Chapter 20
Molecular creativity, genius and madness

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1. INTRODUCTION

Creativity has always been important for the survival of individuals, the group and society in general, and its importance is likely to grow with time. Energy becomes more and more concentrated in chemical and physical systems. Political, executive, and military powers can be canalized electronically by a light touch of a button in an increasingly complex high-tech world. Just one unimaginative political, industrial, or military leader may, under the worst possible conditions, cause havoc, where the consequences of similar acts would previously have taken a more local and less damaging form. Large modern technical corporations now go broke by a few unwise decisions, and others prosper with a single stroke of genius and enjoy previously unheard-of profit. Researchers are now pressed to identify creativity and teachers to cultivate it. The launching of the first Soviet sputnik illustrates the point. Shortly after this event there was a sudden and hectic interest in the U.S.A. to find better ways to promote creativity and put the nation back in its leading role. Systematic comparisons among nations began to appear, leading nations found themselves lagging behind, and everybody agreed that something had to be done. Where are we today?

Over time we see many attempts to define, measure, and promote creativity—to mention but a few, Albert (1992), Amabile (1983), Cattell (1903), Cattell and Drevdahl (1955), Cox (1926), Glover, Ronning, and Reynolds (1989), M. Goertzel, V. Goertzel, and T. Goertzel (1978), V. Goertzel and M. G. Goertzel (1962), Guilford (1950), Jackson and Rushton (1987), Kasparson (1978), Lombroso (1901), MacKinnon (1961), Mansfield and Busse (1981), Ochse (1990), Radford (1990), Roe (1953), Sternberg (1985), Terman (1925), Terman and Oden (1947), Vernon (1982), and Zuckerman (1977).

What is the general lesson emanating from this impressive amount of research? Do we see fair agreement about the precise and empirical definition of creativity? Have we achieved a useful identification of relevant causes of

creativity and do we see transparent operationalization of mediating mechanisms? Do researchers agree on the proper level for analyzing creativity, or on the number of analytic dimensions needed to describe it? Do explanations demonstrate unity and coherence? Have the educational systems finally been handed powerful tools for cultivating creativity or, at least, been provided with the means for not standing in the way of promising creativity or seeing itself brutally suppressing it?

I am afraid the answer is: No. Neither can any of the questions can be answered in the affirmative, nor am I the only pessimist. The recent *Handbook of Creativity* by Glover et al. (1989) came to the extraordinary conclusion that creativity research seems to degenerate. There is something terribly wrong here: Creativity increases in importance in modern societies, but research on it stalls. One possible reaction to this situation is to try and redefine the problem and/or to find new ways of studying it.

Both paths are chosen in this chapter. Obviously, such a radical move involves heavy risks and little promise of success. But what is there to loose? The chapter comes in three sections. The first section briefly discusses major obstacles in contemporary creativity research. Section two presents Hans Eysenck's way of attacking them. The last section redefines creativity, genius, and madness in purely physico-chemical terms, and suggests an entirely molecular natural science approach to study them (Nyborg, 1994).

2. OBSTACLES TO CURRENT RESEARCH ON CREATIVITY

Extraordinary creativity often pops up quite unexpectedly in a family, and there are surprisingly few records of families producing two pure geniuses in succession. Genius sometimes blossoms in poverty-ridden places with no good schools, or surfaces in homes entirely lacking in academic tradition. There are moving stories of how extremely creative individuals survived despite growing up in the prohibitive shadow of terrified or disappointed parents, not understanding a word spoken by their strange child, or in strong opposition to orthodox teachers or a church that knows better. The unfolding of genius in such places or under such circumstances remains a complete mystery to contemporary research on creativity, and challenges theories of creativity based on socialization or role-modeling. The many accounts of outrageous repression of true genius illustrate some further points. Extraordinary creativity is often seen neither as a blessing to the person in question, nor to the immediate surroundings. Sometimes no-one profits from it for a long time or ever. The many fascinating life-history accounts of geniuses rarely reveal anything of scientific interest with regard to the springs of extraordinary creativity. Both favorable and oppressive circumstances may befit the unfolding of true genius. It is unfortunate, that while detailed life-history accounts

certainly illustrate the circumstances (favorable or not) under which genius unfolds, they typically reveal nothing about how the genius came to his extraordinary capacity.

The genetic aspect of genius is also essentially untrodden land. Galton's (1869/1978) study led him to believe that inheritance explained much of the individual variability in genius. This conclusion is beset with problems. In fact, Galton studied acknowledged excellence in various areas rather than genius in any precise sense. Worse, Eysenck (1995) found that Galton actually turned the excellence–inheritance argument on its head, so that his results are probably better explained in terms of the environment. Studies specifically designed to reveal the genetic basis for creativity, such as twin studies, give disappointing results. Nichols (1978) found a modest 20% genetic influence, and when Canter (1973) controlled for IQ, the genetic impact shrank to nothing. The few designated genetic studies thus show weak, or no familial transmission of creativity. They neither tell us where extremely high creativity comes from, nor why a genius rarely, if ever, leaves offspring of equal standing. The discussion of "emergenesis" by Lykken (1982) and Lykken, McGue, Tellegen, & Bouchard (1992) might throw new light on why creativity suddenly appears in a family and still may have a significant genetic component, but right now we simply have too few genetic data to say anything of scientific importance on the matter. The study of a common genetic basis for familial aggregation of creativity and psychopathology may hold more promises (see chapters 6, 17, and 19).

Socialization theory fares no better than genetic theory in explaining the facts. There are many books and courses guaranteeing quick and easy progress in the promotion of personal, occupational, scholastic, scientific, or artistic creativity. None of the promises translate into verifiable generalized effects on creative ability or achievement, however. Children as well as adults can be taught how to take creativity tests, and this certainly raises their creativity score, but the training seems not to generalize to anything useful outside the realm of taking the test, quite like training intelligence.

To sum up, high creativity can neither be explained by anecdotal evidence, nor by contemporary genetic, social, or pedagogical theory.

In real science such a confusing state in an important area would have attracted an army of scientists equipped with a healthy taste for stringent basic research. They would eagerly try to map verifiable causes and mechanisms, bring creativity under tight experimental control and then develop proper means for furthering it. Apparently, this is not what happens in traditional psychology and sociology of creativity. Some researchers continue to study biographies or talk to creative people, to see how they themselves think they do their unusual tricks. Others study the childhood of precocious children or adults, or try to establish connections between creativity, personality, ability, and the environment by questionnaires and factor analyses. Still others train

people in "alternative" or "flexible" thinking (often for good money), but rarely follow up on the decisive question of whether the training generalized to creative acts in related or different contexts. Recently, it has become fashionable to put the cart before the horse, that is, to first develop complex theories of multiple intelligence and creativity, and then to defend or excuse these theories in the absence of hard experimental evidence and solid predictions.

The acid test of any approach to creativity is, of course, to skip the grand visions and begin asking pertinent questions about the psychometric properties, the quality of the experimental control, the predictive value, and the pedagogical effect of the work. However, contemporary research does not even offer a clear answer to simple questions like: "Can you, on basis of your theory or practice, tell me how to train the creativity of this particular child or adult, and can you guarantee that the treatment has lasting and generalizable effects?"; "Can you, in retrospect, identify the specific factors that, given your theory, explain beyond reasonable doubt why this particular person became extraordinarily creative, and the other person not?" As mentioned, families with aggregation for psychopathology also tend to show aggregation for creativity. However, the notion of common or codominant genes begs the question: Which genes? To say that creativity or genius is a product of society, active learning or role-modeling is to beg the question of why controlled socialization and specific learning experiments utterly fail in explaining or promoting anything creative. Glover et al. (1989) seem indeed justified in their critical view of much contemporary creativity research. Instead of leading to new glorious heights, it basically produces more anecdotes, more untestable theories, and more excuses for predictive impotence.

3. HANS EYSENCK'S APPROACH TO CREATIVITY

3.1 Introduction

Eysenck entered the area of creativity research in the early 1980s, but he certainly did not start from scratch. Many years earlier he contemplated how creativity squares with intelligence. With his sound habit of posing simple questions in complicated matters, he first inquired into whether creativity is a cognitive ability or a personality trait (Eysenck, 1983). He also wondered whether creativity squares with S. Eysenck's and his own psychoticism (P) dimension (H. J. Eysenck & S. B. G. Eysenck, 1975; Eysenck, 1989, 1993a). In the early 1990s (Eysenck, 1993b), he was finally prepared to sketch a new theory of creativity. Two years later he wanted to deal more extensively with the complexity of the phenomenon, and published a comprehensive review of the field with the most recent statements about his causal creativity model (Eysenck, 1995). The book *Genius: The Natural History of Creativity* is a masterpiece of clarity, and probably one of the best books he has ever written—and that amounts to something. The *Genius* book exposes various

positions on creativity and evaluates them in terms of theoretical importance and degree of empirical support. However, rather than repeating all the details, I will here first outline Eysenck's motives for writing the book, then briefly discuss his creativity model in terms of selected details, and finally evaluate his general approach to creativity.

3.2 In search of answers

Eysenck (1995) struggled for clear answers to questions like: "Can genius be defined and measured?"; "Can creativity be defined and measured?"; "What role does intelligence play in the development of either?"; "What is the contribution of personality?"; "Is there any relation between genius and "madness," and if so what is it?"; "Can we formulate a cognitive theory to account for creativity, and describe the workings of the creative mind?"; "Can we define and measure intuition, as one of the alleged characteristics of the creative person?"; "What is the role of the unconscious, if any?" (p. 2). If a genius–mad connection can be established, then " ... how does pathology [causally] produce creativity? ... . Why ... how ... ?" (p. 123).

