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The "Equal Environments Assumption" in MZ-DZ Twin Comparisons: an Untenable Premise of Psychiatric Genetics?

A. Pam[1], S.S. Kemker[1], C.A. Ross[2], R. Golden[1]

[1]Albert Einstein College of Medicine, Bronx, New York; [2]Dissociative Disorders Unit, Charter Hospital of Dallas Richardson, Texas, USA

Abstract. The comparison of MZ-DZ twins in behavioral genetics has produced what seems like irrefutable evidence for the heritability of many psychiatric disorders. But such research depends on the validity of the EEA – the "equal environments assumption" – as an underlying premise. In this paper, several empirical studies which support the EEA are critically reviewed in terms of methodology and the way data has been processed in a mathematical model called "path analysis". It turns out that studies investigating the EEA appear to be largely inadequate in terms of technique, as well as biased in the inferences drawn. Further, the "heritability" estimate – often taken to mean the influence of trait – specific genes – is merely a statistical abstraction derived from a matrix of correlations; this estimate encompasses many buried environmental effects so that "heritability" does not correspond to any underlying DNA structure. In conclusion, many MZ-DZ pedigree studies have dubious scientific value, given the non-viable premise of the EEA, as well as the misleading operational definition of what has been called "heritability".

INTRODUCTION

One major mode of investigation into psychiatric genetics has been the use of twin studies, consisting of two main types: first, concordance rates in diagnosis for MZ twins compared to same-sex DZ twins, and second, concordance rates for MZ twins reared apart from birth. The latter approach has been relatively unproductive for several reasons: comparable environments for both twins cannot be assured [19]; MZ twins separated at birth are apt to be "throw-away children" or enfants abandonnés [5]; and for uncommon disorders such as schizophrenia, not enough cases can be found [10]. In any event, only rough estimates of heritability can be derived from reared-apart rates of concordance.

Given inherent limitations of research on MZ twins separated at birth, the field of behavioral genetics largely emphasizes MZ-DZ twin comparisons. Such a design has two advantages: enough cases can now be generated to empirically test a pedigree approach to psychiatric disorders, and this approach permits more precise estimates of the extent to which genetic factors may determine intelligence, personality, and psychopathology. However, in such comparisons, it must be recognized that higher MZ than DZ rates of concordance do not necessarily demonstrate that genetic etiology is responsible [12]. For example, investigators initially thought *folie à deux* was involved when delusional symptoms in concordant twins were compared [7, 11]. More recently, two books on MZ-twin symbiosis, on idiot-savants [27] and on elective mutism [31], speak to ineffable communicative bonds between MZ twins for which there appears to be no analogue between DZ twins. In short, since MZ twins may have a more similar environment than DZ twins, one cannot infer a hereditary factor exists when MZ concordance rates are higher. Such a reading of the data would be just another version of the fallacy "If a disease runs in the family, it must be genetic" – a type of error best illustrated by the conviction that pellagra was hereditary since it followed kinship lines, until Goldberger showed in 1915 that a vitamin B niacin deficiency caused a familial disease because everyone ate the same food [17]!

Studies of MZ and same-sex DZ twins are based on the assumption that sbared environment influences these pairs in an equal manner. This view, known in the literature as the "equal environments assumption" [24], will be henceforward referred to as the EEA. If the EEA holds, genetic effects alone can account for the commonly-found greater similarity between MZ twins for traits, abilities, and diagnosis. Thus, in a typical example supporting the hereditary hypothesis, by taking the EEA for granted, one author [18] simply showed that MZ concordance in schizophrenia is almost 50%, whereas DZ concordance is about 15%. Later, a more sophisticated version of this position appeared, termed the "equal trait-relevant environments assumption" [3] but still referred to as the EEA in virtually all papers. This updated EEA allows for unequally shared environments between MZ and DZ twins, provided it is shown that such differences are not pertinent to the trait under study – i.e., that any increased similarity in environment does not contribute significantly to greater phenotypic similarity.

