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THE BIOLOGICAL BASES OF SEX DIFFERENCES.

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THE BIOLOGICAL BASES OF SEX DIFFERENCES

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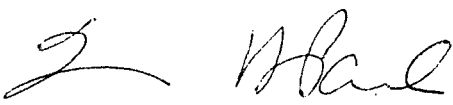
ELIZABETH DINSMORE

A dissertation submitted to the Graduate
Faculty in Psychology in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy, The City University
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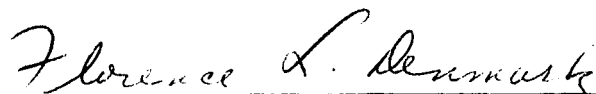
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This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

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PREFACE

To the student of sex differences who wants to know about the unalterable aspects of sexuality, the social science literature offers little of substance. The question of origins is of crucial importance, however, in this historical period of rising consciousness of, and challenges to, traditional sex roles. Therefore, it seems both timely and necessary to turn to the fields of endocrinology and genetics for further direction.

Interest in the biology of sex determination and differentiation has increased greatly during the past two decades, largely as a result of new methods of sex chromosome analysis and advances in our understanding of the central role of fetal sex hormones in differentiation. Although knowledge of these processes is still on a descriptive level, it has been sufficient to generate a rudimentary theory of sex differentiation and a related, coherent body of research on both animals and humans. This "organizational theory" proposes that areas of the central nervous system involved in sexual behavior becomes differentiated and "fixed" under the influence of sex hormones during the fetal (or postnatal) period.

The organizational theory finds its major proponent in an endocrinologist, Ward (1974), who has studied the behavioral

responses of rats to alterations of the early hormonal environment. Its principles have been applied also to the study of gender role development in human intersexes by the psychologists, Money and Ehrhardt (1972) and others.

These data, as well as contributions from developmental psychology, the psychology of sex differences, cross-cultural inquiry, and ethology provide the data for the natural experiment which is the topic of this paper.

The central focus of this paper will be an assessment of the validity of the organizational theory. Chapter I provides a review of non-biological research on gender role differences. This should serve both to inform the reader of the current findings on sex differences and to raise the issue of origins from an evolutionary viewpoint. Chapter II describes the current status of knowledge of sex determination and differentiation and Chapter III assesses the organizational theory as applied to rats. The following chapters extend the inquiry to human intersexes. Principles of gender role development in both normals and intersexes are examined and discussed. A more detailed test of the theory is then made by analyzing the following characteristics in androgenized and unandrogenized intersexes: (1) Tomboyism; (2) Maternalism; (3) Sexual response and eroticism; and (4) Intellectual abilities. Finally, the role of the sex chromosomes is discussed in Chapter VI.

The use of the natural experiment method necessarily involves many problems of experimental control and of general-

izability, which will be discussed in the text. The reader should note at the outset that the data permit conclusions only about gender role, not about gender identity. Although Money and Ehrhardt (1972) use these terms interchangeably (see p. 46), in fact, their data does not probe the private, intrapsychic aspects of gender identity. Secondly, although sex hormones and sex chromosomes appear to be the agents of phenotypic sex differentiation, our understanding of their mode of action is so rudimentary that more subtle and complex processes cannot be discounted.

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CHAPTER I

THE CONTRIBUTION OF NON-BIOLOGICAL APPROACHES TO THE STUDY OF SEX DIFFERENCES

1. Introduction

In recent years, the question of what real differences exist between the sexes has been a subject of intense research. The idea that biology determined the kinds of social behavior and intellectual abilities reflected in cultural stereotypes was a popular one, even though the mechanisms of sexual determination and differentiation were largely unknown. In the absence of biological data, researchers approached this question in three ways.

2. The Contribution of Empirical Research

Most of the empirical psychological research began as a variant of the study of individual differences and so adopted the prevailing method of recording mean differences in behavior, temperament, and capacity (Tyler, 1958). After a half-century of this research few consistent and dramatic sex differences have been identified. Maccoby and Jacklin (1974) made an exhaustive survey of the sex difference literature published between 1967 and 1973 which extended the earlier review of Maccoby (1966). They identified the following

as the sole, "fairly well established" differences:

1. Girls are superior in Verbal Ability (VA), including not only tasks of productivity, fluency, and repetition but also "high level tasks" such as creativity, comprehension, and analysis. This difference obtains only after age eleven, though girls tend to excel in these skills from the age of four. However, the sexes differ by one-quarter of one Standard Deviation, indicating considerable overlap.
2. Boys excell in Visual-Spatial Ability (VSA), both analytic and non-analytic, but not in other spatial skills. Again, the difference appears only after age twelve and amounts to about two-fifths of one Standard Deviation.
3. Boys excell in Mathematical Ability (MA) after early adolescence. The difference is estimated to be less than that found for VSA.
4. Boys are more aggressive, physically and verbally, from the age of two. Aggression declines with age and is more often directed at other boys.

The following were cited as "unfounded beliefs":

- (1) Girls are more "social"; (2) Girls are more "susceptible"; (3) Girls have lower self-esteem; (4) Girls are better at rote and repetitive tasks while boys excell in higher level cognitive tasks and the inhibition of previously-learned responses; (5) Boys are more "analytic" (this is true only for VSA); (6) Girls are more affected by heredity, boys by environment (male identical twins are more similar than

female identical twins but the sexes do not differ in their resemblance to their parents); (7) Girls lack achievement motivation; and (8) Girls are "auditory", boys are "visual".