Eysenck proceeds in the best tradition of psychology to get the answers. He settles for a *cognitive theory* about the workings of the *creative mind,* and looks for the *psychological factors* promoting it. Eysenck prefers measures to postulates, however imperfect, unlike so many psychologists. It worries him that many psychologists search for "facts" without stating an a priori theory. He therefore recommends that they first establish such a theory, however fallible. He explicitly denounces the anecdotal–historical method, because it exposes one to the risk of succumbing to errors of the unaided memory and self-justifying introspections. He further sees a purely psychometric approach as weak because it is more descriptive than explicative. What basically is lacking in most creativity research is, in Eysenck's words: "... a proper reference to the storehouse of knowledge accumulated by experimental psychologists" (Eysenck, 1995, p. 6). His countermove is, accordingly, to draw on psychophysiological, genetic, and psychopathological research on the brain, creativity, and genius.

3.3 Eysenck's model for creativity

Eysenck's model for creativity is illustrated in Figure 20.1.

Briefly, most variables in the model are influenced by genes. The hippocampus is an important physiological formation for creativity. Dopamine enhances, and serotonin reduces, trait creativity by directly affecting cognitive inhibition. Dopamine thus reduces latent inhibition and negative priming, thereby widening the associative horizon. Remote elements can then be easily combined in a creative fashion. However, too high dopamine concentrations

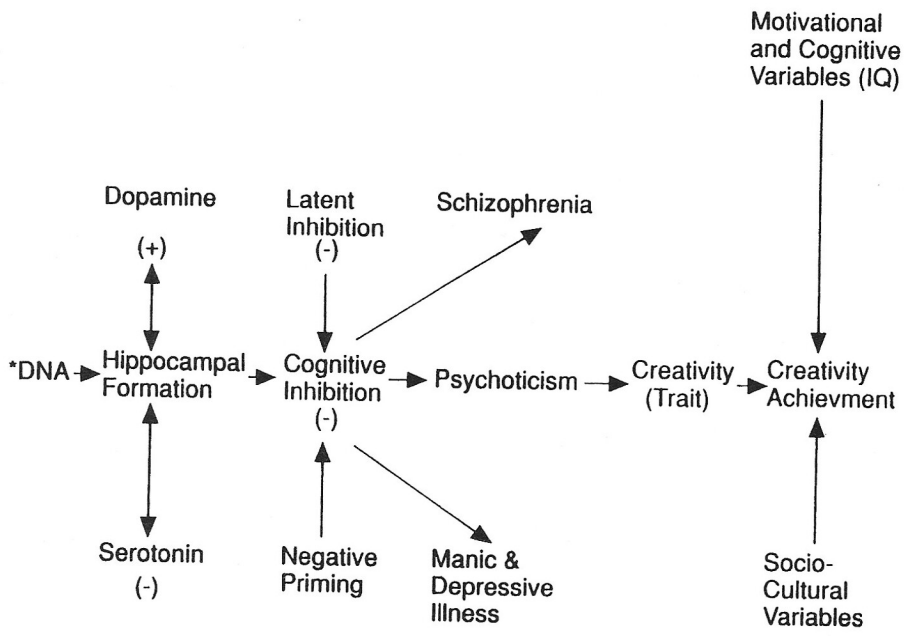

Figure 20.1. Eysenck's causal model for creativity (see text for explanation). From Eysenck (1995) with permission.

link to functional psychoses and low creativity. The impact of increasing dopamine is effectuated along a continuous spectrum, ranging from no creativity to creativity to manic-depressive illness over schizo-affective disturbances to full-blown schizophrenia at very high concentrations. In other words, too little dopamine results in enhanced latent inhibition and negative priming, a steep association gradient, a narrow horizon for combining remote elements, low creativity, low P score, and altruism. Too much dopamine leads to a high P score, lack of inhibition, and negative priming, a flat association gradient, a too wide horizon for orderly combination of remote elements, low creativity, and acute psychosis. Moderate to high dopamine levels result in suitable cognitive inhibition, a moderately tilted association gradient, an optimal association horizon, fairly high P, high creativity, or genius. However, the high dopamine level increases the likelihood of seeing milder psychopathology. Serotonin has the opposite effects.

The left side of Eysenck's model explains, in other words, the circumstances under which individual trait creativity comes to life. But even though *creativity as trait* is a necessary basis, it is not a sufficient basis for proper *creative*

achievement. The right side of the model therefore completes the story. In addition to trait creativity, above average achievement requires an IQ above 120, the presence of personality traits like ego-strength and persistence, and also suitable sociocultural circumstances.

An essential aspect of Eysenck's model is that the factors forming extremely high creativity act synergistically rather than additively. This explains why genius is so rare. The idea is that, if just one of the necessary factors is missing, the product will automatically be only a little creativity and certainly no genius. This is precisely what happens for most of us, according to the model. We may, for example, be exceptionally good at combining remote elements, but if we lack, say, the stamina or guts needed for an almost obsessive follow-up phase, we will never become a genius. The multiplicative formula translates into a J-shaped distribution for creative achievement in a given population. A huge number of nonachievers sleep at the bottom, some high achievers ascend, but only a few isolated geniuses throne at the top.

Eysenck hastens to admit that his theory has not yet passed the acid test—a direct study of genius in accordance with the model. We must at present, therefore, remain satisfied with much encouraging indirect confirmatory evidence emanating from several studies, including some of his own. The next step amounts, in Eysenck's own words, to " ... a lot more work ... before we can hope to articulate a unified theory making possible serious efforts at falsification" (Eysenck, 1995, p. 284).

It is easy to see why Eysenck's half psychological–half brain physiological approach to creativity is superior to most psychological attempts. First, he explicitly takes the scientific route and differs in this from the many intuitively based explanations of genius in terms of divine insight, talents, gifts, deep contemplation, or dreamlike revelations. Eysenck prefers testable hypotheses to the reified concepts some use on a purely descriptive basis as causal variables to explain changes in other reified variables. Instead of undisciplined speculations about multiple dimensions or a myopic focus on abstract or hypothetical variables, Eysenck wants to deal with the task in terms of measurable parameters of brain physiology. Eysenck never remains satisfied with questionnaire data (although he certainly masters the technique), correlations, purely descriptive psychometry, or factor analysis for the sake of factor analysis. He insists on the full exploitation of the power of well-designed experiments.

3.4 PROBLEMS WITH EYSENCK'S MODEL FOR CREATIVITY

If Eysenck admits that the holy grail of genius has not yet been found, then what is still missing? Obviously, one thing is to acknowledge that genes play an important role for creativity, but which genes, which proteins do they code for,

where do these proteins go in the body and brain, and what exactly do they do in the target tissues that affects creativity? Of course, Eysenck is well aware of the problem. This is why he directs our attention to the great research potentials in molecular genetics and biology. Another problem is to pronounce dopamine *the* key factor linking genius, high P score, and increased risk of psychopathology. Dopamine (and serotonin) is probably both heavily implicated in all three, but is it the key factor or a covariate? The recent progress in brain sciences confirms that the brain houses an immensely complex, dynamic, truly interactive molecular chemistry, characterized by multiple reciprocally interactive links between genes, neurotransmitter pathways, and the environment. Too narrow a focus on only one or a few conspicuous agents or links may miss the intricacies of dynamic positive or negative feedback, or feedforward mechanisms. Moreover, much important molecular information is embedded in the release pattern and temporal variations in concentration, inactivation, and receptor sensitivity. This means that simple measures of absolute concentrations may not reflect the full or interesting part of the biological effect of a chemical agent. Eysenck obviously knows this, and his choice to at least start somewhere in a very complex brain and then see where it leads makes perfect sense. Dopamine (and serotonin) might actually not be a bad point of departure. However, accepting for the moment the hypothesis that high dopamine concentration holds the key to an understanding of high creativity, a new problem knocks on the door. Will the dopamine hypothesis explain the remarkable sex-related differences in creativity? As we ascend the ladder leading to genius, we observe still fewer females. Close to the top there are almost no females, in particular in the more technically based areas of science—those drawing heavily on visuo-spatial or mathematical skills. The hypothesis implies that females must have, relatively speaking, much less dopamine (or more serotonin) than males, and therefore score lower P and creativity. They could also have much more dopamine, according to the model, but this would mean a much higher P and many more psychotic females than males. Is there such a marked sex difference in dopamine, and does it explain the sex-related difference in creativity? A fourth problem is to evaluate the proper (causal?) role of P in creativity (see chapter 6). Eysenck insists that his model is dimensional rather than categorial, and that there is a continuum from common to creative to genius to mad. Along that continuum runs an increasingly higher P score. To be sure, P reflects a dispositional variable closely related to, but neither identical with creativity nor with psychosis. Now, to which extent are creative individuals, and at least some of their relatives, increasingly characterizable by traits like: criminal, impulsive, hostile, aggressive, psychopathic, or schizoid (or, periodically, unipolar depressive, affective disordered, or schizo-affective when not in a creative phase, or downright schizophrenic and not ever creative), as opposed to noncreatives showing conformist, conventional, emphatic, socialized, or truly

altruistic traits. Eysenck hastens to regret that we simply have too little good data to be sure, but it certainly is quite easy to point to families with exceptionally creative historical or contemporary individuals, with an increased incidence of psychoticism-like personality traits or psychosis (see chapters 6 and 19). Creativity relates in this way to schizophrenia (Eysenck, 1983, 1987, 1995; Heston 1966; Karlsson 1968, 1970; McNeil 1971), and both Hammer and Zubin (1968), and Jarvik and Chadwick (1973) suggested that there may be a common genetic basis for great potential and for psychopathological deviation (also see Rosenhan & Seligman 1989). Claridge (1990, see also chapter 17) came to the conclusion that creativity may be more closely associated with affective disorders than with schizophrenia, and Jamison (1989) found that 38% of all eminent British authors and artists needed treatment at least once in their life for affective disorder. But then again, there are also examples of healthy families with unaffected highly creative achievers (unless excessive working style is defined as psychopathology). It is hard to know the exact figures, because most reviews of the psychopathology of geniuses are prepared from a particular perspective. The point is therefore not that P has little or nothing to do with creativity, but rather that the connection may not be simple. The psychometric peculiarities of the P scale may also muddy the water (see section 6.6 in this chapter, and also chapter 17).

