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How Can Twin Studies Tell Us About How the Environment Influences Behaviour?

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Twin studies have been a significant method of quantitative genetics and behavioural genetics, which provides an important theoretical basis (i.e., heritability) for educational neuroscience. From a viewpoint of behavioural genetics and human development, the present review will introduce a theory and a methodology of twin studies, identify how the environment in human society make an impact on behaviour as well as on genetic expression, and discuss an educational implication of genetic-environmental influence on children's experience in terms of the potential for an individual to develop in one way or another.

Keywords：twin study, heritability, gene, behaviour, environment

I. Introduction

Twin studies have been one of the major raison d'etre of human behavioural genetics [1) 2) 3) 4)] . They represented a number of scientific attempts to synthesize two different strands of human inheritances: genetic (molecular genetics) and behavioural (quantitative genetics) [5)] .

Recent computational genomics postulated that genes and behaviour are both directed to phenotypic ambiences shared inside and outside of our bodies: the genes create their own ambient pathways in our body, and bodies create behaviours adapted to a variety of environments. Despite the twofold complexity of such a hypothesis, behaviour is socially diverse and "dynamic and changes in response to the environment" [6)] , whereas genes are not so in the same level. Thus, questions are yet to be answered: did genes make us behave as we do? Or is it just a matter of environment? The twin study is a cornerstone for answering those questions.

II. Significance of Twin Studies

Science entails hypotheses and test cycles [7)]. In this respect, twin studies were highly significant for their power to generate falsifiable hypotheses particularly in earlier times when we understood less about genetic mechanisms. Plomin, Owen, and McGuffin (1994) [8)] predicted almost two decades ago:

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> This [quantitative genetic] research will in turn facilitate molecular genetic attempts to identify specific genes that contribute to genetic variance in complex behaviors. The confluence of quantitative genetics and molecular genetics will be synergistic for the elucidation of complex human behaviors.

The scientific allure of twins, therefore, was a natural embodiment of these two levels of ambience distributed in a single population: genomic ambience and "phenomic" [9] ambience (see Figure 1, hereafter, Ambience 1 and Ambience 2, respectively).

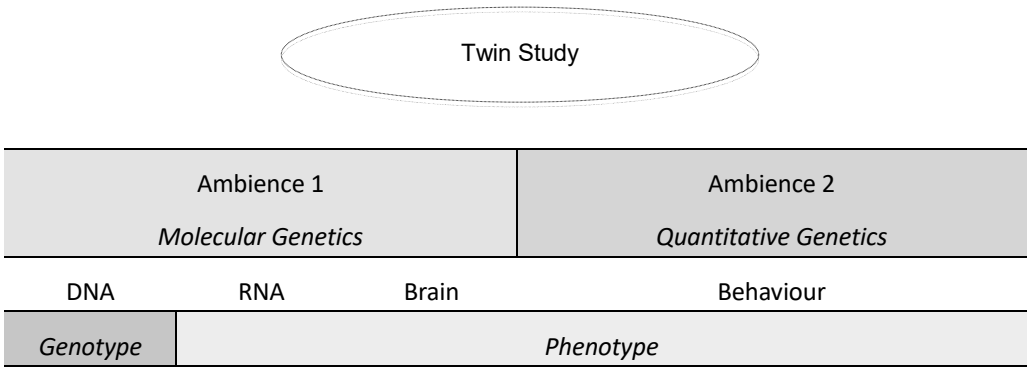

Figure 1 Schematic illustration modified from Plomin and Simpson (2013). Ambience 1 refers to behaviourally unobservable dimension of heredity sought by molecular genetics, whereas Ambience 2 refers to observable behavioural dimension sought by quantitative genetics. Genotype is an individual's combination of alleles at a particular locus, whereas phenotype is an observed characteristic of an individual that results from the combined effects of genotype, Ambience 1, and Ambience 2. DNA refers to deoxyribonucleic acid, whilst RNA refers to ribonucleic acid.

Observation of twins made it possible to calculate molecular genetic phenomena and biopsychological phenomena at once, allowing a valid inference of how genes exert their influence in the field of application such as developmental psychology or social and educational psychology [10) 11) 12)] .

The power of twin studies has also driven our deeper understanding about genomic expression. For example, since Plomin and Daniels (1987) [13)] acknowledged that the greatest social significance from genetic-behavioural research consisted primarily of twin, family, and adoption studies, twin studies directly harnessed the DNA to its own design. It is now evident that the DNA is inherited at the rate of less than 1% of the 3 billion base pairs of DNA [14)] . For years to come, twin study will keep challenging our questions with the synergy of molecular genetics and quantitative genetics.

