes, risk-taking, academic skills, theory of mind
Twins Early Development Study (TEDS)	UK	15,000	1-21 years	IQ, language, academic skills, socio-emotional and behavioural outcomes
Born in Bradford	UK	13,500	0-7 years	academic skills, language, sensorimotor control
Early Childhood Longitudinal Study (ECLS)	USA	14,000	Several cohorts including newborns to 13 years	IQ, language, academic skills, motor skills, socio-emotional and behavioural outcomes, executive functions
Adolescent Brain Cognitive Development Study (ABCD)	USA	12,000	9-12 years	Language, socio-emotional and behavioural outcomes, executive functions, IQ, task-based fMRI (e.g. Monetary Incentive Delay Task)

Note: This list is not exhaustive.

imprecise to developmentalists used to experimental settings. In cohorts, measures are selected for their good psychometric properties (reliability, validity) but also based on practical considerations (e.g. limited number of items due to limited testing time). Indeed, epidemiological studies, in particular longitudinal ones, bring together several research objectives and are not aimed solely at examining cognitive development. The future involvement of developmentalists in the design of cohorts is essential to collect the cognitive measures that will allow them to better address questions that they find of greater theoretical interest.

Lastly, the replicability crisis may affect cognitive epidemiology like other research areas in epidemiology and psychology (Ioannidis, Tarone, & McLaughlin, 2011; Open Science Collaboration, 2015). Analyses of secondary data such as those we presented should be conducted with

care, particularly to decrease the risk of false positive results. Several recommendations have been made to increase the validity of studies using pre-existing data in epidemiological studies (Weston, Ritchie, Rohrer, & Przybylski, 2019). For instance, preregistering the research questions and analyses before analysing the data can help prevent exploratory analyses, postdiction, and p-hacking (Nosek, Ebersole, DeHaven, & Mellor, 2018).

In spite of these limitations, cognitive epidemiology provides fundamental insights on cognitive development and its underlying mechanisms that extend beyond the individual differences perspective. While traditional experimental studies investigate causality by manipulating factors of interest, they often do so at the cost of ecological validity: the intensity, frequency, and duration of exposure of these factors and the overall context of the experiments may not reflect real-life situations, thereby hindering generalisation. Observational studies, capturing natural variations of the same factors (when this is possible), inevitably observe much smaller effect sizes, and generally have greater difficulty inferring causation. Nonetheless, they complement experimental studies with their ecological setting. Consequently, theoretical progress in developmental psychology requires converging evidence from both approaches.

Finally, results from cognitive epidemiological studies can offer practical implications for clinicians, and often form the basis for recommendations in public health and education. Think, for example, of research on the detrimental effects of heavy maternal alcohol consumption during pregnancy on infant cognition, or on the very small impact of screen exposure on children's cognitive abilities (despite all the talk about it). To conclude, we hope that this review will trigger the interest of developmentalists, encourage the use of the abundant data available (see Table 2) – particularly adapted to research conditions in the current COVID-19 pandemic, foster fruitful collaborations, and generate exciting future research.

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