Finally, evidence supporting the following beliefs was either ambiguous or insufficient: (1) Girls have more tactile sensitivity; (2) Girls are more timid, fearful, and anxious (differences are found with ratings but not with observational studies); (3) Boys have a higher activity level; (4) Boys are more competitive; (5) Boys are more dominant; (6) Girls are more compliant; (7) Girls are more nurturant and "maternal"; and (8) Girls are more passive (no differences are found in exploration of a novel environment, response to social situations, or withdrawal).

3. The Clinical Contribution

The clinical and philosophical approaches probed the nature of sex differences by reference to the workings of the Unconscious, thus dealing with the kinds of proof afforded by clinical data and phenomenological observation. Erikson's notion of masculine and feminine modes as expressions of bodily structure and Freud's concepts of penis envy, dimorphic moral and intellectual capacities provide examples of this type of approach. However, these differences--if they are in fact characteristic of the sexes--may be the result of learning. Roy Schaefer (1974) and others have urged reconsideration of these concepts.

4. The Cross-Cultural and Primate Research

The third approach to the study of sex differences was through cross-cultural and primate research. These provided the basis for a kind of biological theory based upon the concept of biological evolution. In brief, this theory views sex differences as the expressions of structural and functional adaptations over the course of human history (and prehistory). As applied to reproductive structure and function the structural explanation is widely accepted. It is not so clear, however, that intellectual capacities, personality traits, and social behaviors not directly connected to reproduction are products of biological evolution.

Proponents of the biological theory point out that regularities of social organization and sex role content may be found both historically and cross-culturally, attesting to the adaptiveness of these forms. The existence of these forms, in turn, is thought to reflect biologically-based "predispositions" or "biases" for universal masculine and feminine modes of behavior (Phoenix, 1974; Hamburg, 1974; Diamond, 1968). Certainly, as regards broad structural regularities, this view has merit. It is a common observation that mammalian social structures are universally organized around the basic reproductive function, resulting in two structural characteristics of societies: (1) the sexual division of labor; and (2) the socialization of the sexes into appropriate gender roles.

However, gender roles vary significantly among cultures. Many examples can be found of the wide variation in gender role content, Mead's (1935) description of sex roles in three New Guinea societies being the most striking (Erikson, 1958; Money and Ehrhardt, 1972; D'Andrade, 1966). Arapesh men and women were unaggressive and cooperative. The Mundugumor, in contrast, were aggressive and "positively sexed." Among the Tchambuli the roles considered normal in male-dominated societies were reversed. Thus, American men would appear "feminine" to the Tchambuli and American women would seem "unfeminine" to the Mundugumor.

Modern genetic theory, on the other hand, emphasizes that "roles have nothing to do with genes" (R. Fehnmann, 1975). The unique and basic characteristic of human evolution, according to Dobzhansky (1973) is human adaptiveness: "to adapt to new environments, mankind changes mainly its cultural inheritance, rather than its genes, as other organisms do" (p. 93). The time span required for biological evolution due to selective pressures is estimated at hundreds of thousands of years. Fehnmann (1975) has observed that technological change since the Roman era is far greater than can be accounted for by genetic changes. Thus, in order to support a genetic basis for sexual dimorphism, it must be demonstrated that significant alterations in sex role occurred over a time span of at least 100,000 years.

The biological theorists hold that, although variations in sex role are found in a significant proportion of cultures,

consistent sexual dimorphisms can be traced from primates through early hominids to present-day peoples. Early primate research, for example, viewed the male as the hub of the social structure. Tiger and Fox (cited in Lancaster, 1973) trace sexual dimorphisms to the original primate division of labor. They propose that the male dominance hierarchy and strong male-male bonds provided the primate group with a stable social organization, sex, and protection. The female role, in their view, was limited to child-rearing.

More recent studies challenge this view of our primate heritage as incomplete and misleading. Lancaster (1973) proposes that social organization is provided by the "matrifocal core" based upon the mother and her offspring: "Both field and captive studies suggest that it is the matrifocal core that provides a primate group with stability and continuity" (Lancaster, 1973, p. 34). These matrifocal units appear to be ranked in the dominance system of the whole troop, suggesting that the male dominance hierarchy may be the result not of individual male characteristics alone but also of the male's "mother's identification and its birth order among its siblings" (Lancaster, 1973, p. 34). Goodall (1971) suggested the same in her chimp research, but she had not been able to identify the matrilineal inheritance of the original chimps.

The existence of the matrifocal subunit was not evident in studies which restricted observation to adult males and to shorter periods of time. Lancaster believes

that ranking in units will be found in all terrestrial primate when long-term observation is done. At present these units have been identified in many species. The discovery of the matrifocal unit suggests that our primate heritage included two, complementary sources of power, the female's power being less conspicuous than the male's but equally influential in the survival of the troop.

Another sexual dimorphic behavior--the stereotype of the sexual privileges of the most dominant male--takes on a different meaning when seen from the female point of view. According to Lancaster (1973) "males are a resource in her environment which she may use to further the survival of herself and her offspring" (p. 35). Among arboreal primates, where protection is rarely necessary, a polygynous mating system is the norm since only one male is needed to impregnate females. Among terrestrial groups, on the other hand, the male role expands to include group protection and the mating system correspondingly expands to bring in other males. Eisenberg suggests that the number of males in a troop "depends upon the advantage of their presence to the reproducing females" (Cited in Lancaster, 1973, p. 35).