III. Theory

According to Plomin and Daniels (1987) [15], the basic idea of twin design is simple. It is theorized by a tenet that individuals vary due to both genotypic—which takes place at the rate about one in every thousand nucleotide-bases difference among individual organisms—and phenotypic reasons that generate a diversity of childhood development even in the same family. This assumption was important because the theory used variance and correlation among relatives on normally (naturally) distributed traits to estimate the role of heredity. By computing and by comparing twin data (including first-degree relatives' or familial data), geneticists calculated the genetic effect and the environmental effect, as well as elaborated additive and non-additive effects.

Assuming that genetic and environment factors are dependent and uncorrelated (thus without covariance), the total variance of phenotypic values (V) is simply additive and expressed by the equation (1) below, where VG is genetic variance and VE is environmental variance—however, note it has also residual variance due to unsystematic errors as well as to measurement errors [16].

$$V = VG + VE \tag{1}$$

Table 1 summarizes the general validation patterns of use in twin studies. If the variation of identical (MZ) twins does not equal fraternal (DZ) twins due to the monozygotic-dizygotic genetic ratio (see (1) in Table 1), or if there is equal variation of MZ reared together to MZ reared apart (see (2) in Table 2), then there must be a genetic effect. For the environmental effect, the opposite combinations should be met; that is, if the observed data shows an equal variation of MZ to DZ, then there must be a violation of the genetic ratio, thus no genetic effect (3), and if the observed data shows an unequal variation of MZ reared together to MZ reared apart, there must be an environmental effect (4).

Table 1

Validation Logic Used in Twin Studies

Genetic validation	
(1) MZ ≠ DZ (MZ to DZ ratio ≈ 2:1)	Plomin et al., 1994; Taylor et al., 2010 [17]
(2) MZ reared together = MZ reared apart	Bouchard, Lykken, McGue, & Tellegan, 1990 [18]
Environmental validation	
(3) MZ = DZ	Plomin, Owen, & McGuffin, 1994 [19]
(4) MZ reared together ≠ MZ reared apart	Plomin, Daniels, 1987 [20]

Note. Adopted children were omitted from the table for simplicity. The source is original.

IV. Method

The linchpin of quantitative trait estimation, which was called "heritability" [21], was a rule of molecular genetics where MZ twin shares 100% of genes, whilst DZ twin shares 50%. Criterion similarity of MZ to DZ denotes that the twofold greater genetic similarity of MZ prescribes the extent to which heredity would affect a trait. That is, rMZ - rDZ = $0.5VG$ (r refers to correlation coefficient, see the equation (2) below), which was a rule of thumb incorporated by behavioural geneticists for a natural experiment into the twin studies. Plomin, Owen, and McGuffin (1994), for example, reported heritability roughly ranged from 40 to 50% for personality (neuroticism and extraversion), vocational interest, scholastic achievement, general intelligence, and some cognition (spatial reasoning and processing speed, but not memory).

$$\begin{aligned} rMZ &= VG + VE \\ rDZ &= 0.5VG + VE \\ \mathrm{r}MZ - \mathrm{r}DZ &= VG - 0.5VG + VE - VE \\ \mathrm{r}MZ - \mathrm{r}DZ &= 0.5VG \end{aligned} \tag{2}$$

Unlike later methods such as genome-wide association studies (GWAS) [22], early twin studies were based only on population-based description. Since the model was originally designed to estimate the genotypic to phenotypic “role” using Fisher’s method of means and variances in a population, this design did not say (or intend to say) anything precise about heritability in terms of a particular polymorphic trait of an individual phenotype. This meant, for example, that it was very difficult to interpret why twin, family and adoption studies all indicated a strong genetic basis for autistic spectrum disorder, since the genes responsible for the heritability (i.e., the role of DNA methylation) could not, at that time, be identified.

Twin study explained to us, however, about a quantitative estimation of distinct proportions of genetic effect and environmental effect by simply comparing the resemblance of a trait (e.g., genetic variance in IQ). Thus, it is important to know that the outcome of early twin design was a statistically converged heritability representing only the static description of, and potential estimate from the current population [23] [24].

As Anastasi (1958) [25] observed more than fifty-eight years ago, the traditional model did not explain how heredity and environmental factors specifically interplay "in the development of behavioral differences," although it could estimate the amount of "how much of variance was attributable to heredity and how much to environment." What Anastasi meant by "development" was indispensable given that there be genotype-environmental (VG x VE) interaction within Ambience 1 and Ambience 2 (or even the strong confounding factor in Ambience 1, such as phenylalanine "Phe" level in phenylketonuria [PKU]). In other words, should it become a more complicated model, eliciting genetic and environmental effects is not

very easy. This flaw was only remediable when a multivariate longitudinal design (e.g., twins early development study [TEDS]) was introduced. Trouton, Spinath, and Plomin (2002) [26], for example, investigated twins during early development across 2, 3, 4, and 7 years of age, focusing on the most common psychological topics, such as communication disorders or behaviour problems, and found increasing heritability of language and cognitive abilities. Chances were higher that longitudinal studies reveal the extent of the environmental influence on the phenotypic expression changed over time. For example, Plomin (1999) [27] pointed out an early genetic link of language impairment to diverse cognition (an example in Ambience 1), as well as the later genotypic-environmental non-verbal behaviours, such as hyperactivity, or activities presenting symptoms of perceived fussiness/callousness in autism (examples in Ambience 2).