The perhaps most serious problem I see with the psychological approach to creativity and genius relates to the notion of a (multiplicative) relationship among causal factors. Like Eysenck, I see several good reasons for a multiplicative rather than an additive relationship among the causal agents producing genius, but it is the psychological approach that begs a crucial question: What multiplies with what? Frankly, it makes no sense in precise experimentally operationalizable terms to multiply a favorable brain dopamine concentration with suitable cognitive inhibition and descriptive phenotypic ego-strength, introversion, and dominance to get the product—high creativity. To multiply fundamentally different chemical (molecular), infered psychological (guessed) and descriptive psychometric (phenotypically observed) factors amounts to committing Rylian category errors!

Obviously, this brief and highly selective excursion pays no justice to Eysenck's general and overwhelming contribution to the area of creativity. The narrow mission was to discuss basic elements in his model for creativity and to see where it raises problems. The more general purpose was to show that his research points to new and basically unmapped directions in an otherwise stagnating research on creativity.

I will in the following reanalyze a series of instructive studies of exceptionally creative scientists by Roe (1904–1991: see Wrenn, Simpson, Gorayska, & Mey, 1991), and then present a natural science model for individual development of

ordinary, creative, genial, and mad states to account for the observations. I see such a solution as just a radical extension of Eysenck's view on the science of human nature.

4. ROE'S STUDIES OF EMINENT SCIENTISTS

Anne Roe was a versatile researcher and clinician, with a professional horizon spanning from clinical work on schizophrenia to mapping personality parameters of importance for various occupations to penetrating research into exceptional creativity and achievement. Despite an obvious psychoanalytic slant, she never forgot to look for data, and I for one have profited greatly from the insight derived from her 1952–1953 series of studies of 64 scientists (1951a, b, 1952a, b, 1953, 1970).

To see what is so special about the most eminent scientists in the U.S.A., Roe examined 22 physicists, 20 biologists, and 22 social scientists (psychologists and anthropologists). They were all selected by a panel of experts to be the best in their respective fields. The impressive list of honorable memberships and prices awarded to them suggests that the judges made a narrow selection.

Roe found large individual differences among these exceptionally creative people and warns that a typical eminent scientist does not exist. She also observed some interesting common features. Most were firstborn sons of professional men. Almost all of them worked hard, devotedly, seven days a week, almost to the brink of displaying an obsession. Many of them admitted openly that their work *is* their life.

Clear group differences arose when the sample was categorized according to discipline. Eminent biologists and physicists contrasted social scientists in most respects (see below). Biologists and physicists tend to be shy and over-intellectualizing. Many were sickly as a child, lonely, "different", and aloof from their classmates. They were only moderately interested in girls, began dating no sooner than college, and married on average at age 27, which is rather late for national standards. Most of them continue to live in stable marriages, behave unusually independently, and have few recreations. The few recreations they have were typically those of a loner, such as fishing, sailing, or solitary walks. They do not care much about family relations, and show little guilt feelings about parental relations. They tend to avoid social affairs, parties, political activities, and religion. Biologists rely strongly on nonemotional and nonaggressive approaches to problems, and physicists show a good deal of free anxiety. Roe found many exceptions, to be sure, but not enough to spoil the general picture of an eminent natural scientist.

The description of the behavioral scientists contrasted that of the physicists and biologists in almost every conceivable way. Behavioral scientists tended to be highly gregarious, and to be socially active at an early age. Often they were acknowledged leaders already in school, where they practiced intense and extensive early dating. They were deeply concerned with human relations, showed many dependent attitudes, much rebelliousness, and considerable helplessness. They tended to be quite openly aggressive, and to experience a high divorce rate (41%).

Roe further noted that very few of these highly gifted scientists came from the South of the U.S.A., none were Catholics, five came from Jewish homes, and the rest were raised in Protestant homes. However, irrespective of background very few scientists had any serious interest in religious matters.

Table 20.1 (from Nyborg, 1991) summarizes, in modified form, Roe's observations of the overall pattern of representation of abilities and personality in the different academic disciplines, and contrasts them with data for blue-collar workers.

The table illustrates how abilities clearly distinguish natural from social scientists. Roe, in fact, even found group differences within these categorizations. To get that far, special tests to map exceptional verbal (V), spatial (S), and mathematical (M) abilities had to be constructed by the Educational Testing Service, as currently available standard tests were much too easy for many of these eminent scientists. The physicists without question scored highest on these demanding tests, but theoretical physicists performed relatively better on verbal tests, and experimental physicists relatively better on spatial and mathematical tests. Among the scientists, the biologists, physiologists and botanists scored relatively higher on verbal, and geneticists and biochemists relatively higher on nonverbal tests. Social scientists obtained a significantly lower overall IQ score than physicists. However, even within this group of scientists, social psychologists and anthropologists performed relatively better on verbal tests, and experimental psychologists better on spatial and mathematical tests. Some of the anthropologists were, in fact, unable to understand the mathematical tasks, whereas the most difficult of these items were too easy for some of the physicists. Here, perhaps, we have identified an important factor in the differential developmental status and sophistication of various scientific areas!

To summarize, eminent physicists and biologists tend to mature slowly, to have a troubled youth, and to feel lonely, shy, and "different" as children. Typically, they are not very interested in girls, marry late, have few children, and live stable solitary lives. They get very high IQ scores, but theoretical physicists do better on verbal ability tests, and experimental physicists do better on spatial tests and in mathematics. Social scientists mature faster, are more popular, begin dating earlier, have more children, and are more likely to

Table 20.1. Body, brain, and behavioral development, and adult personality and ability patterns in exceptionally creative natural and behavioral scientists (modified after Roe, 1952), and in blue-collar workers (from Nyborg, 1991b)

	Natural sciences				Social sciences		Blue collar occupations
	Physicist		Biologist		Behavioral scientists		
	Theoretical	Experimental	Physiologists/ botanists	Geneticists/ biochemists	Social psychologists/ anthropologists	Experimental psychologists	Unskilled workers
Body and brain development							
Early vs. late	Late	Late	Late	Late	Late	Late	Early
Personality							
Troubled youth	+	+	+	+	–	–	+
Shy and lonely	+	+	+	+			
Early interest in girls	–	–	–	–	+	+	+
Stable family life	+	+	+	+			
Emotionality	–	–	–	–	+	+	+
Aggressiveness	–	–	–	–	+	+	+
Ability							
Verbal (V)	V++	V+	V++	V+	V++	V+	V
Spatial (S)	S+	S++	S+	S++	S–	S+	S–
Mathematical (M)	M+	M++	M+	M++	M–	M+	M–

become divorced. They get about as high verbal scores as the physicists, but much lower spatial and mathematical scores. An exception is experimental psychologists. They approach the intellectual pattern of the physicists.

Roe's work illustrates two important points. First, despite the many similarities, extraordinarily high creativity or genius is not a unitary phenomenon; it is to some extent domain-specific. Second, whether a given individual will ever contribute anything creative in a particular domain depends on his ability–personality constellation, everything else equal (e.g., a suitable environment in the broad sense described later).

5. PHENOTYPIC SIMILARITIES AMONG CREATIVE AND HIGH IQ INDIVIDUALS

Roe's description of the extraordinarily creative scientist dovetails nicely with observations of high IQ people. This, actually, is not too surprising. Most of Roe's subjects had IQs above 140, so we could expect to find at least some analogies in the development of high IQ individuals and exceptionally creative individuals. However, the argument to be developed here is not one of complete identity. Creativity and IQ do not correlate above IQ 120, so we are talking about some striking developmental similarities among high IQ and creative individuals, but also a few decisive differences between creatives and noncreatives to be addressed later.

What about sex-related development? High IQ individuals tend to mature slowly, to become slightly taller, and to develop an androgynous body type, relative to the average for their sex (e.g., K. B. Hoyenga & K. T. Hoyenga, 1979, 1993; Nyborg, 1983, 1994). The three groups of highly creative architects, studied by MacKinnon (1962, 1964, 1970), showed an extremely high peak on the Mf (femininity) scale of the MMPI. Studies of highly creative males by Hassler, Birbaumer, and Nieschlag (1992), Kemp (1985), and others, confirm that creative musicians tend to be characterized by psychological androgyny; so do high IQ individuals (Maccoby & Jacklin, 1974).

Postwar (but perhaps not prewar) fertility, as measured by number of offspring, is lower in high IQ individuals (Vining, 1982, 1984), but their life expectancy is higher (Danmarks Statistik, 1985). There is a tendency for high IQ boys to behave less physically aggressive, and for high IQ girls to behave more physically aggressive than the average (Maccoby & Jacklin, 1974). Roe (1952b) noted that exceptionally creative natural scientists tend to have few children, social scientists more, but lower IQ.

What about sociability? Highly creative children in elementary schools tend to feel estranged from their teachers and peers (Torrance, 1962), as do creative adolescents (Getzels & Jackson, 1962) and high IQ children. Cattell and Butcher (1968) found, like Roe, that adult research scientists tend to be

skeptical, withdrawn, unsociable (McClelland 1962; Taylor & Barron 1963; Terman & Oden 1959) critical, precise, apt to express socially rather uncongenial and "undemocratic" attitudes (Van Zelst & Kerr, 1954) associated with dominance (Rushton, Murray, & Paunonen, 1983; see also chapter 19), to hold the belief that most other people are rather stupid, and to show a surprising readiness to face endless difficulties and social discouragement in order to have it their way. Barron (1965) finds that the original individual rejects regulation by others, and has a strong need for personal mastery, involving self-centeredness and self-realization. MacKinnon (1962, 1964, 1970) finds profound skepticism, rebelliousness, self-assertiveness, and independency characteristic for highly creative architects, already manifested clearly in school and onwards (Dudek & Hall, 1984).

