4

The Neuropsychology of Sex-Related Differences in Brain and Specific Abilities

Hormones, Developmental Dynamics, and New Paradigm

Helmuth Nyborg

I. INTRODUCTION

The neuropsychological study of sex differences in specific mental abilities is a minefield of methodological and theoretical problems. We are faced with the paradox that neuropsychology abounds with empirical data and detailed clinical observations but lacks a unifying theory, whereas the study of sex differences abounds with (typically mutually exclusive) theories and correlation coefficients but is meager on good hard data. Smooth integration of neuropsychology with sex differences research is further made difficult by fundamental disagreement about possible reason(s) for differences in specific abilities, and because various putative reasons have not yet materialized into precise operational definitions that allow for formal testing in tight experimental designs. The final and perhaps most critical hurdle for progress is that sex researchers often lean exclusively on group mean data, whereas clinical neuropsychologists typically harvest idiographic information in case studies. Another line of research, psychometrics, often collects data on sex differences in specific abilities and typically uses factor analytic or partial correlation coefficient techniques. These approaches have yet to prove their value as a platform for testing causal hypotheses about brain mechanisms behind specific abilities. The net result of all this is that we have laboriously acquired a huge amount of questionable data about average sex differences in specific abilities

Copyright © 1994 by Academic Press, Inc. All rights of reproduction in any form reserved.

and are in urgent need of accurate theoretical interpretation and meaningful cross-disciplinary integration.

The main thesis of this chapter is that we now need a completely new, coherent, and integrative paradigm in the neuropsychological study of individual and sex differences in specific abilities. The tremendous recent advances in understanding the nuts and bolts of developmental neuroendocrinology suggest that this area holds potential for accomplishing such an ambitious goal. The past 10–20 yr of research in this field has thus produced new and often quite surprising evidence, which promises proper identification of the proximate base(s) for sexual differentiation. It seems possible within this field to tentatively define mechanisms and biological loci of action, and to apply this knowledge outside the field in order to derive rather precise and testable hypotheses about person-specific brain-ability relationships. Obviously, such a radical cross-disciplinary approach presupposes a readiness to accept that sex differences in specific abilities can be handled in ways that differ fundamentally in some respects from the usual psychological, genetic, and psychometric treatment. It presupposes, in other words, methodological as well as theoretical reorientation.

The chapter is divided into two parts. The first part is a selective review of observations made in accordance with the traditional sex-dimorphic approaches, and focuses on problems with these approaches. It is concluded that there probably will be little progress before adequate solutions are found to the following number of problems. It seems to have been a dubious procedure from the beginning to first calculate separate averages for male and female abilities, and then to test for statistical differences between the averages. There is little agreement about how to identify and operationalize the causal agent(s) behind sexual differentiation. The predictive power of specific ability scores for performance outside the test room is modest at best. More importantly, traditional theories of sexual differentiation cannot account for recent evidence of very dynamic gene–experience–brain–ability relationships. The aim of the second part of the chapter is to try and implement a paradigmatic shift in the neuropsychological study of sex differences in specific abilities. A physicochemical model for body and brain development is presented, and its theoretical and evolutionary framework is discussed. It is argued, that the model and the theory ordain revision of basic assumptions behind the traditional nature–nurture interaction model.

II. PART I: A SELECTIVE REVIEW OF SEX-DIMORPHIC STUDIES OF SPECIFIC MENTAL ABILITIES

One of the first written accounts of a sex difference in specific mental abilities is found in an ancient Sanskrit book, in which we are told that "Ten

shares of talk were handed down to Earth—the nine went to the women." This early view is repeated in more recent and systematic reviews of quantitative sex differences in mental abilities. Garai and Scheinfield (1968) and Maccoby and Jacklin (1978) thus suggest that females surpass males in some, but not necessarily in all, areas of verbal ability. With respect to development, girls speak on average earlier and better than boys, and boys are over-represented among stutterers, dyslexics, and general learning-disabled children. Females further outperform males on delicate fine-motor tasks and in some tasks requiring speeded perceptual scanning, whereas males outperform females in gross-motor tasks like throwing accuracy, even when no great force is called for (Jardine & Martin, 1983; Watson & Kimura, 1990), a difference that is better considered in an evolutionary perspective (Kolakowski & Malina, 1974).

One of the most pronounced and robust sex differences is seen in spatial abilities (McGee, 1979), and in the many activities assumed to draw upon good spatial skills. Unfortunately, despite wholehearted efforts (e.g., Smith, 1964) we still have no clear-cut definitions of the various spatial abilities and their interrelations. Nevertheless, however defined, males on average outperform females in most standard and also in not-so-standard tests for spatial abilities. Males excel in simple recollection, visual coding, and disembedding of geometrical shapes and figures, in mental rotation and identification tasks, in geometrical and in particular in mathematical problem-solving tests (but not in numerical tasks), in chess, in tests of direction sense, in visual and walking maze tests, in tactual mazes, in pattern walking, in map reading, in absolute direction tasks, in left–right discrimination, in aiming and tracking, in the rod-and-frame test and embedded-figures test, in geographical knowledge, in Piagetian three-dimensional tasks, in logical conservation, in the water-level test, and in music composition. Harris (1979) has provided an instructive overview of these sex differences. Males also dominate in educational and occupational areas believed to involve spatial abilities, such as architecture, physics, and engineering. It is worth remembering, however, that specific ability scores usually correlate only moderately with achievement in these areas. Finally, at the extreme ends of ability dimensions, there are more geniuses among males than females, but also more idiots. Male abilities tend, in other words, to vary more than female abilities.

Many more differences could be lined up here, but this is not necessary for the present purpose. First, excellent and fairly exhaustive reviews of sex differences are published regularly. More importantly, however, the sex differences already mentioned serve well to illustrate two major points, namely, that they mirror fundamental methodological and theoretical problems in the field, and that these difficulties prevent real progress in the neuropsychological study of sex differences in abilities. I shall first discuss the methodological problems, and then address some serious theoretical problems.

A. Methodological Problems

1. Introduction At least five major methodological problems impair sex-dimorphic studies of specific abilities. The first is that genetic, genital, or self-reported sex is a dubious classificatory variable for abilities. Exclusive reliance on male–female averages tends to obscure the considerable individual variation, and leaves out important information about individual differences. Second, it is a truism to say that the choice of research methods depends on the nature of the phenomenon to be studied. However, with respect to sex differences there is widespread confusion about the cause. This, in turn, makes it difficult to decide on the best (and probably only—see later) method for examining details of the cause(s) of the differences. Third, most specific ability tests show only moderate sex differences, but even a quick glance by the untrained eye indicates that men and women achieve quite differently in all known societies in areas believed to draw heavily on specific abilities, whether in education, occupation, or in top positions in the corporate or political power structures. Can we take the low correlation to mean that specific ability measures are inherently unreliable, or that they are so narrow in scope that they must be supplemented by further information from other areas related to specific abilities to be usable? Fourth, sex differences in specific abilities vary with age. This means that studies failing to consider neuropsychological sex differences in specific abilities within a developmental framework may be at fault. Fifth, ideology and concern over "political correctness" have a significant impact on research on sex differences. This is particularly obvious in the United States, where popular sexual policy at times hampers the rights of researchers to work in the area or makes publication difficult. But even in Denmark, considered by many as a politically and sexually most liberal country, it is almost impossible to fund studies of the biological basis of sex differences in abilities, whereas social learning approaches, questionnaires research, and correlation studies are generously rewarded with million-dollar funding in the absence of previous meaningful results. Even the construction of tests for specific abilities has been perverted so as to better conform to prevailing norms for "political correctness" or to the ill-defined notion of sexual equality. I will first discuss each of these problems in some detail, and then suggest methodological and theoretical corrections in order to promote a more adequate scientific program for the neuropsychological study of specific abilities.

2. Sex Dimorphism People seldom find it difficult to identify themselves or others as either male or female. Self-reported, genital, or genetic sex is, accordingly, an easy way of classifying humankind into two groups. There are, however, several very good reasons for not using this procedure when looking for the basis for difference in abilities. It is, for example, a risky business to go the other way around and tell an individual's sex from

knowledge about his or her specific ability score, as the distributions of male and female ability scores overlap to a considerable degree for all but a few areas. One possible exception is high-level problem-solving mathematics. Here male superiority increases rapidly with increasing item difficulty, so that there is virtually no overlap at the highest levels of ability (Benbow, 1988). But for most other abilities, the large within-group variability in abilities and the overlap in distributions both decrease the probability of finding statistically significant differences, so that the average sex differences in abilities typically hover around a modest 5% figure. This observation gives some researchers reason to conclude that the scientific or practical value of studying sex differences in abilities is low (e.g., Jacklin, 1979; Plomin & Daniels, 1987). Besides the custom of treating individual variation as statistical "noise," there are other reasons for not accepting this conclusion at face value. Whereas extreme within-sex scores counterbalance each other and moderate the group mean, the whole procedure makes it almost impossible to identify the cause(s) for the differences among individuals (Nyborg, 1977, 1983). To give an example, social learning theorists typically explain female superiority in verbal fluency by general or specific sex differences in upbringing. This intuitively understandable assumption is then tested by correlating average type of upbringing with average level of fluency. It is well known, however, that correlation does not prove causation, but the point here is that the relatively lower male and higher female verbal fluency averages usually cover a few high-ability males and low-ability females. The performance of these individuals is nevertheless "explained" by arguments used to explain also the opposite trend. The sex-dimorphic approach becomes a defective tool for unraveling the underlying individual cause(s) of development of a sex difference in verbal fluency, because it by and large misses exact information about within-sex high- and low-ability differences, and because it is unable to deal with causes except in the most general and abstract sense. The explanation, that girls excel in verbal fluency because they are reared in a particular way is compromised by the fact that some similarly reared girls score low on verbal fluency, and that some presumably differently reared boys score high. Sex dimorphism confounds, in other words, different individuals and masks causes. It might be essentially correct to declare, on the basis of available data, that sex differences in specific abilities are trivial, but the nature of the sex-dimorphic approach makes this statement more or less empty. In the ideology section I shall demonstrate that the statement is also perfectly circular. The relevant question is really whether there are proper ways to identify the sources of the tremendous individual and sex-related differences in abilities. In this manner we better substitute the nomothetic sex-dichotomous approach with a person-specific approach. Only by concentrating on individuals is it possible to avoid inadvertently confounding different people and to enhance the probability of identifying the proper causes for differences in specific abilities.

3. Causal Factors Obviously, research methods must fit the nature of the task at hand. If our hypothesis is that genes cause differences in abilities, we use specialized methods from molecular, population, or behavioral genetics. But most sex difference researchers employ one or another variation of the social learning approach. They typically inquire into people's experiences by asking them to fill out lengthy questionnaires, or observe them in various social or field situations. They then interpret the observations in terms of the hypothesis, that differences in social experiences, norms, or stereotypes explain differences in abilities. Investigators with a psychometric inclination collect data by test batteries, and then factor analyze them and derive principal and secondary components, or calculate partial correlations. From this they sometimes make inferences about underlying brain processes. Neuropsychologists typically study clinical cases and judge the results on an individual basis or with reference to more or less representative age, sex, or population norms. Aside from the clinical use of data, neuropsychologists also reason about the implications of reduced performance, as a function of circumscribed lesions in particular brain areas, of medication, or of sex-specific anatomical or functional differences in brain function (e.g., hemispheric lateralization), in order to explain sex differences or to develop a theory of brain–ability relationships in general.

Each of these approaches has greatly enlarged our descriptive knowledge, but with respect to a deeper understanding of the reason(s) for the sex differences in abilities none of them can be said to have enjoyed great success. The situation is rather that various approaches tend to produce mutually exclusive results, while not providing a clue as to which explanation is the scientifically more acceptable. We are faced with the ugly dilemma that literally thousands of studies have neither allowed us to operationalize the proper cause(s) of sex differences in specific abilities, nor to decide on the proper methods, interpretation, and integration of the mountain of data. One solution to this is to continue to shoot in all directions, to try and work out better theories for interaction, or to remain elective and combine the best from all approaches. So far none of these strategies promises rapid progress. Perhaps this view may appear unduly pessimistic to many experts in the field. I think, however, that most will agree with me that real progress depends on our ability to identify and operationalize the proximate cause(s) for sexual differentiation of specific abilities. Such a feat would greatly help us in choosing proper methods for further studies, preferably ones that allow for a rigorous falsification procedure.

4. The Developmental Perspective Everybody with experience in the field of abilities knows how essential it is to keep track of the developmental status of each single individual when studying sex differences. Most sex differences in abilities are rather small before puberty, but multiply within a short time window of a couple of years, take direction, and increase in size, to reach a

maximum during the later phases of puberty. Another interesting feature is that the appearance of differences in specific abilities is to some extent harmonized with the maturational tempo of the body (Petersen, 1976; Petersen & Crockett, 1985; Petersen & Taylor, 1980). Several studies have found that early and late maturation relates to different patterns of ability. Early-maturing individuals tend to end up with higher verbal than spatial abilities, and late-maturing individuals tend to display the opposite pattern of development (Crockett & Petersen, 1985; Waber, 1976, 1977a, 1977b). A controversial aspect of the development of abilities is that more females than males seem to actually regress in spatial abilities around puberty (Nyborg, 1988a; Nyborg & Nielsen, 1977; see review by Nyborg, 1983). The female regression has been explained in terms of increasing social pressure towards conformity in puberty, or by accumulation of sex-specific learning. Other theorists thought that the female regression was due to a subject sampling bias (Witkin, Goodenough & Karp, 1967). Few researchers have dared to ask if a biological explanation was appropriate. The model presented later in the chapter allows us to examine this possibility by generating testable predictions about whether particular females run a higher risk of regressing in spatial abilities around puberty. The model also makes predictions about which males are likely to suffer a similar regression.

5. Modest Ability–Achievement Correspondence Most educations and occupations require different involvement of specific abilities and show differences in the proportion of male and female participation. Some of these differences are large enough to talk about "male" and "female" educations and occupations (Danmarks Statistik, 1985; Ragins & Sundstrom, 1989). Moreover, the strongly biased distribution of males in top positions in almost all areas (Reid, 1982), and in the societal power structure in general (Goldberg, 1977; Wormald, 1982), is too obvious to be easily missed or pronounced trivial. On this background, it is indeed puzzling to realize, that most standard tests show a modest sex difference in the abilities required for achievement. It is equally surprising to learn that metatheoretical analyses of literally hundreds of studies of sex differences in abilities often come out with almost negligible differences (e.g., Linn & Petersen, 1985). As mentioned, these results are taken to mean that sex differences in abilities are "trivially small" (Jacklin, 1979), that sex differences diminish over time (Hyde & Linn, 1986; Jacklin, 1989), or that sex explains less than 5% of the total variability (Plomin & Daniels, 1987). The last mentioned study concluded that the major platform for explaining individual differences in the development of abilities and personality is social and extrafamilial. There are several alternatives to this explanation, however. Perhaps single-ability test scores are less than optimal predictors for later development and achievement. Even though ability tests predict later educational achievement better than teacher evaluation, tests differ with respect to how well they reflect a sex difference in a particular

ability dimension, and the ability measured in a particular study may not be entirely relevant for predicting later achievement in related areas. Ability tests may leave out vital information about *how* a given individual makes use of his or her specific abilities outside the test room. The position taken in this chapter is that ability test scores must be supplemented with covariant measures of other characteristics of the individual in question to gain credibility as a predictor variable. Such an idea is neither new nor without problems. The bold attempt by Witkin et al. (1954) and by others from the group around Witkin (Witkin, Dyk, Faterson, Goodenough, & Karp, 1962; Witkin & Goodenough, 1977; Witkin, Goodenough, & Oltman, 1977) to connect spatial ability, field dependence, and cognitive style with personality, within the concept of psychological differentiation, met with only moderate success, even though field dependence measures correlate with particular types of educational choice (Witkin, 1973; Witkin, Moore, Goodenough, & Cox, 1977). A particularly nagging problem has been that personality measures do not usually correlate well with specific ability scores. There may be a solution to these problems, as suggested later in this chapter. The basic problem may be how to first establish causal connections between specific abilities and particular personality traits, and then to relate these to the development and achievement of a single individual. The model to be presented later strives to formalize these connections in terms of a molecular bridge between genes, experience, the development of ability, body, brain, personality, and achievement.

6. Ideology Run Wild Research on sex differences in specific abilities is a sensitive area, and the ramification of ideology should not easily be dismissed. The field is looked upon by many with suspicion, and not a few believe that the topic is too controversial to be worth the trouble to investigate. Public debates often confuse facts with ideology and "what is" with "what should be," and this sometimes reflects back on science. Personal experience teaches me that public discussions often take the following general forms. There are no sex differences in abilities! OK, there are sex differences, but they are trivial. Well, perhaps some of the differences are important, but they arise from unfortunate socialization practices, are malleable, and will go away with time or a little help from friends. Well, sex differences may be important and genes may be responsible. This only means that we must make an extra effort to change peoples' attitude or society to counter the effects. By the way, do you know how biased ability tests are, and that—what was his name—faked the heritability estimates. In case none of this works, discussants may set unrealistically high scientific criteria for the collection and interpretation of data on sex differences, question the motives or moral integrity of the sex researchers, politicize the observations, or ask not to be bullied, patronized, or discriminated against. In this respect, research on sex and race differences suffers equal fate. At the same time, opponents often present correlations,

anecdotal evidence, or strong beliefs as counterevidence. This unsound semi-professional climate for examining sex differences in specific abilities has soured the soil for many studies, such as those by Benbow (1988), Hampson (1986), and Hampson and Kimura (1988). The major reason for mentioning such problems here at all is, however, that the hostile public atmosphere bounces back with measurable negative effects at all levels of sex research. Test constructors have, for example, felt forced to calibrate their apparatus to conform to dogmas of sexual equality. Preliminary versions of Wechsler's well-known IQ tests showed "unacceptable" male and ethnic advantages in overall IQ, but then certain subscale items, believed to be unfair to "certain minority groups" were removed, so that final versions of the test no longer showed a sex difference in overall IQ (Kaufman, 1975; Vogel, 1990; Wechsler, 1981). Conversely, the common female verbal superiority in the Scholastic Aptitude Test (SAT) was moderated by simply adding to the verbal content side areas outside the humanities—such that typically interest males more—and this favored male performance. However, no attempt was made to counter the male advantage in mathematics. These examples illustrate an important point. The "empirical fact" of no sex difference in *g* is entirely arbitrary. It is based on the ideologically inspired a priori decision that there must be no overall sex difference. It is therefore rather puzzling to see that recent analyses of the American standardization sample of the Wechsler Intelligence Scale for Children—Revised (WISC-R) by Jensen and Reynolds (1983) and of the Scottish standardization sample of the WISC-R by Lynn and Mulhern (1991) both indicate that boys no longer excel in performance IQ (which was expected from previous research), but now obtain a significantly higher full-scale IQ and, surprisingly, also a higher verbal IQ. Matarazzo, Bornstein, McDermott and Noonan (1986) reanalyzed the normative Wechsler Adult Intelligence Scale—Revised (WAIS-R) sample (Wechsler, 1981) and found that males significantly outperformed females on verbal IQ, on performance IQ, and accordingly on full-scale IQ. They find, however, that the differences are trivial, as their magnitude (1 to 2 points) is only half that of the standard errors of measurement on the WAIS-R (i.e., 2 to 4 IQ points) (p. 967). Interesting hypotheses about possible generational changes or changes in tests have been put forward to explain these observations. However, it is perfect circular reasoning to first carefully construct a test to show minimal sex differences, and then to conclude from data collected with that test that there are only trivial sex differences. The large-scale metanalyses of abilities ought really to show no sex difference at all had the test constructor done his work properly. What is at stake is that test construction, and the interpretation is influenced more by ideology than by reality, and that data collection is corrupted by the sex-dimorphic approach. It is actually very easy to construct an intelligence test showing either female or male overall superiority, depending on the number of verbal and spatial items included. The model presented in the second part of this chapter represents an attempt to avoid the

traps of sex dimorphism and ideology by taking its point of departure in different individuals rather than in average sex differences, and by accounting for sex-related variability without supporting any particular sexual ideology.

