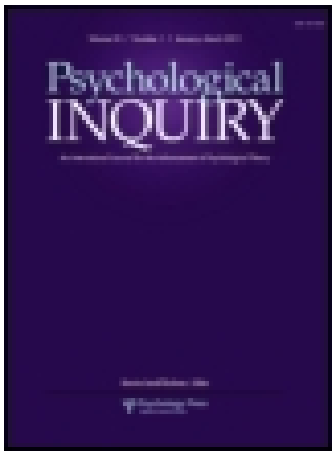

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AUTHOR'S RESPONSE

Creativity and Personality: An Attempt to Bridge Divergent Traditions

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Amabile

I have little to reply to Amabile's very reasonable comments. She is right in saying that my target article "falls short of actually presenting a theory of creativity," but I attempted to offer only "suggestions for a theory." She is also right in stating that "more direct empirical data are needed" in relation to many of my suggestions—and to many deductions that would follow from the theory. Some of her queries are answered elsewhere, if only because the target article was already much longer than usual. Thus, the difference between high–P scorers and psychotics was discussed at length elsewhere (H. J. Eysenck, 1992); there is a large body of evidence relevant to this point, and it could not be dealt with in a paragraph or two.

How does personality give rise to cognitive style, like overinclusion? How, specifically, do both give rise to creativity? Amabile's questions are very much to the point; I can only give a tentative answer. As I mentioned in the target article and described in more detail in my psychoticism article (H. J. Eysenck, 1992), schizophrenia and psychoticism are both quite closely correlated with mechanisms like negative priming (Beech, Baylis, Smithson, & Claridge, 1989; Beech & Claridge, 1987) and latent inhibition (Baruch, Hemsley, & Gray, 1988; Lubow, 1989). These are well-documented mechanisms that produce cognitive inhibitions needed to control overinclusiveness, and diminished or missing in schizophrenics, schizotypals, and high–P scorers. Perhaps here we have the crucial agents that mediate between personality (high P) and cognitive style implicit in trait creativity.

My analysis of product creativity is indeed perfunctory and clearly owes much to Amabile's componential theory; I have little to contribute on that score, and I wanted to get to trait creativity, where I conceive my major contribution to lie. What would constitute testability of my model? It clearly predicts a fairly close relation between trait creativity and (lack of) latent inhibition and/or negative priming; these would be interesting experiments. The literature shows that dopamine antagonists increase and dopamine agonists decrease latent inhibition and negative priming; do these substances have a similar effect on creativity? These and many similar experimental paradigms emerge from my attempt to formulate a testable theory; when they have been tested, we shall know more about its value.

Barron

Frank Barron is an old friend from my early days at Berkeley, when hearing of the Institute for Personality Assessment and Research (IPAR) researches into creativity kindled my interest in the topic through work that still seems to be the best in the field. I know of his argument that "the mark of creativity is elegance" and that, "in mathematics, and in science too, we say of an explanation that it is most elegant when, with a minimum number of postulates, it embraces a maximum number of implications." Is it odd of me to leave this out of account? I have looked at the historical evidence for the belief and have found it wanting. Take the man often regarded as the most creative mathematician of the century, the great Ramanujan (Kanigel, 1991). His production of original ideas was inchoate, lacking proof, volcanic, but certainly not elegant; it needed the much less creative Hardy to bring order and a semblance of elegance into the mess of unfinished business left by Ramanujan. Copernicus set astronomy alight, but his model is an even worse mess than Ptolemy's. Newton's *Principia* may seem elegant, but the elegance was bought by his fudging data (Westfall, 1973). Kepler simply invented many of the data that made his *New Astronomy* appear so elegant (Donahue, 1988). Mendel's account of his work, too, appears elegant because his data were fudged (Fisher, 1936). The aesthetic criterion may sometimes work, but the history of science and mathematics does not encourage reliance on it.