Cattell and Butcher (1968) find, like Roe, that the typical research scientist, and in particular the physical scientist, is introverted, stable, and withdrawn, and characterized by a combination of higher than average ego-strength, high anxiety, and excitability. Moreover, researchers are more self-sufficient, more bohemian, and more radical than are successful administrators and teachers. Cattell and Butcher further find greater susceptibility to nervous disorder among artistic than among scientific geniuses, and that artists and literary men are more bohemian and more emotionally sensitive, than are scientists, in addition to having a higher ergic tension level and a general tendency for greater instability and emotionality.

To summarize, Roe's exceptionally creative high-IQ scientists show a number of similarities with creative high-IQ individuals observed in other studies as well as with noncreative high-IQ individuals. Creative scientists of all colors seem thus to have many important traits in common, but there is a tendency for greater emotional instability and relatively speaking lower intelligence in the social scientists.

These patterns of trait covariance leave us with a number of nagging questions. Which proximal factors are responsible for the striking developmental similarities between exceptionally creative scientists and noncreative high-IQ people. Do these factors relate causally to measurable brain parameters? Do we here find the mechanisms accounting for the supposed lack of correlation between creativity and intelligence above IQ 120? Phrased differently, what makes creative high-IQ people stand out from each other and from noncreative high-IQ individuals? The answer to these questions involves a proper solution to a crucial methodological question: just how many analytic dimensions do we really need to account for the developmental similarities and differences?

6. COVARIANT TRAIT DEVELOPMENT

6.1 The General Trait Covariance (GTC) model

With respect to the question of proximal factors I have argued elsewhere that gonadal hormones are ideally suited for coordinating sex-related development, and formulated a *General Trait Covariance* (GTC) model with 12 principles to account for hormone effects (Nyborg, 1979, 1983, 1984, 1994). Perhaps the hormone principles could explain the above mentioned similarities between creatives and other high-IQ individuals? Variations in hormone balances may even account, at least in part, for domain-specific differences. In any case, the original GTC model needs extension in order to formalize the harmonizing and differentiating effects of genes, gonadal hormones, and environment on creativity development (Nyborg, 1991a; b).

Briefly, the development of an originally sexually neutral fetus is guided by three interdependent factors: genes, hormones, and experience. Hormone production is thus determined by genes as well as by the environment. Hormones exert organizational as well as activational effects on body and brain tissues by modulating accessible genes in the genome, by affecting neurotransmitter systems, and by changing cell membrane characteristics. Gonadal hormones go everywhere in the body, but are biologically active only in hormophilic tissues capable of inducing specific receptors for them. This arrangement makes hormones uniquely suited to selectively co-ordinate and pace body, brain, and behavioral development. The 12 principles account for how the sexually neutral (except for Y chromosome material) fetus metamorphoses into covariant male, female, or "something-in-between" patterns of phenotypic traits.

Figure 20.2 presents a recent version of the original General Trait Covariance (GTC) model for hormonally guided development (for more details, see Nyborg, 1979, 1983, 1984, 1987b, 1988a, b, 1990a, 1991a, 1992a, 1994, 1995, 1997a, b, c).

The model works in the following way. Males and females are first divided (somewhat arbitrarily) in different hormotypes in accordance with their person-specific position on continuous androgen or estrogen dimensions. A male with high plasma testosterone (t) is said to be hormotype A5, and a male with low t is hormotype A1. A female with high plasma estrogen (E_2: 17-β-estradiol) is hormotype E5, and a female with low E_2 is hormotype E1. The advantage of hormotyping is, that the GTC model now generates rather precise predictions about individual covariant body, brain, ability, and personality development. It is actually a bit surprising to see how well the predictions of covariant development fit available evidence (for reviews, see Nyborg 1983; 1984; 1994).

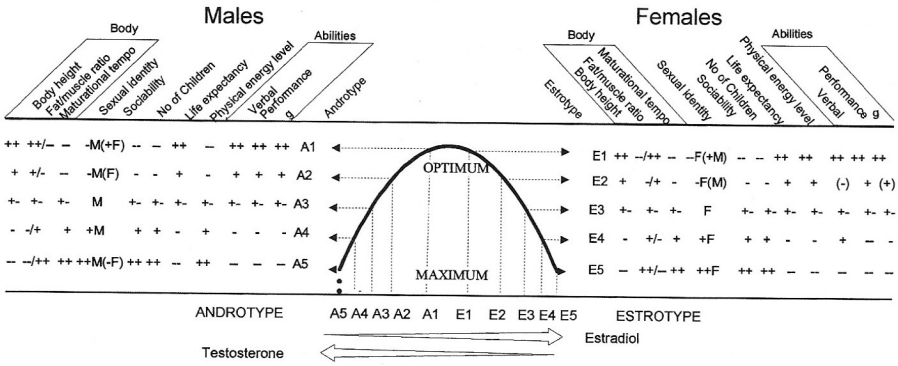

Figure 20.2. The General Trait Covariance (GTC) model for development. The model generates testable predictions about harmonized body, brain, intellectual, and personality development from parental DNA, plasma testosterone/estradiol balance, and experiences. Optimum intellectual and personality development is predicted by moderate and balanced hormone concentrations, but at the cost of sexual differentiation. Maximum sexual differentiation is predicted by high and contrasting testosterone and estradiol concentrations, respectively, at the cost of less than optimal intellectual and personality development. However, even slight variations in sex-related hormone exposure cause deviations from the expected modal pattern of male or female development, because each trait has its own developmental trajectory, time table, and hormone receptor sensitivity. The mechanism for this is probably, that hormones may transiently or permanently "switch" parental genes on or off by modulating their transcription rate (after Nyborg, 1994).

6.2 Hormotypic similarity among creative high-IQ and noncreative high-IQ individuals

Roe's descriptive studies, and those of others, raised the suspicion that creative high-IQ and noncreative high-IQ individuals share common developmental factors that make them deviate from the average person. Gonadal hormones may be these proximal causal factors. Unfortunately, there are no direct hormone studies of high-IQ people, so the hypothesis has to be evaluated in terms of indirect evidence.

Confluent evidence suggests that extraordinary creative scientists and noncreative high-IQ males both tend to have moderate to low plasma *t* (Nyborg, 1991a, b). Let us tentatively assume that there are many hormotype A1 or A2 among them, and then follow the predictions of the model. These males obviously are found in the upper left corner of the full spectrum of hormotype—trait connections in Figure 20.2. The dashed line inside the curve, guides us to their most likely modal development. Bodily, they tend to be slightly taller than average (obviously, familial disposition plays a role here, but then their parents probably were taller than average, too). They would also have an above average fat/muscle ratio (due to catabolic effects of low *t* or surplus E_2, but again relative to family disposition). A1s are expected to enter puberty late and to show minimal early sexual interests, relative to the population average. The low *t* A1 male is predicted to be characterized by an

androgynous sexual identity, with some "feminine" personality traits interspersed among some not too dominant male traits, and by few social interests (i.e., high introversion and occasional loneliness). He would predictably father fewer children, live longer, and have a high threshold for physical (but not necessarily verbal) aggression than the average. His intellectual pattern is high V, high S, and high M skills, and he would thus have a high Spearman's *g*. Even though these predictions for the low *t*, A1 high-IQ males were originally formulated without any thought on creativity, they fit surprisingly well the picture Roe and others give of the exceptionally creative natural scientists.

6.3 Hormones fine-tune differences in abilities and personality

Already the earliest version of the GTC model (Nyborg, 1979) predicted an inverse relationship between S and V as a function of variation in gonadal hormones. This was later confirmed in studies by Hampson (1986, 1990) and by Hampson and Kimura (1988). This inverse relationship may provide a point of departure for generating sets of hypotheses about the causal basis, not only of within-group differences in abilities in extraordinarily intelligent people, but also of group differences among creative natural and social scientists and artists, and even of finer differences among subgroups of natural scientists. All it takes is a few testable assumptions. Circa 50% of intelligence is co-determined by genes; provided equal *g*, the V-gifted male has been exposed to slightly more *t* than the S–M-gifted male; the gifted social scientists and artists have been exposed to more *t* than the natural scientists; provided equal *g*, the theoretically oriented high-V natural scientists have been exposed to slightly more *t* than the experimentally gifted high S–M natural scientists.

Male social scientists may thus qualify as genetically gifted hormotype(s) A4 (or higher). In that case, the GTC model predicts the following trait constellation. The slightly above average intelligent A4 male will be shorter than the highly intelligent natural scientist, and have a lower fat/muscle ratio. The more *t* would make him enter puberty earlier, make him more person oriented, less shy, and more popular among peers than a prospective natural scientist. His sexual identity would develop in a slightly more masculine direction, associated with earlier awakening of sexual interests, though not to the same extent as in the A5 "macho" hormotype. He would marry more often and have more children but, alas, would also divorce more often. He would die earlier than his natural science colleague, due to an increased risk of high blood pressure, circulatory diseases, heart attack, or prostate cancer. He would be more prone to aggressive outfits, and react more emotionally and hostile than the natural scientist, but less than the A5.

There is some evidence suggesting that S and M abilities are more sensitive to variations in gonadal hormones than is V (Gouchie & Kimura, 1990; Nyborg, Nielsen, Nærå, & Kastrup, 1992), even though V may be enhanced by increases in *t*. We can accordingly expect that the slightly higher *t* (and slightly lower E_2) in hormotype A4 male, relative to A2s, would manifest itself in a slight improvement in V (mostly in verbal fluency) and in a decrease in S and M. This would go some way to explain the differences in patterned intelligence between natural and social scientists (again, person-specific familial dispositions obviously also have to be taken into account). The highly creative artists may also be a moderately above average intelligent hormotype A4 or higher.

Most blue-collar workers have definitely higher than average *t* (Dabbs, La Rue, & Williams 1990; Nyborg, 1994), and would qualify as hormotype A4 or A5 males. Their lower than average IQ can be explained in more ways. They either carry a familial disposition for lower than average intelligence, or they show a hormonal depression of a familial disposition for high intelligence. Further deducible from Figure 20.2, they are expected to be shorter, more athletic and muscular, and to mature earlier than average A3s and much earlier than the more intelligent and the more creative A2 individuals (obviously, again seen in the light of family dispositions). A5s will show very early interests in girls, will display a more aggressive, impulsive, and person-oriented style, and may socialize but condition less well than the average A3. A4s and A5s will have more children, but will not live as long as A2s. Their verbal fluency score may be higher than the P, S, and M scores, but then again high *t* is particularly punitive for the expression of both P, S, and M. A5s therefore tend to obtain lower than average scores on heavily *g*-loaded intelligence tests.

