The Non-Shared Developmental Residual: What Twin Variance Implies for a Prenatal-Hormone Account of Correlated Phenotype

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Theoretical synthesis; no new data.

Abstract

Twin studies reveal a robust and under-exploited pattern. Across thousands of human traits, roughly half of phenotypic variance is genetic. Shared family environment contributes little for most traits. A large remaining fraction is non-shared. This is individual-specific variance unexplained by either genes or household. We synthesize the personality and facial-morphology twin literatures. We argue that this non-shared residual is a plausible natural home for a prenatal developmental contribution. Conventionally, this residual is treated as post-natal idiosyncrasy or noise. We distinguish this residual sharply from the missing heritability gap. The two quantities are frequently conflated. They sit on opposite sides of the genes not-genes partition. A developmental cause acting directly can target only one of them. We conclude that a prenatal-hormone account of correlated physical and psychological phenotype is consistent with the structure of twin variance. It is not established by it. We specify the evidence that would move it from consistent to confirmed.

1. Introduction: the pattern hiding in plain sight

Polderman et al. (2015) conducted the largest twin meta-analysis to date, aggregating 17,804 traits from 2,748 publications and 14,558,903 partly dependent twin pairs. This effort essentially covered the entire published twin literature on complex traits. The study reports a mean heritability of 49% across all traits. Critically, for a majority (69%) of traits, the observed twin correlations fit a parsimonious additive-genetic model. In these cases, twin resemblance is consistent with additive genetic variation alone, with no evidence for a substantial influence of shared environment or non-additive genetic effects. The implication is structural rather than incidental. For most traits, the variance that is not additive-genetic is not shared-environmental either. It is non-shared.

Personality heritabilities for the Big Five traits cluster in the ~41-61% band. Jang, Livesley, and Vernon (1996) estimated broad twin heritabilities of 41%, 53%, 61%, 41%, and 44% for Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness respectively. They noted that environmental influence was consistent across all five dimensions, dominated by the non-shared component, with shared environment near zero. We adopt a twin-reference band of ~41-61% taken directly from the Jang et al. (1996) point estimates for benchmarking. The upper bound, 61%, is Openness. Power and Pluess (2015) likewise contrast their own SNP-based estimates against a twin reference of roughly 40-60% drawn from the broader literature, not from these specific Jang et al. point values. Across personality, then, shared environment is small and the non-shared component absorbs the large majority of environmental variance.

The Minnesota Study of Twins Reared Apart (Bouchard, Lykken, McGue, Segal, and Tellegen, 1990) sharpens the same point from the opposite design. Across measures of personality, temperament, interests, and social attitudes, monozygotic (MZ) twins reared apart are about as similar as MZ

twins reared together. Roughly 70% of IQ variance tracked genetic variation. The similarity is genetic. What differs between co-twins is decidedly *not* the household they grew up in.

Facial morphology shares this architectural pattern, yet it does so with higher heritability. Djordjevic et al. (2016) conducted a 3D geometric-morphometric twin study involving 1,380 female twins from the TwinsUK registry. They reported feature heritabilities ranging from 30.5% to 84.8% across scaled principal components, and from 38.8% to 78.5% for unscaled components. Genetic factors account for more than 70% of the variation in facial size, nose dimensions, lip prominence, and inter-ocular distance. Monozygotic (MZ) correlations are roughly twice those of dizygotic (DZ) twins. Common, or shared, environment reached significance for only 4.3% of linear distances, a vanishingly small proportion. The unique, or non-shared, environmental contribution was largest for traits such as horizontal facial asymmetry. Even MZ pairs differ measurably in face shape. Surface superimposition places within-MZ-pair surface deviation at a mean of approximately 0.08 mm, reaching approximately 1.0 mm at the 95th percentile (mean 0.08 $\pm$ 0.25 mm; 1.01 mm at the 95th percentile across 14 MZ pairs; Ozbilen et al., 2023). Identical genomes do not produce identical faces.

2. The residual is not noise - and not "missing heritability"

Two distinct quantities are routinely conflated. Separating them is the analytic core of this paper.

Missing heritability is the gap between *twin-estimated* heritability (~50% for personality) and *SNP-captured* heritability from molecular data. For the Big Five, Power and Pluess (2015) estimated, via GREML on common variants in 5,011 adults, significant SNP heritabilities of **15% for neuroticism and 21% for openness**, with the remaining traits non-significant (extraversion ~8%, conscientiousness ~1%, agreeableness ~0%). These SNP-based estimates capture well under half of the twin-estimated genetic variance. This gap is **still genetic** – it lives in rare variants, structural variation, epistasis, or many small additive effects not yet tagged by genotyping arrays. It is, by construction, *not* environmental.

The non-shared developmental residual represents the fraction of variance that twin studies attribute to factors that are neither genes nor shared environment. This substantial E component persists after accounting for additive genetics and the household. It constitutes a distinct remainder situated on the opposite side of the genes versus not-genes boundary.