B. Theoretical Problems

1. Introduction We have a number of more or less mutually exclusive genetic, social learning, interaction, and cognitive theories to explain sex differences in abilities. This is a clear sign of confusion. In this section I first outline some of the theoretical restraints that give rise to bewilderment, and then suggest a reorientation that is less vulnerable.

2. Genetic Explanations Genetic explanations of specific abilities basically come in three versions: the X-linked recessive-gene hypothesis, the polygene hypothesis, and the multifactorial hypothesis. The X-linked recessive-gene model was originally proposed by O'Connor (1943) and later supported in studies of Stafford (1961, 1963, 1965). The model assumes that a "good" gene for high spatial ability exists with a certain frequency in a given population. The gene is recessive and is linked to the X chromosome. The two sexes have an equal chance of receiving the gene, but males with one X chromosome need receive only one "dose," and females with two X chromosomes need the gene in double "doses" to express high spatial ability. Leaving out details here, the model predicts various familial transmission-pattern frequencies for spatial ability. These predictions found some support in the early studies, but did not hold up in more recent studies (Bouchard & McGee, 1977; Corley, DeFries, Kuse, & Vandenberg, 1980; Nyborg & Nielsen, 1981a, 1981b; Vandenberg & Kuse, 1979). It is now generally acknowledged that the X-linked recessive-gene hypothesis for specific abilities is inadequate.

Polygene models posit that the expression of specific abilities depends on a number of separate genes, each with a small additive effect. Multifactorial models acknowledge that multiple genes and multiple environmental factors combine in the expression of abilities. A major problem with such models is that we still do not know which genes combine with precisely which environmental factors. Neither the causal mechanisms nor the levels at which the effects are mediated are identified. How can the effects of, say, sex stereotypic rearing be transmitted to the brain, and where in the brain do they meet to affect the expression of specific abilities? There is another perhaps more serious problem with genetic models. Theories of population genetics and behavior genetics are applicable at the level of populations only. As discussed later (and elsewhere, Nyborg, 1977; Nyborg & Sommerlund, 1992), this restriction makes it virtually impossible to identify causal factors at the level of the individual, and this is the information we need most. Heritability coefficients are of great value for determining the average impact of genetic transmission, but they reflect a high level of statistical abstraction

of little relevance for understanding the individual. To identify the reasons for the recently observed dynamic perturbations of specific abilities in adult females we have to move to the level of the single individual.

3. Social Learning Theory Undoubtedly, only few learning theorists would insist that social experience is the only determinant of the development of specific abilities, but most emphasize the profound effects of upbringing, of social norms, of stereotypes, and so forth on abilities, at the expense of biological factors. Females are often said to excel in verbal areas because they passively model female role figures, or because they are positively or negatively reinforced to do so. Sherman's (1967) "Bent-twig" hypothesis presumes an early biologically based inclination for superior female verbal ability, and that this "Bent" is later socially forged into the adult sex difference in verbal abilities. We like to do what we do best. Boys have an initial bent for spatial ability, so they foster this ability at the expense of verbal ability. Genetic differences are seen by many social learning theorists as nonexistent or quite irrelevant. In this they follow the lead from the early American version of behaviorism. They further see the brain as sexually undifferentiated or tend to downplay effects of known sex differences in neuroanatomy and brain function on abilities.

The primary reason why socialization theory failed to explain the development of sex differences in specific abilities lies in the fact that putative causal factors like upbringing, modeling, norms, and stereotypes have never been operationalized. Mechanisms for transcribing putative exogenous effects in the social field into "endogenous" norms or attitudes and then again into effects on the expression of specific abilities, are left to speculation (Nyborg, 1983, 1992a, 1992b, 1993a). The principles of social learning theory make intuitive sense, but the empirical basis is essentially nondirectional correlations. As correlations do not prove causation, exclusive reliance upon them precludes further progress with respect to elucidating causes, effects, and direction.

Whereas social learning theory tends to view the brain as equipotential, most neuropsychologists have little problem with accepting the possibility that there may be important sex differences in the way the brain is organized and functions. What, then, is the neuropsychological evidence for sex differences in the brain?

4. Neuropsychological Explanations

a. Absolute Brain Size Studies of sex differences in the size of the brain and of size-specific ability relationships have not been particularly informative until quite recently. Many studies show that males have a larger brain than females, and this was originally taken as proof that males are more intelligent than females. It was then argued that females may actually have the same size

or a relatively larger brain than males, with control for body weight. The notion of a direct brain size–intelligence relationship was under fire during this long-standing and sometimes emotionally colored dispute. Defenders of the brain size–intelligence connection referred to a well-documented relationship between brain size and "intelligence" across species. Critics pointed to the fact that some of the largest brains were found in idiots, and very small brains were observed in highly gifted people. However, various aspects of the techniques for measuring brain size were criticized, and so was the quality of the brain preparations examined.

Until recently it was therefore generally accepted that the sex difference in corrected overall brain size was modest at best, and that size per se probably did not mean much to level of abilities. Then Willerman, Schultz, Rutledge, and Bigler (1991) measured brain size with magnetic resonance imaging (MRI), and were able to correct for individual differences in skull thickness and body size. They found that the high-IQ group (WAIS-R full-scale IQ 130; $M = 136.4$, $SD = 3.95$) had larger brain average ($p < .05$) than had the average IQ group (WAIS-R full-scale IQ 103; $M = 90.5$, $SD = 8.12$). A sex difference in adjusted brain size–IQ correlation was not significant ($p = .08$) but large enough (female $r = .35$ vs. male $r = .65$) to suggest a possible sex difference in brain organization. Johnson (1991) used a method developed by Van Valen (1974) and found an estimated brain size–intelligence correlation of .29. Ankney (1992) reanalyzed data on sex differences in brain size from autopsy records of 1261 25–80-year-old subjects originally studied by Ho, Roessmann, Straumfjord, and Monroe (1980). The reanalysis indicated that the absolute brain mass of white males and females of average height differ by 135 g, and that correction for body size reduces the male lead to 100 g. A similar result was obtained in black female–male comparisons. After correction for stature and weight, Rushton (1992a) found a male lead in cranial capacity (i.e., 1442 versus 1332 cm^3, as derived from external head measurements) in a stratified random sample of 6325 United States military personnel. Finally, Rushton (1992) calculated cranial capacity from external head measurements collected by Jurgens, Aune, and Pieper (1990). He noted a 173 cm^3 male lead in a very large population of 25–45-year-old males and females.

Obviously, these findings will appear controversial to many. The measures, the formulas, and the interpretation undoubtedly will be carefully scrutinized by skeptics. However, the consistency in the data from independent studies presents us with some interesting puzzles. Males seem to have a larger brain than females. Brain size seems to correlate moderately with overall intelligence. Common intelligence tests have been "adjusted" not to show sex differences. Recent large-scale studies nevertheless suggest a slight male advantage in overall intelligence. It is, in fact, possible to formulate various sophisticated hypotheses to make ends meet. My suggestion is simple. The sex-dimorphic approach indiscriminately bundles sometimes very different individuals and prevailing ideology perverts test construction. The cure is to

apply individual-centered approaches and to become aware of and counter the damaging effects of sexual ideology.

b. Brain Anatomy More and more sex differences are found in the neuroanatomical organization of the brain. The right hemisphere may be thicker than the left in males, but not different in females (Diamond, Dowling, & Johnson, 1981). The area of planum temporale is larger in the left than in the right hemisphere, irrespective of sex (Witelson & Pallie, 1973), and relatively larger in females than in males (Wada, Clarke, & Hamm, 1975). The functional significance of this has been related to superior female verbal ability, but the evidence for this is not strong. Animal data (mostly from rats) have long testified to the existence of a sexually dimorphic nucleus in the preoptic area of the hypothalamus (Gorski, 1984; Jacobson, Davis, & Gorski, 1985; Tarttelin & Gorski, 1988), and to sex differences in the corpus callosum, a broad band of fibers that connects the two hemispheres transversally (Denenberg, Berrebi, & Roslyn, 1988). These differences are moderated by exposure to gonadal hormones. There is some confusion as to the functional significance of this anatomical sex difference in the rat brain. It may relate to the regulation of male sexual behavior, although previous sexual experiences of the animal apparently also play a role (Jonge et al., 1989). Recently Swaab and Fliers (1985) reported a similar finding in humans, namely, a nucleus in the preoptic area of the hypothalamus that differentiates according to sex. The differentiation begins at postnatal age 2 when the female nucleus loses cells at a dramatic rate. At age 50 the rate of cell loss again accelerates, but this time in males as well as in females (Hofman & Swaab, 1989; Swaab & Hofman, 1988). The implications of this for sexual behavior in old age or in general are not known, and this particular sex difference has not been demonstrated to relate to specific abilities. A controversial topic is whether there is a human sex difference in the size of the corpus callosum. An early report on a larger female than male corpus callosum (Lacoste-Utamsing & Holloway, 1982) triggered speculations about whether this anatomical sex difference could explain the presumed more bilateral female brain organization and better verbal ability. A number of subsequent studies were unable to confirm the original observation of a sex difference in the corpus callosum, or gave only partial support (O'Kusky et al., 1988), using various measurement techniques, including MRI and cerebral blood-flow approaches. A recent study suggested that there probably is no sex difference in overall anatomical size of the corpus callosum, but there may be a sex difference in its shape (Allen, Richey, Chai, & Gorski, 1991).

Excellent reviews of these and many other sex differences in the brain anatomy can be found in Goy et al., 1980; Kimura, 1983, 1987; McGlone, 1980, 1986; and Swaab, Hofman, and Fisser, 1988. The caveat is that most of these data are collected within the constraints of the sex dimorphic approach, which maintains that genetic, genital, or self-reported sex is a befitting classification variable.

c. Functional Brain Organization The early studies of sex differences in functional brain organization yielded a mountain of puzzling data, and their interpretation is problematic. We know, for example, that a simple functional left–right brain terminology with related absolute sex differences in verbal-performance IQ needs qualification. Early lesion studies indicated that damage to some areas in the right side of the brain lowers spatial abilities more in males than in females, but tends to leave male verbal abilities relatively unaffected, whereas damage to the left side of the brain lowers verbal abilities more in males than in females, but tends to leave male spatial abilities relatively intact. These observations led to the assumption that the male brain was functionally organized more unilaterally, and the female brain organized more bilaterally. It has gradually transpired, however, that degree of functional brain lateralization, kind of ability, level of task complexity, handedness, and sex, are all interrelated. Many attempts have been made to identify the subserving neuroanatomical substrates, and to determine the developmental timetable for the lateralization processes, but newer evidence suggests that the developmental brain architectural–functional relationships are more complex than a simple left–right sex-dichotomous terminology would allow for. It may very well be that both male and female brains are slightly differently lateralized for some relatively automated executive motor functions, and that circumscribed areas in the female left anterior hemisphere may be more lateralized or better organized for verbal skills than the female posterior and the male anterior and posterior left hemisphere (Kimura, 1983; Kimura & Harshman, 1984; McGlone, 1980). Moreover, with increasing task complexity both verbal and spatial performance seem to call upon concomitant neural activity in both hemispheres, although perhaps to a different extent in the two sexes.

Further adding to complexity in the area, Juraska (1984, 1986; Juraska, Fitch, Henderson, & Rivers, 1985) and others have demonstrated sex differences in the effects of experience on neuroanatomical development. Harris (1980) and Nyborg (1983) have, among others, called attention to the possibility of anatomical and/or functional sex differences in subcortical areas with an effect on patterns of selective activation or inhibition of cortical activities.

A relatively new line of research—psychoneuroendocrinology—suggests that it might be a good idea to call upon a more molecular approach in order to elucidate the functional aspects of brain–ability relationships. This is so, because steroids obviously hold exciting potentials for explaining concomitant development of the brain and abilities. Prenatal and later brain effects of hormones may, in fact, constitute one of the most direct ways to experimentally combine structural with functional aspects of brain neuroanatomy and chemistry. To give an example, early steroid treatment affects subcortical development and ability. Pavlides, Westlind-Danielsson, Nyborg, and McEwen (1991) treated neonatal rats with thyroid hormone, a treatment which speeds up prepubertal hypothalamic development and initially improves learning perfor-

mance compared to untreated controls (Schapiro, Salas, & Vukovich, 1970). However, adult rats treated neonatally with thyroid hormone demonstrated defective spatial learning in an eight-arm radial maze, and also showed impaired electrophysiological long-term potentiation in the dentate gyrus of the hippocampus. This was taken to reflect reduced synaptic efficacy and neural plasticity. Other steroid influences will be outlined later in connection with a presentation of psychoneuroendocrinological evidence for sex-related differences in the developmental pattern of the left and right cerebral hemispheres and for different ability patterns in subgroups of homosexual men.

5. Psychometrics Truly quantitative approaches, such as psychometrics, have yielded an invaluable body of descriptive data on individual and sex differences in specific mental abilities. Unfortunately, even the most exact psychometric study runs into problems when it comes to explanations. Some doubt was recently expressed on empirical grounds, whether purely descriptive nonexperimental approaches—such as psychometrics—will ever disclose the nature of putative brain processes subserving specific abilities (Lynn, 1990; Nyborg & Sommerlund, 1992; Vernon, 1990). The only way to disclose whether knowledge about neuroanatomy and functioning applies to specific abilities is through the study of the brains of single individuals (Nyborg, 1977), and we urgently need a coherent theoretical framework that does not confuse different levels of explanation (Nyborg, 1993a).

C. Concluding Remarks

Genetic, social learning, neuropsychological, and psychometric studies have provided invaluable information about average sex differences in specific abilities. The major problem with these studies is, however, that they do not provide us with the information we would like to have most of all. We want to know exactly what are the cause(s) of sex differences in specific abilities. We want to know details of their causal nature, their mechanisms, and their biological locus of action. We want to identify the proper level of description of interactions among the various causal agents involved. We want to establish a precise, comprehensive, and testable theoretical framework in order to better understand relevant observations made in other fields. None of the traditional theories offer that much. The genes, held responsible for sex differences in abilities, have yet to be identified. Too much social learning theory is based on anecdotal evidence, intuition, correlation coefficients, and many results or interpretations are carefully adjusted to conform to sexual norms for equality or for political correctness. Most neuropsychologists operate with empirical data, and some even rise to the level of experimental control, but neither clinically oriented nor experimentally oriented neuropsychologists have yet been able to formulate a comprehensive, coherent, and generally acceptable neuropsychological theory about the brain-specific abil-

ity connections. Psychometrics is strong on descriptive analysis and on quantification, but is in principle and in practice unable to outline the brain mechanisms behind specific abilities.

I suspect that the main problems with traditional theories is that they all accepted at face value the sex-dimorphic strategy. Sex dimorphism is not just a misleading term. It inevitably confounds within-sex high- and low-ability males and females (see also Nottebohm, 1980). This indiscriminate within-sex mixing of sometimes fundamentally different individuals, superficially made similar by sexual classification, may be the most serious obstacle on the way towards proper operationalization of cause(s) and mechanisms. With the possible exception of neuropsychology, most theories further operate simultaneously at several incompatible levels of explanation. Social, genetic, mental, and neural levels are tied together by unspecified interaction processes. These two objections go, I believe, to the heart of reasons for the conspicuous lack of progress in the study of the causal nature of sex differences in specific abilities. We need a fresh approach!

A fresh approach must, first of all, take its point of departure in the study of single individuals (Nyborg, 1987a). The individualized approach enables us to probe directly into those parts of the brain that are believed to be functionally important for the appearance of differences in specific abilities, while at the same time this approach fully appreciates the large individual variation. Only if enough individuals were found to be similar with respect to a given characteristic would we have stumbled over something genuinely general (Nyborg, 1977; see also Allport, 1962; and Runyan, 1983). The within-group variation reflects the extent to which it makes sense to talk about a modal trait. This is the opposite of trying to establish valid principles for individuals on the basis of statistical averages or factors. With respect to sex differences, the study of modal trait development allows for the obvious possibility that some traits show a sex-*related* tendency, but in that case we have to operationalize sex more adequately than phenotypic or self-reported sex allow for (see Section 8). The fresh approach should further enable us to directly address questions about proximate causes for individual and sex-related differences in brain development, and to identify specific mechanisms and loci of biological action. The new approach should allow us to operate at one level of explanation instead of many. This is the way to escape the problem of incompatibility. The approach should be empirically testable at each step of exploration and explanation. The new approach should allow us to empirically examine details of concomitant ontogenetic brain development and associated individual and sex-related differences in specific abilities. The new approach should, finally, facilitate the interpretation of the results within a phylogenetically meaningful perspective of ultimate causes. Obviously, all this means nothing less than a paradigmatic shift in the traditional exploration of sex differences in specific abilities. This shift would imply a rather reserved look at data obtained through sex-dichotomous procedures, and a keen interest in safe

identification of the cause(s) for covariant individual and sex-related differences in body, brain, and ability development.