Sex differences in mobility, moreover, carry different implications when viewed in the female perspective. Even highly territorial males tend to leave their groups at puberty, while females rarely do so. When an adult female does leave it is probably to establish a new troop, since she often takes

all her offspring with her. Terrestrial and semi-terrestrial primates have specific, large home ranges through which they wander in search of food. Some observers of the wandering troop--which is led by some adult males followed by a group of adult females, other males, and infants--have assumed that the males in the lead were the dominant males, making decisions about troop movement and protecting the troop. Rowell (cited in Lancaster, 1973) has proposed, however, that the decisions come from the old females in the center who, by virtue of their long experience in the home range, most likely know the resources of the area. The males in the center group are probably the brothers and sons of "the older females of the dominant genealogies that form the physical and social core of the group" (p. 35).

Finally, females have been observed to form bonds based upon these units. For example, they may form coalitions against individual males in dominance encounters over food or infants. (These encounters do not alter male rankings but do limit the autocracy of dominant males.) Groups of females, furthermore, have been observed to follow daily routines for long periods in the absence of a male, and to continue undisrupted when a male joins the group.

Thus the primate female heritage includes traits of decision-making based upon memory and organization; adult female bonds promoting the organization of daily routines and mutual aid and protection against dominant males; and leadership and influence in troop affairs--all based upon

the fortuitous factor of reproductive difference.

Among societies extant today the majority are organized around males. The evidence to follow supports the view that this pattern is of recent origin and represents a modification of culture rather than biology.

According to traditional theory, it was the male role of hunting that achieved the evolutionary step from superior monkey to man and laid the basis for the organization of societies around males. In fact, Goodall (1971) has observed a modified type of hunting among chimpanzees as well as meat-eating and tool-making--all of which were thought previously to be distinctly human. The only unique human characteristic which remains is language, which Gray and Buffery (cited in Hamburg, 1974) propose may have developed in females through the mother-infant bond. In any case, it is during the evolution of "Man the Hunter" that the "physiological and behavioral adaptations that enhance sexual dimorphism and the sexual division of labor" are thought to have occurred (Hamburg, 1974, p. 378).

Stern et al (1974) suggest, however, that the male hunting role was adaptive because of its contribution to social cohesion and stability through maintenance of a stable dominance hierarchy. This possibility is supported by two observations. Firstly, present-day hunting and gathering peoples, have no descent groups and relations among group members are cooperative. Secondly, though it was previously

thought that the selective pressure for hunting was due to the adaptive value of meat protein in the human diet, it appears that a major source of food supply of these peoples was provided by the females; that is, small game and gathered foods.

It was probably not until human groups became settled in agriculture with cattle and animal husbandry some five or six thousand years ago that social organization--that is, descent group and residence--became centered around males. It was at this time that economic capital came into use, and D'Andrade (1966) has noted that male-centered social organization is importantly related to "both economic capital and the sex bias in the use and control of this capital" (p. 184). In addition, sex typing is more extreme and extensive in cultures characterized by hierarchial authority relationships of the patriarchal type. These arose with the establishment of autocratic agrarian states or kingdoms, and were not characteristic of earlier tribal systems (D'Andrade, 1966). According to Barry, et al (cited in D'Andrade, 1966) large sex differences are found in "an economy that places a high premium on the superior strength and superior development of motor skills requiring strength, which characterizes the male" (p. 330). An emphasis on male skills can only exist in a society where the female is no longer able to contribute importantly to subsistence.

Hamburg (1974) links current sex-role definitions to

those postulated for early hominid and present-day "primitive" societies. However, of the "competency" cluster attributed to males, most of the qualities are seen in primate and early human females: independence, activity, competitiveness, decisiveness, self-confidence, ambition, and leadership. Her style was merely less conspicuous. The sexes have always been interdependent; the dependency characteristic of females in male-dominated societies probably arose from their inability to provide sustenance for themselves and their offspring without the aid of the male, that is, in societies where economic capital became the primary source of sustenance. Thus, it seems likely that current sex role stereotypes have a recent history relative to the time span of human history and are correspondingly based upon cultural evolutionary, rather than biological evolutionary factors.

5. Conclusions

The evidence reviewed in this chapter permits no conclusions about direct biological bases for sexual differences. The indirect effects of biology, on the other hand, are evident: as the means of subsistence changed, sex roles were modified. At first, males and females had separate, overlapping spheres of power--females by virtue of the mother-infant bond which formed the basis of social organization and males by virtue of their dominance hierarchy which provided the social group with protection and stability (by reducing intragroup aggression). In the development of

human hunting and gathering societies, peoples evolved closer interpersonal bonds and habits of cooperation and sharing. As peoples became agriculture and economic capital brought the concept of private property, the female was less able to contribute to subsistence because of her lack of mobility and strength due to childbearing and rearing. Those biological factors which once formed the basis of feminine power and independence--her susceptibility to repeated pregnancy and relative lack of mobility and muscular strength--became the source of her submission and dependency.

However, the female is not at a disadvantage in technological societies, which place a premium on intelligence rather than muscular strength. The development of safe and effective birth control has freed her from repeated childbearing--a change which undoubtedly constitutes a major turning point in human cultural evolution, if not in biological evolution. The increasing movement toward international interdependence requires an awakening of the "feminine" priorities of life over death, dialogue over force, cooperation over competition.

If our stereotypes of sexual differences are true reflections of biological evolution, on the other hand, then major modifications in behavior cannot be expected. The evidence to follow supports the view that human sexual dimorphisms are largely expressions of cultural evolutionary processes based upon a reproductive pattern that was in-

capable of modification until the development of effective birth control during the last century. The process of gender role development, seen in this context, is one in which certain behaviors, attitudes, and abilities are selectively reinforced in order that the child adapt to his particular culture.

