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Performance and Intelligence in Hormonally Different Groups

H. NYBORG

Institute of Psychology, University of Aarhus, Risskov (Denmark)

INTRODUCTION

Males and females differ with respect to both performance and intelligence (for review, see Garai and Scheinfeld, 1968; Maccoby and Jacklin, 1974; Hoyenga and Hoyenga, 1979). Compared to women, men tend to be more assertive (e.g. to behave more independently and self-reliantly), aggressive, and to show high physical energy expenditure (e.g. they demonstrate a preference for "rough-and-tumble" outdoor play and "chasing behavior" in childhood and show more athletic interests in adulthood). Prepubertal play patterns are typically instrumental and object-oriented in boys, while many girls behave in some respects as though they rehearsed a later maternal role. General IQ scores do not differ markedly between men and women but certain subscores and IQ factors tend to do so. Men tend to outperform women on difficult spatial tasks, whereas women generally outperform men on certain types of verbal tasks. Men typically score higher on performance IQ than on verbal IQ, whereas the opposite is true for women. The differences in performance and intelligence between men and women are summarized in Table I.

These commonly observed sex differences have been explained in several ways. "Environmentalists" with a preference for "tabula rasa" models propose that sex differences appear because boys and girls are reared differently. Such workers typically use concepts like "reinforcement", "training", and "imitation", or assume that careless transfer of now inadequate cultural stereotypes can explain their observations. "Genetically oriented" researchers, on the other hand, often resort to less one-sided but sometimes very complex genetic models. A number of "interactionist" paradigms have also seen the light of day (see Hoyenga and Hoyenga, 1979, for review of theories). Unfortunately, neither nurture nor nature or combination models have succeeded in indicating whether specific environmental or genetic factors may be attributed a causal status with respect to the origin of gender differences. Furthermore, the mediating mechanisms have not been identified.

We have also looked for causal factors and their "route of impact" on sex-related personality and intellectual development in genetically abnormal groups. We examined the hypothesis that various karyotypes predispose for abnormal personality and intellectual development. However, after 10 years of work with persons of karyotypes 45,X; 47,XXY and 47,XYY and their normal controls, we could find neither a simple relationship between sex chromosome complement and performance, nor could any existing genetic or environmentalistic hypothesis be supported (Nielsen et al., 1977; Nyborg and Nielsen, 1979, 1981a,b). Some of our data led, in fact, to a rejection of the prominent X-linked, recessive

TABLE I

COMMONLY OBSERVED SEX DIFFERENCES IN GENDER IDENTITY, GENDER ROLE PERFORMANCE, AND INTELLECTUAL PERFORMANCE

	Gender identity	*Assertiveness, independence, self-reliance*	*Level of aggression*	*Physical energy expenditure*	*Prepubertal play preference*		*Maternal interests*	*Spatial ability*	*Verbal ability*	*Verbal (V) — performance (P) score relationship*
					Objects	*Persons*				
Men	Masculine	+	+	+	+	÷	÷	+	÷	P > V
Women	Feminine	÷	÷	÷	÷	+	+	÷	+	V > P

+, high "normal"; ÷, low "normal".

gene theory for spatial ability (O'Connor, 1943; Stafford, 1961). We, therefore, reanalyzed our data, taking into account the fact that the groups were not only genetically abnormal but also abnormal hormonally. Thus, women with Turner's syndrome (i.e. lack of X-chromosome material) produce only minute amounts of sex hormones as their gonads develop improperly, and such women also perform poorly on spatial tasks and in mathematics. The reanalysis unexpectedly showed that Turner's women who had been treated with cyclic estrogen and gestagen for a period ranging from 3 months to 2 years performed at a normal female spatial ability level. In contrast, those who either received no hormone therapy at all or received this treatment for many years (8 years on the average) showed extremely low spatial ability in a number of spatial tasks (Nyborg and Nielsen, 1981a). These observations, the findings of Broverman et al. (1964, 1968) and of Petersen (1976), and the fact that other groups with abnormal sex hormone levels show deviant spatial ability suggest that sex hormones influence spatial ability. Therefore we began to pay more attention to the relationship between gonadal hormones and spatial problem-solving ability. We soon found several lines of evidence for such relations. Firstly, spatial ability apparently varies with the menstrual cycle (Klaiber et al., 1974; Dor-Shav, 1976). Secondly, the development of spatial ability may be related to early and late pubertal maturation (Waber, 1976, 1977a,b; but see also Petersen, 1976). Thirdly, using bodily or gender role criteria, individuals at the extremes of sexual polarity exhibit opposite levels of spatial ability. Thus, masculine men and feminine women show low spatial ability while androgynous men and women show high spatial ability (Maccoby and Jacklin, 1974; Hoyenga and Hoyenga, 1979). Fourthly, there is little difference between the sexes with respect to spatial abilities during the long prepubertal period during which the sexes are much alike as far as plasma hormone values are concerned, but in puberty a significant gender difference in spatial ability suddenly appears shortly before the time when plasma hormone levels differ maximally. Finally, spatial ability differences remain fairly stable throughout adulthood, just as do the sex hormone differences. We interpreted these relationships in terms of an ability-specific hormonal "optimal estrogen range" (OER) model (Nyborg, 1983). Furthermore, a growing body of recent evidence supports the notion that also gender role performance is under the influence of sex hormones. For example, human studies as well as animal studies indicate that dominance, assertiveness, aggression and parental behavior may depend on the pattern of prenatal hormone exposure (for review, see Hoyenga and Hoyenga, 1979). These and other observations recently led to a revision of our OER model in order to account for the general covariant pattern of development of personality and intellectual characteristics in men and women seen in Table I.

