Theory, Scientific Evidence, and the Communibiological Paradigm: Reflections on Misguided Criticism

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In our brief essay, "A Few Comments About Communibiology and the Nature/Nurture Question" (This Issue), we directed attention to misrepresentations of our position and methodological oversights contained in some of the challenges to our work. We thought our comments regarding those challenges would clarify our position. Evidently, that was not the case, Condit's reply to our response ("Toward New 'Sciences' of Human Behavior," Also This Issue) further misrepresents our position and our claims. On the dubious assumption that the merits of conflicting stances can be illuminated as a disagreement unfolds, we turn our attention to a general oversight driving Condit's complaints about communibiology before turning to her inaccurate and misleading statements about our work and quantitative methods.

(Mis)Identifying the Level of Abstraction

From the outset, we have referred communibiology as a *paradigm,* not a *theory.* In a general sense, paradigms consist of sets of assumptions from which theories and hypotheses are derived and tested. According to Reynolds (1971), a paradigm (1) presents a radically different conceptualization of phenomena of interest, (2) suggests new research strategies, although dramatic new research procedures are not described, (3) suggests new research questions, and (4) may explain events previously unexplained. While elements of the first three characteristics were certainly present in our initial work, most of our energies were dedicated to fulfilling the expectations of the latter dimension of paradigms. For instance, our essay focussed on communication apprehension (Beatty, McCroskey, & Heisel, 1998) presented an integration of a sizable corpus of research results that were previously difficult to reconcile. While we discussed implications of a theory based in a temperament paradigm, we advanced no such theory. In a similar way, we integrated the research on trait verbal aggressiveness into a "working model." Although we have worked toward the development of theory, deriving and testing hypotheses from this tentative model (Valencic, Beatty, Rudd, Dobos, & Heisel, 1998), communibiological propositions were not presented as theory. Rather, in our earliest published article (Beatty & McCroskey, 1997), our propositions were called "metatheoretic principles" (p. 448). Accordingly, Condit's preoccupation with the lack of "definite proof" in our work or our failure to "prove scientifically" seems premature given the level of abstraction of our work (Kuhn, 1962).

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Paradigms are not "proved" scientifically or otherwise and few scientific enterprises move from paradigm to fully elaborated, strongly supported theories in under three years (Kuhn, 1962). Einstein contented that gravity warps time and space ten years before a telescope powerful enough to verify the claim was constructed. Galileo, and Copernicus before him, lacked "definite proof" that the sun did not orbit the earth as Aristotle had proclaimed. Kepler could not prove by direct observation that planetary orbits were elliptical rather than circular. Instead, these paradigm shifts were introduced through inferences, hypotheses, and questions arising from gaps between observations and predictions of existing theories. Many paradigm shifts are incremental, occurring over a long period of time but others appear explicitly and dramatically, and when they do, Reynolds (1971) warns that they are "often met with a great deal of skepticism and hostility" (p. 42). Despite our repeated reference to our work as the "communibiological paradigm," a phrase which we include in the subtitles of two of our initial works (Beatty & McCroskey, 1998; Beatty, McCroskey, & Heisel, 1998), some of our detractors continue to inappropriately apply to a paradigm the evidentiary requirements for theory verification.

Condit's concern over our supposed commitment to an indefensible position does not square with the natural process of paradigm development. Refinement and elaboration of unspecified details of an initial version of a paradigm ultimately produce "paradigm variations" or alternative versions (Reynolds, 1971, p. 32). Certainly, we recognized this when we noted that "As further research accumulates some of our ideas will be confirmed, some will be revised, and some will be rejected" (Beatty & McCroskey, 1997, p. 455). When paradigms are introduced they represent an interpretation of events from a particular perspective, which may or may not be shared by most members of a discipline. However, their initial assumptions are rarely preserved in their original form.

The importance of introducing paradigms should not be underestimated. As Kuhn (1962) has pointed out, "In absence of a paradigm or some candidate for a paradigm, all of the facts that could possibly pertain to the development of a given science are likely to seem equally relevant. . . . Furthermore, in absence of a reason for seeking some particular form of more recondite information, early fact gathering is usually restricted to the wealth of information that lie ready at hand" (p. 15). As many of the essays in this special issue indicate, the communibiological paradigms seems to have prioritized fact gathering about communication apprehension. In this way, Condit's complaint that we have relied on convenient "ready at hand" data and her demand for molecular biological studies (i.e., seek particular forms of evidence) is right in step with the stages of paradigm evolution. Unfortunately, the tone of Condit's essays seems to indicate that she does not clearly recognize this point. Perhaps more troubling are the errors contained in her attack.