We explicitly flag a category error that arises from conflating the two. Missing heritability is genetic by definition, so it cannot be explained by a developmental or environmental cause. A prenatal-hormone account properly targets the non-shared environmental residual rather than the missing-heritability gap. Separately, because hormone exposure is itself partly under genetic control, prenatal hormones may mediate a portion of the genetic variance. This is a logically distinct claim that requires its own test and should not be smuggled in here.

3. Why prenatal development is a natural occupant of the residual

We typically explain the non-shared residual through post-natal idiosyncrasies such as peer groups, chance life events, and differential parental treatment. Yet a substantial share of this variance is established before the post-natal environment even exists. Three observations constrain the candidate causes:

(a) Even with identical genomes and largely shared rearing, trait differences between MZ co-twins do arise, and the residual is real rather than an artifact of measured environment.

(b) Although monozygotic (MZ) twins share a uterus, their intrauterine conditions are not identical. Placental sharing is often unequal, cord insertion sites differ, and local hormone diffusion and nutrient supply vary between co-twins. Documented MZ-divergence mechanisms such as unequal placental sharing and chorion-type differences in the intrauterine environment (van Beijsterveldt et al., 2016), twin-to-twin transfusion, amniotic hormone gradients, and post-zygotic epigenetic drift in receptor density (Fraga et al., 2005) all act prenatally or early. We note that the measured effect of chorion-type (placental-sharing) differences on MZ within-pair resemblance is, where tested, small and trait-limited (van Beijsterveldt et al., 2016). We invoke these mechanisms to establish that the intrauterine environment differs between co-twins and can source divergence, not to assert that it dominates the residual.

Distinct temperament is observable in infancy (Buss & Plomin, 1984; Kagan & Snidman, 1991). This occurs before extended post-natal socialization could account for it, establishing that trait-relevant individual differences predate the post-natal environment.

Together, these factors point to the prenatal environment, and specifically the intrauterine hormonal milieu, as a plausible, albeit currently untested, contributor to a non-trivial portion of the non-shared residual. This residual is not a single homogeneous bucket. Rather, the claim is that part of it is sourced prenatally, not post-natally.

4. Discussion: what this does and does not establish

This synthesis establishes consistency, not proof. The structure of twin variance features high heritability, a small shared-environment contribution for most traits, and a large non-shared residual that partly predates the post-natal environment. This pattern is what a prenatal developmental cause would produce. However, it is not unique to that cause. Developmental noise, stochastic gene expression, measurement error, and idiosyncratic post-natal events also live in the E term and would produce a superficially similar variance signature. The argument here narrows the search space. It does not close it.

To move from consistent to confirmed requires evidence that the residual is organized by a prenatal signal rather than random. The decisive test: show that the same intrauterine hormonal variation predicts correlated physical and psychological differences between MZ co-twins, i.e., that the morphological and behavioral residuals co-vary along a hormonal axis, rather than drifting independently. A random-noise account predicts independence; a prenatal-hormone account predicts coupling. We develop the mechanistic model that this test would evaluate, the multi-axis common-cause account built on top of this non-shared residual, in the companion paper (Abdo, 2026, Prenatal Hormonal Milieu as a Common Cause of Correlated Facial and Psychological Phenotype, PsyArXiv preprint, DOI 10.31234/osf.io/akwrg). The present paper's contribution is narrower and prior: it identifies where in the variance budget such a cause must live, and clears away the missing-heritability conflation that would otherwise misdirect the search.

5. Limitations

There are several caveats that limit the strength of the inference, though not its direction.

Heritability estimates are specific to both the population and the method used. Point estimates shift depending on the instrument, such as self-report versus peer-report personality measures, the dimensionality of the data, since 2D and 3D facial morphometrics diverge, and the population itself.

The flagship facial study conducted by Djordjevic et al. (2016) sampled female British twins only. This narrow focus limits generalization across sex and ancestry.

The Equal-Environments Assumption (EEA). Twin-design inference depends entirely on the assumption that monozygotic (MZ) and dizygotic (DZ) twins experience environments of equal similarity with respect to the traits in question. When MZ twins are treated more alike and evoke similar treatment from others, the design attributes their excess similarity to genetics. This process inflates heritability estimates while deflating apparent environmental components, including the non-shared residual that this paper highlights. Critics have long contested the EEA, so we treat it as a live limitation rather than a settled premise. An EEA violation would generally render our central quantity, the non-shared residual, a conservative lower bound because such a violation would shift variance from environmental factors (E) to additive genetic factors (A).

Reared-apart designs face standing critiques. The Minnesota studies (Bouchard et al., 1990) are subject to objections regarding partial rather than full separation, post-placement contact between co-twins, and non-random selective placement, each of which can inflate apparent genetic similarity.

Djordjevic et al. (2016) note that facial asymmetry has the highest unique-environmental contribution among facial traits. They do not provide a specific point value for this figure. The current argument depends solely on this rank ordering, which places asymmetry at the top for non-shared environment, rather than on any precise magnitude.

Competing Interests

The author declares none.

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Data Availability

This work synthesizes findings from prior publications, meaning no new data were generated or analyzed. The primary statistics come directly from the cited sources.

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