III. PART II: THE GENERAL TRAIT COVARIANCE–ANDROGEN/ESTROGEN MODEL

A. Introduction

The general trait covariance–androgen/estrogen (GTC–A/E) model reflects an attempt to formalize effects of genes, gonadal, hormones, and experience, on body, brain, and behavior in a way that overcomes problems associated with the sex-dimorphic approach (Nyborg, 1979, 1983, 1988a, 1988b; Nyborg & Nielsen, 1977). The model represents, in fact, an attempt to explain complex individual and sex-related covariant trait development as a combined function of the reciprocal effects of genes, hormones, and experience on body and brain tissues. The model presumes that males and females are made individual by genes, experience, and hormones, and that useful predictions can be derived after hormotyping them. A complete hormotyping also incorporates family dispositions (heritability estimate of genetic transmission until more precise DNA measures become generally available) and personal life history events (including prenatal experiences), but most of this remains a task for the future. As soon as an individual is defined according to hormotype, the model generates predictions about the most likely development of his or her body, brain, and behavioral trait pattern in a form that can be tested immediately. The present neuropsychological context makes it natural to emphasize the molecular gene–environment–hormone–brain–behavior mechanisms used to explain unfolding of the specific ability patterns. The model is presented graphically in Figure 1. Besides specific abilities this version of the model also deals with a few of the other traits that tend to harmonize with the particular ability pattern of the different hormotypes. A more elaborated version of the model dealing with more traits is presented elsewhere (Nyborg, 1992a, 1992b), as is evidence that the model successfully predicts covariant personality trait development (Nyborg, 1984). The theoretical framework behind the model is called physicology, and is presented in part in Nyborg (1988a, 1989, 1992a), and in detail in Nyborg (1993a).

Only a few of the many molecular mechanisms believed to mediate steroid-based development and function will be outlined here. The field is vast, and grows at an exponential rate, so the discussion will be focused on mechanisms by which gonadal hormones regulate gene expression prenatally and later in life, and on the recently observed capability of gonadal hormones to quickly change nerve membrane characteristics and thus mimic effects of neurotransmitters. Neither do space restrictions allow a detailed overview of hor-

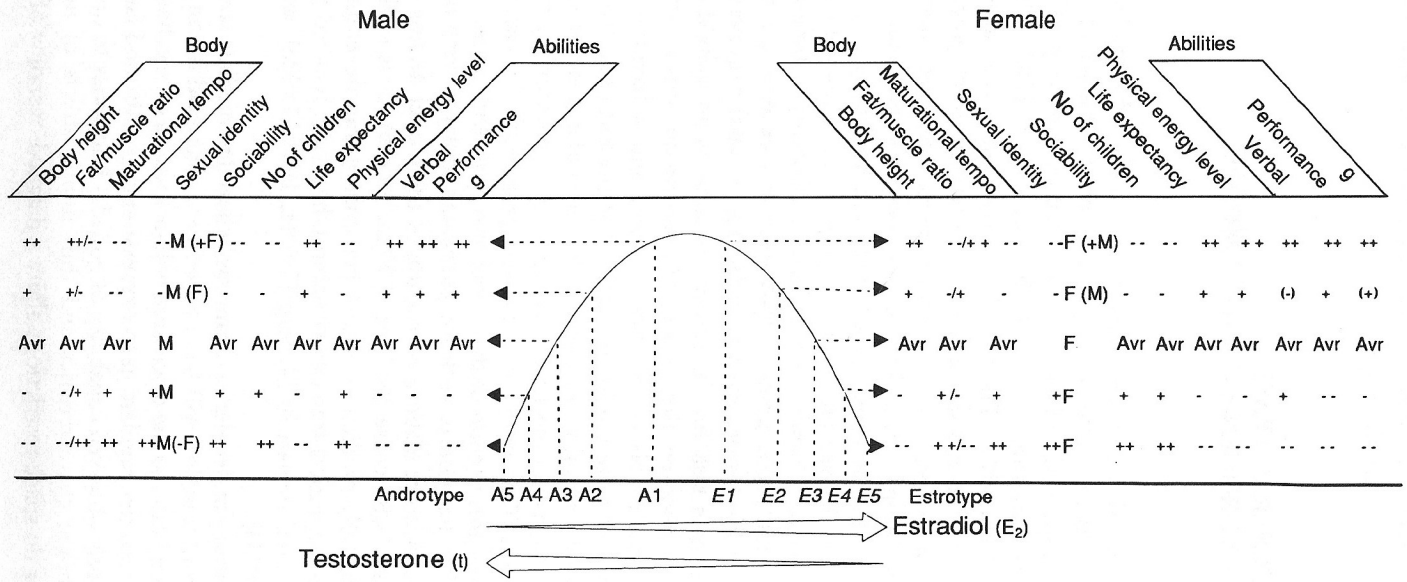


FIGURE 1

The General Trait Covariance–Androgen/Estrogen (GTC–A/E) model for development. Males are classified in accordance with plasma testosterone (t) concentration into hormotypes A1 to A5, where A1 is low t and A5 is high t individuals. Females are classified in accordance with plasma estradiol (E_2) concentration into hormotypes E1 to E5, where E1 is low E_2 and E5 is high E_2 individuals. The GTC–A/E model generates predictions for the various hormotypes with respect to coordinated somatic, psychological, and behavioral trait development (see text for details). The present version of the model is borrowed in part from Nyborg, 1979; 1983; 1984; 1987b; 1988a; 1990b; Nyborg & Nielsen, 1981b.

monal effects on neuronal architecture, but good discussions can be found in Toran-Allerand (1984, 1986). As mentioned previously, the GTC–A/E model readily acknowledges that gonadal hormones do not work in isolation. Various experiences transiently or permanently affect the production of gonadal hormones, as for example under short-term or long-term stress. The causal pathway for experience goes, according to physicology, through physicochemical changes in the peripheral sensory systems as a function of variation in the entirely physical environment (including physical people's physical behavior). The causal pathway also includes hormonally mediated modulation of gene transcription and neurotransmitter changes in the brain, often with an effect on molecular memory systems, and sometimes with an effect on the neuroregulation of executive motor systems leading to overt behavior. Nonetheless, it is constantly under the influence of positive or negative feedback and feed forward adjustment from various physicochemical systems. In this respect the model represents an attempt to explain the extremely complex and dynamic molecular aspects of interactions among experience, gonadal hormones, genes, and the brain.

B. The Dual Role of Hormones in Evolution and the Ontogenesis of Specific Abilities

1. Evolutionary Aspects A brief look at evolutionary theory helps illustrate why physicology considers steroids the most central of ultimate causes in the evolution of life forms with complex central nervous systems and so-called intelligence. The following section outlines why physicology also puts steroids, together with DNA material and experience, in the role as *the* proximate causal agents in the ontogenesis of individual and sex-related differences in specific abilities.

Natural and sexual selection is, according to Darwin, the driving force behind evolution. Selection takes place among different individuals, so it became essential for Darwin to identify the origin of individual differences. Unfortunately, Darwin never opened a letter he received from a then little-known Austrian monk, Gregor Mendel, which explained the rules for heritability of simple traits. Darwin was therefore forced to reluctantly resort to a kind of Lamarkian thinking, which acknowledges that the environment exerts a heritable impact on the individual. However, when Watson and Crick broke the genetic code it became obvious that robust chemical instructions, in the form of particular combinations of four relatively simple nucleic acids, explain stable species-specific ontogenetic development, and that structural and numerical mutations plus gene combinations plus environment superimpose ontogenetic individualization on otherwise remarkably stable species-specific programs.

Gonadal hormones are vital for the evolution of complex life forms in general (Hapgood, 1979; Roth et al., 1982; Symond, 1979; Witzmann,

1981). With respect to specific abilities, there are several good reasons why gonadal hormones become of particular interest (Nyborg, 1992a, 1993a). First, they enable the evolution of the sexual reproductive mode, and this confers a major competitive advantage with respect to superior sexual selection: It became possible to quickly and efficiently combine favorable mutations and new gene constellations for body and brain structures and functions that best subserve survival and reproduction of parents and of the next generation. This is one reason why gonadal hormones became an important ultimate evolutionary cause for successful adaptation to life-threatening challenges in a variable world. Second, besides conferring an evolutionary edge through the appearance of the sexual reproductive mode, gonadal hormones have another long-term advantage. During evolution they exerted profound and very direct effects on the timing of the single individual's ontogenesis, that is, on the unfolding of various anatomical and functional body and brain structures, in close coordination with environmental requirements. The most obvious evolutionary advantage of this is a survival bonus to species that responded fast and adaptively to harsh conditions during upbringing and in later phases of life. In this way reproductive capability could be adjusted effectively with nutritional and stressing conditions to save precious energy. To make a long story short, gonadal hormones constitute, according to the physicological viewpoint, the molecular bridge in the evolution of complex life forms between accumulated effects of numerical and structural mutations and of gene recombination, the adaptive regulation of body and brain development and reproductive behavior, and sexually biased social interaction. To the extent that gonadal hormones and reproductive aspects color so-called social interaction, gonadal hormones constitute the proximate basis upon which all complex forms of society have to be understood, according to physicology. They provide the molecular basis for Freud's speculation that sex penetrates all aspects of culture. The point is that species are certainly made in accordance with specific genetic information in DNA, but the gradual making of maleness and femaleness (or of everything in between) during evolution presumes proximate and rather specific effects of gonadal hormones regulating protein production of the genome in hormophilic tissues. Without gonadal hormones there will be no sex-related differentiation, and the evolution of species with complex central nervous systems would have been slower or even missing completely.

2. Ontogenetic Aspects For a long time it was believed that there are two phylogenetically stable gene programs for sexual differentiation: one for female development and one for male development. The male program was believed to be the female program modified by additional information from the Y chromosome. Femaleness was, in other words, seen as more basic, and maleness as a kind of add-on to the female program. Recent findings indicate that all fetuses more likely begin body and brainwise as sexually undiffer-

entiated, except for the presence of a Y chromosome in male fetuses. The Y chromosome provides for the formation of testicular tissues, and the later androgen output down-regulates genes for female development, and up-regulates genes for male development. It has further been suggested that prenatal female brain development depends on relatively low estradiol (E_2) output, whereas higher output of E_2 masculinizes the brain (Döhler & Hancke, 1978; Döhler et al., 1984). Whether this can be verified remains to be seen, but it is safely established that androgen can be aromatized to E_2 in the brain, and that gonadal hormones regulate genes to promote male and female variations in initially neutral body and brain tissues (McEwen, 1987a, 1988a), in reproductive behavior (Gorski, 1974; McEwen, Jones, & Pfaff, 1987; Romano, Mobbs, Lauber, Howells, & Pfaff, 1990), and in nonreproductive behavior at the most complex levels (Hoyenga & Hoyenga, 1979; Nyborg, 1979). This newer experimental evidence indicates that if there are some kind of phylogenetically stable gene programs for separate phenotypic male or female differentiation, they can be entirely overruled by manipulation of gonadal hormone content during early phases of development, by blocking actions of relevant hormones, or by influencing the induction of specific receptor proteins for gonadal hormones.

Early in the century it was realized that infrahuman and other animal reproductive behavior depends on the presence of gonads and, by inference, on hormones. With improvements in techniques for refining and measuring gonadal hormones it gradually became obvious that not only human body and brain development, but also performance remotely related to reproductive functions, was profoundly affected by hormones. The expression of verbal and spatial abilities, as well as of personality was seen to be significantly influenced by naturally occurring and medically provoked hormone perturbations. These observations line up with the hypothesis that all sex-related differences in body, brain, and behavior are a function of proximate effects of gonadal hormones, and that the sexually most dimorphic traits show the strongest sex hormone involvement (Hoyenga & Hoyenga, 1979). Talking about effects of gonadal hormones it is worth noting that both sexes produce "male" and "female" sex hormones, and that "male" hormones often act as prohormones for "female" hormones in both sexes. This makes terms like "male," "female," and "sex hormone" rather ambiguous. They will, therefore, in the following be substituted with the more neutral expression "gonadal hormone," or be referred to by their family name (e.g., androgen and estrogen), or by their specific name (e.g., testosterone, t, and estradiol, E_2, to mention some of the more important hormones in the present context.

C. Theoretical Implications

The choice of gonadal hormones as *the* ultimate and proximate causes for sex-related differentiation of specific abilities carries with it a number of

theoretical implications. Being an integrated part of the physicological framework, they are better made explicit here before a presentation of the GTC–A/E model. Otherwise, the new paradigm might too easily be confused with elements of the traditional sex-dimorphic view or we might continue to operate at several levels of explanation. For example, it makes absolutely no sense within a physicological framework to assume that gonadal hormones affect abstract mental entities. Gonadal hormones become biologically active exclusively through molecular interactions with genes, other chemicals, or membrane proteins, and this can have an effect on body and brain architecture and function. The GTC–A/E model is designed in accordance with this, and does not operate with concepts like "mind," "mental" or "cognitive" ability, "thinking," or "general intelligence." These abstractions have no place in a modern molecular account of behavior. They may, however, be used (with extreme caution) as convenient shorthand descriptors, as long as it is clearly understood that they refer to complex 100% material processes going on in the brain. Mental concepts are, in other words, completely stripped in physicology for the explanatory value previously ascribed to them in the mentalist framework. They have no causal status whatsoever. This means that hormones are not assumed in the GTC–A/E model to guide mental development, and they have neither "purpose" nor "goal." Gonadal hormones rather increase or decrease the probability that body and brain development takes particular directions, by regulating the expression of species-specific genes of importance for the appearance of particular traits. When reference is made in the following to the development of a positive manifold of abilities, such as for example, the sum of verbal and nonverbal abilities, Spearman's (1904) more neutral designator, *g*, is used, again under the assumption that we mean brain function and not mind.

Another aspect of the new paradigm is that the level of explanation is confined to one and one only, namely, the molecular (Nyborg, 1988b; 1992a; Nyborg & Bøggild, 1989). The emphasis on the material basis of sex-related differences in specific brain abilities makes it possible to provide coherent explanations of their development. There actually may be no other scientifically acceptable way to avoid the classical, anthropocentric, dualistic body–mind trap, but this is not the place to discuss details of the arguments for the physicological point of view, for which the reader is referred to Nyborg (1993a). Suffice it here to state that the choice of gonadal hormones as the proximate agents for sex-related differentiation makes it possible to circumvent most of the problems associated with traditional sex dimorphism. Classification of individuals in accordance with their hormotype (see Section D) solves another problem. Males typically have higher plasma androgen concentrations and lower E_2 than females, but the levels of gonadal hormone in plasma are clearly continuously distributed, and there is overlap between male and female distributions. This encourages a nondichotomous classification of individuals. Add to this that "male" hormones often act as pro-

hormones for "female" hormones in both sexes, and that the rate of conversion from one hormone to another varies on an individual basis, and the advantage of using models that operate with continua on the causal as well as on the effect side becomes obvious. It follows that the tradition of dividing individuals into two sexes is better substituted by the more flexible and individualized classification, and that the expression "sex-related difference" is, if not perfect, a more adequate term than "sex difference." The only reason for not exclusively using the term "individual differences" is that quite often a particular modal pattern of specific abilities actually goes together with self-reported sex. However, the ability pattern of some individuals can be very different from that of their same-sex fellows, and closer to that of the heterotypic sex (depending on genes, experience, and hormotype). In such cases "sex differences" becomes a misleading term.

The new paradigm necessitates a revision of our classical nature–nurture model. This is so, because gonadal hormones regulate the protein production of the genome throughout life, and this violates several basic assumptions of the traditional nature–nurture interaction model. Some implications of this are discussed later.

After these brief remarks about the dual role of gonadal hormones in evolution and ontogeny, I turn to the task of hormotyping. Procedures for classifying individuals are presented first, and then the phenomenon of trait harmonization is alluded to. Examples of trait harmonization are provided with the presentation to illustrate the role of hormones in this orchestration of trait development.

D. Hormotyping

There are two basic ways to classify individuals according to hormotype: (1) indirect estimates through anthropometry, behavioral evaluation, or questionnaires, and (2) direct measures of gonadal hormones in plasma, urine, or saliva.

1. Indirect Estimation Indirect estimates of hormone status can be made on the basis of quantitative or qualitative examination of various objective body characteristics (Marshall & Tanner, 1986) that typically differ among the sexes. Among those used are shoulder and hip width, amount and distribution of fatty tissues, muscular strength, distribution of hair, and deepness of voice (Bourguignon, 1988; Crockett & Petersen, 1985; Farthing, Mattel, Edwards, & Dawson, 1982; Petersen, 1976; Petersen & Taylor, 1980; Waber, 1976, 1977a, 1977b, 1979; Young & Reeve, 1980). Such traits are known to be affected by gonadal hormones (Farthing et al., 1982; Tanner, 1975). Other indirect indicators of hormonal status are aggression and dominance, or self-confidence and sexual (gender) identity, often measured by questionnaires, such as the Bem (1983) Scale or Berzins, Welling, and Wetter's (1978)

Personality Research Form (PRF) Androgyny Scale. The PRF Andro Scale was, for example, used successfully to show that individuals with high masculinity or femininity score (assumed to reflect high androgen or E_2 status, respectively) encountered problems in nonverbal ability tests, whereas androgynous individuals with some high masculinity *and* some high femininity score (assumed to have intermediate hormone values) as well as sexually undifferentiated individuals with low male *and* low female score (assumed to have low hormone status) did well in tests for spatial ability, irrespective of genetic sex (Bøggild & Nyborg, 1992). Berenbaum and Resnick (1982), McKeever (1986), and Petersen (1976), found that physically androgynous males and females score higher on spatial ability tests than do sexually more developed individuals.

Despite some success, neither anthropometric measures nor scores on personality questionnaires can be said to provide very accurate estimates of hormonal status (Knussman & Sperwien, 1988). There are several reasons for this. Individuals differ in sensitivity to circulating hormones, and the sensitivity varies over time, because of changes in the number of hormone receptors induced, which relates to changes in the maturational state of tissues. In addition to gonadal hormones, adrenal hormones also affect body and brain development, and many other factors contribute to biochemical variation. This suggests that indirect estimates of hormotype should be used with caution, and only when direct hormone determination is out of the question.