Both models are based on the fact that males are normally exposed both prenatally, perinatally and postpubertally to more circulating testosterone (T) than are women, whereas women are exposed to higher concentrations of 17β-estradiol (E_2). According to the general covariance (GC) model, circulating T and E_2 act as intervening variables coordinating the development of the gender-related traits seen in Table I. Thus, we assume these two factors to explain concerted prototypic development of the male and female gender pattern of personality and intelligence, in addition to differentiating the body sexually. Stated in its most radical form, the basic tenet of the GC model is that all gender-related characteristics — whether mental or somatic — develop harmoniously as a primary function of circulating sex hormones. Logically, then, adequate hormonal manipulation might be able to overrule whatever gender-differentiating power the sex chromosome complement may have on the phenotype. Furthermore, social impact is considered secondary to gonadal hormone effects. The GC model makes a distinction between the biochemically conditioned development of gender-related characteristics and their phenotypical expressions; the former is a function of

sex hormones while the latter can be inhibited or facilitated by various environmental means that may act via the sex hormones among other mechanisms.

The present paper has two aims: (1) to perform a comparative analysis of gender-related characteristics in hormonally different groups, and (2) to see to what extent the outcome of the comparison can be predicted by the GC model. Material concerning gender role and intellectual performance of persons with various hormonal disturbances and different karyotypes was collected and compared in order to see whether the male and the female patterns indicated in Table I vary with the hormones or with the karyotypes. Persons with a history of "normal male hormone exposure", along with individuals who were exposed to more circulating T than was usual for people with their chromosomal make-up, were pooled in a so-called "T/E" group, and compared to an "E/T" group consisting of persons with a history of "normal female hormone exposure", along with individuals having been exposed to more circulating E_2 than was usual for their chromosomal make-up.

LITERATURE, DATA AND METHODOLOGICAL PROBLEMS

Subjects

Data for inclusion in the comparative analysis were compiled from the literature and from the author's own files on the following 9 hormonally atypical groups (see Table II): (1) individuals with the adrenogenital syndrome (karyotype 46,XX and 46,XY: Money and Lewis, 1966; Ehrhardt and Money, 1967; Ehrhardt et al., 1968a,b; Lewis et al., 1968; Money and Ehrhardt, 1968, 1972; Ehrhardt, 1973, 1975; Perlman, 1973; Baker and Ehrhardt, 1974; Ehrhardt and Baker, 1974, 1975; Lev-Ran, 1974; McGuire and Omen, 1975; Money and Daléry, 1975; Money and Schwartz, 1975; Solomon and Schoen, 1975; Reinisch, 1976; Reinisch et al., 1979), (2) persons with progestin priming early in life (46,XX and 46,XY: Ehrhardt and Money, 1967; Money and Ehrhardt, 1968, 1972; Reinisch, 1976, 1977; Reinisch and Karow, 1977; Reinisch and Gandelman, 1978), (3) men with two Y chromosomes (47,XYY: Nielsen, 1969; Owen, 1972; Nielsen and Christensen, 1974; Nöel et al., 1974; Witkin et al., 1976; Nyborg and Nielsen, 1981b), (4) women with three X chromosomes (47,XXX: Kidd et al., 1963; Tennes et al., 1975), (5) men with two X chromosomes (47,XXY: Nielsen, 1969; Money and Ehrhardt, 1972; Theilgaard, 1972; Witkin et al., 1976), (6) men with protein deficiency leading to kwashiorkor syndrome (46,XY: Dawson, 1966, 1972), (7) individuals insensitive to androgen (46, XY: Money et al., 1968; Masica et al., 1969; Money and Ehrhardt, 1972; Perlman, 1973; Money and Ogunro, 1974), (8) individuals with estrogen priming early in life (46,XX and 46,XY: Dalton, 1968, 1976, 1981; Yalom et al., 1973; Zussman et al., 1975; Ehrhardt et al., 1977; Meyer-Bahlburg et al., 1977; Reinisch, 1977), and (9) women lacking some X chromosome material (45,X or mosaics: Schaffer, 1962; Money, 1964; Money and Alexander, 1966; Ehrhardt et al., 1970; Theilgaard, 1972; Nielsen et al., 1977; Baekgaard et al., 1978; Nyborg and Nielsen, 1979, 1981a). For general discussion of hormone–behavior relationships, see Reinisch (1976), Meyer-Bahlburg and Ehrhardt (1977), Ehrhardt and Meyer-Bahlburg (1979), Hoyenga and Hoyenga (1979) and Nyborg (1983).

