A Few Comments About Communibiology and the Nature/Nurture Question

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In our initial essay, our goals were to reconceptualize communication apprehension in light of genetic and neurobiological knowledge and "stimulate thinking and research" (Beatty, McCroskey, & Heisel, 1998, p. 212). This issue of *Communication Education* does much to advance the latter goal. Although we recognize that much of communibiology is counter to the ways many members of our profession are accustomed to thinking about communication, some of the challenges to communibiology presented in this issue are seriously flawed.

Condit, for instance, rejects our contention that traits are mostly due to genetic inheritance. In particular, she challenges our 80/20 genetics to environment ratio by reviewing our sources. We believe that Condit overlooks several issues central to our speculation about the *potential influence* of genetic inheritance. *First, our 80/20 estimate referred to communication apprehension (CA) and the traits associated with it, not all traits.* Our estimate for the impact of heredity on trait verbal aggressiveness, for instance, was in the .60 region (Valencic, Beatty, Rudd, Dobos, & Heisel, 1998). *Second, we did not contend that each trait is the product of a single gene (If it were it would be almost impossible to discover the CA gene; It is probably hiding behind the other genes).* We said individual differences in thresholds of the *neurobiological systems* responsible for the cluster of behaviors and feelings commonly referred to as introversion (I) or neuroticism (N) *are inherited.* These individual differences in neurobiological sensitivity are distributed across a continuum so that people differ in degrees. Condit argues that similarity of heritability coefficients for combined traits such as CA and the traits of which it consists are suspicious because the probability of inheriting I and N are multiplied to determine the probability of inheriting both traits, and therefore, CA. However, like neurobiological sensitivity, I, N and CA are continuous rather than dichotomous variables. High levels of CA (e.g., upper quartile of the PRCA) can result from a variety of combinations of I and N (i.e., extremely high N and moderately high I; extremely high I and moderately high N; moderately high I and moderately high N; high I and high N). Conclusions based a two independent outcome dichotomous model (e.g., blue eyes and male) are inapplicable to our model.

Suppose that I and N were dichotomous, the multiplicative function Condit describes pertains to the *probability* of inheriting a trait, *not the correlations.* Correlation coefficients are not probability estimates, thus, twins' correlations for a combined trait would not equal the product of the correlations for the primary traits. Probability estimates are often attached to obtained correlations (in the form of significance levels) but these do not represent the probability of a linear relationship between the

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variables. Rather, they indicate the probability of the data pattern for a particular sample size, given the hypothesis is false. The probability estimates needed to calculate the probability of a combined trait (e.g., CA) given two primary traits (e.g., I, N) require calculation of the probability that a particular correlation coefficient indicates a linear relationship (Phillips, 1974). It is the product of those probabilities, not correlations, that must be calculated.

Third, we did not base our perspective on communication apprehension entirely on the results of identical twin research. Actually, anyone who reads our work will note that the twins paradigm *does not* comprise the bulk of the literature we reviewed. Although we cited results from identical twin research as *one* source of evidence, our reanalyses of McCroskey's original propositions regarding communication apprehension (which accounts for over ⅓ of the essay) were exclusively based (no mention of twin studies) on experimental studies of drug effects on and analyses of biochemical markers of activity in targeted anatomical regions of the brain important to our conceptualization (Beatty, McCroskey, & Heisel, pp. 204–211). Our propositions were based on the heritability of organic brain matter.

Fourth, we did not maintain (a) that attenuated correlations in identical twin studies were .80 nor (b) that most psychologists interpret the literature as indicating an 80/20 ratio. We speculated about the potential impact of genetics based on the correlations in light of the psychometric properties of the measures employed (the validity coefficients of Eysenck's personality measures are about the same magnitude as correlations for those measures obtained in the study of identical twins). A few remarks concerning the disparity between Condit's and our interpretation of these correlations is in order. Condit based conclusions on attenuated correlations, which would be appropriate if our interest was in predicting one twin's score on a measure from the other twin's score. However, when statements about the association between the *constructs* that the scores represent, such as in the context of theory construction, attenuated correlations are inadequate. The operational definitions of I and N consist of responses to self-report inventories, the reliability and validity of which are necessarily imperfect (Item pools are incomplete, interpretation of item wording varies among respondents, response options solicit rough estimates of applicability of a particular item, items are imperfectly weighted in summary scores, etc.). Although one component of these scores consists of stable trait variance, another component consists of unstable error variance. The greater the stable trait variance the greater the validity of the measure, which is an important consideration when making statements about the potential degree of association between constructs because the maximum correlation coefficient that can be obtained between two measures cannot exceed the square root of the product of their validity coefficients (Guilford, 1954).