Claridge

Gordon Claridge is another old and valued friend who provided the best evidence there is for a strong link between creativity and psychoticism (Woody & Claridge, 1977); indeed, he managed to "give artistic verisimilitude to an otherwise bald and unconvincing narrative", as the *Mikado* has it. His doubts about the nature of the Psychoticism scale were answered elsewhere (H. J. Eysenck, 1992); I cannot accept his preference for the various schizotypy scales. I recently reanalyzed the correlations between nine schizotypy scales, the P scale, and a small number of other scales—published by Kendler and Hewitt (1992); the major results were:

1. In a group of scales made up almost entirely of schizotypy scales, Psychoticism had the largest communality, in spite of lower reliability, suggesting that P was closest to the intended underlying schizotypy.
2. The schizotypy scales had very high loadings on a Neuroticism (N) factor, suggesting that, unlike the Psychoticism scale, they included a large admixture of N (H. J. Eysenck & Barrett, 1993).

Of course, the point here is that P and N are essentially orthogonal, both theoretically and psychometrically. Such a conclusion emerges not only from our own work (H. J. Eysenck & S. B. G. Eysenck, 1976) but also from many other studies (e.g., H. J. Eysenck & M. W. Eysenck, 1985). J. H. Johnson, Butcher, and H. N. Johnson (1984), to give but one example, factored the whole Minnesota Multiphasic Personality Inventory item pool using two samples of more than 5,000 subjects; the two largest orthogonal factors were P and N. Given that there is strong evidence of independence of P and N, the high correlation usually found between schizotypal scales and N suggests that they are very imperfect measures of P, combining P and N in varying degrees. Their use as measures of psychotic proneness is therefore compromised by a very large addition of irrelevant variance that acts as error variance for the purported use of the scales.

Claridge's criticisms of the P scale may have some substance, but I am unwilling to give up the concept of psychoticism for the reason stated (H. J. Eysenck, 1992).

Csikszentmihalyi

Csikszentmihalyi is a trifle disingenuous and more than a trifle selective when he writes that "less than 3% of the variance in a divergent-thinking test filled out by university students is in common with the variance in their scores on psychoticism (Rushton, 1990)," leaving out the much more elaborate and thorough Woody and Claridge (1977) study, which gave a figure of about 70% of common variance. Altogether, his criticism betrays a much greater interest in achievement creativity than in trait creativity; in my view, both are of interest, and I tried to indicate their relation and to provide a causal theory of the latter. And is it true that "correlations ... tell us nothing about causation or even about process"? Hume already suggested that all we can know about causation is correlation—but I will leave this debate to the philosophers, noting merely that, as I already explained, the theory includes causal elements in the concepts of negative priming and latent inhibition that might account for the overinclusiveness of psychotics and high–P scorers—and of creative people too!

Dudek

Dudek seems to find it reprehensible that the P scale includes no items directly referring to overinclusiveness; this idea fails to do justice to the reasons for creating the scale—namely, to measure not psychotic symptoms but an underlying, normal dispositional dimension. Proof of the postulated relation comes from the correlation of P with overinclusiveness responding on the Word Association Test and with object-sorting tests and the recognized measures of overinclusiveness cited in the target article; it is not clear why Dudek fails to note this confirmation of my hypothesis—a confirmation all the stronger because overinclusive thinking is not mentioned in the P items!

Gedo

I am particularly impressed by Gedo's support from his clinical experience of some of my suggestions; if I did not cite any clinical or psychoanalytic data in the target article, this was due entirely to my reliance on experimental or psychometric evidence. The problem with clinical evidence is that it is so often contradictory; Bleuler prominently included "creative"in his description of the schizoid. Gedo denies any such relation. Who is right? We clearly need evidence from carefully planned, large-scale empirical studies before deciding between conflicting opinions.

I trust that Gedo is not serious in thinking that my theory demands or suggests "that P is always caused by the same factors and that these are exclusively genetic." No geneticist could possibly believe anything of the sort, and our attempts to discover the genetic architecture of P (Eaves, H. J. Eysenck, & Martin, 1989) have tried to sort out the relative contributions to P of many factors, environmental as well as genetic. What is most directly relevant to Gedo's hypothesis is the absence of any evidence for between-family (shared-environment) environmental influences; this would seem to

seriously contradict his view.

However that may be, my theory should in principle be testable clinically on the kind of population Gedo is relying on, and his support is all the more welcome as it comes from the other side of the tracks—however you like to interpret this remark!