There are not many exceptionally creative females. It is food for thought that the few female artists with a well-deserved recognition as exceptionally creative, show a tendency to write poetry and novels about people, or paint flowers and other natural motives. The very few eminent females in natural science are typically found in the biological disciplines. There are almost no females qualifying as exceptionally creative mathematicians, musical maestros or composers, chess masters, engineers, or architects. The GTC model suggests that this is due to a slightly reduced P, S, and M score, relative to V, caused by their relatively high E_2/t balance. The hormonal modulation of personality may also be important for creativity. The lesser willingness of most females to pursue a typical male obsessive pathway towards some remote goal for years and years, and to pay what may seem to most women an unacceptably high social and personal fee, may be related to their lower *t*. Let it be clear, that the GTC model cannot provide final answers to the question of why so few females pursue an exceptionally creative path. The model stresses an important point, however, with respect to the importance of hormonal perturbations prenatally

and at puberty. Even slight variations in the hormone balances in these two periods may decisively alter the pattern of female (and male!) abilities and personality in ways that make highly intelligent females (and males!) less likely to join the creative elite or compete without compromises in the gruesome power play for top positions. Note, however, that the model actually predicts that some of the low-numbered estrotypes will make it all the way to the top. E1 or E2 females, exposed to higher than average *t* (medically or physiologically, prenatally or at puberty, or briefly at menopause), may be more inclined to begin or pursue a professional career path than average females (e.g., Purifoy & Koopmans, 1979).

More generally, the model suggests that high levels of homotypic hormones (*t* for males and E_2 for females) are incompatible with scientific, artistic, or occupational achievement. The high levels seem to depress the particular abilities and personality traits called for in creative achievement in these areas. Moreover, they elevate social inclination and caring attitudes in females, and overt physical aggression in males. None of these traits are particularly valuable assets in creative achievement. In very high (or low) doses hormones may, in fact, not only disturb sensitive hormophile brain functions but can even accomplish massive systematic neural cell death (Nyborg 1991b; 1992; 1994; 1997a).

6.4 Hormones fine-tune differences in brain structure and function

It has not been possible to identify with a sufficient degree of certainty the specific brain structures subserving exceptionally high IQ and creativity (see chapters 11, 12, and 14). Large areas of the brain are active during problem solving, but there are large individual, regional, age- and sex-related differences in task-related metabolic rates.

Hormones are important for the brain. In addition to coordinating body and brain maturation, they affect neural plasticity and regulate the electric activity of the brain. Let us consider the "simple" hypothesis that neural plasticity is the missing link between low-numbered hormotypes and higher than average creativity (Nyborg, 1991a; b).

The neural plasticity hypothesis for creativity has three components: a temporal, a structural, and a functional. The temporal aspect refers to the notion that an exceptionally intelligent adult brain is a brain that retains much of its childhood plasticity long after puberty (for a critical discussion of neoteny, see Nyborg, 1994, chapter 13). Neural plasticity thus becomes a necessary, though not a sufficient, condition for above average intelligence. The structural aspect refers to the notion that plastic brain tissues are more likely than fully mature and solidly established tissues to reconfigure as a function of use. The functional aspect relates (perhaps) to Eysenck's notion of a wide association horizon.

Hormones causally affect neural development, plasticity, and functionality, so that only moderate prenatal and pubertal surges are compatible with high adult intelligence and creativity.

Nottebohm (1981, 1989) has provided interesting animal support for the hypothesis that gonadal hormones regulate the neural plasticity and associated "creativity" in birds. Briefly, only males sing in many bird species, and then almost exclusively during the mating season. They are then silent again until next season. Nottebohm demonstrated that the neural song system of a bird is highly sensitive to variations in plasma *t*. As *t* secretion increases gradually up to the mating season, several neural song nuclei begin to form in the bird brain and grow in volume, and probably also in number of neurons, and in richness of synaptic connections. This gradual process at first allows the bird to draw upon a primitive song repertoire with few vocals. With further increases in *t,* the bird enters a period of "plastic song." When *t* is at its zenith it finally reaches the full song stage with highly reliable song performance. This coincides in time with the bird being sexually fully mature and in need of effective means for attracting the attention of female birds for reproductive purposes. The interesting part of this story is, that the "plastic song" stage is the time, when the bird is most capable of learning new song variations. With *t* at its maximum, the bird cannot but sing in a stereotypic way. Nottebohm explains the plastic or creative song phase with optimum neural plasticity at moderate levels of *t*. As *t* declines, and the mating season is over, there is a reduction in the number of synaptic connections in the song nuclei, and the bird's song repertoire becomes partly wiped out by "forgetting." A moderate increase in *t* marks the overture to the next season, and allows new flexible synaptic connections to be established, which enables the bird to create new constellations of vocals and multicolored creative song.

Can Nottebohm's bird hormone model for neural plasticity serve as a human model? What moderate *t* concentrations mean to bird-song nuclei and potentials for "creative" song variations could roughly match what moderate concentrations of gonadal hormones mean to human brain plasticity, intelligence, and creativity. The hypothesis would provide a much-needed temporal–structural–functional perspective on the Eysenckian notion of wide association horizons in creatives. Most likely not only *t* per se, but also the aromatization of *t* to E_2 is involved, but an account of this complicated technical story cannot be given here (see Nyborg, 1994, chapter 8)

Covariant body and brain maturation follows a person-specific developmental timetable. Most children show natural ease in new learning, and often combine old and new elements in "unexpected" ways throughout childhood. Alas, this flexibility, and in particular the ability to combine elements in unusual ways, often dissipates, in many to a dramatic extent, as children reach puberty. Like for canaries, "childhood creativity" is supplanted by more robust but sex-stereotypic adult performance, and the more so the earlier and larger

the hormone surge. There are, to the best of my knowledge, no published studies specifically addressing the question of covariant human brain plasticity, intelligence, and creativity development during childhood. Presently, we have to remain satisfied with bird and rat evidence, showing that high levels of hormones negatively affect adult neural plasticity. Pavlides, Westlind-Danielsson, Nyborg, & McEwen (1991) thus demonstrated, that neonatal hyperthyroidism simultaneously disrupts hippocampal long-term potentiation (LTP) and adult spatial learning. We took this to mean that the hormonally conditioned reduction in neural plasticity disrupts the capacity to figure out and remember where to find food in the eight-arm radial maze. Also relevant is Shapiro's (1968) observation, that thyroid hormone treatment of neonatal rats speeds up body and brain development and learning capacity before puberty but, unfortunately, the early maturation and initial hormonal boost of learning capacity has to be paid for with lower neural plasticity and inhibited learning after puberty. Given that rat, bird, and human hormones and receptors are chemically identical we might perhaps be justified in assuming that this is a good animal working model for human development, the main difference being, of course, that the hormones modulate partly identical/partly species-different genes through similar mechanisms, in various animals.

In addition to the narrow hormone–brain plasticity hypothesis for creativity, hormones could also account for broader covariant trait development by monitoring the tempo of body and brain maturation, and in this way harmonize body development with neural plasticity, intelligence, personality, and creativity. High levels of pubertal hormones speed up body maturation, reduce neural plasticity, hamper the expression of familial intelligence, and reduce creativity. The hypothesis would account for the observed relationship between early–late maturation and different patterns of intelligence and creativity in Roe's samples of exceptional scientists in terms of variations in gonadal hormones and their effects on neural plasticity. We know that high hormone concentrations at puberty cause early closure of the growth zones in the long bones, leading to low final body height. They also seem to reduce neural plasticity. It is this covariance hypothesis that allows for quite specific predictions from hormotype over brain mechanisms to which kind of children will most likely suffer selective reduction of childhood creativity at puberty. This application of the GTC model is discussed in more detail elsewhere (Nyborg 1991a; 1994). It is a sad fact, however, that there are several definitions of neural plasticity, each referring to complex and partly unexplored brain conditions. Without a much better understanding of the details of the hormone–brain connection we will not be able to test the hypothesis.

6.5 What makes creative high-IQ individuals stand out from just high-IQ individuals?

Section 6.2 demonstrated that high-IQ individuals share many developmental characteristics with exceptionally creative high-IQ people that make both groups differ from the average person. What makes creative high-IQ people stand out from noncreative high-IQ individuals?

The tentative psychometric or factor-analytic answer is, that particular differences in covariant combinations of intellectual and personality traits make for the distinction, and that this also explains domain-specific differences among scientific geniuses. This description is totally vacuous, however, unless we can transcend the phenotypic surface description, and identify details in the differences in various supporting molecular brain devices that lead up to differences in psychometric intelligence, personality, and creative achievement. To facilitate this explorative process, we better first recapitulate which psychometric trait combinations fit which domains.

Unusual stamina, that is, an almost obsessive devotion to work despite adversities is a must for any genius, according to Roe (1952b) and many others. Above average intelligence is, too. Her study further indicates that eminence in various disciplines requires different combinations of body and brain development, abilities, and personality. Theoretical physics, physiology, and botany may call for a combination of late maturation, introversion, and a V/S–M balance. Experimental physics, genetics, and biochemistry calls for a combination of late maturation, introversion, and an S–M/V balance. Experimental psychology calls for the combination of moderately late maturation, moderate introversion and IQ, and a S–M/V balance. Social psychology and anthropology call for slightly above average intelligence, slightly later than average maturation, moderately high extraversion, and a V/S–M balance. Nonprofessional areas do not require high IQ, and neither early maturation nor extraversion is a hindrance.