These groups were categorized in accordance with whether they had a history of "usual high" or an "unusual high" (i.e. hypernormal) hormone level of T (the T/E group) for either endogenous (e.g. adrenogenital syndrome) or exogenous reasons (e.g. due to the effect on the fetus of androgen treatment of the pregnant woman), or of "usual high" or an "unusual high"

TABLE II

HORMONALLY ABNORMAL GROUPS CONSIDERED IN THE PAPER

Group *	*Karyotype*	*Origin of anomaly*	*Hormonal deviation*	*Phenotype*	*Treatment*
1. Adrenogenital syndrome (congenital adrenal hyperplasia)	46,XX 46,XY	Autosomal, recessive	Excessive adrenal androgenic output, pre- and postnatally	Virilized Normal	Cortisone ± surgery Cortisone
2. Prenatal progestin induction	46,XX 46,XY	Androgenic hormone treatment of mother during pregnancy	Prenatal androgen priming	Virilized Further masculinized	Surgery None
3. Men with double-Y chromosomes	47,XYY	Non-disjunction during meiosis in the father	Perhaps increased androgen levels and/or abnormal metabolism	Tall, masculine	—
4. Women with triple-X chromosomes	47,XXX	Non-disjunction during meiosis	Sometimes early menopause and menstrual irregularities	Delayed development	—
5. Men with double-X chromosomes (Klinefelter's syndrome)	47,XXY	Non-disjunction during meiosis	Low androgen output	Tall, small testes, feminized	Hormone therapy
6. Men with kwashiorkor syndrome	46,XY	Severe protein deficiency causing liver damage etc.	Elevated plasma estrogen values	Feminized	—
7. Androgen-insensitive persons	46,XY	X-linked, recessive	Receptors insensitive to androgen	Tall, little pubic hair, no internal sex organs, distinctively feminine	—
8. Prenatal estrogen induction	46,XX 46,XY	Estrogenic hormone treatment of mother during pregnancy	Prenatal estrogen priming	Normal Shorter, some genitally abnormal	—
9. Women lacking X-chromosome material (Turner's syndrome)	45,X and mosaics	Non-disjunction or other loss of X-chromosome material	Very low plasma hormone values	Short and sexually infantile	Hormone therapy

* For literature sources, refer to the text.

level of E_2 (the E/T group) relative to their chromosomal sex. Groups with hormonal variations within a physiologically more normal range were also included in the study. These non-clinical individuals were categorized hormonally according to their bodily appearance, because heavy masculinization depends on ample plasma T and feminization on the presence of E_2.