Gough

Gough, as one would expect, as another friend of my Berkeley days, remains eminently factual along psychometric and experimental lines. His own work, like that of Barron, has been one major source of inspiration for my own model building. His major concern is with the content of the P scale; I have already suggested that the answer to such objections will be found in my psychoticism article (H. J. Eysenck, 1992), and I will not add to that. But Gough's remarks that there is an obvious opposition with the rigidity and lack of creativity of psychotics cannot go unchallenged. My theory states categorically that P is a trait predisposing high scorers to develop psychotic disorders under specific environmental stress; psychosis adds something qualitatively different to high P, and it is this added something that is destructive of creativity. (The same point is made by Gedo.) Lack of creativity in psychotics is no criticism of a theory linking high creativity with high psychoticism.

Haensly and Reynolds

I have little to disagree with in Haensly and Reynolds's comments. They recognize that my target article was intended as "a doable research agenda", not as a means to control or predict without error. Are the data only correlational, and should we look for "definitive causation"? Cause is not an easy concept, as Hume discovered; can we ever go beyond correlation? We can certainly test experimentally some of the deductions from my theory, such as the facilitative or depressant action on creativity (via latent inhibition or negative priming) of dopamine antagonists and agonists; if many such deductions can be verified, we might use causal language even though sanctification of such language by philosophers may still be withheld.

Is the testing of my "causal chain" really all that problematic? Each step already has some evidence to legitimate it, and, although I would agree that the evidence provided for some of the steps is rather thin, there is no problem in principle in firming it up in more detailed, larger scale, better controlled studies. I think this is true of each link in the chain; our own interest as a department has lain in the development of the theory linking schizophrenia and psychoticism with the processes of latent inhibition and negative priming (about which more anon). If these are indeed responsible for the inhibitory control of our associative processes, and these in turn determine the degree of creativity, as seems possible, we would have a direct experimental approach to the problem of creativity—and pathology! It is, I think, the lack of such a measure that has stymied research for so long; if the target article does nothing but attract researchers to look at the relation between creativity and latent inhibition, efforts in putting it together will be amply rewarded. This stress on process, experimentally defined, may also lessen the doubts expressed concerning "the identification of what constitutes process and what constitutes contributing components in the process."

Haensly and Reynolds seem to agree with Spearman that all processes underlying *g* are noegenetic and hence creative—producing new content. But clearly, fluency is partly independent of *g* and hence must have different antecedents and consequences. Divergent thinking and convergent thinking are both modes of thinking, but they are clearly differentiated; the new or novel product created by the one is new only in the sense of being implied directly by the terms of the problem—but not yet explicitly expressed—whereas the other introduces a much wider range of implications, some of which might never (or only rarely) have been expressed. Of course, most thinking in real life includes elements of both, but experimentally they can be clearly divided.

Harrington

Harrington's first comment, that "new studies are needed," is one with which I obviously concur. Any (somewhat) novel theory rearranging existing data in a different fashion must make a large number of testable predictions in order to qualify for scientific attention; inevitably, only a few of these many possible deductions will already have been tested. Harrington is obviously right in drawing attention, as other critics have done, to the piecemeal nature of the evidence, the lack of crucial bits and pieces, and the doubtful nature of some research data. All this is inevitable, because few if any of the studies cited were done with a (non-existent) theory in mind. It was indeed my hope that publication would lead to stringent testing of many of the deductions that can be made from the theory; thus, I welcome Harrington's comment.

On some specific points, I cannot agree with Harrington. Gough's word-association data are the least convincing of those I cited; the MacKinnon data I quoted are much more convincing. The greater correlation with creativity of unusual but not singular responses I would consider supportive of my theory; associations must be relevant and unusual. I agree that problem finding is as important as problem solving, but I have found no studies linking problem finding with P;

I certainly do not consider such processes as less "basic." Last, Harrington complains about the heterogeneity of the P scale, but this is inevitable in a higher order factor (H. J. Eysenck, 1992). Such a factor is produced by the empirical fact of the correlations appearing between primary traits (including the Agreeableness and Conscientiousness factors Harrington mentions—H. J. Eysenck, 1991). Like many American investigators, Harrington may prefer to work at the primary level, but there are arguments in favor of using higher order concepts (H. J. Eysenck, 1992).

Harrington's criticisms of the P scale are based on very ancient sources dating back to the years immediately following publication of the scale; he disregards the hundreds of studies on the nature of the P scale (H. J. Eysenck, 1992). This evidence should be considered before rejecting the scale.