Specific Misrepresentations of Our Position

Misinterpretation of statistical analyses. In her reaction to our response, Condit advanced a set of factually incorrect assertions to depict our stance on the heritability of extraversion and neuroticism as "oversimplified." First, Condit attributes our "misinterpretation" of her use of "multiply" to our social science bias. Instead of the "narrow," "mathematical" sense as in multiply two numbers, "as a critical scholar," Condit meant "multiply" in the broader sense, such as in "the multiplication of a population" (Footnote 2). Actually, however, Condit did not say "multiply," she said

"multiplicative." Specifically, she said "The biological problem with using correlations of data from monozygotic twins to estimate general heritabilities of complex traits lies in the *multiplicative effects* of gene *interactions* (emphasis ours)" Critical scholars consider context when ascribing meaning: The context of this sentence is not "population growth." Condit is describing the complexity of determining the probability of an outcome event (a complex trait) when the effect of one variable on that outcome is contingent on the value or characteristic of another variable, which is precisely what is meant by "multiplicative interaction" in the mathematical sense. When scientists, social or biological, describe a process as a "multiplicative interaction," multiplying the quantitative values of the predictor variables is *exactly* what they mean. We did not misinterpret Condit: Condit misinterpreted a quantitative feature of the genetics literature.

Second, attempting to clarify her argument, Condit proposes that "if the high N-contributing allele #6 is located on the chromosome relatively close to the high E-contributing allele #8, the two are more likely to co-segregate during sexual reproduction than if one is located more distantly from the same chromosome" (This Issue). Now, unless as a critical scholar, Condit means something other than the normal meaning of "more distant" and "more likely," she has written a quantifiable, testable hypothesis that, contrary to her assertion, does not require a complex statistical model. According to Condit's explanation, for instance, co-segregation equals distance between alleles. Measuring the distance between alleles might be tedious, but the statistical test would be rather simple. Complex statistical models are not required to test for complex effects.

In fact, examples of the statistical analysis used in molecular genetics studies contradict Condit's claim that complex statistical models are necessary. While at the Center for Development and Health Genetics (Pennsylvania State University) Plomin and Saudino (1994) (also cited by Condit), compared allelic frequencies for children in high and low-activity groups (determined by mothers' ratings of their children on a Temperament Inventory). They analyzed the data by calculating chi-square, hardly a complicated statistic, for a 2×2 contingency table. Aside from obtaining a significant chi-square, our computation of an *phi* coefficient for the data was .47. Keeping in mind that the reliability of the observational ratings was far from perfect (although we did not perform a correction), and that genetic correlations are not squared, these results do not support Condit's position at any level, especially when we consider that "activity" is a component of extraversion (Eysenck, 1986, p. 11).

Finally, as a ploy to avert discussing the specific statistical errors contained in her essay, Condit opts to paint us as adhering to dogma rather than applying appropriately complex analyses to variables. Specifically, Condit tells us that she is "not opposed to the use of statistics per se, but merely to oversimplifications based on adherence to a particular set of dogmas . . ." Apparently, Condit is unaware that our analysis was not based on a single "dogma." The formula for calculating the probabilities we referenced is from Bayesian statistics, correlation coefficients are rooted in the Pearsonian tradition, and the attachment of significance levels is classic Fisherian "dogma." Thus, we drew from three separate and often conflicting traditions or "sets of dogma" in an effort to fit the analyses to the problem at hand (Readers will also take note of the explicit rationale for the data analytic approach taken by Beatty & Valencic's piece, which was peer reviewed for this issue).

It is not enough to "not be opposed to statistics" when the literature of interest is inherently quantitative. Although molecular genetics holds great potential to contribute to the study of human behavior, any meaningful summary of the state of genetic science must be based on an understanding of the language employed and the mathematical models underlying the quantitative analyses upon which researchers verbal conclusions are based. We, too, are against oversimplication, whether "statistical" or "critical" in nature. As will become evident later in this essay, Condit's interpretation of the genetics-behavior research is not universally shared, even by molecular biologists.

Dismissing our citations. In a single broad-brushed pass at our citations, Condit inaccurately claims that "The McCroskey and Beatty team repeatedly gives long lists of citations about genetics and psychological states, but the careful and industrious reader will find that these are almost exclusively citations to (sic) other members of their team, citations to (sic) pen and pencil studies, including attitude scales and self-reports, twin studies, theorization without empirical support from psychologists (much of it out-dated), and descriptions of biological but not genetic systems." The "careful and industrious reader" might also consider the following, however. First, the qualifier "almost exclusively" is important because (1) even assuming Condit's description of our citations is true (which we will show it is not), and (2) even if the characteristics listed by Condit were cause for rejecting our claims (which they are not), the citations missed by Condit's load of buckshot would be sufficient to introduce a new paradigm. Kuhn (1962) notes that paradigms can be sparked by as little as a single scientific achievement.