While it is true that most of the correlations obtained in twins studies are between .30 and .60, the validity estimates for the personality measures employed typically do not exceed .60. In fact, Cronbach (1970) in an oft cited observation noted that in the behavioral sciences "It is unusual for a validity coefficient to rise above .60" (p. 135). Although validity coefficients of this magnitude are sufficient for large sample studies, especially when statistical significance is considered an acceptable outcome, the unstable error variance must be considered when making statements about the true degree of relationship between variables. Given what is known about the restrictive effects of validity on correlation coefficients (e.g., Guilford, 1954), an 80% contribution of genetics in a identical twin study would be observed as a correlation

of approximately .45 (about the midpoint of correlations in the twins studies) given a validity coefficient of .60 for the measure. In her assessment of the correlations, Condit misattributed error in personality scores to error in the predictiveness of genetic models of traits.

We are not advocating that knowledge claims should be based on corrected correlations. However, the extent to which a particular effect size represents impure measurement rather than conceptual incompleteness is important in directing our research efforts. Moreover, the attenuating effects of measurement error on correlations in the twin literature can be shown in ways other than correcting correlations for attenuation. Lykken (1995), for instance, pointed out that "The average heritability of psychological traits seems to be about 50%, based on single measurements, and perhaps 75% when based on estimates of the stable components of traits (e.g., on the means of repeated measures)" (p. 108). Our position has been that larger inter-class correlations between twins' scores will follow improvements in trait measurement, which is why our concluding paragraph contained the phrases "It may turn out" and "with psychometric refinement" (p. 213). It turns out that when reasonably stable measures are employed in twin studies, the findings approximate our initial projections. Since the publication of our initial article, for example, Horvath (1998) reported a heritability coefficient of .74 for sociability and .62 for the relaxed dimension of social style. The relationship of these variables to communication apprehension seems especially noteworthy.

Condit compounds the aforementioned errors in two ways. First, effect sizes from different studies are compared but they were not put on a common metric. This is problematic because the literature is uneven with respect to the statistical form in which heritability is reported (e.g., at times, *r* is reported as the estimate of shared variance, at other times *r*-square is used [*r* is correct, see Jensen, 1971; Ozer, 1985]; at times estimates are based on heritability formulas, at other times, raw correlations are reported; some report average *r* for multiple administrations while others are based on single administrations; sometimes correlations are corrected for attenuation, sometimes not). Second, Condit's assertion that the formulas employed to estimate heritability from twin correlations inflate estimates by a factor of 2 overlooks an important statistical detail: Determining the shared variance between two variables *x* and *y* (e.g., twins' scores) due to a common latent variable *z* (e.g., common genetics) for sets of respondents requires a different mathematical model (and, therefore, set of operations) than does determining the variance in *y* attributable to *x* in a repeated measure design. Although a full treatment of this issue is beyond the scope of this essay (interested readers are directed to Falconer, 1989, and Ozer, 1985), the point is that the formulas employed are not "inflationary." In sum, the methodological oversights we have pointed to are precisely the mistakes that led to premature disillusionment with "nature" models of social behavior and development forty years ago, before knowledge of basic measurement theory was widespread among serious social science scholars (For a discussion and historical perspective see, Eysenck & Eysenck, 1985).

While efforts of researchers attempting to document methods for reducing CA (in this issue) are laudable, we simply repeat that inferences about the effects of treatment on traits must be made cautiously when every participant has been pre-tested (whether or not a control group is used). Self-report indices often sensitize participants to the purpose of studies and threats to validity due to pretest-treatment

interaction cannot be ruled out (Campbell & Stanley, 1963, p. 8). Shifts in the unstable component of self-reports can be experimentally induced without affecting lasting changes in CA (e.g., Beatty, Behnke, & McCallum, 1978).

Our initial proposal was that differences in traits are due to individual differences in parameters of neurobiological functioning. We presented evidence that neurobiological parameter differences (not traits) were mostly inherited. Accordingly, we described in detail the neurobiological systems responsible for avoidance, approach, and fight responses. At least two points are worth considering when evaluating our claims regarding the heritability and role assigned to environment. First, our comments pertained to emotionally-based traits (e.g., CA and verbal aggressiveness). The neurobiological systems (e.g., limbic system) underlying emotional reactions *are* primarily inherited and relatively impervious to learning. Naturally, as our attention turns toward processes presumed to be under more "rational" control, other brain regions (e.g., cerebral cortex) become increasingly important. Second, "environment" referred to social learning theory (not any possible conception of external force), and "trivial" referred to (a) effect sizes obtained in research and (b) growing evidence that some of the supposed environmental effects contain genetic influences (e.g., Phillips & Matheny, 1997; Saudino & Plomin, 1997; Scarr, 1992).

Whether social behavior turns out to be purely genetic in origin or 51% genetic does not in the long-run matter to us. After carefully sifting through all of the available evidence and making the uneasy decision to abandon a twenty year commitment to social learning theory, revising communibiology to reflect new research findings presents little challenge. Thomas Kuhn's (1962) forewarning about reactions to paradigm shifts notwithstanding, we hope that our colleagues in the field will describe communibiology accurately and completely, and that evidence regarding it will be collected and interpreted according to conventions for scientific inquiry.

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