Martindale

Martindale has made many important and original contributions to the study of creativity, and his approach throughout has seemed to me to resemble mine; hence, I am particularly gratified by his ready acceptance of my major theoretical proposal. I would agree with him in his suggestion that important elements of my theory are not new; the "degeneration" theories of Morel, Lombroso, and Nordau do, as he points out, have some similarities to the notion of "psychoticism" that would be idle to deny. Of course, there are also important differences; in psychotics, the "weakening of higher, inhibitory brain centers," which "allows lower, more primitive functions to emerge in an uncontrolled fashion"—which Martindale describes—is indeed "uncontrolled," whereas in psychoticism there is a degree of control (ego-strength?) that allows creativity or genius to flourish, instead of psychosis.

But of course the major contribution I would claim to have made is metrological. I have attempted (a) to provide a proper measure of this hypothetical degenerative factor and (b) to provide empirical evidence to support the underlying theory that separates psychosis from psychoticism (H. J. Eysenck, 1992). Thus, the theory has become testable and hence enters into the realms of science; the degenerative theory was never truly testable because it lacked the possibility of measurement. My theory is now in a form that makes disproving it possible; that is its major virtue. Also, of course, the theory links the personality disposition with numerous distant and proximal antecedents and consequences, many of which are relevant specifically to creativity and provide it with a (testable!) modus operandi, such as latent inhibition.

Martindale refers to the astonishing fact that "creative achievements tend to occur in waves" and suggests social factors that, he says, "are quite consistent with what we would expect from Eysenck's model of creativity." Actually, there exists a well-supported theory, going back to the Russian historian, Chizhevsky, who produced data to show that revolutions tend to occur cyclically at times of great sunspot activity. Ertel (1991), a German psychologist, suggested that periods of scientific and artistic creativity should then fall at times of low sunspot activity. He convincingly demonstrated the truth of this deduction, not only in Western culture, but also in Chinese, Arabic, and Persian cultures, using completely objective measures of cultural activity to link with sunspot activity. I merely mention this extremely well argued and supported theory to indicate a possible link between the ups and downs in creative manifestations and natural physical occurrences that may also be connected with personality—a possibility perhaps too outrageous to contemplate, but not impossible to test.

Richards

Richards's contribution seems the most helpful and insightful in trying to understand before criticizing and in considering sympathetically even conceptions regarded as erroneous. I think disagreement centers on one major point only—namely, that entitled by Richards "Categories of Psychiatric Disorder." Richards argues that "evidence is strong for distinct families or spectra of psychiatric disorders. Twin-concordance, adoptee, and family-risk studies all support a distinct separation between, for instance, bipolar mood disorders and classical schizophrenia." Of course, this is the classical Kraepelinian dogma, already seen by Kraepelin to be inadequate because of the large number of cases clearly not fitting into it. Crow (1986, 1990) surveyed the literature from the psychiatric point of view and strongly argued for a continuum of functional psychoses, with schizophrenia at the most afflicted end, schizoaffective disorders next, and bipolar disorders least afflicted (very much as in Figure 4 in the target article).

My own position, after reviewing the literature from a psychological point of view, is intermediate between Crowe and Richards (H. J. Eysenck, 1992). Having surveyed eight different sources of evidence, I gave points (from 0 to 5) in accordance with the evidence provided for a general factor of psychotic disorder or for specific disorder factors. Table 1 shows the result; clearly, both sources of variance are present, as indeed I had originally postulated (H. J. Eysenck, 1972). Psychoticism is conceived as the dispositional variable underlying general theories of psychosis; specificity refers to specific genetic and environmental factors making for specific disease processes. Both are clearly present.

One further point may be of interest. Richards refers

Table 1. *Empirical Evidence Favoring Continuum (General) or Categorical (Specificity) Theories of Psychosis*

Source of Evidence	Theories of Psychosis	
	Continuum (General)	Categorical (Specificity)
Distribution of Symptoms	4	1
Symptom Similarity	4	1
Outcome	3	2
Medication	1	4
Biological Abnormality	2	3
Genetic Research: Markers	2	3
Genetic Research: Familial Incidence	2	3
Diagnostic Stability	2	3
Total	20	20

to "extracreativity factors, related to manifest bipolar disorders themselves—including a driven, 'obsessoid,' work-orientation ..." There is good evidence linking obsessional states with schizophrenia and with psychoticism, via negative priming (Enright & Beech, 1990). Similarly, when we (H. J. Eysenck, Barrett, & Jackson, 1993) recently looked at obsessionality traits in a 21-trait complex, using multidimensional scaling techniques, obsessionality lay halfway between the Neuroticism cluster and the Psychoticism cluster.