These phenotypic patterns equip us with a preliminary answer to the question of what makes exceptionally creative individuals stand out from each other, from noncreative high-IQ individuals, and from the average person. The answer depends, in other words, on which area we are talking about, and at least five different ability–personality combinations are needed to explain Roe's between-group differences in Table 20.1. An interesting implication of this is that—as the genius can neither select his own intelligence nor personality nor his person-specific combination of abilities—we are forced to conclude that the genius does not choose his scientific discipline at his own discretion. It is rather the other way round: The domain "selects" him in accordance with an evolutionary-like process involving selective pressures. If his strong sides mismatch a particular domain, he and the domain waste time and energy.

The preliminary nature of this hypothesis of domain-specific selection for genius is obvious. It might, nevertheless, facilitate the search for appropriate causal models for creativity, genius, and madness. But what is an appropriate causal model? Most likely, it is a model that accounts in strictly causal terms for the complex processes and interactions *behind* the phenotypically observable covariant patterns.

6.6 How many analytic dimensions are needed?

This raises a fundamental question: Is it possible—within the framework of a single analytically and causally coherent model—to examine how particular combinations of genes, hormones, neural plasticity, neurotransmitters, intelligence, personality, and environmental circumstances play together to produce domain-specific behavior like extraordinary intelligence and creativity? The solution to this problem presumes that it is possible to identify a unitary causal level at which genes, hormones, and brain plasticity transform intelligence and personality into creativity and genius, in the presence of an ever-important environment. Neither traditional psychological analyses nor contemporary gene–environment interaction analysis will allow us to go that far (Nyborg, 1987a, 1989, 1990b, 1994, 1997a; Nyborg & Bøggild, 1989; Wahlsten, 1990). Eysenck has, as usual, seen the problem already, and recommends that we go beyond the empty lexical definitions, surface factor analyses, and descriptive psychometry, and begin to look in the direction of brain physiology and chemistry. In the following section I will take his advice to its logical extreme, and settle for just one analytic dimension with three analytic windows.

7. THE NONLINEAR, DYNAMIC, MULTIFACTOR, MULTIPLICATIVE, MULTIDIMENSIONAL MOLECULAR (ND4M) MODEL FOR EXISTENCE

7.1 Introduction

The descriptive GTC model (Figure 20.2) was primarily designed to help formalizing covariantly developing body and brain parameters in order to become able to predict ability and personality patterns as a function of the trinity of DNA–body chemistry–environment interaction (Nyborg, 1983, 1994, 1997a).

To progress, the GTC model was next extended to account in proximal causal terms for the nonlinear, dynamic, multifactor, multiplicative, multidimensional molecular interactions leading up to the existence of people and all other existing configurations, and to their disappearance. The result of this megalomane endeavor is a new ND4M model. A specialized version of the model is presented in Figure 20.3, where it is adapted to focus narrowly on human development, behavior, and society exclusively in terms of molecular

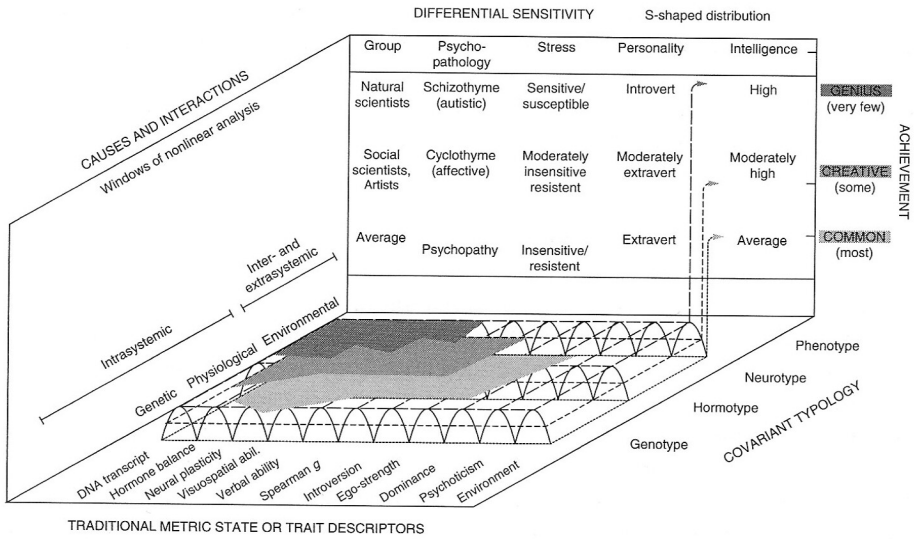

Figure 20.3. A nonlinear, dynamic, multifactor, multiplicative, multidimensional molecular (ND4M) model for common, creative, genial, or pathologic development. Creativity/sensitivity/susceptibility is a combined function of particular gene constellations, moderate plasma sex hormones, low sexual differentiation and high adult neural plasticity. Schizothyme development is a function of low hormone and incomplete subcortical development, whereas cyclothyme development is a function of high hormone and cyclic instability among molecular parameters. The model mimics multidimensional mass molecular space–time–phase (x, y, z + time + phase) changes over long phylo- and shorter ontogenetic periods.

interactions. It is further restricted to illustrate how intelligence, creativity, genius, psychopathology, and society can possibly be subjected to a single all bottom-level molecular analysis. In that, the ND4M model differs radically from the usual multilevel psychological approach as well as from the linear, additive, and statistically based multivariate nature–nurture model for average differences around a population mean.

The name of the model is indeed ackward, but each of the adjectives refers to vitally important interrelated aspects of development. The following sections first briefly describe what the adjectives refer to, and then discuss the interaction with a emphasis on creativity, genius, and psychopathology.

7.2 A molecular account of human nature and society

It has been argued, to the horror of some psychologists and philosophers, that (1) man is basically a molecular constellation, evolving, developing, acting, and disappearing again like all other molecular constellations in a basically molecular earthly world embedded in a larger molecular universe, that (2) all

this can sensibly (although, of course not exhaustibly) be analyzed in terms of molecules, and that (3) this is presently the only scientifically acceptable way to try and account for covariant human (and societal) development. The research program behind this view is presented in details, and related to a notion of our exclusively molecular evolutionary past in Nyborg (1994).

Briefly, molecular DNA instructions are transcribed during ontogeny into protein molecules that either amass intrasystemically into body and brain tissues or affect their functionality when first formed. Environmental impacts are more or less systematic changes in extrasystemic physico-chemical parameters having an effect on intrasystemic molecular parameters through the digestive and perceptual systems. One particularly important class of extrasystemic impacts, social interaction (love, etc.), is defined as intersystemic physico-chemical (molecular) interaction among systems made slightly dissimilar by, among other agents, hormones. This exclusively molecular view of man, society, and the universe is named physicology. The neologism refers to a research program designed primarily to entangle molecular causes, interactions, and effects in very complex systems, be that of organic or inorganic origin. The presence or absence of carbon atoms is really not essential to the analysis. The molecular level of analysis is chosen entirely for practical reasons. Molecules have sizes and effects that place them conveniently in between the remote small-scale elementary particle level and the large-scale level of the cell or organs. Molecules are sufficiently close to the human scale to be of practical value in the causal study of development and function, whereas elementary particle physics would entangle the analysis in the small-scale peculiarities of quantum mechanics. The cell structure level would leave out of view many intricate fluid processes within the cell. Effects of hormones can, for example, be analyzed entirely in terms of molecular concentration, affinity, and time–space–phase coordinates if one wishes. So can nerve cell membrane characteristics. Membranes are conglomerates of molecules "frozen" temporarily in space in accordance with their stereotaxic characteristics and environmental circumstances.

The left z-axis in the ND4M model in Figure 20.3 identifies the focus for a given causal analysis. The first practical step in the analysis of systemic molecular causes, interactions, and effects is to open an analytic window to either the intra-, inter-, or extrasystemic aspects of molecular interactions. Ideally, all windows should be opened at once, but presently we do not even have proper tools for keeping track of everything going on in one.

Where the psychological analysis inevitably involves a tangled hierarchical web of surface, top-down, and bottom-up analyses, in the futile attempt to connect incompatible material and abstract spheres, the molecular account of human nature involves a nonhierarchical all-bottom approach to the examination of covariant molecular mass-actions separated more or less clearly in space and time and defined by phase (see also chapter 25).

7.3 Multidimensionality

The front *x*-axis in the ND4M model in Figure 20.3 provides an overview of some of the metric state or trait parameters, that are often related to creativity in the literature on genius. Three aspects of the model deserve mentioning in that connection.

As already said, it is a very general model covering all aspects of becoming, being, and breaking apart again (i.e., developing, living, and dying in the animal and plant cases). Had the model not been adapted here to focus on creativity, the metric indicators at the front *x*-axis would have had different names, and would have referred to other molecular causal processes.

Second, the metric indicators are pure surface names with only descriptive value. A common sin in the psychometrics of intelligence and personality is to see such indicators as genuine causes, but this is a dangerous method of explaining away what goes on intrasystemically. As mentioned previously, it makes no sense in causal terms to say that genes interact additively or multiplicatively with hormones, intelligence, and personality traits like ego-strength, introversion, or social factors to produce intelligence or creativity. Those who say so are multiplying apples with pears, and what do they get?

Third, the model circumvents the fatal category error problem inherent in all hierarchical psychological, cognitive, or rational analyses. Each of the descriptors in the ND4M model refers to a more or less well-defined molecular mass-action process. These mass actions may share important serial or parallel processual community, but the analysis still amounts to just one-level molecular interaction in a truly cause–effect sense.

It would, for obvious reasons, be wrong to say that the physicological program behind the model refers, in fact, to a traditional behavioral program. Many of the decisive molecular interactions do not show up immediately in phenotypic behavior, and some only much later in life. Physicology is therefore rather a program for the study of the molecular dimensions *behind* behavior, even though behavior is obviously an expression of interacting molecules moving collectively in space–time coordinates. In other words, each descriptive indicator tentatively lined up along the front *x*-axis of the ND4M model refers to important events in a particular molecular dimension. The left *y*-axis indicates the most likely space–time–phase coordinates for the interaction of these molecular events.