V. Discussion

So how does the environment influence behaviour? From the behavioural point of view, twin studies gave an implication for non-heritable factors in two distinct levels of interaction between environment and gene: a biological level, which affords behaviourally unobservable ambience (DNA, RNA, and brain in Ambience 1), and an individual level, which is available as behaviourally observable ambience (Ambience 2) (see Figure 1). Interestingly enough, the impact of genetic-environmental traits, such as personality (e.g., extraversion, neuroticism), psychopathology (e.g., autism), or cognitive development (e.g., dyslexia), were larger than that of genes in medical disorders (e.g., idiopathic epilepsy, peptic ulcer, rheumatism) (Plomin, Owen, & McGuffin 1994 [28]). One explanation of this is that we are relatively more amenable to individuals' psychology and behavioural response than we are to the biology and genes' activities within individuals.

An analogous example of this is the biometric traits (e.g., height, hair, colour, etc.), which may be too diverse for us to discern, but for categorization we share as human cognition; we cognitively tend to ignore hairbreadth margins of biometric traits (e.g., light grey, pale grey, pinkish grey, etc.) because the crude classifications suffice. Plomin, Owen, and McGuffin (1994) pointed out that there is a propensity of genetic variation in psychological behaviours. Statistically speaking, this is arguable because psychological outcomes expressed by the phenotypic process in Ambience 2 are more susceptible to the natural environment (i.e., normality) than those biometric outcomes, which has been genomically processed by genetic-environmental effect within individuals (e.g., a mutating process of genes in Ambience 1, such as allele frequency).

Importantly, one of the main findings of twin, family, and adoption research [29] [30] [31] was the environmental property that accounted for most of the non-genetic (not genetically evoked) influence on

individual variation; it was not, as had been expected, that of the shared environment, but instead that of the non-shared environment. In fact, Plomin et al. (2001) [32] pointed out this "non-shared environment" as the most important discoveries from genetic research.

The non-shared environment component in the model is defined as the term of variance that heredity (*VG*) and shared family environment (*VSE* [+]) does not explain (unless interaction and error are posited). The equation (3) expresses this term as *VSE* [-] (the negative sign in the brackets means not sharing an environmental variance, whereas the positive sign means sharing the variance).

$$V = \quad VG \quad + \{VSE\,[+] + VSE\,[-]\} \tag{3}$$

However, the clarification of what constituted this non-shared environmental effect was complicated not only by the residual errors in the model, but also by the definition of what is ‘shared’: we could infer the effects of variation in children and adults. It is not specific events occurring to the individual children but the accumulated effects observed in an average event. For example, the difference between identical twins allows us to estimate how much a trait comes from non-shared environment since such a difference by definition must be generated by no other than *VSE* [-] (see (4) in Table 1) in those twins; it indicates nothing more than that.

The Minesota study of twins reared apart [33] demonstrated this effect. Bouchard and his colleagues, using more than 100 sets of twins across ten countries such as the UK, the USA, China, etc., revealed that 70% of the variance in IQ was associated with genetic variation, regardless of whether they were reared together or apart, which suggested a strong heritability of most psychological traits. However, one of their conclusions denoted the non-shared environmental effect, stating, "MZA [reared] twins are so similar in psychological traits because their identical genomes make it probable that their effective environments are similar" (p. 227). This meant there was a gene-environment interaction during development, noticeably, from non-shared environment to psychology. Should the genes interact with a variety of behaviours in non-shared environment, the educational impact on children's experience would be soon becoming stronger than the impact on the familial experience, and advancing more rapidly as well.

Parents may suppose that the shared environment (VSE [+]) might be found to have a strong effect in making children growing up in the same environment similar, due to many potential causes such as socioeconomic status, parenting, or familial ambience (e.g., a parent's hobby, job, etc.) On the contrary, genetic research findings showed that offspring resembled their parents not because they shared such an environment provided by parents or siblings, but because they shared heredity. That was the main reason why Plomin and Daniels (1987) emphasized the role of familial ambience that yielded tremendous

diversity for children in the same family during development, in addition to the research in education [34) 35)] —both family and school represent a type of Ambience 2.