A number of important reservations must be made about the present survey. There is little consensus about how to define gender variables, and no metric scale exists for their measurement. The various authors' definitions of the variables were accepted on face value in the comparative analysis, as were the various approaches by which they chose to study them. Obviously, experimental studies of the effects of sex hormone variations in humans are not feasible for ethical reasons. The survey relies therefore mainly on the outcome of relatively few, small-scale clinical studies typically including less than 50 subjects each. Most of the observations reported here reached statistical significance, but in some cases positive trends in the direction expected from the results of other studies were accepted as well even if not tested for statistical significance because of too few subjects or because the measuring scale or the test applied precluded quantitative treatment. A further problem with some studies of prepubertal children is that they take the absence of significant hormone–intelligence relationships to mean that sex hormones do not influence intelligence. This conclusion is unwarranted, because the usual sex differences in intelligence typically do not appear before puberty anyhow. Other factors that tend to obscure hormone–behavior relationships if not taken into consideration are: that some children are more sensitive to hormone treatment than others as seen in differences in genital development; that treated individuals may be pooled despite different time of onset and length of treatment, and that dosage and type of hormones applied differed; and that prenatal sex hormone effects may differ radically from postnatal effects. Furthermore, it is the exception rather than the rule that details of medical treatment are given and that plasma sex hormone values were measured, especially in the early studies. But even if exact measures were at hand, they would probably tell only a small part of the story about the relationship between circulating sex hormone values and their biological effects, because plasma sex hormones may be bound, aromatized or degraded long or shortly before action. It can, accordingly, be misleading to draw conclusions from plasma values to central effects. In order to solve these problems via studies of hormonally abnormal individuals, we need large-scale, internationally coordinated, cross-disciplinary studies, in which the researchers use identical methods.

GENDER ROLE PERFORMANCE AND INTELLIGENCE

The T/E group is characterized by a masculine or an androgynous gender identity (see Table III). In general, T/E individuals show high self-reliance and physical energy expenditure but are not particularly aggressive. They appear to be object-oriented rather than person-oriented, and to show relatively little maternal interests. This characterization applies regardless of sex chromosomal make-up. Table IV indicates that the general IQ is variable in the T/E group, in that both high and low IQs are compatible with a T/E balance. With the exception of the first two masculine groups, spatial ability was high, and performance IQ scores were equal to or better than verbal IQ scores. Verbal ability was also variable between the groups. No systematic relationship between chromosomal gender and phenotypic traits was observed.

TABLE III

PERSONALITY CHARACTERISTICS IN THE TESTOSTERONE/ESTROGEN (T/E) GROUPS

Groups			*Gender identity*	*Gender role performance*					
Characteristics	*Chromosomal sex*	*Genital sex*		*Assertiveness, independence, self-reliance*	*Level of aggression*	*Physical energy expenditure*	*Prepubertal play preference*		*Maternal interests*
							Objects	*Persons*	
Men with extra Y material	47,XYY	♂	Masculine	?	+ +*	+ +	+	÷ ÷	÷ ÷
"Masculine" men**	46,XY	♂	Masculine	+ +	+ +	+ +	+ +	÷ ÷	÷ ÷
Adrenogenital syndrome	46,XX	♀ or mixed	Androgynous	+ +	+	+ +	+ +	÷ ÷	÷ ÷
	46,XY	♂	Masculine	+	+	+ +	+	÷	÷
Prenatal progestin induction	46,XX	♀ or mixed	Androgynous	+ +	+	+ +	+ +	÷ ÷	÷ ÷
	46,XY	♂	Masculine	+	+	+	+	÷	÷
"Normal" men	46,XY	♂	Masculine	+	+	+	+	÷	÷
Androgynous men	46,XY	♂	Androgynous	+ +	+	+	+ +	÷ ÷	÷
Androgynous women	46,XX	♀	Androgynous	+ +	+	+	+ +	÷ ÷	÷ ÷

+ +, unusually high for their sex; +, high "normal"; ÷, low "normal"; ÷ ÷, unusually low for their sex; ?, not determined or disputable.

* Perhaps impulsive rather than aggressive.

** For definition, see Broverman et al. (1964); Petersen (1976); for discussion, see Maccoby and Jacklin (1974); Hoyenga and Hoyenga (1979).

TABLE IV

INTELLECTUAL CHARACTERISTICS IN THE TESTOSTERONE/ESTROGEN (T/E) GROUPS

Groups			*Gender identity*	*Intellectual performance*			
Characteristics	*Chromosomal sex*	*Genital sex*		*General IQ*	*Spatial ability*	*Verbal ability*	*Verbal (V) — performance (P) score relationship*
Men with extra Y material	47,XYY	♂	Masculine	÷ ÷	÷ ÷	÷ ÷	?
"Masculine" men *	46,XY	♂	Masculine	+	÷ ÷	+ +	V > P
Adrenogenital syndrome	46,XX	♀ or mixed	Androgynous	+ +	+ +	+ +	P = V **
	46,XY	♂	Masculine	+ +	+ +	+ +	P = V **
Prenatal progestin induction	46,XX	♀ or mixed	Androgynous	+ +	+ +	+	P = V
	46,XY	♂	Masculine	+ +	+	+	P > V
"Normal" men	46,XY	♂	Masculine	+	+	÷	P > V
Androgynous men	46,XY	♂	Androgynous	+	+ +	+	P > V
Androgynous women	46,XX	♀	Androgynous	+	+ +	+	P > V

+ +, unusually high for their sex; +, high "normal"; ÷, low "normal"; ÷ ÷, unusually low for their sex; ?, not determined or disputable.