7.4 Multiplicativity

The ND4M model is based on the notion of multiplicativity, quite like Eysenck's model. The state or trait descriptors at the *x*-axis were selected basically because they typify creative individuals or geniuses, and because none of them is likely to be missing in the description. Creative achievement (right *z*-axis) inevitably suffers if an individual has optimum scores for all state or trait

descriptors at the *x*-axis apart from one. High Spearman *g* would, for example, be wasted for creative achievement if ego-strength was missing (of course, this obscure psychoanalytic term is taken here to reflect some kind of long-term molecular consistency, perhaps related to *t*!). Neither can physico-chemical factors like a suitable prenatal environment or nutrition be missing from the formula for a genius. Even learning, memory, and social interaction can be defined in molecular terms (Nyborg, 1994) and may then enter the molecular formula for a genius.

Eysenck (1995, p. 49ff) assumes that intellectual and creative achievement is best described by a J-shaped distribution (like so many other psychological and socio-economical phenomena: Allport, 1934; Burt, 1943; Nyborg, 1991b; Walberg, Strykowski, Ronai, & Hung, 1984). Price (1962; 1963) found, however, that the distribution is better described in terms of an S-shape. This makes sense, as nothing grows perfectly. I will follow Price's advice, and inscribe the asymptotic multiplicative cause–effect on creativity in the ND4M model in terms of an S-formed distribution. The total creative brain potential is, accordingly, seen as an exponential product of a limited number of key factors, each representing evolutionarily optimized molecular mass actions. Some of the factors represent fairly stable aggregations of molecules forming sensitive nerve cell membranes or other structures; other factors reflect mobile neurotransmitters, peptides, or hormone–receptor complexes, and still others represent robust DNA structures.

One implication of the notion of an S-shaped creativity achievement distribution is that adding more and more optimally adjusted key factors to the multiplicative formula means little to the expression of genius, as long as the number of original key factors equals or surpasses an absolute lower number. Further fine-tuning of factors in the existing genius may broaden his domain specificity, however.

Another important characteristic of the model is that concomitant optimum tuning of all factors is seen as a rare and probably rather unstable situation. The loss or severe mistuning of just one key factor may spell a dramatic deterioration in the performance of a genius. On the other hand, the extremely rare occasion of a "divine stroke of genius" in an otherwise unremarkable person may reflect a sudden lucky optimum tuning of all factors. Life-span analyses may illustrate temporary shifts. Eminent physicists typically realize their most creative potentials while young (sometimes even before the age of 25, even though Nobel prizes are typically given to old men). Age-related loss of creativity may be due to, say, the inevitable (and highly regrettable!) decrease in *t* production with age (e.g., Ellis & Nyborg, 1992). Of course, many other age-related factors are involved, too. Perhaps the decreasing *t* levels relate to loss of persistence (ego-strength or willpower, if you must!). Such temporal shifts in molecular tuning could explain the sad fact that even the most extraordinary creativity lessens a bit with time. On the other hand, a

genius first flourishing at middle-age might actually reflect that he had too much *t* to unfold full genius in young adulthood, where he overshot the optimum. Elsewhere I hypothesized that most young adults loose their "childhood creativity," because the considerable pubertal surge in sex hormones reduces neural plasticity while enhancing sexual differentiation of the body and brain (Nyborg, 1991a).

In summary, the absence of one or more key factors marks the difference between genius and not genius. Fine-tuning of one or a few (ability or personality?) key factor(s) explains the domain specificity of extraordinarily creative individuals. More dramatic mistuning of one or more factors threaten creative achievement in general. Although the model is basically multiplicative, the tuning of single key factors affects the weight by which they enter the formula for creativity and genius.

Which weight should be attributed to social factors in the fomula? Actually, little if any, as there is no hard experimental evidence proving that unspecific rearing or social engineering affects extraordinary creativity. The closest we come to documentation for a socialization effect is Zuckerman's (1977) observation that Nobel laureates tend to seek the working company of those who already got the prize. However, this could either mean that clever domain-specific people prefer the company of likes, or that creative role modeling really works wonders. We simply do not know. The ND4M model nevertheless remains fully open to any experimentally documented environmental effect, though it has no space for loosely defined and poorly documented social parameters like prevailing norms, cultural stereotypes, or passive role modeling without a physico-chemical address. In contrast, factors such as the chemical interaction between a pregnant woman and her fetus, nutrition, and the modulation of neurotransmitters by stress, or learning from "significant others" can and should be measured and entered into the multiplicative formula for molecular interactions among factors (Nyborg, 1994). Eysenck, as usual, strikes the truth when he says that much hard work remains to be done.

7.5 Creativity, psychoticism, and psychosis

Eysenck found an apparent paradox in the *t*-creativity-psychoticism connection in the GTC model (but he might not find it again in the ND4M model). Eysenck's own creativity model associates high creativity with moderately high dopamine and *high* P (and androgyny). The GTC model seems to associate high IQ (and creativity) with low *t,* ample neural plasticity and *low* P, and low creativity with high *t*, little neural plasticity and *high* P [females differ from males in this respect: here, high creativity associates to high *t* (or low E_2), ample neural plasticity, and high P—see Nyborg, 1994—but this need not concern us here].

To explain the paradox, Eysenck wondered (1995, p. 276) whether genius is the exception where "high testosterone levels and 'cognitive androgyny' may be negatively correlated in the general population, but is closely associated in a small sub-group of creatives." Perhaps so. The human brain actually aromatizes some *t* to E_2, there are large individual differences in aromatase activity, and the mechanism might bear on Eysenck's suggestion. Unfortunately, we know next to nothing about the possible effect of conversion on neural growth and plasticity, brain function, creativity, and P. Another possibility is that the brains of geniuses show reduced sensitivity of hormones.

However, many observations definitely speak against the high *t*–androgyny–genius hypothesis. For example, high *t* relates negatively to both IQ and introversion, and significantly so; high *t* further associates positively to early somatic maturation, intense sexual and social interests, physical aggression, and a stereotypic sexual identity. The covariant body–intelligence–personality pattern of an A4 or 5 stands in contrast to the common description of a genius.

There is a way to solve the apparent paradox, however. Let us for a moment contemplate the hypothesis that high P refers to different disorders in high and low *t* males with high IQ. This hypothesis has two important implications. It threatens Eysenck's notion of a smooth dimensional causal continuity between normals, affectively disordered, and schizophrenics. It might partly resolve the previously mentioned problem of the psychometric irregularity of the P scale. To see how, we have to simplify complex matters considerably.

Using the nonlinear molecular dynamics of the ND4M model, I propose the following three developmental hypotheses:

1. Extremely low prenatal and pubertal *t* disposes for slow body development characterized by incomplete sexual differentiation, and a vulnerable neural development with abnormal migration and/or incomplete (primarily subcortical?) dendritic aboreation. The result is incomplete (sub)cortical development, a tendency for enlarged ventricles already at birth, abnormally high neural plasticity, and/or nervous instability and sensitivity (and perhaps high dopamine), with resulting confused thinking and disturbed perception, as seen in schizophrenia. Low *t* is, according to this hypothesis, made partly responsible for the tendency of male schizophrenics to mature late and show a neotenic and somewhat demasculinized sexual development, with little interest in girls, a tendency for attaining a linear body build (as noted by Kretchmer), extreme introversion and reduced contact with reality, and high P score.
2. The less extreme cases of low *t* males would still be characterized by slow neural and somatic development, but now associated with optimum migration and dendritic aboreation, unusual synaptic connectivity, and optimum neural plasticity, sensitivity and a wide association horizon (read: molecular covariation). The lucky combination of suitable parental DNA–

moderate hormone exposure could dispose for the effective development of a large brain with above average intelligence (at least above IQ 120), and result in the creative hormotype A1 or A2 natural scientist with higher M–S than V scores seen in Table 20.1. This creative low-*t* individual might nevertheless earn a high P score. Extreme sensitivity, due to an exceptional overall brain state with optimally tuned parameters, may already in childhood result in eccentric behavior matching that of a prospective noncreative postpubertal schizophrenic patient. Negative reactions from significant others may further stress such a child enough to raise his P score. Obviously, this speculation calls for chemical rather than psychoanalytic testing.

3. A male with DNA favorable for high IQ, but now combined with the much higher prenatal and/or pubertal *t* exposure of an A4 would also earn a high P score, although now for quite different reasons. The relatively high *t* would dispose for brain development characterized by general neural overexitation, some neural plasticity, sensitivity, and creativity, but also for early body and brain maturation, extraversion, and a social inclination. This could be the genesis of the social scientist or artist in Table 20.1, with a higher V then M–S balance. Perhaps *t*-related neural overexitation represents, in extreme cases, an unstable brain condition alternating between mania and depression. This would explain why a surprisingly large number of eminent high V authors suffer from affective disorders and score high P. The fact that acute affective psychotic states relate to periods with low creativity comes as no surprise.

The hypotheses suggest, in other words, that creative social scientists, artists, and authors may score as high on the P scale as the creative natural scientist, but for entirely different endocrine and neural reasons. This interpretation obviously does not fit Eysenck's dimensional idea of an underlying continuum from normality over affective to schizophrenic conditions. It rather suggests that P, creativity, and psychopathology covary as a function of hormones and, of course, genes and enviromental factors. One thing is sure: There are presently too few good data to take a firm stance in the matter.