Much of the research mentioned by Richards has obvious bearing on my theory, usually but not always supporting it. I have a feeling that gradually a consensus is developing that may in due course give us a better understanding of these complex and difficult matters.

Rothenberg

Rothenberg throughout confuses psychoticism and mental illness, in spite of my efforts to point out the major differences between a dispositional trait and a developed illness. He also seems to hold some odd views on correlation. "As the Eysenck Psychoticism scale is based on unusual traits and behaviors, it will be highly correlated with a source of creativity defined as unusual associations or any other type of unusual content." It seems perfectly reasonable to imagine a person with highly unusual, creative associations whose behavior is perfectly normal; if a negative correlation is found, that is an empirical finding, not a tautology. It is curious to have Rothenberg argue that Shakespeare and Mozart lived highly conventional, unrebellious lives. This clearly is untrue of Mozart, and, of course, so little is known of Shakespeare's life that nothing can be proved by citing him.

Rothenberg keeps jumping from trait creativity (which I tried to link with P) to achievement creativity, which I only remotely linked with P.

A last point. Rothenberg insists that, in his research subjects, "the manifestations of the illness invariably interfered with rather than facilitated their creative work." The doubtful syntax does not disguise the fact that, even if true, the observation does not contradict my hypothesis, which centers on psychoticism, not actual psychiatric illness. Is the observation correct? Richards, in her commentary, seems to find exactly the opposite, as does Andreason (1987). The issue is neutral as far as P is concerned but might be of interest in considering the influence of the actual disease process.

Runco

Runco, like Richards, makes a thoughtful contribution. His comments on causality are well taken; there are possibilities other than the one presented in the target article, but, as there appears to be little empirical evidence, I preferred not to discuss them. Some of his points are of more semantic than substantive interest, such as the either/or description of personality and cognition. There is a dichotomy between personality and intelligence; P clearly belongs to the personality side. Many people have argued that creativity belongs to the intelligence-ability side; I am suggesting that cognitive style may be an aspect of personality. But all these terms are used in such different ways by different psychologists that any meaning attaching to them must be fuzzy.

I don't think that the trait view "[plays] down the impact of situations and intrapersonal variability"; clearly, one cannot be creative in every situation or without special abilities or knowledge relevant to a given task. I tried to make this clear in Figure 2 of the target article. The distinction between problem solving and problem definition is important but might be artificial—I might have to solve the problem of how to define a problem. But, as I have pointed out before, one is constrained by the existing evidence, and I know of no relevant studies looking at the relation between P and defining a problem. The same applies to Runco's remarks about nonverbal associative tendencies—my theory would make predictions here also, but none have been tested. However, it is not true that I "dismiss" divergent-thinking tests. For instance, I place much weight on the Woody and Claridge (1977) study, which used such tests. I am merely suggesting that they might not be the perfect answer to our needs for a test of creativity.

Runco's doubts about appropriateness/relevance are well taken. Such concepts are difficult to define and measure, although in the extreme they might not be so. Gough gives good examples in his commentary, and

they could be multiplied. When I am thinking about the Schwartszchild horizon defining a black hole, ideas about the causes of the Pelopennesian wars or about Haydn's habit of plagiarizing would be irrelevant. However, specific measurement in specific test situations obviously presents great problems, and, in science generally, paradigm differences often lead to differences in considerations of relevance. In the debate between Linus Pauling and Moertel on the curative effects of vitamin C, Pauling considered the absence of citotoxic treatment essential, whereas Moertel considered it irrelevant (Richards, 1991). Who is right? Only the future will tell—and you cannot base the scoring of a test on what the future may hold!

Last, I fully agree with Runco's concluding remarks on education. In the United Kingdon, there has been an unholy alliance between trendy professors of education and left-wing politicians aiming to make education "child-centered," by which is meant emphasizing creativity without content and refusing to impart factual knowledge and training. The result was a wholesale breakdown in education, leading to an adult population illiterate, innumerate, and totally uncreative! Runco's warning is only too necessary to prevent this type of educational bandwagon from rolling too far!