CHAPTER II

SEXUAL DIFFERENTIATION OF THE PHENOTYPE

1. Introduction

If sexual dimorphisms are reflections of biological evolution the potentialities for masculine and feminine behavior must be established by those biological factors which are responsible for the sexual differentiation of the phenotype. Detailed reviews of the physiology of sexual differentiation are found in Federman (1967), Mittwoch (1967), and Money and Ehrhardt (1972). The present description presents only the basic facts concerning the process of sexual differentiation.

2. Sexual Differentiation of the Reproductive System

Our knowledge of the laws by which sexual differentiation of the reproductive anatomy occurs is still at the descriptive level. Sex determination in the strict biological sense occurs at conception when the nuclei of egg and sperm combine to reinstate the full complement of 46 chromosomes. The egg carries an X sex chromosome along with 22 autosomes (which are identical in the sexes); the sperm carries either an X or a Y sex chromosome and 22 autosomes. The pairing of XX produces a genetic female; XY produces a genetic male.

However, the zygote is not properly male or female but only potentially so (Crew, 1965). As Sinott et al (1958) state: "It would seem that the function of the sex chromosomes in man is to switch the embryo into one or the other channel of sexual development" (p. 306). The role of the sex chromosomes will be considered in depth in Chapter VI.

Indeed, regardless of sex chromosome constitution, the primordial Anlagen of reproductive anatomy are identical in the sexes. Phenotypic differentiation occurs in three stages. First, the indifferent gonad becomes either an ovary or a testis depending on the presence of XX or XY sex chromosome constitution, respectively. Thereafter the role of the sex chromosomes is eclipsed by the sex hormones, which in vertebrates mediate the effects of genetic sex on the phenotype (Mittwoch, 1967). In both sexes, the same pair of primordial duct systems are laid down in the early weeks. The ducts differentiate during the second stage, which occurs between 9-1/2 and 12-1/2 weeks. In the genetic male, the testis secretes various androgens--mainly testosterone--"as a matter of course" (Mittwoch, 1967). These induce the development of the Wolffian ducts into vas deferens, seminal vesicles, and ejaculatory ducts while a locally-acting, nonhormonal Mullerian Inhibiting Substance (MIS)--also produced by the testis--causes the Mullerian structures to regress and atrophy. In the genetic female (or in the fetus which has at least one X sex chromosome, such as X,0) it is the absence of these testicular secre-

tions which allows the Mullerians to proliferate into uterus, fallopian tubes, and upper two-thirds of the vagina and the Wolffians to diminish and atrophy. The ovaries and the adrenal cortex also produce androgens but in relatively small amounts and possibly only after the thirteenth week when ductal differentiation has been completed (Zaaijer and Price, 1971). In any case, the external female phenotype will develop in the absence of all gonadal hormones since X,0 (Turner's Syndrome) genotypes develop as females despite the absence of gonads.¹

The third stage of differentiation completes the external genitals. Until the eighth week, both sexes possess identical Anlagen: the genital tubercle, urogenital split, uretho-labial folds, and labioscrotal swellings (Money and Ehrhardt, 1972). The tubercle forms the glans and corpus cavernose of both penis and clitoris. Testicular hormones bring about the expansion of the tubercle into a penis, the closure of the labioscrotal swellings to form a scrotum, and the fusing of the folds to enclose the urethral tube which opens through the tip of the penis. In the relative absence of androgens (or of all hormones), the tubercle diminishes to become a clitoris, the labioscrotal swellings become labia majora, and the urethral folds become labia

¹The external female genitals which develop in the absence of fetal hormones are "immature," however (Federman, 1967).

minora and lower vagina with the urethral opening in the female position.

3. Differentiation of the Hypothalamic Gonadotropin Secreting Mechanism

In addition to differences in reproductive structure, mammalian males and females possess dimorphic temporal patterns of gonadotropin production after puberty. Gonadotropins are released from the pituitary gland according to an elaborate feedback mechanism which is regulated by neurohumoral secretions from nuclei situated in the adjacent hypothalamic loci (Barraclough, 1967; Money and Ehrhardt, 1972). In the female, Follicle Stimulating Hormone (FSH) induces the reopening of the ovarian follicle which secretes estrogens; in the male, FSH--or Interstitial Cell Stimulating Hormone (ICSH) as it is sometimes called--promotes spermatogenesis in the seminiferous tubules of the testes. Luteinizing Hormone (LH) causes the corpus luteum (shell of the burst follicle) to proliferate and secrete progesterone (P); in males, it stimulates the Leydig cells of the testes to produce androgens. In each case, the feedback of gonadal hormone output to the hypothalamus alerts the pituitary to produce FSH or LH.

This sexually differentiated pattern appears to be established during a brief sensitive period during ontogeny. In rats, the period is postnatal. Evidence of the role of testicular hormones in this process comes from early work on

gonadal transplants. Neonatal transplants of testicular tissues in females produced the Persistent Estrus Syndrome (PES) in adulthood, namely, constant vaginal cornification and follicle development but failure of ovulation and corpus luteum formation (Pfeiffer, 1936). Males castrated in adulthood and given ovarian transplants developed follicle growth but did not ovulate (Pfeiffer, 1936). Pfeiffer concluded that the mechanism for gonadotropin regulation was still undifferentiated in new-born rats. Harris (1964) demonstrated that neonatally castrated male rats, when given ovarian transplants in adulthood, developed corpora lutea and ovulated while intact males did not (Harris and Levine, 1965). Thus, it appeared that the neural structure which regulates gonadotropin secretion "becomes differentiated and fixed in its function under the influence of androgens in early life" (Harris, 1955).²

In primates, however, this effect is not seen. Goy and Phoenix (1971) produced eight female hermaphrodite rhesus monkeys by prenatal injections of Testosterone Propionate (TP). Despite masculinization of the genitals and late menarche, all eight females cycled normally in adulthood (Phoenix, 1974).