* See ** in Table III.

** However, very high IQ range: V > P.

TABLE V

PERSONALITY CHARACTERISTICS IN THE ESTROGEN/TESTOSTERONE (E/T) GROUPS

Groups			*Gender identity*	*Gender role performance*					
Characteristics	*Chromosomal sex*	*Genital sex*		*Assertiveness, independence, self-reliance*	*Level of aggression*	*Physical energy expenditure*	*Prepubertal play preference*		*Maternal interests*
							Objects	*Persons*	
"Normal" women	46,XX	♀	Feminine	÷	÷	÷	÷	+	+
"Feminine" women *	46,XX	♀	Feminine	÷ ÷	÷ ÷	÷ ÷	÷ ÷	+ +	+ +
Women with triple-X syndrome	47,XXX	♀	Feminine	÷ ÷	?	?	?	?	?
Men with Klinefelter's syndrome	47,XXY	♂	Demasculinized	÷ ÷	?	÷ ÷	?	?	?
Men with kwashiorkor syndrome	46,XY	♂	Demasculinized	?	÷ ÷	÷ ÷	?	?	?
Androgen-insensitive persons	46,XY	♀	Feminine	÷ ÷	÷ ÷	÷ ÷	÷ ÷	+ +	+ +
Prenatal estrogen induction	46,XX	♀	Feminine	÷	÷ ÷	÷	÷	+	+
	46,XY	♂	Demasculinized	÷ ÷	÷ ÷	÷ ÷	÷ ÷	+ +	?
Women with Turner's syndrome	45,X and mosaics	♀	Feminine	÷ ÷	÷ ÷	÷ ÷	÷ ÷	+ +	+ +

+ +, unusually high for their sex; +, high "normal"; ÷, low "normal"; ÷ ÷, unusually low for their sex; ?, not determined or disputable.

* See ** in Table III.

TABLE VI

INTELLECTUAL CHARACTERISTICS IN THE ESTROGEN/TESTOSTERONE (E/T) GROUPS

Groups			*Gender identity*	*Intellectual performance*			
Characteristics	*Chromosomal sex*	*Genital sex*		*General IQ*	*Spatial ability*	*Verbal ability*	*Verbal (V) — Performance (P) score relationship*
"Normal" women	46,XX	♀	Feminine	+	÷	+	V > P
"Feminine" women	46,XX	♀	Feminine	+	÷ ÷	?	?
Women with triple-X syndrome	47,XXX	♀	Feminine	÷ ÷	?	?	?
Men with Klinefelter's syndrome	47,XXY	♂	Demasculinized	÷ ÷	÷ ÷	÷ ÷	?
Men with kwashiorkor syndrome	46,XY	♂	Demasculinized	÷ ÷	÷ ÷	+ +	V > P
Androgen-insensitive persons	46,XY	♀	Feminine	+	÷	+	V > P
Prenatal estrogen induction	46,XX	♀	Feminine	+	÷	+	V > P
	46,XY	♂	Demasculinized	+	÷ ÷	+ +	V > P
Women with Turner's syndrome	45,X and mosaics	♀	Feminine	+	÷ ÷	+	V > P

+ +, unusually high for their sex; +, high "normal"; ÷ low "normal"; ÷ ÷, unusually low for their sex; ?, not determined or disputable.

In the E/T group a "feminine" or a demasculinized gender identity predominates (see Table V). Self-reliance tends to be low, as do aggression and physical energy expenditure. Object play preference is low, while preference for playing with dolls and other "girlish" toys tends to go together with maternal interests in the E/T group. There is a downward trend in spatial ability in the E/T groups, whereas verbal IQ scores tend to exceed performance IQ scores (see Table VI).