Simonton

Simonton has for a long time seemed to me the most outstanding individual to have concentrated on research into creativity and genius, comparable only to the joint efforts of IPAR in its effectiveness. His measured praise for my efforts, therefore, are all the more welcome. When I criticized his and Campbell's chance theory of creativity, I felt guilty of lèse majesté, issuing in feelings of distress, guilt, and anguish—at least as far as this is possible in someone with a score of zero on Neuroticism! I am therefore very happy to read Simonton's reorientation or—as he would no doubt prefer to put it—clarification. The point at issue is a vital one for any theory of creativity, and I find it odd that hardly anyone other than Furneaux has given much thought to it—with results very similar to the Campbell–Simonton theory. Simonton's present formulation of the theory is certainly much more similar to mine and leaves both of us with the task of operationally defining *relevance,* or whatever corresponds to it in Simonton's account (I assume that *configuration* assumes that role).

What seems to me new in the present formulations of his theory is Simonton's concept of "problem space." It is the apparent absence of this notion of what is relevant that I missed in his original presentation (as well as in Campbell's and Furneaux's theories); its inclusion certainly makes my criticism outdated. I don't think I misconstrued Simonton's use of the mental elements (as he calls them), which enter into the selection process. As regards *chance,* Simonton's present explanation of his use of the term is perfectly acceptable to me, but, rereading the original presentation of his theory, I don't feel that one can easily read the present meaning into it. However accurate or inaccurate my original interpretation of Simonton's theory, I feel greatly relieved that our paths seem to converge; one does not enjoy being opposed to Galton's successor in the creativity-genius field!

Sternberg and Lubart

Sternberg and Lubart note many similarities between their theory and mine. The criticisms they put forward do not appear to me very crucial. Is the theory clearly stated? It is stated sufficiently clearly to give rise to testable deductions; that is surely a virtue. But quite generally in science, novel theories cannot be stated "clearly"; by definition, the evidence to do so is not available. I have discussed the difference between "weak" and "strong" theories in some detail (H. J. Eysenck, 1960, 1985). Mine, like nearly all psychological theories, is a weak theory and has to be judged by criteria other than those applying to strong theories, like those in the hard sciences.

Psychoticism is criticized on semantic grounds, as putting off laypersons. Sternberg and Lubart may call the trait what they like; the name is justified on scientific grounds (H. J. Eysenck, 1992), and that should be enough—public-relations concerns seem irrelevant to me.

Are the tests used by Woody and Claridge (1977) to measure creativity "trivial"? That P correlates both with these tests and with true creative achievement seems notable to me and does seem to support my theory.

What Sternberg and Lubart say about genetic aspects of creativity seems rather curious. They write: "It is important to remember that even attributes with a partially genetic basis can be developed." Who ever doubted it? My target article was written for psychologists, not for laypersons who might harbor odd thoughts about that. Equally odd is the comment about "a person who believes that most psychological attributes have a genetic explanation". The evidence is pretty conclusive that there is genetic involvement in pretty much everything we do. Would Sternberg and Lubart deny it? But nobody denies that environmental factors also play a part. So what does their sentence imply? Are they suggesting some sort of prejudice? I would certainly not deny that "the attributes of creativity are susceptible to development"; I only wish we knew how to do that!

Torrance

Torrance is another of the outstanding pioneers in this field, and he is one of the few who has tried to answer this question creatively. His work is well known, and his recent book, *The Incubation Model of Teaching,* certainly suggests possible ways of meeting the problem. It is too early to judge its success; does it really increase creative achievement? It is possible to argue that the creative person, with a high P score, might actually benefit from having early struggles with an uncomprehending public—teachers, parents, peers. Whatever the truth, I agree with Torrance that a process focus is of great value in approaching creativity, and I also find that this focus is quite close to personality. After all, negative-priming and latent-inhibition research is process research deriving its interest from personality! Spearman has certainly tried to combine the two, although few American psychologists are aware of any of his writings other than those concerned with psychometric *g;* I am happy that Torrance shares my admiration for Spearman.