Second, it is impossible for "the team" to "give a long list of citations" that are "almost exclusively citations to (sic) other members" of our team. Except for four co-authors (i.e., Dobos, Heisel, Rudd, Valencic) involved in two of the four essays on communibiology published prior to this issue of *Communication Education, there are no other members of our "team."* Unless as a critical scholar, Condit means something other than people who work together by "team," her remark makes no sense.

Third, the attack on paper-and-pencil measures seems unjustified. Some of the research cited, such as the Plomin and Saudino study cited above, did not rely on "paper and pencil" variables. Eley (1997) (cited by Valencic, Beatty, Rudd, Dobos, & Heisel, 1998) reported that "genetic factors accounted for 70% in aggressive symptoms" (p. 94). Other research not yet published when we wrote our initial paper, also conflicts with Condit's indictment of our proposition. For example, Boomsma, Anokhin, and de Geus (1997) collected electroencephalogram (EEG) and event-related potential (ERP) output, in response to several tasks. These researchers found that "individual differences in brain electrical behavior are heritable" (p. 107), Specifically, 50% of the variance was attributable to genetic factors. Moreover, before glossing over the work of prominent personality theorists (e.g., Eysenck, Zuckerman, Telligen, and Costa & McCrae), Condit should note that many of the measures have achieved some measure of predictive validity, and not just in terms of other paper and pencil measures. Dismissing findings merely because self-report data are examined was fashionable in the 80's, but since that time many of the self-report inventories employed in the research cited have undergone successful validation processes.

Fourth, although Condit is not alone in her stance that heritability cannot be inferred from twin studies, it is far from a consensual view. Research articles

continue to appear in recent issues of prestigious mainstream journals such as the *Journal of Personality and Social Psychology* and *Current Trends in Psychological Science,* in which authors (many of whom also conduct research at the molecular level) derive estimates of genetic influence from identical twin designs (e.g., Eley, 1997; Hamer, 1997; Phillips & Matheny, 1997).

Finally, with respect to the wide spectrum of complaints about our references, we redirect Condit to the quotation from David Lykken (1995) that we provided in our response: "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). Indeed, the results of Plomin and Saudino's (1994) study, discussed earlier, linking alleles to single scores derived from observational ratings of children's activity, Boomsma et al.'s (1997) link between genetics and brain activity, and Eley's (1997) study of aggression symptoms are consistent with Lykken's estimate from twin's studies based on single measures and with the average attenuated correlation for traits reported in the twins' literature. Keep in mind that (a) the researchers cited here are psychologists, (b) more than paper and pencil variables were employed, (c) both twin studies and a molecular study are included, (d) none of the citations is profoundly out-of-date, and (e) although we admire the work of Lykken, Plomin, Saudino, Eley, and Boomsma et al., none is a member of "our team."

If we were as blinded by our "enthusiasm" for the paradigm as Condit alleges, Condit would be able to point to scores of communibiological articles by "our team" (reviewed by like-minded narrow, diluted, but enthusiastic team members) appearing in *Communication Monographs,* under Beatty's editorship. The record, however, stands to the contrary although numerous manuscripts supporting communibiology have been received. Our "enthusiasm," and "good intentions" have not overwhelmed our judgment or our respect for the orderly process of scientific development.

Misunderstanding of measurement principles. Our primer in basic measurement principles moved Condit away from the suggestion that we misrepresented the twins research and toward a different but equally untenable argument. According to Condit, correcting correlations for attenuation "presupposes, however, that there is a narrowly coherent underlying variable that is stable, unidimensional, and unaffected by the 'noise' of context, environment, and other biological variables . . . the so-called 'measurement error' is really an accurate indicator of the somewhat diffuse character of the underlying cluster . . ." Condit contends that by assuming what we attempt to measure the argument is "circular." As a preface to our specific responses, we admit that when researchers design measures, they assume there is something to measure.

First, if we worked from Condit's assumption that an underlying variable is diffuse and unstable, the assumption could be confirmed on the basis of a poorly designed and unreliable measure, whether not the underlying variable is diffuse. Clearly, such a posture is logically untenable. Consider, for example, Condit's response to our calculation of the probability of inheriting both extraversion and neuroticism, which we discussed previously. Recall that she did not immediately conclude that co-segregation is a "fuzzy" concept simply because our response to her statement was unanticipated. Instead, Condit retained her belief that co-segregation is a clear concept. She attributed the "misunderstanding" to differences in language use

between social scientists and critical scholars and attempted to repair the problem by further elaborating what she meant by "multiply." Condit could also have tried substituting "proliferation" or "expansive" for "multiply." Researchers, too, must rely on words in the collection of data, whether in the form of self-report questionnaires or observers' descriptions of events. And, like Condit, researchers sometimes need to revise word choices.