VI. Summary

In sum, twin studies have demonstrated the presence of heritability, which was a significant discovery of behavioural genetics that combined molecular genetics with quantitative genetics. They offered us helpful and far-reaching validation models (including more recent bioinformatics technology) to understand the relationship of the genetic biology with individual human behaviour. Concurrently with the twin-study shortcut in behavioural science, the heritability told us that genes (the DNA sequence) primarily matter for everything in our biological environment.

On the face of it, this might give us rather mixed pictures about the environment. When genetics is combined with environment, let alone social policy, a gloomy shadow appears. Parents and teachers may resort to a dubious fatalism, which tells children's (thus our) future is helplessly decided by the genes. However, this is not what gene and twin studies have told geneticists about heritability.

From the viewpoint of behavioural genetics and human development, the expression of genes is not necessarily omnipotent (e.g., PKU) but rather, in almost all cases, offers the potential for an individual to develop in one way or another (e.g., adoption, family, school, etc.) [36) 37) 38)] . For these reasons, to conclude, we need to consider educational neuroscience that brings us a broader and steady vision that the environment including education also exerts its effect on our behaviour.

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References and Notes

1) R. Plomin: The role of inheritance in behavior. *Science, 248* (1990) 183-188.

2) R. Plomin: Genetics and general cognitive ability, *Nature, 402* (1999) C25-C29.

3) R. Plomin, & D. Daniels: Why are children in the same family so different from one another? *Behavioral and Brain Science, 10* (1987) 1-60.

4) R. Plomin, & M. A. Simpson: The future of genomics for developmentalists. *Development and Psychopathology, 25* (2013) 1263-1278.

5) R. Plomin, J. C. DeFries, G. E. McClearn, & P. McGuffin: *Behavioral genetics. (4th ed.)*. (New York, Freeman,

2001).

6) the same as the note 1), pp. 183.

7) S. T. Kuhn: *The structure of scientific revolutions (3rd ed.)*. (London, University of Chicago Press, 1996).

8) R. Plomin, M. J. Owen, & P. McGuffin: The genetic basis of complex human behaviors. *Science, 264* (1994) 1733–1739, p. 1739.

9) Plomin and Simpson (2013) used this bioinformatics term "phenome" as homologous to genome, both at the two ends of an entire pathway (see Figure 1) put for understanding individual differences.

10) the same as the note 4).

11) R. Plomin, & S. O. Walker: Genetics and educational psychology. *British Journal of Educational Psychology, 73* (2003) 3-14.

12) A. Trouton, F. M. Spinath, & R. Plomin: Twins early development study (TEDS): a multivariate, longitudinal genetic investigation of language, cognition and behavior problems in childhood. *Twin Research, 5* (2002) 444-448.

13) the same as the note 3).

14) the same as the note 4).

15) the same as the note 3).

16) S. Purcell: Statistical methods in behavioral genetics. In R. Plomin, J. C. DeFries, G. E. McClearn, & P. McGuffin, *Behavioral genetics. (4th ed.)* (New York, Freeman, 2001) pp. 327-371.

17) J. Taylor, A. D. Roehring, B. S. Hensler, C. M. Connor, & C. S. Schatschneider: Teacher quality moderates the genetic effects on early reading. *Science, 328* (2010) 512-514.

18) T. J. Bouchard, D. T. Lykken, M. McGue, N. L. Segal, & A. Tellegen: Sources of Human Psychological Differences: The Minnesota Study of Twins Reared Apart. *Science, 250* (1990) 223-228.

19) the same as the note 8).

20) the same as the note 3).

21) the same as the note 8), p. 1734.

22) Harlaar, N., Meaburn, E, L., Hayiou-Thomas, M. E., Wellcome Trust Case Control Consortium 2, Davis, O. S. P., Docherty, S., Hanscombe,K. B., Haworth, C. M. A., Price, T. S., Trzaskowski, M., Dale, P. S., Plomin, R. (2013). Genome-Wide Association Study of Receptive Language Ability of 12-Year-Olds, *Journal of Speech, Language, and Hearing Research, 57*, 96-105.

23) the same as the note 1).

24) the same as the note 2).

25) A. Anastasi: Heredity, environment, and the question "How?". *Psychological Review 65* (1958) 197-208, p. 197.

26) the same as the note 12).

27) the same as the note 2).

28) the same as the note 8).

29) the same as the note 18).

30) the same as the note 3).

31) the same as the note 8).

32) the same as the note 5), p . 296.

33) the same as the note 18).

34) the same as the note 11).

35) the same as the note 12).

36) T. J. Bouchard: Genetic influence on human psychological traits: A survey. *American Psychological Society, 13* (2004) 148-151.

37) D. T. Lykken: The mechanism of emergenesis. *Genes, Brain and Behavior, 5* (2006) 306-310.

38) the same as the note 4).