In either form of the EEA, the heritability estimate is derived from the difference between the concordance for MZ and DZ twins, or $r_{MZ} - r_{DZ}$, and experience has shown that this figure will usually be fairly substantial. Further, a technique of "path analysis" has been devised which parcels out the variance in a matrix of MZ-DZ concordance rates into the three components which comprise the etiological spectrum: additive genetic effects, common twin environment, and the unique environment of each twin. But such statistical elegance is contingent upon the legitimacy of the EEA, which allows $r_{MZ} - r_{DZ}$ to be attributed solely to hereditary factors.

This paper presents a critical appraisal of the EEA, the validity of which determines whether MZ-DZ twin comparisons can scientifically stand or fall. Although a full literature review of existing research is beyond the scope of this paper, many studies supporting the EEA will be critiqued for representative procedural errors and bias. The mathematical model used in such research will also be scrutinized to determine if its underlying assumptions have been met in such studies. Finally, we will examine some implausible results in studies which used the standard methodology.

Research relating to the EEA

To date, we know of no literature review of empirical research on the EEA; however, many papers use the MZ-DZ design in studies, usually providing some survey of the current status of EEA research in their text. In perhaps the best such survey thus far available [16], we will track the four independent lines of data the authors present in favor of the EEA, in their opinion adding up to an overwhelming case for its validity.

Physical similarity does not create personality similarity

It is usually presumed that perceived physical similarity leads to similarity of treatment, in turn inducing greater similarity of behavior. If this is so, problems are posed for the viability of the EEA. In refutation of this proposition, the survey [16], cited three studies. In the first study, a group of researchers [23] administered questionnaires to parents of three year-old twins, asking them to rate the twins for similarity of appearance and of personality. The mothers rated the MZ twins as more different in personality the more they physically resembled each other! The conclusion was that, although there was unequal environment between MZ and DZ twins because of similarity of appearance, this situation did not bias twin studies in the direction of inflated heritabilities. However, in review of the study, we disagree with this conclusion: there was poor inter-rater agreement between father and mother ratings, the "personality" being rated was that of very young children, and parents could have readily surmised that the research was about treating the twins as separate people and so may have stressed individuation in their perceptions of the two youngsters.

The second study [21] rated twins, ages 3-13, for similarity in resemblance and dress. There was a range in both MZ and DZ twins, but MZ's were far more likely to look alike, whereas no significant difference was found between groups for being dressed alike. Ratings for resemblance and dress were then rank-ordered and correlated with scores on cognitive, perceptual, reading, speech, and personality measures. The researchers concluded that neither looking nor being dressed alike had any bearing on the "bulk" of their measures. However, the word "bulk" was used because there were several exceptional findings, and even one inverse correlation, so that the results are hard to interpret.

The last study [14] divided the NAS-NRC Twin Registry into three groups, based on close, moderate, and lesser resemblance of MZ twins. It was reasoned that environment would be most similar for MZ twins who looked alike – they should have the highest concordance rates for psychopathology if similarity of environment accounted for their conjoint problems. No significant difference was found in the three groups in rates for schizophrenia, hence, the researcher argued that similar environments had no bearing on concordance. However, bonds between MZ twins are not only the product of a physical resemblance which induces others to treat them as a unit, but may also arise due to intimate contact and common experience – even if the twins can be told apart on sight!

In summary, evidence that physical similarity does not create similarity in milieu thus far seems too unclear to uphold what most clinicians are still apt to regard as a counterintuitive thesis.