We start with the assumption that the construct is viable until evidence indicates otherwise because we want to avoid avoid forming impressions of the construct on the basis of inadequate measurement. Because sources of measurement error can be detected and measures can be revised, researchers are not stuck with the initial or existing versions of measures, and therefore, they are not locked into a circular argument. Items can be deleted, rephrased, added, or differentially weighted in the computation of single scores. While it is true that some constructs are likely to be unstable and diffuse, just as some hypotheses are false, Condit forgets that assumptions can be examined empirically. Before deciding that a construct is diffuse or unstable, however, evidence indicating that the problem resides in the construct rather than the measure should be examined.

Sometimes a measurement problem resides in the response-foils. Again, the extent to which this is so can be checked. Take, for instance, Eysenck, Eysenck, and Barrett's (1985) measure of psychoticism. When we administer the measure to two samples with one form featuring a five-point Likert-type format and the other featuring a Yes-No option, the alpha reliability coefficient is approximately .60 for the Likert-type version and .80 range for the Yes-No version. These reliability coefficients do not indicate that "psychoticism" is more diffuse in one case and more unidimensional in the other. Rather, measurement error has been reduced. Furthermore, if we were to add more items of equal quality to the initial set, the reliability would be further improved in a predictable manner not because "psychoticism" has become less diffuse between administrations of the measure but because reliability coefficients are mathematical functions of both the average correlation among items and number of items (Guilford, 1954).

With respect to undimensionality, psychometric properties sometimes indicate that a measure is tapping a multidimensional construct, consisting of two or more "narrow, unidimensional" subconstructs rather than one diffuse, unstable one. Again, the assumption can be checked. For instance, although the Verbal Aggressiveness Scale (VAS: Infante & Wigley, 1986) has been quite useful in its unidimensional form, our factor analytic investigations (Beatty, Rudd, & Valencic, in press) have produced two stable factors. When entered as separate predictors in a regression equation, significantly higher criterion validity coefficients are obtained than when the unidimensional version of the VAS.

Second, Condit's argument does not explain basic empirical observations. The notion that psychometric properties are indicators of the nature of a construct does not account for (1) why the magnitude correlations in the twins research generally corresponds to the magnitude of the reliability coefficients of the measures, or (2) why higher correlations result when computed on the average of several estimates of a trait rather than scores based on a single test, or (3) why different measures of the *same construct* display *different reliability coefficients*. If the underlying construct is diffuse and unstable rather than the measures being imprecise, none of these empirical

outcomes would be observed because the "diffuseness" of the construct should not by chance covary with the reliability of the measures.

In our short essay, we explicitly stated that "We are not advocating that knowledge claims should be based on corrected correlations." Instead, we presented corrected correlations to illustrate the potential impact of common biology on traits. Sources of measurement error can be and have been identified and reduced in many of the measures we employ and, as the literature shows, such improvements result in larger estimates of heritability.

Oversimplifying the gene behavior link. We never suggested that single genes determine specific behavior. However, as Boomsma and associates (1997) point out, while "identifying the effects of single genes on behavior" may be difficult, "It may be easier to identify the effect of a gene on a more elementary neurobiological trait" (p. 106). Communication apprehension, neuroticism and extraversion are traits, not behaviors, and we suggested that "traits are not inherited but the neurobiological structures are mostly due to heredity" (Beatty & McCroskey, 1998, p. 48). We proposed that "Interpersonal behavior" was "principally due to individual differences in neurobiological functioning" (Beatty & McCroskey, 1998, p. 52). The point was not that genes determine behavior but that because behavior was due to neurobiological functioning, it is not a direct function of environment as defined in social learning theory. Genes, however, heavily influence the development of neurobiological functioning. For example, Hamer (1997), a molecular biologist (Laboratory of Biochemistry, National Cancer Institute) describes research in which "There was a clear correlation between the variation in the serotonin transporter gene and neuroticism . . . people with the highest levels of neuroticism had the shortest version of the gene" (pp. 113–114). In addition to elaborating the anatomical components of the neurobiological systems involved in emotional traits, getting at "how" not just "how much" biology is involved, we explicitly described behavior, not as a direct effect of genes, but as a complex function of the neurobiological systems triggered in response to stimuli. Furthermore, we described the neurobiological components of the interface between environmental stimuli and neurobiological activation.