THE GENERAL COVARIANCE MODEL FOR GENDER DEVELOPMENT

The major pattern that emerges from the findings is that the majority of T/E individuals exhibited the male gender repertoire, whereas most E/T individuals had the female gender repertoire. These observations support the main predictions of the GC model, namely that sex hormones act as the primary determinants of whether a prototypic male, female, or mixed mental development will take place, whereas the karyotype is a poor predictor of gender development. I believe that the observations point to the need for a re-evaluation of research into gender differences, calling for an analysis that is based neither on traditional environmentalist nor on available genetic theories. Such an analysis should be able to specify and quantify the major factors responsible for gender differentiation; primarily those biochemical variables that can account for the fact that gender-related traits show continuous, overlapping distributions that tend to cluster around the male and the female prototypic developmental pathways (summarized in Table I) despite considerable environmental and genetic variation. Any new analysis must account for the causal chain of the biochemical variables, for stability as well as for flexibility in gender development, and should have individuals rather than statistical group means as its target. Finally, a new analysis has to address the problem why gender differences appear at all.

The GC model is conceived in accordance with these requirements, but may be too simple to meet them fully. The GC hypothesis views sex hormones as the fingers on the physiological switchboard for gender differentiation. More specifically, the GC model assigns to sex hormones the ultimate biochemical responsibility for producing not only gender-related differences in sensory modality priorities, but also in interests, cognitive style, gender role differences, physical energy expenditure, androgenization of the muscles and fat distribution, and in other gender-related somatic characteristics. All these traits would depend on whether or not the sex hormones were present at the right place, and at the right time, and in the right amount.

But what is the right place, right time, and the right amount for sex hormone actions? How can biochemical signals be translated into gender-specific behavioral patterns, and what is the purpose of this translation? Which mechanisms mediate the processes, and what specific endogenous and exogenous factors influence the hormonal systems? What keeps the system in balance and how great is its flexibility? Unfortunately, there are no satisfactory answers to these vital questions today, but systematizing the fragments of relevant information already available may enable us to formulate testable hypotheses.

Only those aspects of the GC model which are relevant for understanding how the gender repertoires described in this paper became a reality will be discussed.

The right place

Let us assume that the sites of sex hormone uptake in the brain correspond to their site of biological action. High uptake of sex hormones occurs in the preoptic-hypothalamic and the limbic systems (McEwen, 1976). This is especially interesting from a cognitive point of view, because it might indicate that traditional explanations for gender-related differences in verbal–spatial ability based upon left–right brain lateralization fail to incorporate an important cortical-*subcortical* dimension. However, there are also sex hormone receptors in other parts of the brain, and our knowledge about their significance depends to a large extent on our techniques for detecting the effects of uptake of circulating gonadal hormones in these areas. Furthermore, sex hormones interact both peripherally with each other, and centrally with neurotransmitters (Dörner, 1978), where they also influence pre- and postsynaptic membrane characteristics (Moss and Dudley, 1984). E_2 is biologically very active in the cell nucleus, whereas T seems effective in the nucleus only if aromatized to E_2. According to the GC model, E_2 is the most important hormone for gender-related actions in the central nervous system.

The right time

The GC model allows some tentative inferences to be made about when and how sex hormones exert their effects on phenotypical characteristics. Gender role traits such as physical energy expenditure, aggression and "nurturant" behavior appear long before puberty (e.g. Brindley et al., 1973), and can accordingly be ascribed to relatively permanent, prenatal organizational actions of sex hormones which do not need pubertal activation. In contrast, gender differences in the intellectual pattern do not usually appear before puberty, and probably they depend on pubertal activation in addition to early organizational effects.

The right amount

Peaks in T levels occur prenatally as well as perinatally in boys. Thereafter, differences are scarcely detectable in plasma sex hormone levels between boys and girls during the prepubertal period using current radioimmunoassay techniques. After puberty, however, boys produce about 10 times more T than do girls, whereas girls have about 3 times more plasma E_2 than do boys (moreover, their E_2 levels vary with the menstrual cycle). The GC model supposes that these average values result in the typical male or female gender repertoires. However, in order to account for individual variations in prototypic development, the GC model is designed explicitly as a person-specific, threshold model. Thus, the effect of E_2 is assumed to be curvilinearly related to spatial ability as illustrated in Fig. 1. Letters A–D and E–H in Fig. 1 signify two groups of individuals who are differently sensitized to sex hormones (and/or have a different brain organization) during the pertinent period. Letters A, B, C, and D represent in this case males who had been prenatally primed with T, and who therefore are relatively insensitive to their comparatively low levels of circulating E_2, and who scored just below their optimum level for spatial ability. The GC model assumes that a further increase in E_2 will enhance their spatial ability, and will influence their gender role performance and their body development in an androgynous direction. This explanation can account for covariant personality, intellectual, and somatic development. The curvilinear relationship shown in Fig. 1 predicts that increases in E_2 above the optimum level will inhibit the expression of spatial ability. This can explain the low spatial ability and the gender