MZ twins act on the environment to treat them alike, rather than being alike because they are treated alike

An early and very crude version of this second proposition, for good reason uncited in the survey [16], involves the contention that the environmental similarity of MZ twins is the result and not the cause of their psychological similarity. It was attempted [33] to refute environmental explanation of greater MZ than DZ concordance for schizophrenia as follows: "If MZ twins create a similar environment through their greater similarity, they do so because of the greater inherited similarity in their appearance and response modes. Thus, in a roundabout way, we still come back to the importance of heredity". This older approach acknowledged that psychological similarity in twins is influenced by similarity in their treatment by the social surround, but this factor is seen as an epiphenomenon brought about by their degree of resemblance. It has been criticized by pointing out that similarity of treatment is an environmental response to a biological stimulus – the two should not be confounded. Put plainly, it is specious to treat "environment" as a mere dependent variable which is called forth by "genetics" so that social factors can be more or less dismissed as etiological agents in their own right [9]. In addition to this issue, a paradoxical problem with the older approach is that it comes so perilously close to compromising the EEA – by allowing that environments are not equal – that it had to be modified, although residues of such thinking are still amply sprinkled throughout the EEA literature.

The more sophisticated modern approach involves observation of twins in the family setting. Granting that the milieu tends to treat twins as a "unit" a distinction was made [20] between events where the family arranges such similarity for twins (the "imposed environment") and events where the family is responding to behavior initiated by the twins (the "elicited environment"). A group of researchers [22] followed up this distinction by checking similarity of early environments of 343 same-sex twin pairs against their scores in adulthood on neuroticism, anxiety, and depression scales. As these investigators expected, MZ twins were treated much more similarly than DZ twins – a finding which, if taken at face value, would quickly invalidate the EEA. But, in their judgement, such an interpretation would be misleading: their sample demonstrated no correlation between imposed similarity and subsequent intra-pair psychopathology but some significant correlations between elicited similarity and later psychopathology. They then reasoned that since similarity imposed by the environment had no effect on concordance but similar treatment elicited by the twins' behavior did have some effect, the latter result was arguably "a consequence of their genetic identity". In short, they reached the inference that the EEA is valid: in ways conducive to the development of psychiatric disorders, the twins induce the milieu to treat them similarly! We find such an inference dubious since it is based on the supposition that one can discern which behavior is "imposed" or "elicited" – but any family therapist will insist that child-parent encounters are interactional. Moreover, twin behavior which "elicits" similar treatment from significant others is no longer a self-evident product of common genetic constitution just as soon as the two infants grow into children with a joint history and mutual interests.

Similarity in childhood milieu does not correspond to later concordance for personality, intelligence, and psychopathology

Since MZ twins have been repeatedly found to have a more similar environment both in childhood and adulthood than DZ twins, any violation of the EEA should be detectable

by rates for psychiatric conditions commensurate with environmental similarity. According to the survey [16], various studies for personality and intellectual differences, anxiety, depression, and alcoholism have not reflected this postulated relationship and thus support the EEA because, for the psychiatric disorders in question, the relevant aspects of the environments were either comparable or differences did not matter. However, the survey also reported contrary findings, but these incompatible studies are all dismissed on technical grounds.

In sum, given the lack of uniformity in the findings of a series of studies which are based on different measures of shared environment, it seems fair to say that the results from this line of evidence are still inconclusive.

Twins with "mistaken zygosity" are alike in personality in accordance with genetic endowment rather than social attribution

The fourth line involves a comparison of trait similarity as a function of both "real" zygosity, as assessed by investigators, and "perceived" zygosity, as reported by twins or their parents. Since virtually everyone expects MZ twins to be more similar than DZ twins, and if this expectation influences trait similarity, then studies of falsely-classified MZ and DZ twins can provide a pivotal measure of social influences versus true genetic endowment. A research team [24] reviewed investigation of "mislabeled zygosity," occurring in 40% of pairs in the major study they cited [20]. They noted that those who falsely thought they were MZ or DZ twins resembled their biological groups on intelligence and personality tests more than their groupings by attribution – a finding which runs counter to what environmental theory would have predicted. They argued that the environmental variable of "labeling twins as identical or fraternal has little effect on their behavioral similarity". However, while the MZ twins who thought they were DZ were more alike than correctly-classified DZ twins, they were also less alike to about the same degree than correctly-classified MZ twins, which constitutes an equivocal result. In contrast, DZ twins who thought they were MZ were more dissimilar on personality tests than either correctly-classified MZ or DZ twins – a puzzling result which throws into question any simple inference from this data. Their conclusion that labeling has little effect on similarity seems to be an overgeneralization based on one more or less favorable study.