²The pituitary itself is not differentiated, as Pfeiffer thought. Cross-sex pituitary transplants caused no changes in either sex, but pituitary transplants from androgenized females to normal females produced full reproductive capacity in the normal females if contiguous to the hypothalamus (Segal and Johnson, 1959, cited in Money, 1965; Money and Ehrhardt, 1972).

The same is true of androgenized human females. Females with the Adrenogenital Syndrome (AGS) who are subject to high repeated doses of adrenal androgens throughout the fetal period and/or postnatally begin to ovulate and menstruate within months of regulation on cortisone. Females exposed to synthetic progestins prenatally, sufficient to masculinize their genitals, spontaneously menstruate at the normal age of puberty. Menarche may be delayed in AGS females treated soon after birth with cortisone, but this may be an effect of cortisone treatment (Money and Ehrhardt, 1972). (See Table 6 for description of human intersex syndrome.)

CHAPTER III

AN ASSESSMENT OF THE VALIDITY
OF THE ORGANIZATIONAL THEORY
IN ANIMALS

1. Introduction

In addition to morphologic and hypothalamic changes, the presence or absence of critical period androgens has been associated with alterations of typical mating behaviors in adulthood in many non-human mammalian species and with juvenile sexually dimorphic play patterns in Rhesus monkeys. These findings have suggested to some researchers that the principles of morphologic and hypothalamic differentiation may apply to the brain as well. In the present chapter, the notion of brain dimorphism--termed by Beach (1971) the "organizational theory"--will be evaluated in terms of the evidence on rats. Chapters IV and V will be devoted to an assessment of its validity as applied to humans.

2. The Organizational Theory

Ward (1974) has become the principal spokesman for the organizational theory:

We now understand that hormones operating during critical periods of perinatal development control the differentiation not only of sexual morphology

but of sexual behavior as well. If androgen is present during these very specific periods of early development masculinization and defeminization of behavior potentials, reproductive physiology, and morphology occur. In the absence of androgen, morphology and behavior are feminized. (p. 3).

The theory is based upon the concept of "organizers," described by Beach (1971) as chemical substances which bring about the organization of specialized structures from undifferentiated tissues, regardless of the presumed fate of the tissues. Such organization renders the tissues permanently sensitive to the organizing substances in later life.

Proponents of the theory hold that specific areas of the central nervous system which are destined to mediate sexually dimorphic behavior are originally bipotential or undifferentiated like the reproductive system. Androgens present during critical periods of early development are believed to organize these regions into "centers" or "pathways" controlling male behavior and to disorganize the substrate for female behavior. In the absence of androgens, the female behavior controlling region is thought to become organized.

A major variant of the theory is the idea proposed by Money and his colleagues, whose work has been with human intersexes. They suggest that bipotentiality persists in the relative or total absence of early androgens--that is, in the female phenotype--but it is "resolved in favor of unipolar masculinity of mating behavior" when the early hormonal

environment is androgenic (Money and Ehrhardt, 1972).

Numerous modifications of the theory have appeared in the literature in response to the many complexities and contradictions of the data. These modifications concern: (1) the specificity of hormone action; and (2) the identification of specific sensitive periods during which differentiation of the various morphological, physiological, and behavioral components of mating behavior occur. However, with a few exceptions, these modifications left unchallenged the central assumption that the brain itself becomes differentiated.

3. Male Mating Behavior in Male Rats

Male mating responses are defined in terms of four elements, two of which characterize Incomplete Responses (IR) and one of which is termed the Complete Response (CR). The fourth element is also a CR, but is distinguished in the literature as the Ejaculatory Response (ER):

Incomplete Response (IR)

Mount with Clasp: posterior mounting of the lure female

Mount with Thrust and Palpitation: Clasp plus palpitation of the female's flanks with forepaws

Complete Response (CR) or Intromission (Int.)

Mount with Intromission: Same as above but with more vigorous thrusting and a final "backward lunge"; dismount is usually followed by genital grooming

Ejaculation: Same as above except final thrust lasts longer than simple intromission; dismounting is not accompanied by a backward lunge; post-ejaculatory pause of four to five minutes before copulation is resumed

Many studies have shown that males deprived of androgens--either by postnatal castration or by injection of the anti-androgen Cyproterone Acetate (CA) pre or postnatally--are markedly deficient in mating behavior when given Testosterone Propionate (TP) in adulthood. Typically, CRs decline--mounts with intromission diminish and ejaculatory capacity is lost--while IRs, that is, mounts with clasp and mounts with palpitation and thrusts remain at normal levels or increase in frequency (Stern, 1969; Beach, 1971; Hart, 1972; Levine and Mullins, 1964; Ward, 1974; Gerall, et al, 1967; Nadler, 1969). The decline in ERS and Int. is temporary in normal males castrated in adulthood and given TP and does not occur when neonatal castrates are given replacement TP at the time of castration (Beach, et al, 1969; Whalen and Luttge, 1971).

However, the characteristic pattern seen in these unandrogenized males does not necessarily reflect a loss of behavioral responsivity to androgens. Levine and Mullins (1964), for example, characterized such males as having "a great deal of sexual vigor." Beach (1971) found no deficiency in responsivity of neonatally castrated male rats to females. As compared to rats castrated after the sensitive period (day 21) day 1 castrates were more responsive both be-

fore and after TP treatment. However, they were significantly deficient in CRs and Ejaculations.