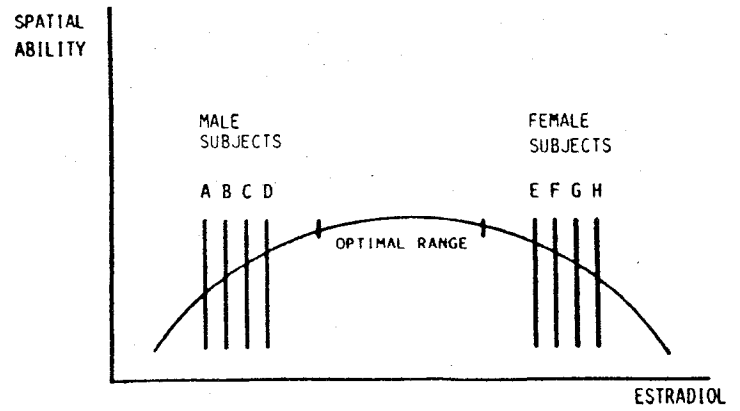

Fig. 1. The "optimal estrogen range" (OER) model for hormone-spatial ability relationship. (Modified from Nyborg, 1983.)

development both in demasculinized men with Klinefelter's syndrome and in men suffering from the kwashiorkor syndrome. These same principles may also explain what happens in distinctively "masculine" men, i.e. they aromatize part of their high plasma T to E_2 centrally and thereby exceed the optimal E_2 range, while at the same time being bodily androgenized, perhaps by direct action of T on somatic tissues (for details, see Nyborg, 1983). Letters E, F, G, and H here represent females who have relatively high plasma E_2 values and who are situated somewhat below their optimum level for spatial ability. The GC model predicts that a prolonged decrease in their E_2 levels will enhance their spatial ability, influence their gender performance in an androgynous direction, and enable T to androgenize their body while not being much antagonized peripherally by E_2. A transient decrease in their E_2 levels will also temporarily enhance their spatial ability, but will not influence their gender identity or body markedly, thus explaining why spatial ability is high in the estrogen-low phases of the menstrual cycle. On the other hand, a further increase in E_2 would lead to even more repressed spatial ability, in addition to feminizing their performance and their bodies. Thus, a curvilinear model can explain why spatial ability is optimal when gender identity and gender role performance are most alike in the two sexes, and why spatial ability will be low both in distinctively feminine women and in masculine men. The model also provides a basis for understanding the behavioral status of various hormonally deviant groups. Furthermore, the model explains some observations in Tables III, IV, V, and VI, that apparently run counter to what would have been expected from a simple T/E versus E/T classification of subjects. This points to important methodological constraints in traditional studies of gender differences. Identical changes in plasma sex hormone levels may lead to facilitation of personality and intellectual parameters in one subject but to inhibition of these same parameters in another subject. The GC model is person-specific and this makes it more useful than traditional dichotomic group mean approaches by allowing for individual evaluation of quantitative variation on the biochemical as well as on the behavioral side.

Mediating mechanisms

The GC model assumes at least 3 interrelated avenues for the effects of sex hormones: (1) by prenatal priming of sensitivity to pubertal sex hormones, (2) by early permanent "organizational" effects on developing neural tissues, and (3) by transient "activational" effects on the functioning of the mature nervous tissues. With regard to prenatal effects, Döhler and Hancke (1978) have suggested that absence of E_2 in the fetus will leave its brain

sexually undifferentiated, that a moderate amount of E_2 will feminize its brain, and that large amounts of E_2 lead to masculinization (see Döhler et al., 1984). Massive amounts of E_2, however, have a neurotoxic effect leading to a less differentiated status of brain tissues. It is likely that E_2 exerts some of its growth-promoting effects on neural tissues (Toran-Allerand, 1976). This can partly explain gender-typing of the brain by "organizing" the specific neuronal circuits that are believed to be essential for the appearance of gender-related behavior. As E_2 influences genomic expression it modulates the production of proteins considered essential for neural development, so that the effects of E_2 in the central nervous system may show up long after the hormone itself has disappeared.