Before accepting Condit's assertion that we characterize humans as simple machines, we recommend that the "careful and industrious" read our actual discussion (Beatty & McCroskey, 1998, esp., pp. 52-54) of the complex interaction among neurobiological systems, which is consistent with contemporary thinking that complex behaviors are "influenced by multiple neurobiological components" (Boosma, Anokhin, & de Geus, 1997, p. 106). All sorts of biological processes such as the dramatic activity of sperm (some attempting to penetrate, others blocking "rival" sperm) during the fertilization, which are just as complex and full of wonderment as communication, if not more so, occur automatically and without our control. The more one reads about human biological functions, the less one is likely to argue that taking a biological reductionist stance equals simplistic conceptualization.

Although Condit disqualifies herself from addressing neurobiological features of our paradigm, they are essential to appreciate our assumptions about heritability. Gray (1991) maintained that the major dimensions of personality (extraversion, neuroticism, and psychoticism) represented psychological manifestations of individual differences in "parameter values" for neurobiological systems. These parameter differences have been described by Strelau (1994) as "sensitivity to neurons'

postsynaptic receptors or sensitivity in their synaptic transmission, the amount of neurotransmitters being released, the reactivity of the neural structures (including receptors), to different kinds of stimuli, all taking part in determining the individual differences in traits" (p. 135). "Postsynaptic" refers to the neuron driving the impulse away from the synapse, and "neural structures" usually refers to dendrites but cell bodies can receive impulses also (Adams & Victor, 1993). Our initial interest in the genetic contribution to communication resided in the observation that the cell bodies of 12 billion neurons, the building blocks of neurobiological systems, which conduct the electrical charges that in turn stimulate the production of neurotransmitters necessary for synapses among neurons, contain genetic material contributed by the individual's parents. The rate at which a the current travels among axons (an extension from the cell body which varies in diameter and forms the junction with processes such as dendrites from a nearby neuron) conforms to basic laws of physics (e.g., The resistance to an electrical current decreases as the cable diameter increases) (Adams & Victor, 1993). As mentioned, genes seem to influence properties of electrical activity (Boomsma, et al., 1997). However, when we suggested that the smallest unit of communication is neither the dyad nor the individual, but perhaps the neuron, we were thinking in terms of a paradigm (Beatty & McCroskey, 1998), not a theory, not an hypothesis, and certainly not a fact.

We do not believe it unreasonable to question the potential of human adaptability. Certainly, some individuals are quite adaptable, but adaptability varies within the species. The nature of many of our more serious social problems might indicate a lack of adaptability. Prisons are overcrowded with humans who have failed to adapt to cultural systems. Indeed, stress, which results from poorness of fit between individuals and their environments seems pervasive and the widespread self-medication through the (ab)use of alcohol, cigarettes, illegal drugs and prescription medication such as Prozac to supplement human coping efforts seems inconsistent with natural adaptability. Although we do not offer these general observations as scientific proof that adaptation is over-estimated, accurate description of our nature requires consideration of the full range of our behavior.

Concluding Remarks

In our original essay (This Volume), we offered teaching theory and principles of communication, providing information to assist students achieve a better compatibility in romantic relationships, and identifying occupations that might better fit their temperaments (also known as good career advising) as instructional implications of communibiology. We couched our ideas in the form of information or content, not policy. We did not suggest that students cannot or should not expect intellectual growth through college courses. We merely questioned the relative potency of intellectual and emotional forces in shaping action. Nothing remotely resembling Condit's dark vision of a communibiological world can be found in our paper.

In Beatty and Valencic (This Issue), pharmacological options for treating stage fright are mentioned but not advocated. Even as Condit labels such approaches as "futuristic," we note a "health note" in *Parade Magazine* (September 12, 1999, p. 7) announcing that "The FDA has approved the drug Paxil as the first treatment for people with social anxiety disorder. For sufferers, situations like interviewers and public speaking can induce a debilitating self-consciousness and fear of humiliation." Although we are not advocating a "brave new world," as a discipline we need to be brave enough to face the new world. In contrast to Condit's unfounded anxiety

about the debilitating effects our work might have for those interested in biological approaches to communication, we believe our work will stimulate the studies Condit waits to see. Although molecular biology will undoubtedly enhance the study of communication, methodological complexities plague that field also.

Like all other paradigms, communibiology will most assuredly will spawn paradigm variations, theories, and self-examination of our assumptions about our discipline. It is our hope that this essay has clarified the state of our work, and does in fact stimulate research and theory.

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