This evidence would suggest that the deprivation of androgens during the sensitive period has permanently affected male mating behavior, perhaps because of a failure of masculine differentiation of the central nervous system. Furthermore, since IRs are characteristic elements of female mating behavior in many species, one could argue that these rats have differentiated a feminine behavior controlling center.

An alternate hypothesis is that deprivation of androgen may affect functioning at the periphery. Beach (1971) explored this possibility in his studies on the effect of sensitive period androgens on penile growth. In the rat the genital tubercle is still undifferentiated at birth. Castration within hours of birth allows the development of a small but morphologically complete phallus. Beach castrated male rats on day 1, 21, 50, 100, 150, or 350 and injected TP in adulthood. The penile growth of day 1 castrates in response to TP in adulthood was significantly diminished, that of day 21 castrates less so, and growth was unaffected after day 100. Next, Beach replaced TP on two days between the first two weeks postnatally in day 1 castrates. When these rats were given TP in adulthood, those given TP on days 1 and 2 or 3 and 4 produced the greatest growth response, and those given no replacement TP showed the least growth. In the absence of TP

injections no systematic effect of TP replacement in infancy was found. Beach concluded that the capacity for penile growth under exogenous androgen in adulthood is systematically related to the time after birth that TP was administered. Furthermore, he found a high covariance as a function of age that TP was given in infancy between penis weight ratio (penis weight/body weight) and mounts with intromission.

Another effect of the lack of early androgen may be to reduce sensitivity of the penis. Aronson and Cooper (1966) desensitized the glans penis of adult, sexually experienced cats by nerve section, leaving erectile capacity unimpaired. Males readily mounted estrus females but could not achieve intromission. Mounting, however, increased in frequency and duration. The authors concluded that loss of penile sensations prevented both orientation to the female and sensory feedback necessary to maintain sexual arousal. In a similar study, Beach (1971) found the identical patterns in normal, sexually experienced male rats after removal of a small section of the os penis, though not in control animals in whom the bone section was cut but not removed.

The similarity in the pattern of male mating behavior in these different cases suggests that the crucial factor in the display of CRs is penile growth and sensitivity. Beach (1971), for example, attributes the loss of CRs to the reduction in frequency of intromission, which provided insufficient genital stimulation to elicit ejaculation. Nadler

(1969), Gerall et al (1967), and Money and Ehrhardt (1972) have drawn the same analogy between phallic development and CRs. Nadler (1969) observed that the lower frequency of intromission responses in CA-treated males compared to day 1 castrates and control males was consistent with the difference in phallic size. CA males had phalli like the normal female clitoris, while day 1 castrates' phalli were like those of prenatally androgenized females.

Males need not be deprived of early androgen to produce these effects. Injections of Estradiol Benzoate (EB) or Estradiol Dipropionate (ED) during the perinatal period also bring about permanent loss of CRs and ejaculations. In addition, EB-treated animals mounted bizzarrely, suggesting that sensory feedback was not merely diminished but disorganized (Levine and Mullins, 1964; Harris and Levine, 1965).

Whether this deficit is the result of inadequate penile size and sensitivity alone or of peripheral metabolic changes as well (Van de Weile, 1974) remains to be seen. Gerall et al (1967) suggested that early androgen may "permanently affect metabolic processes in the somatic organs and may determine their capacity to provide adequate feedback necessary to exceed thresholds for evoking various response systems and consummatory reflex mechanisms" (p. 212). In either case, no "additional assumption of deficiencies in hypothetical mechanisms within the central nervous system" is necessary (Beach, 1971, p. 260).

Proponents of the organizational theory point to the permanence of the effects of early androgen deprivation as evidence that the masculine controlling centers have been irreversibly disorganized (Ward, 1974). Examination of the reproductive organs, however, indicates that all of the treatments which cause the "unmasculinized" pattern of mating described above also cause characteristic changes in the reproductive organs. The internal accessory organs fail to develop normally in males treated with estrogens or CA, or castrated early in development (Harris and Levine, 1965; Beach, 1971). Estrogen was considered to have a "direct" effect on the testes causing a "partial functional castration" and diminishing the development of the os penis (Levine and Mullins, 1954; Harris and Levine, 1965). Penile growth is permanently stunted under these conditions (Beach, 1971). Castration in adulthood induces atrophy of the testes and accessory organs--effects which are only partially remediable by replacement with TP, Testosterone (T)¹, or Androstenedione (A) (Whalen and Luttge, 1971; Luttge and Whelan (1970); Harris and Levine, (1965)). Adult castration also produces structural changes in the glans skin, reducing sensitivity (Aronson and Cooper, 1966).

Further support for the peripheral hypothesis comes

¹The naturally occurring free alcohol form. See pp. 35-36.

from reports that CRs and Ejaculations "indistinguishable from the normal male" are observed only in those female rats who have been androgenized both pre and postnatally (Ward, 1974). Pre and postnatal androgen treatment is the sole schedule that renders the phallus equal in size and position (in terms of ano-genital distance) to that of the normal male (Ward, 1969). The behavior and morphology of androgenized females will be discussed at length in Part 6.

4. Female Mating Behavior in Male Rats

Although most proponents of the organizational theory now acknowledge that the brain centers mediating masculine behavior appear to be functionally organized despite the absence of sensitive period androgens (or the presence of estrogens), they interpret the finding of enhanced female receptive behavior and cyclic hypothalamic gonadotroping secreting mechanism in such males as evidence that the brain has been "masculinized but not defeminized"--as it is, presumably, in males with normal sensitive periods (Phoenix, 1974).