2. Direct Measures Techniques for direct measurement of plasma gonadal hormones have improved considerably over time, but there are still problems. Blood sampling is a stressing event to some, and stress affects the endocrine system. One sampling of blood is the minimum, but a series of consecutive blood samples is preferred because of the pulsatile nature of hormone secretion. Particular individuals may refuse to participate in studies requiring blood sampling. Laboratories often use different techniques or chemicals for assaying, which means that data from one laboratory cannot safely be compared with hormone data from another laboratory without a common quality control. Hormone content is related to age, weight, medication, nutrition, and race, and techniques for analysis undergo constant improvement, which complicates the evaluation of longitudinal measures. Hormone content can further be estimated by analyzing urine, typically collected over a 24-hr period. Urine sampling is less stressing than collecting blood, and allows compensation for diurnal variations in hormones that are secreted in brief pulses followed by long intervening periods with little secretion. A drawback is that the estimation of plasma hormone concentrations from urine typically is based on measurement of metabolites of the plasma hormones. It has become possible to measure some gonadal hormones in saliva (Landman, Sanford, Howland, Dawes, & Pritchard, 1976; Read, Riad-Fahmy, Walker, & Griffiths, 1982; Sannikka, Terho, Suominen, & Santti, 1983). Sampling saliva

is not particularly stressing, and saliva provides a good measure of the biologically free fraction of t. However, most other hormones cannot yet be determined with sufficient accuracy in saliva. Thus, care has to be taken when estimating an individual's hormotype, irrespective of whether direct or indirect methods are used.

3. Classification Young females typically have 3–5 times higher E_2 than males, and can be ordered linearly according to plasma E_2, a biologically very active member of the estrogen group. The E_2 distribution can, for example, be a priori divided in five arbitrarily chosen intervals, and the intervals are named E5 to E1. E5 and E1 in that case represent females with the 20% highest and lowest E_2 values, respectively, and E3 refers to the 20% females with close to average values.

Males can be hormotyped in the following way. They are linearly ordered according to individual t (a biologically very active member of the androgen group). The t distribution is a priori divided into five arbitrarily chosen intervals named A5 to A1, where A5 and A1 represent males with the 20% highest and lowest t values, respectively, and A3 represents the 20% individuals with close to average values. Young males typically have much higher plasma t concentration than young females. In addition to the problems mentioned with hormotyping females, the hormotyping of males has its own problems, however. For reasons given elsewhere (Nyborg, 1979, 1994b) the GTC–A/E model assumes that E_2 is the most important steroid in the brain for understanding hormone-ability relationships. Moreover, E_2 may feminize the brain in low concentrations and masculinize it in high concentration (Döhler & Hancke, 1978). So, the body may essentially be feminized by E_2 and masculinized by t or their metabolites, but for the brain E_2 might be the more important hormone. It complicates matters that females aromatize some of their brain and peripheral t to E_2, but males do so to a larger extent. Obviously, an evaluation of the effects of a particular hormone on the brain is made difficult when the hormone measured in plasma, say t, might masculinize the body and at the same time actually act as a prohormone for E_2 with effects on the brain that may differ according to dose. There is now a number of animal study groups looking into these problems. They use, among other techniques, radioactive labeling of gonadal hormones and follow the pattern of uptake and the biological and behavioral effects with rather sophisticated techniques. This chapter is not the place to review the evidence, and the reader is referred to a brief discussion of how the GTC–A/E model handles limited aspects of this complex problem in Nyborg (1983). To illustrate general principles of male adult hormotyping it suffices here to mention that as boys pass through the various pubertal phases, they begin to differ markedly with respect to plasma t concentration. A complete hormotyping also includes evidence for familial dispositions. A probabilistic estimate of what genes can be expected to be modulated by hormones is called for, as is

exact information about possible permanent prenatal hormone effects on body and brain development. There are many possibilities. Atypical fetal hormone secretion, variations in the mother's hormone status due, for example, to stress or medication, or even the hormone secretion of a cotwin (in particular if the cotwin is male) may affect the fetus. Major socioeconomic and life-history events should also be recorded in connection with hormotyping. There are not many studies of the heritability of hormone status, but those at hand suggest that the hormotype is under considerable genetic control. Meikle, Stringham, Bishop, and West (1986) thus found that about 40% of the variability in t in males can be explained by a familial disposition. In an earlier study of monozygotic and dizygotic twins, Meikle, Bishop, Stringham, and West (1986) found that genes regulate between 25% to 76% of the total variation of plasma t, E_2, estrone, 3α-diol G, free t, luteinizing hormone (LH), follicle-stimulating hormone (FSH), but only 12% in dihydrotestosterone (DHT) and <1% in sex-hormone-binding-globulin (SHBG). Moreover, the male hormotype seems to remain fairly stable over time, as repeated measures of t show short-term stability (Couwenbergs, Knussman, & Christiansen, 1986; Knussman & Christiansen, 1986) and remains fairly constant over a year (Smals, Kloppenborg, & Benraad, 1976). However, there are systematic circadian and circannual changes in plasma t content (Nieschlag, 1974; Reinberg, & Lagoguey, 1978; Reinberg, Lagoguey, Chauffournier, & Cesselin, 1975), which should be taken into account when sampling. The female hormotype also seems to remain reasonably stable over the reproductive period, as repeated measures of plasma E_2 tend to give similar readings from one E_2 high phase to another and from one E_2 low phase to another over consecutive menstrual cycles.

Obviously, the proposed hormotyping represents the simplest possible solution to an immensely complex problem. It is assumed, for example, that female plasma E_2 content does not correlate with plasma t content, and that male plasma t does not correlate with E_2 content. However, preliminary analyses suggest that E_2 and t do correlate significantly in 8 and 10 yr old girls (r = .57 and .69, respectively) but not in boys of similar age, and that the correlation remains positive but not significant thereof (Nyborg, 1994c). No doubt further research will lead to more sophisticated ways to classify individuals hormonally, but in complex matters it sometimes pays off to first see if simple solutions work. Genetic, genital and bodily sex, and sexual identity typically (but certainly not always) coincide with a predominant t or E_2 exposure. Effects of extreme hormone exposure are discussed at length by Hoyenga and Hoyenga (1979); Nyborg (1984, 1990a); and Nyborg and Nielsen (1977, 1981a, 1981b).

4. Trait Covariance As said before, the primary goal of the GTC–A/E model is to account for harmonization of traits during development. Gonadal hormones are ideal for this purpose, because they go everywhere in the body.

Unlike many other substances in plasma, gonadal hormones easily transcend the blood–brain barrier, so they can affect the body and the brain at one and the same time. On the other side, gonadal hormones become biologically active only in target tissues that are able to induce hormone-specific receptors. This arrangement puts gonadal hormones in a unique position. Despite their simultaneous availability in all body and brain tissues, they nevertheless can exert precise and highly circumscribed effects only in designated target areas. The task of the GTC–A/E model is to formalize the developmental and functional implications of this arrangement. By simultaneously and selectively influencing body and brain target tissues, gonadal hormones become capable of precise coordination of widespread but circumscribed aspects of body and brain development and functioning. Variations in gonadal hormones may force single bodily or behavioral traits to appear, disappear, or reappear in the phenotype, or change intensity, frequency, or direction of the expression of traits. More importantly, they concomitantly orchestrate the various aspects of sex-related body, brain, and behavioral development, including specific abilities, and may thus profoundly modify ontogenetic timetables for development. The GTC–A/E model is devised to explain why various hormotypes develop different combinations of body, brain, and behavioral traits, why pubertal tempo differs among hormotypes, why different hormotypes exhibit different degrees of adult sexual differentiation of the body and the brain, and thus why specific abilities and personality come to differ among hormotypes in adulthood.

E. Model Predictions

Figure 1 is a graphical representation of the GTC–A/E model. The present version is restricted to explain only individual development of specific abilities and a few other characteristics. It will be remembered that hormotypes A3 and E3 represent individuals with close to average male t values or average female E_2 values, respectively, and average sexual differentiation. A4 and E4 designate a male with somewhat higher than average t, or a female with somewhat higher than average E_2 concentrations, both showing a higher degree of sexual differentiation. A2 and E2 mirror a male or a female with somewhat lower than average t or E_2, respectively. Hormotype A2 and E2 approach so-called androgynous males and females, that is, males who in addition to ordinary masculine traits also show some clearly feminine traits, or females who in addition to the usual feminine traits also show some clearly masculine attributes. There are problems with proper definition of the dimensions of masculinity, femininity, and of androgyny (e.g., Lenney, 1979; Spence & Helmreich, 1979), but this is not the place to take up that discussion.

The GTC–A/E model generates person-specific testable predictions about the most likely trait combination as soon as an individual is classified according to hormotype. An individual with a moderate plasma concentration for his

or her sex like a male A2 or a female E2 is expected to be tall and show either a fatty (A2) or a leptosome (E2) body build. They are expected to be late maturers, to have a late sexual debut, and to display an androgynous sexual identity. There might be a slightly increased tendency for bi- or homosexuality in A2 males (with low prenatal t concentration, or during the first few years after birth; Swaab & Hofman, 1988), and for E2 females with high prenatal t (Ellis & Ames, 1987; Ellis, Peckham, Ames, & Burke, 1988; Feder, 1984; Money, Schwartz, & Lewis, 1984; Swaab, Hofman, & Fisser, 1988; Whitam & Mathy, 1991). This, however, is probably far from the rule, and needs further investigation (Gooren, 1988). A2s and E2s are not very sociable. They tend to be loners and prefer books and work over people. They marry late, and typically get one or few well-in-advance planned-for children. They live longer than average for their respective sex. In addition to these similarities, there are also predictable differences among A2s and E2s. A2 males are expected to have been less physically aggressive during upbringing than most boys, whereas E2 females are expected to have behaved physically more aggressive than usual for girls. A2s and E2s may, nevertheless, be rather high in verbal aggressiveness and score high on persistence. With respect to specific abilities, both A2s and E2s are expected to score higher than average, after correction for family dispositions, of course. They typically get high performance scores, but E2s are expected to get a slightly lower than average verbal score than A2s, for reasons to be discussed.

A4s and E4s with higher than average homotypic plasma gonadal hormone concentrations for their sex are expected to show a developmental pattern that in many respects represents the diametrical opposite of that of the A2s and E2s. A4s and E4s tend to be short, stocky, or athletic early-maturing individuals. A4 males show extensive bodily masculinization and E4 females display a pronounced feminization of the body. Sexual identity is unanimously and stereotypically in line with their respective sex. A4s and E4s are expected to prefer extensive social activities at the expense of book reading, to marry early, and to have many children. They may show shorter life expectancy than A2s and E2s, perhaps due to bad health consequences of high plasma hormone content (Ellis & Nyborg, 1992). A4 males are expected to display an increased level of physical aggression, relative to A3s, but female E4s most likely display more submissive behavior than E3 females (Baucom, Besch, & Callahan, 1985). With respect to abilities, A4s and E4s are expected to show overall below-average *g*, and significantly lower than that of the A2s and E2s. Although A4s and E4s may excel in some of the more automated aspects of verbal skills, such as verbal fluency, and in motor skills, relative to E3s and A3s, they are expected to display a severe depression of information and nonmotor performance subscale score. This, obviously, detracts from overall *g*, which is defined as the sum of verbal and performance scores.

Socioeconomic background is often claimed to determine achievement and vocational status, and to explain why low *g* individuals tend to cluster in

low-status occupations. An alternative explanation is that *g* codetermines achievement in certain occupations (Harrell & Harrell, 1945; Jensen, 1980; McCall, 1977; Terman, 1925; Waller, 1971), obviously, together with other factors. To the extent that specific abilities are actually called upon in certain high-status jobs, the GTC–A/E model predicts that the lonely, relatively infertile (Vining, 1984) androgynous A2 and E2 individuals are more likely to be found near the top of the educational, occupational, and political pyramid (Editorial, 1976; Hingley & Cooper, 1983). The model further predicts that the more socially inclined A4 and E4 individuals will excel in areas requiring physical power, aggression, or dominance rather than high *g* (Dabbs & Morris, 1990; Dabbs, de La Rue, & Williams, 1990; and section A.5).

F. Mechanisms

The literature on mechanisms through which gonadal hormones exert their many and varied effects on body and brain target tissues expands rapidly. Excellent overviews are provided by, among others, Döhler et al., 1984; Goy et al., 1980; McEwen, 1987b; Nottebohm, 1981, 1989; Nottebohm, Nottebohm, and Crane, 1986; Toran-Allerand, 1984; and other chapters in the book edited by Vries, Bruin, Uylings, and Corner (1984). The literature suggests that gonadal hormones have profound effects on cell proliferation, migration, and differentiation, as well as on the functionality of mature cell assemblies. The general causal pathway seems to be as follows. After secretion, gonadal hormones are carried around in the bloodstream. In this phase most hormones are biologically inactive by being associated to binding proteins, SHBG, with such high affinity that most steroids appear in plasma in a bound form, leaving only a few percent active. Figure 2 (from Norman & Litwack, 1987) illustrates the mechanisms of action of hormones on the genome.

The figure shows that unbound hormone molecules enter the target cells and are bound there to unoccupied receptors (2) to form an inactivated complex (3). It is not clear whether this happens in the cytoplasm or in the nucleus, so two pathways are indicated in the figure. The steroid-receptor complex is accordingly either activated while still in the cytoplasm (4) and translocated to the nucleus (5), or activated while in the nucleus (4). In either case the steroid-receptor complex exhibits an increased affinity for DNA (6), and binds to presumptive high-affinity binding sites upstream from the genes (6), with the result that the gene is regulated by the hormone. As the transcription rate of the gene increases, the thus accomplished mRNA is translocated (7) to the cytoplasm for translation. The final result of steroid action on the gene is, in other words, that the protein production of particular genes is modulated. The enhanced level of the new proteins can then be utilized for multiple purposes, such as to build up specific body and brain tissues during development, to induce neurotransmitters or influence their working condi-

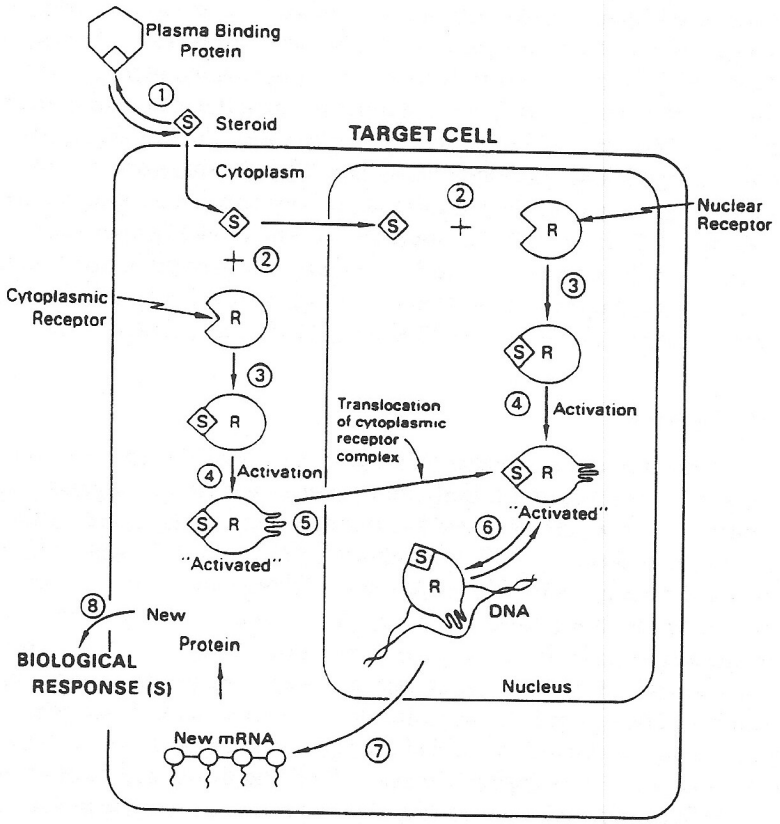


FIGURE 2

Diagram of how plasma sex hormones regulate the expression of genes. Most of the sex hormone (S) is chemically bound by protein in plasma (1), but the free fraction can enter the target cell (2) and is bound to a receptor (R), that is either in the cytoplasm or in the nucleus (3). After activation (4) the *cytoplasmic* hormone-receptor complex enters the nucleus (5) and then binds (6) to a part of the gene with high affinity. Alternatively, the *nuclear* hormone-receptor complex is activated (4) and then binds directly (6) to the gene. There is some discussion as to where the intracellular hormone receptor is situated, and accordingly which way the receptor-hormone complex goes, but in either case the result is gene transcription, new messenger RNA, and a new protein that can be utilized in body and brain tissues for structural or functional ends. (Reproduced with permission from Norman & Litwack, 1987, p. 23.)

tions, or to alter the metabolism of various remote tissues and organs with further multiple effects for other organic systems. Understanding of the details of these intricate processes is still incomplete. Do receptors for gonadal hormones reside in the cytoplasm or in the cell nucleus in the absence of hormones (Toran-Allerand, 1984)?

Another question is whether gonadal hormone work exclusively through

the regulation of gene products, or affect each other and neurotransmitters? Gonadal hormones sometimes have an effect with such a short time delay that they could not possibly work exclusively through the rather time-consuming actions on the genome (McEwen, 1988a, 1991b). It appears that some steroids and their metabolites are capable of modulating the release of neuroactive substances, chloride flux, and the distribution of oxytocin receptors in the hypothalamus via membrane receptors within a time span of minutes (McEwen, 1991a, 1991b). Moss and Dudley (1984) have illustrated how E_2 may affect nerve cells in a manner similar to that proposed for certain neurotransmitters by a membrane-mediated effect (see Figure 3).