Waller, Bouchard, Lykken, Tellegen, and Blacker

Waller et al.'s comments and their genetic data on the part emergenesis plays in the genetic determination of trait creativity constitute valuable additions to the model I am trying to develop. The differentation between the heritable and the familial in trait development is important and should be more widely known; epistasis is an unknown quantity for most psychologists! Waller et al. contrast the genetic architecture of creativity and psychoticism, but they do not mention more recent studies (e.g., Heath, Eaves, & Martin, 1985) that underline the existence of a more complex genetic structure of P than that assumed by them. However, there is no doubt that they have indicated the existence of a problem and furnished us with a welcome new model of analysis and explanation—of the previously difficult-to-understand very low DZ correlations for personality traits and now for creativity.

General Remarks

So much for specific replies. I would also like to make one or two more general points. Several commentators have remarked on the somewhat unusual nature of my target article; this has been due to two major views I have long held about the scientific method and its application to psychology in particular. In his recent book, Danziger (1990) traced three research models that eventually developed into the widely accepted paradigms for psychological research—the experimental (Wundt), the psychometric (Galton), and the clinical (Binet?). Unfortunately, as Cronbach (1957) argued so long ago, there has been little mutual recognition of the Galton and Wundt traditions, and the clinical tradition is even further removed from the other two than they are from each other. Actually, there is a fourth model that has been left out unaccountably from Danziger's list—namely, the sociobiological, relying on evolutionary insights (Darwin), psychophysiology (Helmboltz), genetics (Galton), and conditioning (Pavlov).

I have always agreed with Cronbach that a scientific psychology cannot exist as a house divided against itself, and I have tried to combine as far as possible in any research all four strands. Hence, the superimposition in my target article of elements from all four areas—a combination that will appear strange to all those who have worked consistently in one discipline alone.

For these reasons, I put much emphasis on the section entitled "The Causal Chain From DNA to Creativity"—in spite of the open derision with which Sternberg and Lubart treat it. I feel sure that a proper theory of creativity has to take some such form as that suggested, even though the details may be completely misconceived and subject to change. Of course, the attempt to reconcile four such very divergent (and often hostile) traditions, methods of arguing, and empirical procedures is strewn with problems and difficulties, and no easy success can be expected. Trying to fit together date of birth, negative priming, dopamine agonists, primary process, schizotypy, additive genetic variance, word association, artistic excellence, overinclusiveness, and many more seems from the beginning a hopeless undertaking, but I feel strongly that nothing less will do to rescue work on creativity from one-sidedness and neglect of important variables and concepts from outside the theorist's own paradigm. No doubt others will do the task better, but a first attempt seemed worthwhile. We will have to learn one another's language, look at one another's data, and try to understand one another's paradigm. The alternative is the Tower of Babel atmosphere that characterizes so much of modern psychology.

My second remark concerns the important difference between weak and strong theories (H. J. Eysenck, 1960), to which I have already alluded. Consider the nature of a scientific experiment (Cohen & Nagel, 1936). Cohen and Nagel take as their example Foucault's famous experiment in which he showed that light travels faster in air than in water. This was considered a crucial experiment for deciding between two hypotheses: Hypothesis 1 is that light consists of very small particles traveling with enormous speeds, and Hypothesis 2 is that light is a form of wave motion. Hypothesis 1 implies Proposition 1 (that the velocity of

light in water is greater than the velocity of light in air), and Hypothesis 2 implies Proposition 2 (that the velocity of light in water is less than the velocity of light in air). According to the doctrine of crucial experiments, the corpuscular hypothesis of light should have been banished to limbo once and for all. However, as is well known, contemporary physics has revived the corpuscular theory in order to explain certain optical effects that cannot be explained by the wave theory. What went wrong?

As Cohen and Nagel (1936) pointed out, in order to deduce Proposition 1 from Hypothesis 1 and in order to perform Foucault's experiment, many other assumptions, *K*, must be made about the nature of light and the instruments we employ in measuring its velocity. Consequently, it is not Hypothesis 1 alone that is being put to the test by the experiment—it is Hypothesis 1 and *K*. The logic of the crucial experiment may therefore be put in this fashion. If Hypothesis 1 and *K*, then Proposition 1; if our own experiment shows Proposition 1 to be false, then either Hypothesis 1 is false or *K* (in part or complete) is false. If we have good grounds for believing that *K* is not false, Hypothesis 1 is refuted by the experiment. Nevertheless, the experiment really tests both Hypothesis 1 and *K*. If, in the interest of the coherence of our knowledge, it is found necessary to revise the assumptions included in *K*, the crucial experiment must be reinterpreted, and it need not then decide against Hypothesis 1.