A more recent study [8] using this same approach found "mislabeled zygosity" in 15% of female twin pairs but no evidence for a significant influence of perceived zygosity on major depression, generalized anxiety disorder, phobia, bulimia, and alcoholism. One limitation of the study was the small number of cases in each of the five diagnostic categories because only 15% of this sample were classified as "mislabeled zygosity" as compared to the rather incredible 40% in the eartier study [28]. An ironic problem involved possible errors in investigator-assigned zygosity because blood specimens were not always taken!

In review of the "mistaken zygosity" research, this line of evidence has been the most productive for behavioral genetics so far, but again we do not see how one could claim at this time that it constitutes a compelling argument for the validity of the EEA.

Overall evaluation of the literature review

We are struck not only by tendentious interpretation of data but also by the omission of important research findings within psychiatric genetics which, while not directly testing the EEA, still show that the impact of environment has been underestimated in twin studies. Several reports have noted double to triple rates of schizophrenia for DZ twins as compared to singleton siblings [19]; since genetic heritage of both sides of this comparison are equal, only environmental diversity can explain such results. Moreover, when studies of same-sex DZ twins to opposite-sex DZ twins were also compared, again genetics being held equal, the same-sex twins had more than twice the rates of schizophrenia, once more indicating that the operative explanation is difference in environment. The immediate implication for the EEA of these findings is that differences in family milieu are fraught with consequences for the genesis of psychopathology – indeed, comparing differences in MZ-DZ concordance when genetics are held constant to differences when environment is (presumed) held constant, the effect attributable to variations in milieu is far greater and more unequivocal. In other words, these findings point to shared life events as the crucial determinant of joint vulnerability to schizophrenia, and by logical extension, to other psychiatric disorders. As in nearly all current reviews of the EEA, the survey [16] leaves such contrary data out of account, instead insisting that the EEA is being confirmed in study after study. Another reviewer of this reserach [4] has added that the literature on EEA frequently glosses over the fact that, when r_{MZ} is less than .50 (a not uncommon result in twin research), nongenetic factors must predominate in the causal pattern, with environment actualizing whatever modest risk the implicated genes may convey.

Further, we regard the conclusion reached by the survey [16] in their literature review as inherently untenable. The combination of the two EEA approaches, under the very same name, presents logical problems. Note that although the two forms of EEA express the same hypothesis, the new form contradicts the old by no longer positing that MZ and DZ twins have comparable environments. Oddly, surveys of current evidence for the EEA tend to combine the two approaches, when, in our estimation, the "equal trait-relevant environments assumption" could just as reasonably be called the "unequal-environments-don't-matter assumption". Moreover, if literally understood, the "equal trait-relevant environments assumption" suggests that for a given phenotype, appropriate parameters of early environment can be identified and held constant in MZ-DZ twin comparisons; yet, seldom are the exact etiological parameters for a psychiatric condition known.

Worse yet, the "equal trait-relevant environments assumption" soon renders academic the issue as to whether the EEA holds or not.

Research which was intended to establish whether MZ and DZ twin pairs are treated in an equivalent manner is readily transformed into an inquiry as to whether we need the EEA after all. We agree that, scientifically speaking, this is a legitimate inquiry and it may be that the old EEA is wrong but the new EEA is right. However, mathematically speaking, the strict aim of all the studies reviewed should have been whether environment was in fact equivalent or not, for only when it is verified that MZ and DZ twin environments are comparably uniform can the statistical model which apportions respective estimates of etiology apply. In contrast, the survey [16] of empirical data mainly

concerns studies which violate this basic assumption and yet still utilize the statistical model. Nor is this the only way used to get around doubts about the premise of the EEA: one authority [15] has assured researchers whose data showed there had been violations of the EEA which might invalidate their studies that fortunately "... statistical methods are now available to determine the role of genetic and other familial environmental factors, adjusting for these violations". To our way of thinking, such methods amount to having your statistical cake and eating it too.