Estrogen molecules can interact with receptors on the cell membrane to stimulate the intracellular production of cyclic adenosine monophosphate (cAMP), which then binds to an intracellular receptor and affects other metabolic processes in the nerve cell. According to this model, E_2 needs not actually enter the cell during this process, and bypasses the time-consuming translocation to the nucleus, the subsequent transcription of gene proteins, and the alteration in protein synthesis, but results in a change in cell metab-

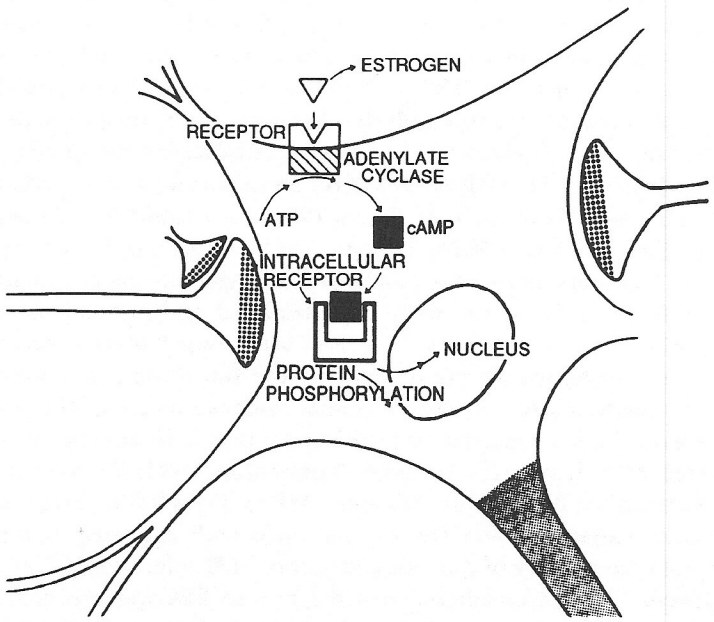


FIGURE 3

Diagrammatic representation of a membrane-mediated mechanism of estrogenic action, which is similar to that proposed for neurotransmitter action. ATP, adenosine triphosphate; cAMP, cyclic adenosine monophosphate. (From Moss and Dudley, 1984.)

olism. These details of mechanisms are interesting by themselves, but do not affect the general argument made here. It is sufficient for an understanding of the GTC–A/E model to know that gonadal hormones profoundly affect the anatomy and functionality of numerous bodily and brain tissues in various ways. They can permanently alter the neuronal architecture and function. They can change the organization or secretional pattern of glands remotely situated from where the chain of molecular events began. The reactions may take place within seconds, hours, days, or months. The fact that gonadal hormones initiate cascades of local effects as well as guide more remote molecular interactions, explains how even minor perturbations in gonadal hormones are capable of profoundly influencing the orchestration of rather different body, brain, and behavioral functions, depending, among other things, on the maturational status of the organism. Moreover, these cascades of causal effects often unfold in nonlinear ways.

Another important point in steroid chemistry is that gonadal hormones often show almost opposite effects in low and high doses. E_2 exerts strong neurotropic effects even in tiny doses (Toran-Allerand, 1984), but it may have neurotoxic effects in high or very high concentrations. The possibility of circumscribed or diffuse neurotoxic reactions to long-term medication with steroids (Nyborg & Nielsen, 1981b), or to constantly high physiological concentrations (Ellis & Nyborg, 1992) should not be underestimated. The concept of natural cell death has become increasingly popular in recent years (Bowen, 1981; Hinchliffe, 1981). It is based on the observation that the organism produces many more cells during development than is needed, so an important question is which mechanisms accomplish the systematic eradication of surplus cells. Steroids seem to play an important role in programmed cell death (Gould, Woolley, & McEwen, 1991; Greenwald & Martinez-Arias, 1984; McEwen, 1988a, 1991a; Truman, 1985; Truman & Schwartz, 1984).

Until a few years ago it was customary to divide effects of gonadal hormones into two groups: the early organizational and permanent neuroarchitectural effects on immature body and brain target tissues, and the late activational effects, transiently influencing the functioning of mature body and brain tissues. However, recent animal evidence indicates that the adult brain of some bird species remains sufficiently plastic to undergo major anatomical seasonal changes (DeVoogd & Nottebohm, 1981; Nottebohm, 1981, 1989; Nottebohm, Nottebohm, & Crane, 1986). The adult rat brain responds to hormone variations over the estrous cycle with dendritic changes and alteration in the number of synaptic connections (Woolley, Gould, Frankfurt, & McEwen, 1990). Considering that the human EEG pattern changes dynamically over the menstruating cycle (Becker, Creutzfelt, Schwibbe, & Wuttke, 1980; Klaiber, Broverman, Vogel, & Kobayashi, 1974), as a function of the female brain being exposed to varying concentrations of gonadal hormones, and that the use of oral contraceptives has a stabilizing effect on the human EEG (Matsumoto, Sato, Ito, & Matsuoka, 1966), it may very well also

be that the adult human brain responds with microscopic neuroanatomical or molecular changes to natural or provoked hormone perturbations.

IV. NEUROPSYCHOLOGICAL APPLICATION OF THE GTC–A/E MODEL

A. Early Structural Effects

Gonadal hormones appear to condition differences in the development of the left and right cerebral hemispheres. Irrespective of sex, the right hemisphere grows larger than the left in several species, including the human (Kolb, Sutherland, Nonneman, & Whishaw, 1982), and it has been suggested that t is responsible (Diamond et al., 1981; Geschwind & Galaburda, 1987; Steward & Kolb, 1988). However, the male right hemisphere becomes slightly thicker than the female (Diamond, 1988). The functional significance of this anatomical asymmetry for specific abilities is not transparent, and the sex-dimorphic nature of the studies may explain mixed results in the area. We already know of early significant asymmetries in the number of hormone receptors in the two brain halves (Sandhu, Cook, & Diamond, 1986) and also subcortically, and that receptors come and go with age. Moreover, there are complex region, sex, and developmental asymmetries in those brain-aromatization processes by which t is metabolized to E_2 (Ziegler & Lichtensteiger, 1992). We now need data from large-scale studies of hormonally different individuals rather than from sexually classified groups to learn about details of the origin, developmental significance, and proper meaning of the observations for specific abilities.

B. Developmental Dynamics

1. Prepubertal Development As stressed previously, it is essential to keep a developmental perspective on the evaluation of differences in specific abilities. It is generally acknowledged that most sex-related differences in abilities do not appear, or typically are of moderate size, before puberty (Maccoby & Jacklin, 1978). One exception is the early and persistent female lead in some verbal areas. With respect to spatial abilities, a few studies even report a slight prepubertal female lead. However, these observations may be explained by the generally more advanced prepubertal female body and brain development.

2. Pubertal Development Ability patterns change at puberty according to sex-dimorphic studies, but these studies often raise more questions than they answer. One unanswered question is whether boys continue to improve in spatial abilities during late puberty, whereas girls' performance reaches an early asymptote, or whether boys' spatial abilities reach an asymptote at

puberty and girls' spatial abilities actually decline. It was concluded in an earlier review (Nyborg, 1983) that many girls (and some boys) actually regress in spatial abilities, but ideology sometimes prevents a proper interpretation of this phenomenon (Witkin et al., 1967). The GTC–A/E model allows us to formulate specific and testable hypotheses to answer the above question. Recall that the model predicts low spatial abilities in females with high-E_2 plasma content, and high spatial abilities in females with low E_2 content. Let us for a moment use the model as a tool for examining developmental dynamics, and exploit its curvilinear dose–response characteristics. First we map female biological age along the X-axis in Figure 1, in parallel with the one for individual age-related variation in plasma E_2 concentration. Girls enter puberty in accordance with a gradual increase of plasma E_2, but there are large individual differences in the size of the E_2 surge. These differences can be used for prediction. The moderate increase in E_2 in hormotype E2 girls partly explains why she matures late and why she might show signs of virilization. She does not "overshoot" the optimum brain E_2 range by much, so the prediction is that her family disposition for spatial ability will be only slightly depressed, if at all. A girl with more ample age-related increase in plasma E_2, such as hormotype E3, matures at the average female age, and displays average female sexual differentiation. She "overshoots" the optimum brain E_2 level more than an E2 girl, so the prediction is that she suffers more depression of familial disposition for spatial abilities than an E2 girl. A girl with a considerable surge in plasma E_2 at puberty, that is hormotype E4, is expected to mature at an early age and to show a high degree of secondary sexual feminization. She "overshoots" the optimum range by a wide margin. The prediction is that highly feminized E4 girls suffer the most severe depression of spatial abilities. The model further predicts that the depression in spatial abilities takes place at different chronological ages in the various hormotypes. E4s are expected to show the earliest depression and E2 the latest and least, with E3 in between. For girls in general the regression seems to take place some time between ages 12 and 14 (Nyborg, 1983, 1988a, 1990a; Nyborg & Nielsen, 1977), but we need studies aimed specifically at testing these predictions.

The GTC–A/E model generates, in other words, testable predictions about who regresses in spatial abilities, when, why, and how much. The basis for this is quite simple. Examine covariant hormonal, bodily, brain, and ability development in terms of the effects of hormones, and keep the analysis on a person-specific basis. It then becomes obvious that the highly feminized E4 girl runs a particular risk of early and severe depression of nonverbal abilities, that the moderately feminized E3 girl suffers less and later depression, and that the androgynous E2 girl shows little or no depression of spatial abilities. The traditional sex-dichotomous approach averages the scores of these three groups, and thereby blurs the picture. Obviously, nothing in the GTC–A/E model speaks against an even finer differentiation of individuals into more

than five hormotypes. Such fine-grained analyses are best made in forthcoming specific and uncompromising testing of the above-mentioned predictions, preferably in the form of rigorous longitudinal or cohort-sequential designs (e.g., Nyborg, 1994c).

Low plasma t prepubertal boys grossly "undershoot" the optimum brain E_2 level because there is little brain t to aromatize to E_2. As they approach puberty, some boys (hormotype A2s) exhibit only a moderate increase in t, delayed body and brain maturation, and an androgynous body type and sexual identity. A2 boys "undershoot" the brain E_2 optimum, and the low t gives more allowance for E_2 to compete for receptors sensitive to both hormones. The result is almost full expression of spatial abilities in A2s. Boys with an average surge in plasma t at puberty (A3s) "overshoot" the brain E_2 optimum due to aromatization, but not nearly as much as the E3s. They suffer slight depression of spatial abilities. A3s display average masculinization of the body and sexual identity. A4 boys with a considerable plasma t surge at puberty "overshoot" the brain E_2 optimum by a wide margin, suffer a significant depression of spatial ability, become highly masculinized with respect to body characteristics, and display a stereotypic male sexual identity.

These examples suggest that classification of individuals according to hormotype gives room for meaningful differentiation among individuals, prenatally, at puberty, and later in life, whereas the sex-dichotomous approach confounds this important within-sex differentiation. The hormotypic approach suggests that when males and females are low in androgens and estrogens, respectively, they also differ least in somatic sexual differentiation and in their specific ability pattern. Conversely, when males and females are high in plasma gonadal hormones they also differ most in sexual differentiation of the body and abilities.

C. Menstrual Dynamics

The model predicts inverse changes in specific abilities as a function of cyclic changes in E_2. Remember that E3 and, in particular, E4 females are assumed to "overshoot" the optimum brain E_2 by a wide margin with resulting pubertal depression of the spatial abilities and enhancement of the verbal abilities (see Figure 4, from Nyborg, 1979; modified, 1992).

We can exploit the fact that the GTC–A/E model sees the expression of specific abilities in adulthood as a dynamic rather than as a static phenomenon as in sex-dimorphic models. The model predicts that if plasma and brain E_2 concentration changes in an adult female, the balance between the spatial and verbal abilities will be affected in a quite specific way. They must change in perfectly opposite directions, or the model would be in error.

Dynamic effects of menstrual variation in plasma E_2 on spatial abilities have, in fact, been observed in several independent studies (Anderson, 1972; Dor-Shav, 1976; Hughes, 1983; Klaiber, Broverman, Vogel & Kobayashi,

1974; Komnenich, Land, Dickey, & Stone, 1978; Silverman & Phillips, 1991). Woodfield (1984) administered the Embedded Figures Test to pregnant women and again after childbirth. Spatial ability was low in the last high-E_2 weeks before birth and increased in the weeks after, when E_2 returned to normal values. Progesterone, which varies slightly out of phase with E_2, is probably not responsible for the hormone effects on abilities (Hampson, 1990). Gordon, Corbin, and Lee (1986) intravenously administered LH-releasing hormone (LHRH) and LH, two feminizing hormones, to males and found that scores for verbal production went up, whereas mental rotation spatial scores decreased.

Some studies are unable, however, to find a consistent effect of cyclic gonadal hormones on specific abilities (Gordon et al., 1986; Ho, Gilger, & Brink, 1986; Woodfield, Whitehead, & Asso, 1987). Moreover, positive studies do not always find a hormone effect in equivalent spatial tests. Small sample size, inaccurate timing of hormone sampling, measurement error, and large within-sex variability probably all contribute to the partly inconsistent picture. Moreover, the dynamic effects of sex hormones on specific abilities may not be of dramatic proportions, even though Hampson (1988) found that 18% of the individual variability in the Paper Folding Test could be explained by absolute differences in plasma E_2. Obviously, further research is needed to reveal the precise nature of cyclic hormone effects on spatial abilities.

The apparent noncyclic nature of male hormones may camouflage that

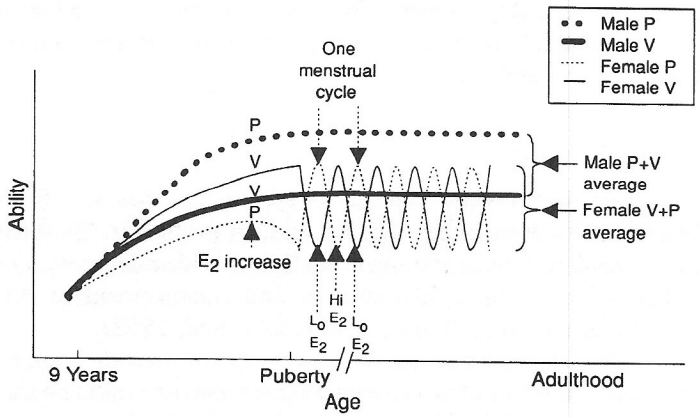


FIGURE 4

Sex differences in adolescent unfolding of verbal (V) and performance (P) abilities. At puberty male V and P abilities reach an asymptote, whereas female P is inhibited. During the female reproductive period, V (and fine-motor skills) will cycle in opposite phase with P (and with visual-spatial skills), as a function of effects of cyclic menstrual changes in the sex hormone estradiol (E_2) on the brain. Lo E_2 = Perimenstrually low plasma estradiol (E_2) concentration. Hi E_2 = Midcycle high plasma estradiol (E_2) concentration. (Nyborg, 1979, 1992.)

gonadal hormone perturbation also affects specific abilities in males. Kimura (1991) thus found that male spatial ability is higher in the spring, when t tends to be low, than in the autumn when t is higher.

D. Adult Male Abilities

I plotted best-fitting polynomial curves for a number of intelligence test scores as a function of plasma t in 3654 middle-aged white American males (raw data from Centers for Disease Control, 1988, 1989) (Nyborg, 1994a). Males with higher than average t (i.e., ≥ 678 ng/dL) got lower general technical scores the higher the t on the Army Classification Battery (ACB) and lower WAIS Information subtest, and WAIS Block Design scores. Males with below average t got higher scores on the same tests the lower their t. Scores on the General Technical Aptitude Test and number of years of formal schooling also followed this pattern. Log t level was significantly inversely related to age in this large sample (Ellis & Nyborg, 1992). The data further suggested that the major races differ with respect to the influence of variation in t concentration on abilities, but are equally sensitive to the effect of high t on depression (Nyborg, 1992). Also, Shute, Pellegrino, Hubert, and Reynolds (1983) found that spatial abilities were best in low t males and high t females, but there might have been problems with the antibody used for t determination (see Gouchie & Kimura, 1990). Gouchie and Kimura also noted better spatial and mathematical abilities in low t males and in high t females than in the reverse, but they were unable to see an effect of t on tests that usually favor females or show no sex difference.

Christiansen and Knussman (1987) found that t correlates positively with measures for spatial abilities, and negatively with verbal abilities, and Tan and Akgün (1992) noted a positive correlation between t and scores on Cattell spatial reasoning in a small sample of young right-hand, right-eye-dominant males, but mixed-dominant males and young females showed no relationship. These observations suggest that it is important to determine exactly where on the curvilinear position of the GTC–A/E model the subjects under examination are located.

Undoubtedly, detailed information about genetic dispositions for abilities and laterality, agreement about which test to use for comparison, uniform assaying techniques, and a protocol for when to sample blood, urine, or saliva, would greatly help to clarify details of the somewhat mixed pattern of the t–ability relationship. Guidelines for exact values of where to cut gonadal hormone distributions into meaningful hormotypic intervals would also be useful. Based on the axiom that all sexually differentiated traits show proximate hormonal involvement, and more so the more differentiated they are, it further becomes of interest to determine which modal "male" and "female" traits will cycle as a function of changes in t and E_2 in addition to spatial, verbal, fine-motor and gross-motor skills. In general, traits that require only

pubertal hormone activation can be expected to cycle. Traits that require both prenatal hormone priming and later pubertal activation can also be expected to cycle. Traits for which prenatal hormone exposure is sufficient to appear may not cycle in adulthood. Our present knowledge of which traits need which kind of hormonal action to be fully expressed is insufficient.

E. Educational and Occupational Aspects

As mentioned previously, specific abilities hold moderate power for predicting achievement in educational and occupational areas. Does hormotyping fare any better in this respect? The question is really whether the hormotype, with its associated covariant body, brain, ability, and personality development, constitutes a more reliable platform for predictions than do abilities or genetic sex? Indirect support for this notion would be if hormotypes appear with different frequencies in particular educational and occupational areas. In fact they do. Purifoy and Koopmans (1979) found that professional women have more plasma t than housewives. In the language of the GTC–A/E model this means that a late-maturing, androgynous, high-ability E2 female enjoys an advantage in occupational achievement, whereas the early-maturing feminized social E4 female cares more for children than for a professional career. The model would by implication, at least partially, explain the fact that *g* differs among occupations (see Section A.5). Schindler (1979) observed higher t in female attorneys than in athletes, nurses, or teachers. The observation of low t in female athletes should come as no surprise because hard physical work depresses t. Dabbs et al. (1991) classified several smaller and one large population of 4462 middle-aged males (same database as used by Nyborg, (1992, 1994a; and by Ellis & Nyborg, 1992) in accordance with occupational criteria, and then looked for differences in t. Ministers had the lowest, and blue-collar workers, football players, and actors the highest plasma t concentrations. It even seems that specialization within a given occupational area is related to differences in plasma t, and that there is an inverse relationship between t and socioeconomic status. Dalton (1976, 1979) found that girls exposed in utero to progestin (often with androgenlike effects) had more academic success than had untreated control sisters. Girls exposed to more than normal prenatal t tend to do better in math and science than their nonexposed sisters (for an early overview, see Hoyenga & Hoyenga, 1979).

We need more research to fully explore connections between hormotype, abilities, and educational and occupational achievement. The advantage of drawing upon models like the GTC–A/E model for this task is essentially that the hormotype and associated trait combinations offer a precise platform for testing predictions of achievement in particular educational or occupational areas. Ability scores and genetic sex by themselves are not enough.