In both sexes, the tendency to show heterotypical behavior differs individually, across strains and species, and under certain stimulus conditions (Levine, 1966; Young, 1961; Beach, 1965). Even in avian and amphibian species, nearly all elements of mating behavior are displayed by both sexes, though differing in frequency (Beach, 1965). Bisexual behavior has been noted occasionally in normally

in normal male rats (Beach, 1971). Normal males may spontaneously display female receptive responses: ". . . The frequency of its occurrence in the absence of any endocrinological pathological condition is of interest for the discovery of the determination of patterns of sexual differentiation. . ." (p. 1179, Young, 1961).

The evidence put forward in support of the organizational theory emphasizes the marked quantitative difference in receptive responses between normal males and males who are castrated during the sensitive period or given CA prenatally.² Unandrogenized rats typically display normal levels of female receptive behavior in response to injections of estrogen and progesterone in adulthood. (Nadler, 1969; Feder and Whelan, 1965; Beach et al, 1969; Harris, 1964; Ward, 1974). When TP is replaced during the first five postnatal days, however, little receptive behavior is elicited in adulthood (Harris and Levine, 1962). By day 9 or 10, TP replacement is less effective, and by day 13 or 14, no inhibitory effect of TP on Lordosis is obtained (Beach et al, 1969). Males castrated in adulthood show the low levels typical of the normal male, and castration on day 10 is too late to enhance the capacity for Lordosis (Grady et al, 1965).

This data would seem to support the hypothesis that the intrinsic female behavior controlling center has become

²See part 5 for a description of female mating behavior.

organized or fixed in the absence of early androgen. However, other evidence suggests that the capacity for female behavior is functionally organized in the normal male brain and can be elicited with the proper hormonal regimen. This hypothesis is supported by the findings of Davidson (1969) and Aren-Engelbrektsson et al (1970).

Davidson (1969) administered large daily doses (200 mcg.) of EB to three groups of normal adult male rats: (1) intact males; (2) adult male castrates one week post-operatively; and (3) adult male castrates six weeks post-operatively. In addition, one group of spayed adult female rats were treated. The male rats failed to respond to the schedule of E and P that elicits normal receptive behavior in spayed females but all males responded at levels comparable to normal females when given EB alone. Intact males reached Lordosis Quotients (L.Q.--Lordosis/Mounts x 100) or 60+% by the 14th day of testing. Males in groups 2 and 3 responded with L.Q.'s of 72.5 by the 7th day of testing, but the latter group showed a steeper slope and higher initial response. Ovariectomized females reached levels identical to the latter groups of castrate males by the 7th day of testing. Davidson concluded that: (1) females are more sensitive to estrogen than males; (2) females are more consistent in their response to estrogen; and (3) males' response to estrogen is not facilitated by progesterone--the sole qualitative response.

In the experiment of Aren-Engelbrektsson et al (1970),

some 73 to 90% of males castrated prepubertally (day 30) showed lordosis after 26 daily injections of EB begun on day 73. Doses were lower, ranging from 10 mcg.--160 mcg. Though lordosis was not qualitatively equal to that of day 1 castrates or estrus females, the frequency of responding was related to dosage, as was latency to first response.

One possible explanation for the low L.Q. found in prepubertal castrates may lie in some as yet unidentified factor that contributes to "attractiveness." The authors noted that the subjects were less attractive and less responsive to the stimulus males: the relative infrequency of mounting "remained a source of trouble" (p. 185). (Unlike estrus females who may lordose to the presence of the male, these males had to be mounted in order to display lordosis. Even when mounted, they were passive and displayed no ancillary responses.)

"Typical" male levels of Lordosis differ considerably across studies, as measured by the behavior of control males. In Nadler's study (1969), for example, control males (castrated day 60) were mounted as often as day 1 castrates and CA-treated males (and control females). Though 30% of control males lordosed as compared to 89-100% of treated males, the L.Q. of those responding (79.3) was not significantly different from treated males and fell within the normal female range. Ward (1972) reported a L.Q. of 40 for control males, but Lordosis was seen on one-third of the testes. Whether

these effects are due to strain differences, age at castration, or other experimental variables is not clear.

Taken together, these findings indicate that the neural substrate for Lordosis is functionally organized in male rats despite the presence of androgens during the perinatal period. In both males and females, the "responsiveness of these mechanisms to external stimulation is temporarily increased under the immediate influence of estrogen" (Beach, 1971, p. 267). Finally, it seems that the main effect of early androgen on female behavior potentials is indirect; that is, androgenized animals are not responsive to progesterone.

In Part 7 of this chapter, we shall consider in more depth the factors which may account for these behavioral findings. That will require an understanding of the way in which early androgens affect the behavior of genetic females, to which we now turn.

5. Female Mating Behavior in Female Rats

Female rats require estrus hormones to display mating responses. Both the female who is not in estrus and the spayed female do not show lordosis. However, replacement of E and P will elicit normal levels of behavior in spayed females. During estrus the female shows an increase in running and darting, ear-wiggling (the ancillary or soliciting responses), and Lordosis--the "crouched, rump-up, tail-

deflected posture" which permits mounting and intromission by the male (Money and Ehrhardt, 1972, p. 66). The most common measure of female mating behavior used in the literature is the Lordosis Quotient (L.Q.), that is, Lordosis/Mounts times 100. A L.Q. between 55 and 97 encompasses nearly all control females (Clemmens et al, 1969); nevertheless, L.Q.'s of less than 80 are often interpreted as "low". Mounting with or without thrusts are part of normal female mating behavior (Young, 1961; Beach, 1971; Money and Ehrhardt, 1972). For the sake of clarity, however, mounting behavior will be discussed in Part 6.