Problems in the concept and measurement of EEA

While all research has its flaws and limitations, drastic shortcomings cannot be overlooked when they threaten to distort the data or misrepresent the results. We contend that the research done on the EEA has such shortcomings, and we will now address three major problems: the questionnaires and rating scales utilized to measure "environment", biological differences between twins based on prenatal and neonatal events, and the pragmatic "fit" of the path model used to segregate genetic and environmental effects.

Are the measures which quantify "similarity of environment" valid?

How is an investigator to measure a variable as complex, subtle, and mutable as "environment," let alone "similarity of environment," other than by some self-report questionnaire or scale which thereby becomes its operational definition? The research then utterly depends on the validity of the measure used – it can be no more accurate than its instrument and only captures an arbitrary dimension of the variable studied. An operational definition is after all a purely human construction, epitomized in a circular but seriously-intended definition of "intelligence" as "what IQ tests measure" [2]. We contend that measures of environment in research on the EEA are far from satisfactory in many respects: they are crude, insufficiently pilot-tested for validity, and tend to be literally accepted as representations of "reality." Consider the scale in a study [30] on inherited cognitive ability: 28 items measure the variable "similarity of environment" – typical examples of the scale are such questions as "Does one twin eat more?"; "Joint ownership of dog?"; "Would you like to have twin children when married?"; and "Go to movies together?" Much more revealing questions could have been asked; for example, another survey [12], cited data as to whether the twins had ever spent a day apart and found that 43% of MZ twins had not, compared to 26% of DZ twins; whether they had the same friends, finding 76% of MZ twins had compared to 52% of DZ twins; or usually studied together, reporting that 40% of MZ twins did compared to 15% of DZ twins. Given that the scale used in the above questionnaire [30] is rather superficial, it will hardly be surprising that only 7 of 28 items significantly discriminated between MZ and DZ twins. Nevertheless, rather than acknowledging that this "twinness" scale was a very gross measure, the researcher accepted his self-report inventory as valid and made far-reaching inferences from his data. Noting that 4 of 6 cognitive abilities he measured did not correspond to how his scale measured "similarity of environment," he concluded that equivalence in twin situations is not a cause of equivalence in mental abilities, and then used this finding as retroactive evidence in support of the EEA, fur-

ther speculating that personality variables would prove to be equally unaffected by similarity of environment.

The thrust here is not that rating scales can be improved; it is that any pen-and-paper instrument will be more or less limited in measuring the relevant mutual experiences which bear on "similar environments." Moreover, technical questions arise: should twins or parents be polled, and if there is disagreement, which generation gives the more accurate rating? Do the twins themselves agree, and if not, why does this happen, and does a consensus score resolve the difference? As can be seen, before even using such measures as a self-report questionnaire, a series of pilot studies are first necessary to work out manifold reliability and validity issues.

Is there prenatal and neonatal "equal environment" between MZ and DZ twin-pairs?

A team of researchers [1] warn of limitations to the EEA which arise out of unequal prenatal twin experience – a factor seldom considered in such research. They questioned mothers of 2-3 year old twins and found that half the children were born prematurely at an average birth weight of only 88 ounces, with comparable findings for both MZ and DZ twins. They also found a high incidence of prenatal and neonatal complications, involving about two-thirds of their sample, which suggested that twins could not be readily compared to singletons. Further, they found notable differences in birth weights between MZ twins, and still more so between DZ twins, placing the same set of twins at very different risk for impediment to their physical and emotional development. Thus, the womb is scarcely an "equal environment" for even the same set of twins. Going beyond intrapair differences to interpair differences, it has been noted [4] that 70% of fetal MZ twins share the same chorion tissue of the maternal placenta (an environmental factor), whereas DZ twins never do. This biological phenomenon highlights the fact that prenatal environment is already much less comparable for DZ than MZ twins. In short, measuring the "environment" is not a simple matter to be addressed by a rating scale focused on mutual twin social experiences.