Sex hormones may also have rapid effects within the central nervous system. For example, E_2 alters the metabolism of neurotransmitters and also influences their actions by changing pre- and/or postsynaptic membrane characteristics (see Hutchison and Steimer, 1984; Moss and Dudley, 1984). Much more needs to be learned about antagonistic and agonistic effects of sex hormones on the CNS, but promising advances have already been made, as a number of chapters in this book testify.

Hormone–behavior translation

Like genes, hormones cannot produce behavior directly. The GC model states, in accordance with Hoyenga and Hoyenga (1979), that sex hormones promote gender-related tendencies to experience and to behave relatively consistently in different situations. Such tendencies have appeared in the past under names such as "drives", "subconscious motives", "traits", and "sex-stereotyped behavioral response patterns". According to the GC model, sex hormones color perceptions so that men and women put a different emphasis on given aspects of the surroundings, as with gender-related focus of interests. More specifically, the GC model assigns the sex hormones the biochemical responsibility for inducing gender-related differences in the way sounds and visual information are processed by the brain (e.g. McGuinness, 1974, 1976; McGuinness and Lewis, 1976). Gonadal hormones also influence whether visual or somatosensory information will be taken as a primary reference for determining the perception of the upright in a visual-vestibular (-somesthetic) conflict situation such as the rod-and-frame task (Nyborg, 1977; Nyborg and Nielsen, 1981a). Sex hormones can promote gender differences by the way perceptual input (verbal-communicative, social, or visuo-spatial information) is handled intellectually. In this way different tasks may be differentially rewarding to men and women, and may reinforce the expression of gender differences. However, the GC model also assumes that sex hormones can influence even more subtle behavioral patterns, such as toy preferences, and parents learn readily which toys their child prefers most, and act in accordance with this knowledge. It follows from the GC model that differential gonadal hormone conditioning is the prime mechanism for conservation of social institutions that guarantee, for better or for worse, the continuation of gender-related differences in child rearing. Obviously, this notion runs directly against prevailing environmentalist explanations of gender differences. According to the GC model, we are neither "tabula rasa" abstractions thrown helplessly at the forces of sex-typing, nor are we predetermined biological beings developing our genetic potentials in a cultural vacuum. We are more specialized than behaviorists believe, but less specialized than radical adherents to instinct theory claim. According to the GC model, sex hormones mediate behavioral readiness to perceive and respond in certain ways. Sex hormone-conditioned responses can, of course, be either positively or negatively reinforced according to cultural prescriptions.

Stability–flexibility of the development of prototypic gender patterns

The GC model assigns to the sex hormones the role of being important mediators between the organism and its environment. Although genes are crucial for sex hormone production, it is now generally acknowledged that sex hormone production is also influenced markedly by environmental factors such as nutrition, stress, experience of social pressure (dominance–submission), and the presence of sexually attractive partners. Adaptability of hormonal production to environmental requirements is secured in this way. A dynamic balance between genetic and environmental factors is essential for adequate gender behavior to appear. It is of interest to know whether certain gender-related parameters remain stable when environmentally induced changes in sex hormone parameters occur. If gender role performance is geared mainly by the prenatal "organizational" effect, then it will show only limited flexibility to environmental impacts. On the other hand, intellectual differences that also require pubertal "activation" can be expected to show greater flexibility to environmentally induced postpubertal hormonal changes.

The "why" of hormonal actions

It is of interest to speculate whether any evolutionary advantages might be associated with having sex hormones bear the major responsibility for gender-linked brain differences. It is generally acknowledged that the sexual mode of reproduction confers an evolutionary advantage in the form of increased genetic variability. The different gender roles must have been subjected to different selective pressures through time. Perhaps the constellation of a feminine behavioral repertoire (a low level of aggression and physical energy expenditure, a "preference for persons" and for early rehearsal of a maternal role, combined with high communicative skills) has optimized the reproductive success of women faced with successive child births, feeding and rearing of their offspring in primitive times. Perhaps the constellation of a "male" behavioral repertoire (independent, energetic, a certain amount of aggression, and high spatial ability) conferred men with an advantage in hunting and warfare, while not being unduly distracted by maternal interests. Thus, the gender roles mediated by sex hormones may have had survival value in primitive societies, and may thereby have become built into human behavior. According to the GC model, the genetic potentiality for developing male, female, or mixed behavioral repertoires exists in both sexes, and the phenotypic actualization of these potentials depends primarily on the kind of sex hormone signals available to the physiological switchboard.

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