7.6 The molecular dynamics of the ND4M model

The ND4M model is build on molecular dynamics, and this is the basis upon which the version of the model accounts for the development, continuity, and the eventual disappearance of creativity and genius. This means that creativity and genius are seen as states rather than traits, even if the states prevail for a long time, given stable molecular circumstances. Extraordinary creativity, or something like it, can be observed in some eccentric children before puberty, as their brains have the considerable neural plasticity and capability needed to

combine remote processes in unexpected and sometimes productive ways. However, large pubertal surges of sex hormones may at first speed up the tempo of maturation, but then put an end to long bone as well as brain growth potentials. The higher the surges, the sooner the termination of body and brain growth, and final neural plasticity. The creative child will remain creative after puberty only if he or she is exposed to low to moderate hormone concentrations prenatally and at puberty, or if the brain was primed prenatally by hormones to low sensitivity to adult hormone concentrations. Whether childhood "creativity" gets a further boost with brain growth at puberty or will be inhibited depends, in other words, on the right gene–hormone concentration combination, but also on hormone-binding globulins, receptor sensitivity, and a favorable environment. Only neotenic children with moderate amounts of sex hormone will retain their childhood "creativity." Some data speak in favor of this hypothesis (Hassler, Birbaumer, & Nieschlag, 1992). Other data also suggest that molecular brain processes subserving IQ are sensitive to hormone molecules: Spearman *g* is definitely negatively correlated with *t* in males (Nyborg, 1994), as are visuo-spatial abilities in high E_2 females (Hampson, 1986, 1990; Hampson & Kimura, 1988; Nyborg, 1979, 1983).

The dynamic aspects of the ND4M model extend far beyond puberty. The model predicts, for example, that a woman will show slightly enhanced creativity shortly after menopause, relative to her creativity during the reproductive period. The material basis for this prediction is straightforward. When ovulation stops, the pituitary reacts to the drop in plasma E_2 with increased gonadotropin release. This stimulates the adrenals to secrete more substances with androgenic effects for a couple of years. Many postreproductive females have, in fact, relatively speaking quite high androgen and low E_2 status (lower than many men of comparable age!), and this ought to show up in a short-lived increase in physical energy, nonverbal IQ and, according to the model, creativity. Hormones further affect brain processes of relevance for personality parameters. As *t* drops with age (Ellis & Nyborg, 1992), we can expect less neural plasticity and disturbed molecular brain processing, lower ego-strength, dominance, psychoticism, and perseverance. Loss of one or more of these factors means loss of the state of genius, and a dramatic reduction in creativity for the rest of us.

As said before, the ND4M model and the physicological research program presume that the last variable to the right on the *x*-axis, the environment, is as much a purely physical–chemical parameter as is the transcription of DNA material. Environmental molecular parameters like prenatal fetal exposure to maternal hormones or viral infections, birth complications, nutrition, stress of all kinds, systematic changes in molecular brain parameters caused by perception or learning, and intersystemic (social!) interaction must all find their proper place and weight in the formula for creativity and genius. However, social conditions in general, and systematic creativity training in

particular, must be rigorously defined in operational terms before they can be allowed to enter the formula for genius. This is evidently not the case now, and there is even some evidence to suggest that opposing social conditions may stimulate some geniuses to work even harder.

This brief discussion is not meant to cover the many dynamic possibilities for enhancing or inhibiting molecular brain processes of relevance for phenotypic creativity. It suggests, however, that it might be worth our while considering the brain as a complex molecular system at the brink of instability (Nyborg, 1997a). In fact, this may be the only scientifically acceptable way to approach creativity, genius, madness, and organic existence in general.

7.7 Nonlinearity

Molecular actions and reactions typically unfold in nonlinear interactions in most biological systems. A slight increase in the concentration of a given chemical species gives a linearly graded response, but further increases often result in nonlinear responses. Very high concentrations may turn the effect into its opposite or into something quite different.

The state of genius thus presumes rare DNA combinations predisposing for optimum flexible brain development and functioning. Genes for moderate hormone secretion, and a favorable environment (prenatal or otherwise—not exposing the fetus to unusually high or low levels of natural gonadal hormones, stress hormones, or artificial hormones), is also a must, as is moderately low pubertal hormone secretion. Abnormally low hormone concentrations negatively affect neurogenesis; moderate hormone levels relate to optimum neural plasticity; high hormone levels to overactivation of neural tissues. Studies, summarized in Nyborg (1984; 1990a; Nyborg et al., 1992) even suggest that the general karyotype (XX or XY) is less important phenotypically than is hormone exposure with respect to covariant body, brain, intelligence, and personality development, even though in most cases karyotype and hormotype go together. E_2 may, for example, feminize the brain in weak concentrations, masculinize it in larger doses, and have neurotoxic effects in high doses. The duration of exposure is also important. For example, short-term increase in stress hormones may have beneficial effects, but long-term surges in stress hormones may cause systematic cell death in sensitive brain tissues.

The ND4M model connects the nonlinear molecular effects with phenotypic behavior in fairly simple ways. The different layers in Figure 20.3 refer to differences in the tuning of the various molecular systems. A person's level is determined by covariant interactions along the left y-axis among the many nonlinear molecular subsystems lined up along the front x-axis as metric state or trait descriptors. Intermediate effects can for convenience be expressed in broad terms like genotype, hormotype, neurotype, and phenotype along the right y-axis. Starting with DNA transcription, the first factor to the left on the

x-axis, protein production by specific genes may be much too high or low to benefit brain structures subserving the personality of the genius, or it may disturb proper functioning of critical brain areas underlying, say, intelligence. In either case the DNA transcription factor would be missing in the multiplicative formula, the end product of the molecular formula would be zero and there would be no genius. For cases, where the optimum molecular levels are either over- or undershot, the level of achievement would approach the second or third levels. The state of genius (first level) is actualized only in the extremely rare case where all molecular processes play pretty close to the optimum at each of the inverted U-curves. Proper DNA transcription of proteins will then relate to optimum hormone balance, ample brain development with rich neural plasticity, and other suitable modes of molecular functionings subserving the genius. Most of us either under- or overshoot the top of one or many of the curves, and our creativity therefore hovers somewhere in the area between the second and third levels.

Multiplicativity, nonlinearity, affinity, and space–time coordinates are the tools by which the model accounts for the molecular dynamics of creativity, and genius is the rare case where all parameters are optimally tuned. Domain-specific differences among genius, like those observed by Roe (Table 20.1) arise if one or a few of the parameters, such as hormone balances, varies slightly around the optimal fine-tuning, furthering in some cases V, in other cases M or S abilities, and at the same time covariantly fine-tuning body and personality parameters.

The third layer in the model represents an inhomogenous majority. Some may suffer from familial transmission of genes not favorable for the development of an intelligent brain; others may be the victim of unhappy emergenetic recombinations; still others may secrete too few or too many sex hormone molecules to fit creative development. These, plus many other conditions such as accidents and illness explain low third layer achievement.

Hormones have been attributed much weight in the previous sections. It is worth remembering, however, that they too are only intermediary buffers in the complex interplay between DNA, the brain and the environment—quite like dopamine and serotonin.

8. THE FUTURE

Creativity research has so far been dominated by three major approaches: the anecdotal–historical, the psychometric, and the psychological–sociological. The harvest of studies conforming to these traditions is not impressive. Serious problems remain, the explanatory power is low, and some specialists in the area of creativity now talk about signs of degenerating research. Then Eysenck entered the scene and suggested that experimental and physiological tools

supplemented the psychological approach, and research began to move again. Eysenck's creativity model generates testable brain hypotheses, instead of trying to excuse failing predictions, and this is a significant improvement.

My only quarrel with Eysenck's approach is that he is not going far enough in the right direction. His hybrid cognitive–physiological approach still keeps too close to that of a classical psychologist to comfort, even if, over time, it looks more and more like a bottom-up than a surface or top-down approach characteristic of much contemporary psychology. More precisely, what really worries me is that largely undefined (and perhaps ultimately undefinable) terms like *mind, cognitive inhibition,* and *ego-strength* still play an important role in a causal network, so that abstract mental and solid material variables feature side by side in a multiplicative model for creativity, genius, and madness.

With the recent progress in the brain sciences, the time may be right to skip the uncensored use of hypothetical psychological constructs and intervening variables. We can now begin to take our first faltering steps along perhaps the only proper scientific avenue to the study of creativity, genius, and madness, namely, in terms of all-bottom causal analyses. This becomes possible only when DNA, the body and brain, intelligence, personality, creative achievement, and the environment are all defined in terms of molecular mass interaction. We need no a priori theory to accomplish this. All we have to do is to map or guide where molecules go, and then see what they do when they meet—for example, when leading up to or away from the states of genius or madness. This may actually be the only acceptable definition of becoming, being, and going away again. It is very fortunate that we do not have to map the fate of each molecules for this, because that would have made the task entirely impossible. Identifying differences in stereotaxic affinity, concentrations, uptake, and biological action, plus real-time picturing or mathematical or molecular modeling of mass-concentration effects in time–space coordinates may suffice.

The physicological research program and the GTC and ND4M models are based on this view. Even though Eysenck thinks favorably of it, he finds that I go too far (Eysenck, 1996). He may be perfectly right, but so help me my molecules, I see no other way around. Any solution that tries to combine abstract psychic or cognitive with material factors is bound to sink into intractable body–mind problems and trap the researcher into committing inexcusable category errors. Physicology steers free of both kinds of problems, by first resolving that the body–mind problem is a philosophical pseudoproblem, and then by resorting to a unitary all-bottom analysis (Nyborg, 1994). There is no reason to deny, however, that the physicological research program faces a serious problem. This problem has less to do with theory than with methodology. Even the most sophisticated contemporary natural science methods cannot deal properly with complex nonlinear

(molecular) dynamics. Where the futile anachronistic reference to abstract psychic, mental and cognitive entities, philosophical body–mind divisions, and the unlimited generation of hypothetical causal variables have impeded the behavioral science, including creativity research, for centuries, also physicology will stall as long as we have access only to rather primitive tools for representing minute variations over time in the DNA–biochemistry–environment formula for people, the environment, society, and existence in general (see chapter 25). Progress in (creativity) research now depends critically on our ability to examine and control the nonlinear dynamics of molecular processes. We must become able to simulate data-dense real-time molecular mass-action processes by massive parallel/serial computing, simplifying graphics or, preferably, for real, or we might not be able to clearly see the nuts and bolts in the processes leading up to states we call social, intelligent, creative, genial, or mad. Eysenck's *Genius* book shows, that he is one of the most glorious fighters here, allowing his inner "Catherine wheel" to spin and spark once again, and ready to move as fast as ever in new directions.

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