Is the statistical model valid?

The path analysis by which a matrix of MZ and DZ correlations are transformed into estimates of genetic and environmental factors now needs to be examined. On the basis of a theoretical model, heritability is calculated by the formula $h^2 = 2\,(r_{MZ} - r_{DZ})$; this result is labeled additive genetic effects. Environmental influence is divided into two components: common environmentability (within-family variance), calculated by the formula $c^2 = 2r_{DZ} - r_{MZ}$); and unique environmentability (between-family variance), computed by the formula $u^2 = 1 - r_{MZ}$ since MZ discordance can only be attributed to unshared milieu. It has been claimed [3] that the assumptions of the genetic model appear to hold best when r_{MZ} falls between .40–.50 for MZ twins and is about twice r_{DZ}, u^2 approximates .50, and c^2 is less than .10 results of most empirical studies more or less fit these criteria. Given such typical outcomes, the factor of c^2 has virtually vanished on the bottom line, implicitly suggesting by its absence that common environment is of little moment in accounting for concordance in twin behavior.

In criticism of this model, h^2 represents a more ambiguous biological or even

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quasi-biological factor than any current studies acknowledge. Mathematically, h_2 is not a statistical representation of specific DNA input into a trait – rather, it is a calculation from a set of MZ-DZ correlations and is that proportion of the variance unique to MZ twins. Thus, h^2 is determined by all the shared genes of the MZ twins and not necessarily by a predisposing gene or group of genes. Moreover, h^2 does not convey a pure "heritability" estimate anyway; instead, it is a product of factors directly or indirectly attributable to the entire genome. Hence, social factors tied to genetically-caused traits will also be picked up in the statistical comparisons: there is no way to eliminate reactions by the milieu to physical or other similarities of MZ and DZ twins from the reported correlations, so that h^2 always includes some element of common environmentability. In sum, we contend that h^2 captures not only genetic contributions but also environmental responses in relation to the biological stimuli of MZ or DZ "twinness." Accordingly, much of "common environmentability" can be expeeted to be incorporated – but hidden – within the h^2 value in path analysis, leading to inflated estimates of "heritability."

In addition, a group of statisticians [13] have pointed out technical problems in path analysis when applied to genetic epidemiology. They noted that the model is linear and additive, asking whether "... path analysts seriously suggest that such variables as 'genotype,' 'culture,' or 'family environment' could be transformed meaningfully to commensurate scales that combine additively," and stating that in such research, "natural processes (e.g., assortative mating, transmission via a major gene, cultural transmission) are inherently nonlinear". Put bluntly, they charged that the inherent restrictions of the "path" model were being ignored in psychiatric research. In rebuttal, another group led by a psychiatrist [6] defended path analysis as heuristic, pointing out that it has been useful in studies of lipoproteins and human blood pressure, but not offering a single example of its utility within the psychiatric domain. In a second rebuttal of the above-mentioned study the founder of the technique of path analysis [32]defended the role of statistical models in research, but was queasy about the EEA: data from MZ and DZ twins "present, in principle, a means of separating out... heredity, but unfortunately require controversial assumptions with respect to possible environmental differences".