F. Homosexuality

The precise cause(s) for homosexuality is(are) not known, so explanations range from exclusive genetic determination, over abnormal hormone conditions in the prenatal (but not in the adult) period (e.g., Ellis & Ames, 1987), to exclusively social determination. Homosexuality does not ordinarily figure in neuropsychological literature, and certainly not in connection with a discussion of specific abilities. There might be good reasons for making the connection, however, because of its potential for revealing details of connections between prenatal hormone exposure, brain development, and the unfolding of specific abilities. McCormick and Witelson (1991) thus inquired into the neurobiological etiology of homosexuality and specific abilities. They reasoned that if male homosexuality is due to lower than normal t exposure during the prenatal period, and further if handedness and specific abilities also reflect differences in prenatal t exposure, then there might be differences in ability patterns among left- and right-handed male homosexuals, as compared to matched male and female heterosexual controls. They found that the mean performance of homosexual males on three spatial tests and one verbal fluency test fell between those of the heterosexual males and females, but was not significantly different from that of the control females. In this they corroborated the findings of others. They further noted significantly more not-consistently right-hand users in the male homosexual group who, like other such handed individuals, also showed a tendency to obtain lower spatial relative to verbal fluency scores. McCormick and Witelson explain these observations in terms of an effect of low male prenatal t on brain organization, abilities, and handedness. Female fetuses, exposed to abnormally high levels of t (e.g., girls with the adrenogenital syndrome or through medication) later display an increased probability of left-handedness (e.g., Nass et al., 1987), a "male" pattern of high spatial ability (e.g., Resnick, Berenbaum, Gottesman, & Bouchard, 1986), and an increased probability of a bisexual or homosexual inclination (Ehrhardt et al., 1985; Money, 1987). Females with abnormally increased prenatal t exposure would probably qualify as hormotype E2 or E1, and males with abnormally low prenatal exposure would count as hormotype A2 or A1. These hormonal variations over a broad physiological range suggest interesting ways to exploit predictions of the GTC–A/E model with respect to hormonally mediated neuronal links between sexual orientation, laterality, and specific abilities.

G. Evolution

Section B.1 briefly outlines two good evolutionary reasons for selection of gonadal hormones during evolution: the advent of the sexual reproductive mode, and the adaptive ontogenetic timing of sexual maturation as a means to conserve energy by not producing much offspring during hard times. There

is a third good reason for selection of hormones in connection with selection for specific abilities. The general form of this argument is, briefly, that males and females became selected for different abilities early in evolution in accordance with a practical division of labor. Males were selected for good spatial abilities on wide-range hunting expeditions, whereas females became selected for traits like sociability, empathy, and verbal abilities. The trait combinations of a dominant, muscular, quiet male hunter with excellent spatial and gross-motor skills, and a subordinate, nutritive, sociable, and talkative child-breeding female was, according to this view, a highly competitive and well-matched arrangement that would be strongly selected for. Views differ, however, about at which level selective forces took effect. Some think they worked at the level of the single gene, others that they worked at the level of specific behavioral traits including specific abilities, or at the level of the individual, or at the level of the population, or at the level of society or culture. Sociobiologists speak of gene–culture coevolution. Not many researchers consider evolution a matter of pure physics, or assume that selective forces can have only an entirely physical target (Atkins, 1981; P. M. Churchland, 1984; P. Churchland, 1986; Jacob, 1982a, 1982b; Monod, 1975). Physicology is an attempt to apply the physical point of view in behavioral fields like psychology. Physicology is defined as the physicochemical study of very complex carbon-based so-called living systems (Nyborg, 1993a). Physicology maintains that it is highly unlikely that physical selective forces can have an effect on mental entities or that behavior as such can be a target for physical forces. Behavior is conceived of as an intermediate product of a series of complex endogenous physicochemical processes interacting constantly with exogenous physicochemical processes as long as so-called life processes take place. Aggression or spatial ability as such cannot, according to this view, be selected for during evolution, but the molecular constellations that give rise to such traits and to their combinations can. The point is, that selective forces in the environment, and this includes other people, are of a purely physical nature, and so are the developmental factors and processes responsible for the unfolding of specific brain-based abilities and the body. Physicology substitutes the psyche with movement of molecules. This brief discourse returns us to the suggested classification of individuals in accordance with their hormotype. Early organisms lacking steroids were doomed to depend on primitive predominantly nonsexual modes of reproduction, which are not particularly beneficial with respect to fast and efficient combinations of genes favorable for the evolution of complex brains and related abilities. Then, some kind of primordial steroid precursors appeared on the evolutionary scene, most likely as by-products of a series of accidental mutations. The path was then set for selection to eventually favor organisms capable of composing a primitive egg full of nutrition and later primitive egg-and-sperm arrangements. The capability to reproduce partly or fully sexually, gives a decisive competitive edge. In other words, the physicochemical basis for the evolution of complex brains and their associated

abilities is, according to physicology, repeated unplanned DNA mutations and later selection for steroids that regulate the expression of the DNA material. It is generally acknowledged that evolutionary processes stabilize at some optimum level, depending in part on the selective characteristics of the ecological niche. The physicological explanation of the origin and relative stabilization of today's prevalent trait combinations, represented by the various hormotypes, is that they reflect cost-benefit rudiments of ancient selection processes. Moderately low-hormone E2 and A2 individuals have superior abilities, but they get few children (Vining, 1982, 1984), and successful reproduction and survival of fertile offspring is the final evolutionary test for the continuation of a species. On the other side, low abilities in high-hormone E4 and A4 individuals is "compensated" for by an edge in high reproduction rate. Depending on the nature and changes of the ecological conditions in a particular evolutionary niche, one or the other hormotype will be selectively favored. From a neuropsychological perspective, this means that the particular central nervous organization and the associated ability pattern of the hormotypic variations of modern Homo sapiens sapiens reflects selective pressures of the past. One of the implications of this is troubling. The high-reproductive–low-ability trait constellation in A4 and E4 individuals may have served well and was favored in primitive times, when physical force and surplus children were necessary prerequisites for the survival of the species. Unfortunately, these very same characteristics seem to be dysgenic in a modern world, where reproductive restraints and good abilities are required. Under such ecological circumstances, A2 and E2 individuals will be favored, educationally, occupationally, and otherwise, and this leaves us with a serious problem.

H. The Nature–Nurture Question

We now know that gonadal hormones regulate genes throughout the life span. The regulation takes a dramatic form prenatally, when the female or male developmental program is switched on by hormones. This regulation raises, in fact, serious doubt about the adequacy of the traditional nature–nurture model, which is based on Fisher's analysis of variance model. Genetic and environmental components are assumed to each make an independent contribution, and together they add up linearly to a 100% phenotypic appearance. Less impact of one factor automatically means more of the other. Such models cannot accommodate for the fact that a change in the environment may induce a change in gonadal hormones, which may permanently activate or deactivate parts of the genome (McEwen, 1988c, 1988d; Nyborg, 1989), but this happens time and again. It happens prenatally if the hormonal milieu of the fetus is affected by medication of the mother. It also happens when the mother becomes severely stressed, or as a function of maternal or placental hormone perturbations, or even via the hormonal contribution of a

cotwin. Environmentally mediated changes of the expression of the genome take place after birth when stimulation of the peripheral sensory system and the brain affects the endocrine and other systems. In each of these cases environmental factors can and do accomplish a permanent change in the genetic contribution to ontogenetic development. Finally, besides linear and additive effects, hormones probably also exert nonlinear multiplicative effects. The traditional nature–nurture model is not at all geared for all these possibilities, and this raises an urgent need for revision for hormonal (Nyborg, 1989, 1990b) as well as for other reasons (Wahlsten, 1990).

I. Race Differences in Abilities

There is in the sex-dichotomous literature fair agreement about the size of race differences in abilities, but little agreement about the reasons for the differences. Some argue that tradition, cultural factors, discrimination, victimization, or lack of learning opportunities explain the low spatial abilities observed in many Blacks, the relatively high spatial abilities seen in many Asians, and the intermediate spatial abilities typically observed in Whites. Others argue in favor of a genetic or a gene–environment interaction explanation. The physicological approach differs from these explanations on two accounts. First, it requires a precise operationalization of relevant causal factors and mechanisms. Second, it requires an examination of individuals and their hormotype rather than remaining satisfied with averages. With respect to causal factors there is evidence that young Black males have significantly more t than White males (Ross et al., 1986), and with respect to individual differences it is equally clear that there are large within-race differences in t (Ellis & Nyborg, 1992). Less is known about Asians, but they are believed to have lower hormone levels than others (Purifoy, 1981). In terms of hormotyping we can make a very preliminary classification: Many Black males will qualify as A4s. Many White males will be A3s. Many Asian males will be A2s (Nyborg, 1987b). Given these assumptions, the GTC–A/E model predicts that many Black males will "overshoot" the optimum brain E_2 range, due to aromatization of surplus t, with a resulting marked depression of spatial abilities. Many White males will display a slight depression of spatial abilities, but most Asian males will show little or no pubertal depression. Blacks are expected to show the highest degree of sexual differentiation, Asians the least, with Whites in between. Race differences in verbal fluency and motor skills can be expected to show the exact opposite order, that is, higher verbal than spatial ability in Black males, slightly lower verbal than spatial ability in White males, and significantly lower verbal than spatial ability in Asians. There is some evidence in favor of some of these predictions, but the picture is far from clear. There are several reasons for this. The custom of averaging abilities for a whole race causes problems similar to those of averaging according to sex. Moreover, besides effects of actual person-specific environmental conditions

(e.g., severe protein deficiency) on hormone status, patterns of inheritance also ought to be taken into account. Geographically determined effects of living for extended periods of time in particular ecological niches most likely play an important role for the gene pool (Nyborg, 1987b, 1994b). Points worth keeping in mind when considering these predictions are that within-race differences are larger than between-race differences, and also that the within-race differences are partially explained in terms of hormotype. Instead of continuing to look for significant average race differences we should perhaps rather compare the frequencies of certain hormotypes within and between races and use formal models to explain what we see in both instances.

V. GENERAL DISCUSSION

The main thesis of this chapter is that the traditional sex-dichotomous study of sex differences in specific abilities is beset with serious problems, and that it is time to introduce new paradigms. The one suggested here provides methodological and theoretical alternatives, and the question then becomes whether the new approach solves all the traditional problems. To answer this question I will briefly review each of the old problems in the light of the proposed solutions.

Does hormotyping actually solve the basic problem with the sex-dichotomous approach? The answer is essentially yes. Hormotype classification is made on the basis of an identified proximate cause, the value of which is distributed along a continuous dimension. The fact that the model for cause–effect relationships is curvilinear does not affect the general argument. We can classify any population into as narrow intervals on the hormone scales as deemed necessary. Neither is the positive answer affected by the fact that minor changes in gonadal hormones can lead to truly tremendous nonlinear cascadelike effects along the causal pathways towards the guidance of development and function of remote tissues. We actually seem to have found a way around the sex-dichotomous Procrustean bedstead. Hormotyping may partly solve another long-standing controversy in psychology: the clinical single case versus population average approach, or the nomothetic versus the idiographic approach (Allport, 1965; Cronbach, 1975; Eysenck, 1976). Male and female averages typically characterize only a few individuals within each of the two groups, and the large overlap in sex distributions makes it difficult to interpret the mean difference. Implicit in the use of sex averages is further the presumption that most males have in principle similar patterns of abilities, and so have females. However, high-and low-ability scores tend to outbalance each other within sex, large variability reduces the probability of finding statistically significant differences, and averages lead to loss of valuable information about individuals. The solution to this problem is to ban the almost exclusive use of the individual-differences approach, and to promote the use of the

different-individuals approach, if we want to look for causes. Sex hormones give rise to continua in effects, and the observation of enough similar individuals may signal the possibility of a common biological basis. To be sure, the individual-differences and the different-individuals approach need not be mutually exclusive at the descriptive level. An average may signal that something general may be lurking behind, but the causal status of that something can be explored only in the study of individuals. The only exception to this rule would be the unlikely case that everybody is identical. Hormotyping provides a more individualized basis for predictions about educational and occupational achievement than sex- or average-specific abilities, because gonadal hormones are predisposed for individual harmonization of bodily, brain, and behavioral development. Several studies, even though not using hormotyping, have already demonstrated the value of taking hormones into account as predictors of education and occupation. This line of research can be expected to develop at a fast rate in the near future. Puberty is the time when sex-related differences in hormones and abilities really show up. However, research indicates that the prenatal period is very important for understanding these differences, and that also other phases of life characterized by hormone perturbations are relevant. The critical attitude towards research on race differences is to some extent based on the argument that within-group differences typically are larger than between-group differences and that causes for individual differences may not explain group differences. The hormotypic approach strives to explain the large within-group differences on an individual basis, while at the same time acknowledging that the frequency of certain genes and hormotypes may differ among racial groups. It is, in brief, argued that the focus on causal agents and on the individual saves hormotyping from much of the critique that deservedly is raised against the sex-dichotomous approach and against the exclusively genetic study of race differences.

It is only fair to state that the GTC–A/E model is nothing more than a preliminary heuristic tool for circumventing traditional problems in the neuropsychological study of individual differences in body, brain, and specific abilities. However, the model has its positive sides. It suggests links between ontogenetic development and evolution and facilitates the understanding of primordial sources (or ultimate causes) of contemporary sexual differentiation of specific abilities. The GTC–A/E model reveals shortcomings in Fisher's classical variance model for analyzing nature–nurture interactions during ontogenetic differentiation of specific abilities and suggests improvements. The model incorporates rapid advances in neuroendocrinology in the reformulation of flexible and person-specific models for nature–nurture interaction. The model can explain recent observations of dynamic effects of sex hormones on abilities, as well as covariant body, brain, and specific-ability development. The model is potentially falsifiable at each step of exploration and explanation. I believe that these characteristics will be minimal requirements for any new model striving to associate modern brain research with devel-

opmental neuropsychological research on individual and sex-related differences in specific abilities. The GTC–A/E model and the framework of physicology might provide the key to developing better paradigms in the field.

REFERENCES

Allen, L. S., Richey, M. F., Chai, Y. M., & Gorski, R. A. (1991). Sex differences in the corpus callosum of the living human being. *The Journal of Neuroscience, 11,* 933–942.

Allport, G. (1962). The general and the unique in psychological science. *Journal of Personality, 30,* 405–422.

Allport, G. (1964). *Pattern and growth in personality.* London: Holt, Rinehart and Winston.

Anderson, E. (1972). Cognitive performance and mood change as they relate to menstrual cycle and estrogen level. *Dissertation Abstracts, 33,* 1758-B.

Ankney, C. D. (1992). Sex differences in relative brain size: The mismeasure of women too? *Intelligence, 16,* 329–336.

Atkins, P. (1981). *The Creation.* San Francisco: W. H. Freeman & Company.

Baucom, D., Besch, P., & Callahan, S. (1985). Relation between testosterone concentration, sex role identity, and personality among females. *Journal of Personality and Social Psychology, 48,* 1218–1226.

Becker, D., Creutzfeldt, O., Schwibbe, M., & Wuttke, W. (1980). Electrophysiological and psychological changes induced by steroid hormones in men and women. *Acta Psychiatrica Belgica, 80*(5), 674–697.

Bem, S. (1983). Constructing a theory of the triple typology: Some (second) thoughts on nomothetic and idiographic approaches to personality. *Journal of Personality, 51,* 566–577.

Benbow, C. (1988). Sex differences in mathematical reasoning ability in intellectually talented preadolescents: Their nature, effects, and possible causes. *Behavioral and Brain Sciences, 11,* 169–232.

Berenbaum, S., & Resnick, S. (1982). Somatic androgyny and cognitive abilities. *Developmental Psychology, 18,* 418–423.

Berzins, J., Welling, M., & Wetter, R. (1978). A new measure of psychological androgyny based on the Personality Research Form. *Journal of Consulting and Clinical Psychology, 46,* 126–138.

Bøggild, C., & Nyborg, H. (1992). The development of spatial abilities: Activity, personality, and sexual identity. Unpublished manuscript.

Bouchard, T., & McGee, M. (1977). Sex differences in human spatial ability: Not an X-linked recessive gene effects. *Social Biology, 24,* 332–335.

Bourguignon, J. (1988). Linear growth as a function of age at onset of puberty and sex steroid dosage: Therapeutic implications. *Endocrine Reviews, 9,* 467–488.

Bowen, I. (1981). Techniques for demonstrating cell death. In I. Bowen & R. Lockshin (Eds.), *Cell death in biology and pathology.* London: Chapman and Hall.

Centers for Disease Control (1988). Health status of Vietnam veterans. I: Psychosocial characteristics. *Journal of the American Medical Association, 259,* 2701–2707.

Centers for Disease Control (1989). *Health status of Vietnam Veterans, Vol. I: Synopsis.* Atlanta: Centers for Disease Control.

Christiansen, K., & Knussmann, R. (1987). Sex hormones and cognitive functioning in men. *Neuropsychobiology, 18,* 27–36.

Churchland, P. M. (1984). *Matter and consciousness A contemporary introduction to the philosopy of mind.* Cambridge, MA: MIT Press.

Churchland, P. (1986). *Neurophilosophy: Towards a unified science of the mind-brain.* Cambridge, MA: MIT Press.

Corley, R., DeFries, J., Kuse, A., & Vandenberg, S. (1980). Familial resemblance for the identical blocks test of spatial ability: No evidence for X linkage. *Behavior Genetics, 10,* 211–216.

Couwenbergs, C., Knussmann, R., & Christiansen, K. (1986). Comparisons of the intra- and inter-individual variability in sex hormone levels of men. *Annals of Human Biology, 13,* 63–72.

Crockett, L., & Petersen, A. (1985). Pubertal status and psychosocial development: Findings from the early adolescence study. In R. Lerner & T. Foch (Eds.), *Biological–psychosocial interactions in early adolescence: A life span perspective.* Hillsdale, NJ: Erlbaum.

Cronbach, L. (1975). Beyond the two disciplines of scientific psychology. *American Psychologist, 30,* 116–127.

Dabbs, J., de La Rue, D., & Williams, P. (1991). Salivary testosterone and occupational choice: Actors, ministers, and other men. *Journal of Personality and Social Psychology, 59*(6), 1261–1265.

Dabbs, J., & Morris, R. (1990). Testosterone, social class, and antisocial behavior in a sample of 4,462 men. *Psychological Science, 1,* 209–211.

Dalton, K. (1976). Prenatal progesterone and educational attainments. *British Journal of Psychiatry, 129,* 438–442.

Dalton, K. (1979). Intelligence and prenatal progesterone: A reappraisal. *Journal of the Royal Society of Medicine, 72,* 97–399.