We may now indicate the relevance of this discussion to our distinction between weak and strong theories. Strong theories are elaborated on the basis of a large, well-founded, and experimentally based set of assumptions, *K*, so that the results of new experiments are interpreted almost exclusively in terms of the light they throw on Hypothesis 1, Hypothesis 2 ... Hypothesis *n*. Weak theories lack such a basis, and results of new experiments might be interpreted with almost equal ease as disproving a hypothesis or disproving *K*. The relative importance of *K* can of course vary continuously, giving rise to a continuum; the use of the terms *strong* and *weak* is merely intended to refer to the extremes of this continuum, not to suggest the existence of two quite separate types of theories. In psychology, *K* is infinitely less strong than it is in physics, and, consequently, theories in psychology inevitably lie toward the weaker pole.

Weak theories in science, then, generate research the main function of which is to investigate certain problems that, but for the theory in question, would not have arisen in that particular form; their main purpose is not to generate predictions the main use of which is the direct verification or "infirmation" of the theory. It is for this reason that I have assembled a multitude of empirical facts that are relevant to my theory but that cannot directly either prove or disprove it. It is noteworthy that eminent people and psychotics tend to be born at the same time of year; how strongly does this support a theory linking psychoticism to creativity and genius? Would a negative association disprove the theory? How relevant is the finding, and how can we nail it down more closely? These are the kinds of questions that arise from weak theories; such theories are to be judged as progressive or degenerating (Lakatos, 1968; Lakatos & Musgrave, 1970), not as true or false. Unfortunately, psychologists still follow the siren song of the outdated "received view" (i.e., the logical positivist position), despite the almost universal rejection of that view (Suppe, 1974).

Of course, even strong theories are nothing like as strong as the adoring multitude outside seems to think. Newton and Kepler claimed to have achieved almost perfect accuracy in their predictions, but, as I have pointed out, they achieved this by fudging or inventing data to suit their predictions (Donahue, 1988; Westfall, 1973). Even in the hard sciences, theory testing is never remotely as simple as many people imagine.

One important deduction from this view of theories as weak or strong is that, for weak theories, positive results are much more important than negative results (*pace* Popper), because a positive result from testing a deduction from a theory suggests that both hypothesis and *K* are correct, whereas a negative result is difficult to interpret, being quite likely due to *K*. For strong theories, negative results are much more important, as the role of *K* has been much reduced—hence, disconfirmation of the hypothesis is much more likely.

This brief historical excursion is relevant to a point made by several commentators—namely, that many of the "facts" I have adduced to support my theory are weak, that alternative explanations are possible, or that their relevance is in doubt. All this is true, but such facts do serve to create that "nomological network" that is required to produce construct validity (Cronbach & Meehl, 1955; Garber & Strassberg, 1991). Such a network can be strengthened or weakened by single experiments, and it is to be hoped that, in doing so, those undertaking the work will remember that we are dealing with a weak theory, so that alleged disconfirmation may be due to faulty assumptions about *K* rather than faults in the construction of the hypothesis.

To conclude this discussion, I want to stress that what to many commentators appear to be serious faults in the structure of the target article are the conscious outcomes of a philosophy of science that has developed over many years and that has benefited from discussions with many experts, from Lakatos to Feyerabend. This approach demands the kind of wide-ranging, inclusive (possibly overinclusive!), comprehensive listing of possible or likely associations and causal links

that I have presented. Of course, it is not a closely knit, highly specific theory amenable to direct testing; such a theory is not possible at the present time. The best we can do is to suggest lines of research that may throw new light on the complex phenomenons of creativity, help explain some of the empirical findings and associations, and, above all, tie together the divergent theories deriving from experimental, psychometric, biological, and clinical backgrounds.

All these points are, of course, debatable; I have mentioned them to indicate that, if my development of the theory of creativity is faulted, the faults are not accidental but intentional. They derive from a philosophical position that seems defensible even though it may not find many friends in psychology. But, if our aim is a unified science of psychology, we must really pay some attention to Cronbach's advice.

Note

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