Ultimately, the twin-study path method is just a statistical model – an idealization – whose parameters are assigned values to fit a particular set of numerical data but does not necessarily provide an adequate approximation to the real world. As readers of these studies may not always realize, a model in any given application cannot be expected to be literally correct; it has been said by statisticians that all models are wrong but some are more wrong than others! For the twin-study path model in psychiatry, we must accept in advance, for example, that various assumptions concerning linearity, additivity, and uncorrelated residuals will not be perfectly satisfied – that is, for any trait there will always be some degree of "assumption departure." Still, the mathematical question to be answered is: to what extent can the assumptions of the model be faulty and yet produce parameter estimates that are accurate enough for practical purposes? This question can only be dealt with by studies which show that the model has predictive power with respect to the actual parameters of heritability as measured, say, by a genetic marker study. However, in the absence of any objective standard by which to measure the proximity of results from path analysis, the predictive power of the model, despite violation of certain assumptions such as the EEA, remains untestable. Such a statement on our part

does not imply that genetic findings based on use of the path model are false, but it does mean that the burden of proof is on the investigators to show that the model is valid – and there is simply not enough information available to know under what conditions it may be.

Some arresting and troubling examples in research findings

It is alarming to us just how unreflective some studies can be in accounting for human social behavior on genetic grounds, without any questioning of methodology even when implausible results occur. For example, some researchers [8] stated: "Even within families, it is clear that DZ twins hold religious and political views that are significantly more diverse than MZ twins... [hence] genetic differences between people are partly responsible for the distinction between godly and ungodly and between liberal and conservative". Our view is that common sense, plus some knowledge of sociology, would suggest that experimental procedures could be fallacious which lead to such inferences about inheritance of social, religious, and political values.

In another example, this time about psychological-traits, a researcher [25] studied MZ-DZ differences in "perceived parenting." In relation to the parameter of "Acceptance-Rejection" by parents, lack of similarity in shared environments did not differentiate between MZ and DZ twins for how they perceived parenting by fathers, but did for how they perceived parenting by mothers. He struggled with this genetic finding with respect to fathers, but an environmental finding with respect to mothers, as follows: "One interpretation of this result is that genetic factors partly determine the perceptions of the father. However, it is difficult to imagine genetic factors that would influence only [Acceptance-Rejection Fathers] ... [but] researchers should be alert to the possibility that perceptions of maternal versus paternal A-R may have different etiologies". The researcher did not stop at this point but soon revisited the topic of "perceived parenting" in a later paper [26]. Using a different measure, he now found that the trait "Acceptance-Rejection" of a child by both mother and father fit a genetic model!

Such preposterous findings indicate to us that the methodology of MZ-DZ research in the field of behavioral genetics is seriously flawed since positive results are reached for virtually anything examined. As declared in a paper critical of such research [4], given an approach based on interpair MZ-UZ differences, one could demonstrate that demonic possession, or almost any human conception or trait is essentially a product of genetic predisposition.

Does this research area meet scientific standards?

Any critique of this area must first give generous credit for serious efforts on the part of researchers in behavioral genetics to come to grips with the EEA. A fair number of articles now bear on the empirical validity of the EEA, and even when the MZ-DZ twin method is only a technique used to study a specific ability, trait, or disorder, many researchers do a literature review on the EEA and carefully appraise their own data as an

indirect check on its validity, leading to what appears to be an impressive cumulative body of work. Nevertheless, our considered judgment is that much of the research supporting the EEA does not meet scientific standards, is often sophistical in the way inferences are drawn from data, and slanted in terms of objectivity.

Our criticism of the field does not imply that genetic factors do not operate in psychiatric disorders. We do insist, though, that they are far less influential than much of the current literature has led us to believe – although it is still impossible to say how much less without research being redone, using a more rigorous methodology. Some biologically-oriented researchers [29] have reached much the same conclusion for different reasons: based on studies of discordant schizophrenic and manic-depressive MZ twins, they suspect that an intra-uterine virus or developmental abnormality may be more etiologically important than genes, though heredity might still play some role. For our part, we would add the wish that investigators shift more of their efforts to social aspects of etiology because environmental risk-factors causing neurological and/or psychological damage appear in our assessment of current research to be more prepotent than genetics in almost every psychiatric disorder so far studied by the twin methodology.

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Correspondence: Dr. Alvin Pam, Director of Psychology, Bronx Psychiatric Center, 1500 Waters Place, Bronx, New York 10461, USA.