Danmarks Statistik (1985). *Kvinder & Maend* (Women & Men). København: Danmarks Statistik.

Denenberg, V., Berrebi, A., & Roslyn, H. (1988). Sex, brain, and learning differences in rats. *Behavioral and Brain Sciences, 11,* 188–189.

DeVoogd, T., & Nottebohm, F. (1981). Gonadal hormones induce dendritic growth in the adult avian brain. *Science, 214,* 202–204.

Diamond, M. (1988). *Enriching Heredity.* New York: Free Press.

Diamond, M., Dowling, G., & Johnson, R. E. (1981). Morphological cerebral cortical asymmetry in male and female rats. *Experimental Neurology, 71,* 261–268.

Döhler, K., & Hancke, J. (1978). Thoughts on the mechanism of sexual brain differentiation. In G. Dörner & M. Kawakami (Eds.), *Hormones and brain development.* Amsterdam: Elsevier/North-Holland Biomedical Press.

Döhler, K., Hancke, J., Srivastava, S., Hofmann, C., Shryne, J., & Gorski, P. (1984). Participation of estrogens in female sexual differentiation of the brain: Neuroanatomical, neuroendocrine and behavioral evidence. In G. Vries, J. Bruin, H. Uylings, & M. Corner (Eds.), *Sex differences in the brain: Progress in the brain research* (pp. 99–117). Amsterdam: Elsevier Biomedical Press.

Dor-Shav, N. (1976). In search of premenstrual tension: Note on sex-differences in psychological-differentiation as a function of cyclical physiological changes. *Perceptual and Motor Skills, 42,* 1139–1142.

Editorial, (1976). More divorce and less salary for women science PhDs. *New Scientist, 69,* 130–131.

Ehrhardt, A., Meyer-Bahlburg, H., Rosen, L., Feldman, J., Veridiano, N., & Zimmerman, I. (1985). Sexual orientation after prenatal exposure to exogenous estrogen. *Archives of Sexual Behavior, 14,* 57–77.

Ellis, L., & Ames, M. (1987). Neurohormonal functioning and sexual orientation: A theory of homosexuality–heterosexuality. *Psychological Bulletin, 101,* 233–258.

Ellis, L., & Nyborg, H. (1992). Racial/ethnic variations in male testosterone levels: A probable contributor to group differences in health. *Steroids, 57,* 72–75.

Ellis, L., Peckham, W., Ames, M., & Burke, D. (1988). Sexual orientation of human offspring may be altered by severe maternal stress during pregnancy. *The Journal of Sex Research, 25,* 52–157.

Eysenck, H. (1976). Ideology run wild. *American Psychologist, 31*(4), 311–312.

Farthing, M., Mattel, A., Edwards, C., & Dawson, A. (1982). Relationship between plasma testosterone and dihydrotestosterone concentrations and male facial hair growth. *British Journal of Dermatology, 107,* 559–564.

Feder, H. (1984). Hormones and sexual behavior. *Annual Review of Psychology, 35,* 165–200.
Garai, J., & Scheinfeld, A. (1968). Sex differences in mental and behavioral traits. *Genetic Psychology Monographs, 77,* 169–299.
Geschwind, N., & Galaburda, A. (1987). *Cerebral lateralization: Biological mechanisms, associations, and pathology.* Cambridge, MA: MIT Press.
Goldberg, S. (1977). *The inevitability of patriarchy.* London: Temple Smith.
Gooren, L. (1988). An appraisal of endocrine theories of homosexuality and gender dysphoria. In J. Sitsen (Ed.), *Handbook of sexology. Vol. 6: The pharmacology and endocrinology of sexual function.* Amsterdam: Elsevier Science Publishing.
Gordon, H., Corbin, E., & Lee, P. (1986). Changes in specialized cognitive function following changes in hormone levels. *Cortex, 22,* 399–415.
Gorski, R. (1974). The neuroendocrine regulation of sexual behavior. In G. Newton & A. Riesen (Eds.), *Advances in Psychobiology* (pp. 1–58). New York: John Wiley & Sons.
Gorski, R. (1984). Critical role for the medial preoptic area in the sexual differentiation of the brain. In G. Vries, J. Bruin, H. Uylings, & M. Corner (Eds.), *Sex differences in the brain: Progress in the brain research* (pp. 129–146). Amsterdam: Elsevier Biomedical Press.
Gouchie, C., & Kimura, D. (1990). The relation between testosterone levels and cognitive ability patterns. *Psychoneuroendocrinology, 16,* 1–30.
Gould, E., Woolley, C. S., & McEwen, B. S. (1991). Naturally occurring cell death in the developing dentate gyrus of the rat. *The Journal of Comparative Neurology, 304,* 408–418.
Goy, R., McEwen, B., Baker, S., Beatty, W., Czaja, J., Dörner, G., Ehrhardt, A., & Fox, T. (1980). *Sexual differentiation of the brain. Based on a work session of the neurosciences research program.* Cambridge: MA: MIT Press.
Greenwald, I., & Martinez-Arias, A. (1984). Programmed cell death in invertebrates. *Trends in NeuroSciences, 7,* 179–181.
Hampson, E. (1986). Variations in perceptual and motor performance related to phase of the menstrual cycle. *Canadian Psychology, 27,* 268.
Hampson, E. (1988). Variations in sex-related cognitive abilities across the menstrual cycle. Research Bulletin, 669. Department of Psychology, University of Western Ontario, Canada.
Hampson, E. (1990). Estrogen-related variations in human spatial and articulatory-motor skills. *Psychoneuroendocrinology, 15,* 97–111.
Hampson, E., & Kimura, D. (1988). Reciprocal effects of hormonal fluctuations on human motor and perceptuo-spatial skills. *Behavioral Neuroscience, 102,* 456–459.
Hapgood, F. (1979). *Why males exist: An inquiry into the evolution of sex.* New York: William Morrow and Company.
Harrell, T., & Harrell, M. (1945). Army General Classification Test scores for civilian occupations. *Educational and Psychological Measurement, 5,* 229–239.
Harris, L. (1979). Sex-related differences in spatial ability: A developmental psychological view. In C. Kopp (Ed.), *Becoming female, perspectives on development* (pp. 133–181). New York: Plenum Press.
Harris, L. (1980). Lateralized sex differences: Substrates and significance. *The Behavioral and Brain Sciences, 3,* 236–237.
Hinchliffe, J. (1981). Cell death in embryogenesis. In I. Bowen & R. Lockshin (Eds.), *Cell death in biology and pathology.* London: Chapman and Hall.
Hingley, P., & Cooper, C. (1983). The loners at the top. *New Society, 65,* 467–470.
Hofman, M. A., & Swaab, D. F. (1989). The sexually dimorphic nucleus of the preoptic area in the human brain: A comparative morphometric study. *Journal of Anatomy, 164,* 55–72.
Ho, H., Gilger, J., & Brink, T. (1986). Effects of menstrual cycle on spatial information-processes. *Perceptual and Motor Skills, 63,* 743–751.
Ho, K. C., Roessmann, U., Straumfjord, J. V., & Monroe, G. (1980). Analysis of brain weight: I. Adult brain weight in relation to sex, race, and age. *Archive for Pathology and Laboratory Medicine, 104,* 635–639.
Hoyenga, K., & Hoyenga, K. (1979). *The question of sex differences: Psychological, cultural, and biological issues.* Boston: Little, Brown and Company.

Hughes, R. (1983). Menstrual cycle influences on perceptual disembedding ability. *Perceptual and Motor Skills, 57,* 107–110.

Hyde, J., & Linn, M. (1986). *The psychology of gender.* Baltimore: Johns Hopkins University Press.

Jacklin, C. N. (1979). Epilogue. In M. A. Wittig & A. C. Petersen (Eds.), *Sex-related differences in cognitive functioning: Developmental issues* (pp. 357–371). New York: Academic Press.

Jacklin, C. (1989). Female and male: issues of gender. *American Psychologist, 44,* 127–133.

Jacob, F. (1982a). *The logic of life: A history of heredity.* New York: Pantheon Books.

Jacob, F. (1982b). *The possible and the actual.* New York: Pantheon Books.

Jacobson, C., Davis, F., & Gorski, R. (1985). Formation of the sexually dimorphic nucleus of the preoptic area: Neuronal growth, migration and changes in cell number. *Developmental Brain Research, 21,* 7–18.

Jardine, R., & Martin. N. (1983). Throwing accuracy and the genetics of spatial ability. *Behavior Genetics, 13,* 331–340.

Jensen, A. (1980). *Bias in mental testing.* New York: Free Press.

Jensen, A., & Reynolds, C. (1983). Sex difference on the WISC-R. *Personality and Individual Differences, 4,* 223–226.

Johnson, F. W. (1991). Biological factors and psychometric intelligence: A review. *Genetic, Social, and General Psychology Monographs, 117,* 3, 313–357.

Jonge, F. D., Louwerse, A., Ooms, M., Evers, P., Endert, E., & Van de Poll, N. (1989). Lesions of the SDN-POA inhibit sexual behavior of male Wistar rats. *Brain Research Bulletin, 23,* 483–492.

Juraska, J. (1984). Sex differences in developmental plasticity in the visual cortex and hippocampal dentate gyrus. In G. Vries, J. Bruin, H. Uylings, & M. Corner (Eds.), *Sex differences in the brain: Progress in the brain research* (pp. 205–214). Amsterdam: Elsevier Biomedical Press.

Juraska, J. (1986). Sex differences in developmental plasticity of behavior and the brain. In W. Greenough & J. Juraska (Eds.), *Developmental neuropsychobiology* (pp. 409–422). New York: Academic Press.

Juraska, J., Fitch, J., Henderson, C., & Rivers, N. (1985). Sex differences in the dendritic branching of dentate granule cells following differential experience. *Brain Research, 333,* 73–80.

Jurgens, H. W., Aune, I. A., & Pieper, U. (1990). *International data on anthropometry.* Geneva, Switzerland: International Labour Office.

Kaufman, A. (1975). Factor analysis of the WISC-R at 11 age levels between 6 and 16 years. *Journal of Consulting and Clinical Psychology, 43,* 135–147.

Kimura, D. (1983). Sex differences in cerebral organization for speech and praxic functions. *Canadian Journal of Psychology, 37,* 19–35.

Kimura, D. (1987). Are men's and women's brains really different? *Canadian Psychology, 28,* 133–147.

Kimura, D. (1991). Sex differences in cognitive function vary with the season. Research Bulletin #697. Department of Psychology, University of Western Ontario, Canada.

Kimura, D., & Harshman, R. (1984). Sex differences in brain organization for verbal and non-verbal functions. In G. Vries, J. Bruin, H. Uylings, & M. Corner (Eds.), *Sex differences in the brain: Progress in the brain research* (pp. 423–441). Amsterdam: Elsevier Biomedical Press.

Klaiber, E., Broverman, D., Vogel, W., & Kobayashi, Y. (1974). Rhythms in plasma MAO activity, EEG, and behavior during the menstrual cycle. In M. Ferin, F. Halberg, R. Richart, & R. Wiele (Eds.), *Biorhythms and human reproduction* (pp. 353–367). New York: John Wiley.

Knussmann, R., & Christiansen, K. (1986). Relations between sex hormone levels and sexual behavior in men. *Archives of Sexual Behavior, 15,* 429–445.

Knussmann, R., & Sperwien, A. (1988). Relations between anthropometric characteristics and androgen hormone levels in healthy young men. *Annals of Human Biology, 15,* 131–142.

Kolakowski, D., & Malina, R. (1974). Spatial ability, throwing accuracy and man's hunting heritage. *Nature, 251,* 410–412.

Kolb, B., Sutherland, R., Nonneman, A., & Whishaw, I. (1982). Asymmetry in the cerebral hemispheres of the rat, mouse, rabbit, and cat: The right hemisphere is larger. *Experimental Neurology, 78,* 348–359.

Komnenich, P., Land, D., Dickey, R., & Stone, S. (1978). Gonadal hormones and cognitive performance. *Physiological Psychology, 6,* 115–120.

Lacoste-Utamsing, C., & Holloway, R. (1982). Sexual dimorphism in the human corpus callosum. *Science, 216,* 1431–1432.

Landman, A., Sanford, L., Howland, B., Dawes, C., & Pritchard, E. (1976). Testosterone in human saliva. *Experienta, 32,* 940–941.

Lenney, E. (1979). Androgyny: Some audacious assertions toward its coming of age. *Sex Roles, 5,* 703–719.

Linn, M., & Petersen, A. (1985). Emergence and characterization of sex differences in spatial ability: A meta-analysis. *Child Development, 56,* 1479–1498.

Lynn, R. (1990). Negative correlations between verbal and visuo-spatial abilities: Statistical artifact or empirical relationship? *Personality and Individual Differences, 11,* 755–756.

Lynn, R., & Mulhern, G. (1991). A comparison of sex differences on the Scottish and American standardisation samples on the WISC-R. *Personality and Individual Differences, 12(11),* 1179–1182.

Maccoby, E., & Jacklin, C. (1978). *The psychology of sex differences Volume I: Text Volume II: Annotated bibliography.* Stanford, California: Stanford University Press.

Marshall, W., & Tanner, J. (1986). Puberty. In F. Falkner & J. Tanner (Eds.), *Human growth* (pp. 171–209). New York: Plenum Publishing Corporation.

Matarazzo, J. D., Bornstein, R. A., McDermott, P. A., & Noonan, J. V. (1986). Verbal IQ vs. performance IQ difference scores in males and females from the WAIS-R standardization sample. *Journal of Clinical Psychology, 42,* 965–974.

Matsumoto, S., Sato, I., Ito, T., & Matsuoka. A. (1966). Electroencephalographic changes during long term treatment with oral contraceptives. *International Journal of Fertility, 11,* 195–204.

McCall, R. (1977). Childhood IQs as predictors of adult, educational and occupational status. *Science, 197,* 482–483.

McCormick, C. M., & Witelson, S. F. (1991). A cognitive profile of homosexual men compared to heterosexual men and women. *Psychoneuroendocrinology, 6,* 459–473.

McEwen, B. (1987a). Observations on brain sexual differentiation: A biochemist's view. In J. Reinisch, L. Rosenblum, & S. Sanders (Eds.), *Masculinity/Femininity: Basic perspectives* (pp. 68–79). Oxford: Oxford University Press.

McEwen, B. (1987b). Steroid hormones and brain development: Some guidelines for understanding actions of pseudohormones and other toxic agents. *Environmental Health Perspectives, 74,* 177–184.

McEwen, B. (1988a). Actions of sex hormones on the brain: "Organization" and "activation" in relation to functional teratology. In G. Boer, M. Feenstra, M. Mirmiram, D. Swaab, & F. V. Haaren (Eds.), *Progress in brain research* (pp. 121–134). Amsterdam: Elsevier.

McEwen, B. (1988c). Genomic regulation of sexual behavior. *Journal of Steroid Biochemistry, 30,* 179–183.

McEwen, B. (1988d). Steroid hormones and the brain: Linking "nature" with "nurture." *Neurochemical Research, 13,* 663–669.

McEwen, B. (1991a). Our changing ideas about steroid effects on an ever-changing brain. *Seminars in the Neurosciences, 3,* 497–507.

McEwen, B. (1991b). Steroids affect neural activity by acting on the membrane and the genome. *Trends in Pharmacological Sciences, 12,* 141–147.

McEwen, B., Jones, K., & Pfaff, D. (1987). Hormonal control of sexual behavior in the female rat: Molecular, cellular and neurochemical studies. *Biology of Reproduction, 36,* 37–45.

McGee, M. (1979). Human spatial abilities: Psychometric studies and environmental, genetic, hormonal, and neurological influences. *Psychological Bulletin, 86,* 889–917.

McGlone, J. (1986). The neuropsychology of sex differences in human brain organization. In G. Goldstein & R. Tarter (Eds.), *Advances in Clinical Neuropsychology* (pp. 1–29). New York: Plenum Press.

McGlone, J. (1980). Sex differences in human brain asymmetry: A critical survey. *The Behavioral and Brain Sciences, 3,* 215–263.

McKeever, W. (1986). The influences of handedness, sex, familial sinistrality and androgyny on language laterality, verbal ability, and spatial ability. *Cortex, 22,* 521–537.

Meikle, A., Bishop, D., Stringham, J. D., & West, D. (1986). Quantitating genetic and nongenetic factors that determine plasma sex-steroid variation in normal male twins. *Metabolism, 35,* 1090–1095.

Money, J., Schwartz, M., & Lewis, V. (1984). Adult erotosexual status and fetal hormonal masculinization and demasculinization: 46,XX congenital virilizing adrenal hyperplasia and 46,XY androgen-insensitivity syndrome compared. *Psychoneuroendocrinology, 9,* 405–414.

Money, J. (1987). Sin, sickness, or status? Homosexual gender identity and psychoneuroendocrinology. *American Psychologist, 42,* 384–389.

Monod, J. (1971). *Chance & Necessity.* New York: Knopf.

Moss, R., & Dudley, C. (1984). Molecular aspects of the interaction between estrogen and the membrane excitability of hypothalamic nerve cells. In G. Vries, J. Bruin, H. Uylings, & M. Corner (Eds.), *Sex differences in the brain: Progress in brain research* (pp. 3–22). Amsterdam: Elsevier Biomedical Press.

Nass, R., Baker, S., Speiser, P., Virdis, R., Balsamo, A., Cacciari, E., Loche, A., Dumic, M., & New, M. (1987). Hormones and handedness: Left-hand bias in female congenital adrenal hyperplasia patients. *Neurology, 37,* 711–715.

Nieschlag, E. (1974). Circadian rhythm of plasma testosterone. In J. Aschoff, F. Ceresa, & F. Halberg (Eds.), *Chronobiological aspects of endocrinology* (pp. 117–128). New York: Schattaur Verlag.

Norman, A. W., & Litwack, G. (1987). *Hormones.* San Diego: Academic Press.

Nottebohm, F. (1980). A continuum of sexes bedevils the search for sexual differences? *The Behavioral and Brain Sciences, 3,* 245–246.

Nottebohm, F. (1981). A brain for all seasons: Cyclical anatomical changes in song control nuclei of the canary brain. *Science, 214,* 1368–1370.

Nottebohm, F. (1989). From bird song to neurogenesis. *Scientific American, 260*(2), 56–61.

Nottebohm, F., Nottebohm, M., & Crane, L. (1986). Developmental and seasonal changes in canary song and their relation to changes in the anatomy of song-control nuclei. *Behavioral and Neural Biology, 46,* 445–471.

Nyborg, H. (1977). *The rod-and-frame test and the field dependence dimension: Some methodological, conceptual, and developmental considerations.* Copenhagen: Dansk Psykologisk Forlag.

Nyborg, H. (1979, June). *Sex chromosome abnormalities and cognitive performance V: Female sex hormone and discontinuous cognitive development.* Paper and hand-out presented in the Symposium on Cognitive Studies at the Fifth Biennial Meeting of the International Society for the Study of Behavioral Development, Lund, Sweden.

Nyborg, H. (1983). Spatial ability in men and women: Review and new theory. *Advances in Human Research and Therapy, 5*(whole issue), 39–140.

Nyborg, H. (1984). Performance and intelligence in hormonally-different groups. In G. D. Vries, J. D. Bruin, H. Uylings, & M. Corner (Eds.), *Sex differences in the brain. Progress in brain research* (pp. 491–508). Amsterdam: Elsevier Biomedical Press.

Nyborg, H. (1987a). Individual differences or different individuals? That is the question. *Behavioral and Brain Sciences, 10,* 34–35.

Nyborg, H. (1987b, June). *Covariant trait development across races and within individuals: Differential K theory, genes, and hormones.* Paper presented in the Symposium on Biology-Genetics at the Third Meeting of the International Society for the Study of Individual Differences, Toronto, Canada.

Nyborg, H. (1988a). Change at puberty in spatioperceptual strategy on the rod-and-frame test. *Perceptual and Motor Skills, 67,* 129–130.

Nyborg, H. (1988b). Sex hormones and covariant body, brain and behavioral development. *Neuroendocrinology Letters (Abstracts), 10,* 217.

Nyborg, H. (1989). The nature of Nature–Nurture interaction. *Behavior Genetics, 20,* 738–739.

Nyborg, H. (1990a). Sex hormones, brain development, and spatio-perceptual strategies in Turner's syndrome. In D. Berch & B. Bender (Eds.), *Sex chromosome abnormalities and human behavior: Psychological studies* (pp. 100–128). Boulder, CO: Westview Press.

Nyborg, H. (1990b). Good, bad, and ugly questions about heredity. *Behavioral and Brain Sciences, 13,* 142–143.

Nyborg, H. (1994a). Individual, sex, and race differences in body, brain, and specific abilities: The General Trait Covariance–Androgen/Estrogen model for development. (Submitted).

Nyborg, H. (1994b). *Hormones, sex, and society: The physicological approach.* New York: Greenwood Publishing Co. (in press).

Nyborg, H. (1994c). Gonadal hormones, body, brain, intellectual, and personality development at puberty: A 14-year cohort-sequential study of 8–18-year-old, unselected Danish children. Unpublished manuscript.

Nyborg, H. (1992, April). *Genes, hormones, maturation, and the etiology of psychoses: An application of the GTC–A/E model.* Paper presented at the International Symposium on Rate of Maturation, Brain Development, and Behaviour," University of Trondheim, Geilo, Norway.

Nyborg, H., & Bøggild, C. (1989). Mating behavior: Moves of mind or molecules? *The Behavioral and Brain Sciences, 12,* 29–30.

Nyborg, H., & Nielsen, J. (1977). Sex chromosome abnormalities and cognitive performance. III: Field dependence, frame dependence, and failing development of perceptual stability in girls with Turner's syndrome. *Journal of Personality, 96,* 205–211.

Nyborg, H., & Nielsen, J. (1981a). Spatial ability of men with karyotype 47,XXY, 47,XYY, or normal controls. In W. Schmid & J. Nielsen (Eds.), *Human behavior and genetics* (pp. 85–106). Amsterdam: Elsevier/North-Holland Biomedical Press.

Nyborg, H., & Nielsen, J. (1981b). Sex hormone treatment and spatial ability in women with Turner's syndrome. In W. Schmid & J. Nielsen (Eds.), *Human behavior and genetics* (pp. 167–182). Amsterdam: Elsevier/North-Holland Biomedical Press.

Nyborg, H., & Sommerlund, B. (1992). Spearman's *g,* the verbal-performance balance, and brain processes: The Lynn-Vernon debate. *Personality and Individual Differences, 13*(11), 1253–1255.

O'Connor, J. (1943). *Structural Visualization.* Boston: Human Engineering Lab.

O'Kusky, J., Strauss, E., Kosaka, B., Wada, J., Li, D., Druhan, M., & Petrie, J. (1988). The corpus callosum is larger with right-hemisphere cerebral speech dominance. *Annals of Neurology, 24,* 379–383.

Pavlides, C., Westlind-Danielsson, A., Nyborg, H., & McEwen, B. (1991). Neonatal hyperthyroidism disrupts hippocampal LTP and spatial learning. *Experimental Brain Research, 85,* 559–564.

Petersen, A. (1976). Physical androgyny and cognitive functioning in adolescence. *Developmental Psychology, 12,* 524–533.

Petersen, A., & Crockett, L. (1985). Pubertal timing and grade effects on adjustment. Special Issue: Time of maturation and psycho-social functioning in adolescence: I. *Journal of Youth and Adolescence, 14*(3), 191–206.

Petersen, A., & Taylor, B. (1980). The biological approach to adolescence: Biological change and psychological adaptation. In J. Adelson (Ed.), *Handbook of Adolescent Psychology* (pp. 117–155). New York: John Wiley & Sons.

Plomin, R., & Daniels, D. (1987). Why are children in the same family so different from one another? *Behavioral and Brain Sciences, 10,* 1–60.

Purifoy, F. (1981). Endocrine-environment interaction in human variability. *Annual Review of Anthropology, 10,* 141–162.

Purifoy, F., & Koopmans, L. (1979). Androstenedione, testosterone, and free testosterone concentration in women of various occupations. *Social Biology, 26,* 179–188.

Ragins, B., & Sundstrom, E. (1989). Gender and power in organizations: A longitudinal perspective. *Psychological Bulletin, 105,* 51–88.

Read, G., Riad-Fahmy, D., Walker, R. F., & Griffiths, K. (Eds.) (1984). Immunoassays of steroids in saliva. *Proceedings of the Ninth Tenovus Workshop,* Cardiff, Wales: Alpha Omega Publ.

Reid, I. (1982). Vital statistics. In I. Reid & E. Wormald (Eds.), *Sex differences in Britain* (pp. 29–58). London: Grant McIntyre.

Reinberg, A., & Lagoguey, M. (1978). Circadian and circannual rhythms in sexual activity and plasma hormones (FSH, LH, testosterone) of five human males. *Archives for Sexual Behavior, 7,* 13–30.

Reinberg, A., Lagoguey, M., Chauffournier, J., & Cesselin, F. (1975). Circannual and circadian rhythms in plasma testosterone in 5 healthy young Parisian males. *Acta Endocrinologica, 80,* 732–743.

Romano, G. J., Mobbs, C. V., Lauber, A., Howells, R. D., & Pfaff, D. W. (1990). Differential regulation of proenkephalin gene expression by estrogen in the ventromedial hypothalamus of male and female rats: Implications for the molecular basis of a sexually differentiated behavior. *Brain Research, 536,* 63–68.

Ross, R., Bernstein, L., Judd, L., Hanisch, R., Pike, M., & Henderson, B. (1986). 1986 serum testosterone levels in healthy young black and white men. *Journal of the National Cancer Institute, 76,* 45–48.

Roth, J., LeRoith, D., Shiloach, J., Rosenzweig, J., Leisniak, M., & Havrankova, J. (1982). The evolutionary origins of hormones, neurotransmitters and other extracellular chemical messengers. *The New England Journal of Medicine, 306,* 523–527.

Runyan, W. (1983). Idiographic goals and methods in the study of lives. *Journal of Personality, 51,* 413–437.

Rushton, J. P. (1992). Cranial capacity related to sex, rank, and race in a stratified random sample of 6,325 military personnel. *Intelligence, 16,* 401–413.

Rushton, J. P. (1992b). Evidence for race and sex differences in cranial capacity from International labor office data. Research Bulletin, 712. Department of Psychology, University of Western Ontario, Canada.

Sandhu, S., Cook, P., & Diamond, M. (1986). Rat cerebral cortical estrogen receptors: Male-female, right-left. *Experimental Neurology, 92,* 186–196.

Sannikka, E., Terho, P., Suominen, J., & Santti, R. (1983). Testosterone concentrations in human seminal plasma and saliva and its correlation with non-protein-bound and total testosterone levels in serum. *International Journal of Andrology, 6,* 319–330.

Schapiro, S., Salas, M., & Vukovich, K. (1970). Hormonal effects on ontogeny of swimming ability in the rat: Assessment of central nervous system development. *Science, 168,* 147–150.

Schindler, G. L. (1979). Testosterone concentration, personality patterns, and occupational choice in women. *Dissertation Abstracts International, 40,* 1411A, (University Microfilms No. 79-19,403).

Sherman, J. (1967). Problem of sex differences in space perception and aspects of intellectual functioning. *Psychological Review, 74,* 290–299.

Shute, V., Pellegrino, J., Hubert, L., & Reynolds, R. (1983). The relationship between androgen levels and human spatial abilities. *Bulletin of the Psychonomic Society, 21,* 465–468.

Silverman, I., & Phillips, K. (1991, August). *Effects of estrogen during the menstrual cycle on spatial performance.* Paper presented at the Meeting of the Human Behavior and Evolution Society, McMaster University, Hamilton, Ontatio, Canada.

Smals, A., Kloppenborg, P., & Benraad, T. (1976). Circannual cycle in plasma testosterone levels in man. *Journal of Clinical Endocrinology and Metabolism, 42,* 979–982.

Smith, I. (1964). *Spatial ability: Its educational and social significance.* San Diego, California: Robert R. Knapp.

Spearman, C. (1904). General intelligence, objectively determined and measured. *American Journal of Psychology, 15,* 201–293.

Spence, J., & Helmreich, R. (1979). On assessing "Androgyny." *Sex Roles, 5,* 721–737.

Stafford, R. (1961). Sex differences in spatial visualization as evidence of sex-linked inheritance. *Perceptual and Motor Skills, 13,* 428.

Stafford, R. (1963). An investigation of similarities in parent-child test scores for evidence of hereditary components. Educational Testing Service, Princeton.

Stafford, R. (1965, April). *Spatial visualization and quantitative reasoning: One gene or two?* Paper presented at Eastern Psychological Association, Atlantic City, NJ.

Steward, J., & Kolb, B. (1988). The effects of neonatal gonadectomy and prenatal stress on cortical thickness and asymmetry in rats. *Behavioral and Neural Biology, 49,* 344–360.

Swaab, D., & Fliers, E. (1985). A sexually dimorphic nucleus in the human brain. *Science, 228,* 1112–1115.

Swaab, D., & Hofman, M. (1988). Sexual differentiation of the human hypothalamus: Ontogeny of the sexual dimorphic nucleus of the preoptic area. *Developmental Brain Research, 44,* 314–318.

Swaab, D., Hofman, M., & Fisser, B. (1988). Sexual differentiation of the human brain. *Neuroendocrinology Letters (Abstracts), 10,* 4, 217.

Symond, D. (1979). *The evolution of human sexuality.* New York: Oxford University Press.

Tan, Ü., & Akgün, A. (1992). There is a direct relationship between nonverbal intelligence and serum testosterone level in young men. *International Journal of Neuroscience, 64,* 213–216.

Tanner, J. (1975). Growth and endocrinology of the endocrinology of the adolescent. In L. Gardner (Ed.), *Endocrine and genetic diseases of childhood and adolescence* (pp. 14–64). Toronto: W. B. Saunders.

Tarttelin, M., & Gorski, R. (1988). Postnatal influence of diethylstilbestrol on the differentiation of the sexually dimorphic nucleus in the rat is as effective as perinatal treatment. *Brain Research, 456,* 271–274.

Terman, L. (1925). Mental and physical traits of a thousand gifted children. In L. Terman (Ed.), *Genetic studies of genius.* Stanford, CA: Stanford University Press.

Toran-Allerand, C. (1984). On the genesis of sexual differentiation of the central nervous system: Morphogenetic consequences of steroidal exposure and possible role of alpha-fetoprotein. In G. Vries, J. Bruin, H. Uylings, & M. Corner (Eds.), *Sex differences in the brain: Progress in the brain research* (pp. 63–98). Amsterdam: Elsevier Biomedical Press.

Toran-Allerand, D. (1986). Sexual differentiation of the brain. In W. Greenough & J. Juraska (Eds.), *Developmental neuropsychobiology* (pp. 175–211). New York: Academic Press.

Truman, J. (1985). Hormonal approaches to the study of cell death in a developing nervous system. In G. Edelman, W. Gall, & W. Cowan (Eds.), *Molecular bases of neural development* (pp. 531–545). New York: John Wiley & Sons.

Truman, J., & Schwartz, L. (1984). Steroid regulation of neuronal death in the moth nervous system. *The Journal of Neuroscience, 4,* 274–280.

Van Valen, L. (1974). Brain size and intelligence in man. *American Journal of Physical Anthropology, 40,* 417–424.

Vandenberg, S., & Kuse, A. (1979). Spatial ability: A critical review of the sex-linked major gene hypothesis. In M. Wittig & A. Petersen (Eds.), *Sex-related differences in cognitive functioning: Developmental issues* (pp. 67–95). New York: Academic Press.

Vernon, P. A. (1990). The effect of holding *g* constant on the correlation between verbal and nonverbal abilities: A comment on Lynn's "The intelligence of the Mongoloids" (1987). *Personality & Individual Differences, 11,* 751–754.

Vining, D. J. (1982). On the possibility of the reemergence of a dysgenic trend with respect to intelligence in American fertility differentials. *Intelligence, 6,* 241–264.

Vining, D. (1984). Subfertility among the very intelligent: An examination of the American mensa. *Personality and Individual Differences, 5,* 725–733.

Vogel, S. (1990). Gender differences in intelligence, language, visuo-motor abilities and academic achievement in students with learning disabilities: a review of the literature. *Journal of Learning Disability, 23,* 44–52.

Vries, G. D., Bruin, J. D., Uylings, H., & Corner, M. E. (Eds.) (1984). *Sex differences in the brain: Relation between structure and function.* Amsterdam: Elsevier Science Publishers.

Waber, D. (1976). Sex differences in cognition: A function of maturation rate. *Science, 192,* 572–574.

Waber, D. (1977a). Sex differences in mental abilities, hemispheric lateralization, and rate of physical growth at adolescence. *Developmental Psychology, 13,* 29–38.

Waber, D. (1977b). Biological substrates of field dependence: Implications of the sex difference. *Psychological Bulletin, 84,* 1076–1087.

Waber, D. (1979). Cognitive abilities and sex-related variations in the maturation of cerebral cortical functions. In M. Wittig & A. Petersen (Eds.), *Sex-related differences in cognitive functioning* (pp. 161–186). New York: Academic Press.

Wada, J., Clarke, R., & Hamm. A. (1975). Cerebral hemispheric asymmetry in humans. *Archives of Neurology, 32,* 239–246.

Wahlsten, D. (1990). Insensitivity of the analysis of variance to heredity–environment interaction. *Behavioral and Brain Sciences, 13,* 109–161.

Waller, J. (1971). Achievement and social mobility: Relationships among IQ score, education, and occupation in two generations. *Social Biology, 18,* 252–259.

Watson, N., & Kimura, D. (1990). *Nontrivial sex differences in throwing and intercepting: Relation to psychometrically defined spatial functions.* (Research Bulletin #693). London, ON: Department of Psychology, University of Western Ontario.

Wechsler, D. (1981). *WAIS-R Manual: Wechsler Adult Intelligence Scale-Revised.* New York: Psychological Corporation.

Whitam, F., & Mathy, R. (1988). Childhood cross-gender behavior of lesbians in four societies: Brazil, Peru, the Philippines, and the United States. *Archives of Sexual Behavior, 20*(2), 151–170.

Willerman, L., Schultz, R., Rutledge, J. N., & Bigler, E. D. (1991). In vivo brain size and intelligence. *Intelligence, 15,* 223–228.

Witelson, S., & Pallie, W. (1973). Left hemisphere specialization for language in the newborn: Neuroanatomical evidence of asymmetry. *Brain, 96,* 641–646.

Witkin, H. (1973). The role of cognitive style in academic performance and in teacher-student relations. *Educational Testing Service Research Bulletin RB-73-11.* Princeton, NJ.

Witkin, H., Dyk, R., Faterson, H., Goodenough, D., & Karp, S. (1962). *Psychological differentiation.* Potomac, MD: Erlbaum.

Witkin, H., & Goodenough, D. (1977). Field dependence and interpersonal behavior. *Psychological Bulletin, 84,* 661–689.

Witkin, H., Goodenough, D., & Karp, S. (1967). Stability of cognitive style from childhood to young adulthood. *Journal of Personality and Social Psychology, 7,* 291–300.

Witkin, H., Goodenough, D., & Oltman, P. (1977). Psychological differentiation: Current status. *Educational Testing Service Research Bulletin RB-77-17,* Princeton, NJ.

Witkin, H. A., Lewis, H. B., Hertzman, M., Machover, K., Meissner, P. B., & Wapner, S. (1954). *Personality through perception.* New York: Harper.

Witkin, H., Moore, C., Goodenough, D., & Cox, P. (1977). Field-dependent and field-independent cognitive styles and their educational implications. *Review of Educational Research, 47,* 1–64.

Witzmann, R. (1981). *Steroids: Keys to Life.* New York: Van Nostrand Reinhold.

Woodfield, R. (1984). Embedded figure test performance before and after childbirth. *British Journal of Psychology, 75,* 81–88.

Woodfield, R., Whitehead, M., & Asso, D. (1987). Performance on particular tests of spatial functioning during the early follicular and peri-ovulatory periods of the menstrual cycle. *Gynecological Endocrinology (Abstracts), 1,* 85.

Woolley, C., Gould, E., Frankfurt, M., & McEwen, B. (1990). Naturally occuring fluctuations in dendritic spine density on adult hippocampal pyramidal neurons. *The Journal of Neuroscience, 10,* 4035–4039.

Wormald, E. (1982). Political participation. In I. Reid & E. Wormald (Eds.), *Sex differences in Britain* (pp. 175–203). London: Grant McIntyre.

Young, M., & Reeve, T. (1980). Discriminant analysis of personality and body-image factors of females differing in percent body fat. *Perceptual and Motor Skills, 50,* 547–552.

Ziegler, N. I., & Lichtensteiger, W. (1992). Asymmetry of brain aromatase activity: Region- and sex-specific developmental patterns. *Neuroendocrinology, 55,* 512–518.