The organizational theory holds that exposure to androgens during the sensitive masculinizes the brain and inhibits the differentiation of neural regions which control feminine behavior. However, considering the data on lordosis in male rats, we may hypothesize that early androgen may simply reduce sensitivity to progesterone and raise the threshold of responsivity to estrogen.

Many studies have established that female rats who are androgenized during the first ten days of life display "abnormal" female mating patterns in response to E-P in adulthood, whether the ovaries are present or removed pre-pubertally (Barraclough and Gorski, 1962; Harris and Levine, 1962; Grady et al, 1965; Ward and Renz, 1972; Ward, 1969). However, the abnormality pertains not to the quality of response but to the temporal pattern--at least at low doses.

A single dose of 10 micrograms TP given on day 5 permanently disrupts the cyclic hypothalamic gonadotropin regulating mechanism in more than 70% of animals (Barracclough, 1967). The resulting Persistent Estrus Syndrome (PES) renders such females anovulatory and therefore sterile. However, persistent vaginal cornification maintains continual receptivity. Androgens are not the only substances which disrupt cyclicity; sensitive period estrogens interfere with normal receptivity in much the same way (Feder and Whelan, 1965; Levine and Mullins, 1964; Barracclough, 1967). Neonatal injections of ED as small as 5 micrograms on day 5 produce the same PES (Barracclough, 1967).

As regards mating behavior, Barracclough stated: ". . . no general statement can be made about the receptivity of PE animals to the male" (1967). These animals "mated readily" but displayed "bizarre" temporal patterns uncorrelated with vaginal cycles--for example, receptivity for 9 consecutive days or every other day. Much higher doses of over 100 micrograms TP or ED on day 5 are required to produce the rejecting behavior characteristic of diestrus females. However, unlike diestrus or castrated females, females treated in this way cannot be induced to respond even when given high doses of E-P in adulthood (Harris and Levine, 1965; Barracclough and Gorski, 1962). Permanent diestrus (PDE) induced by high doses results either in extreme passivity or active rejection of the mounting attempts by males (Whelan and Nadler, 1969; Levine and Mullins, 1964).

One important study, however, indicated that female mating behavior is unaffected by high doses of postnatal androgens despite masculinization of the external genitals and cycling mechanism. Luttge and Whelan (1970) administered one of three androgens--Dihydrotestosterone (DHT), Androstenedione (A), or the free-alcohol form of Testosterone (T)--in doses of 0-800 micrograms on day 2, 3, 4, or 6 to female rats. Though A and T produced typical dose-dependent sterility and masculination of the genitals, female sex behavior was not inhibited even at the highest doses. L.Q.s of 96.7 and 98.5 for T and DHT, respectively, and 95.4 for A-treated females were obtained. The range of L.Q.s was between 90 and 100 in the first two groups and between 50 and 100 in the A group. Paradoxically, control females' L.Q. was relatively low; namely, 67.8 with a range of 0 to 100. The authors discussed the possibility that the naturally-occurring free alcohol form of T, which replaced the propionate ester of T (TP) used in most research, is not a potent steroid. However, it seemed unlikely that 50 micrograms of free T could induce the anovulatory PES while 800 micrograms did not suppress lordosis. Additionally, since 25 micrograms daily revived mounting behavior in male castrates, it seemed unusual that sensitive period doses of 800 micrograms would have no effect on the neurons controlling sexual behavior--"if, in fact, the mechanism of behavioral differentiation is one of neural stimulation by testosterone during the sensi-

tive period" (Luttge and Whelan, 1970, p. 279).

On the other hand, if responsivity to the normal pattern of E-P has been affected by early androgens, one would predict that (1) high repeated doses of EB or ED alone would elicit Lordosis comparable to that seen in normal males so treated; and (2) responsivity to P would be diminished in androgenized females. Apparently, no one has treated androgenized females with high repeated doses of estrogen alone. Davidson (1969) elicited normal lordosis in spayed normal females with 200 micrograms of estrogen injected for seven days. The possibility that normal males would be more responsive than androgenized females to high doses of estrogen is unlikely.

Regarding the second prediction, Clemmens et al (1969) did find that even low doses (10 micrograms) of early TP affected adult responses to P. They injected female infant rats on day 1, 2, 4, 6, or 10. Females treated on days 1, 2, or 10 lordosed at normal levels, but those treated on day 4 or 6 showed significant suppression in response to E-P. Ward and Renz (1972) also found reduced sensitivity to P in TP-treated females.

However, the conclusion that early exposure to androgen permanently alters responsivity to P may be premature. Clemens et al (1969) found that the provision of a two-hour adaptation period produced a significant increase in L.Q. in females androgenized on days 4 and 6; L.Q.'s fell within the normal range. Further tests with control females revealed that the

facilitating effect of preadaptation was related to minimal levels of P. High levels or no progesterone produced little adaptation effect. The authors surmised that some pheromonal or other perceptual phenomenon controlled by P accounted for these results.

In summary, the responses of androgenized and estrogenized females are sufficient to indicate that the neural basis for female behavior is functionally organized despite masculinization of the genitals and of the cyclic gonadotropin secreting mechanism. The relative reduction in Lordosis to E-P may be accounted for by reduced sensitivity to P in adulthood as a function of early androgen or estrogen. However, it should not be overlooked that behavioral sensitivity was not reduced when T, rather than TP, was the androgenizing agent and when a long period of pre-adaptation was provided.

6. Male Mating Behavior in Female Rats

Incomplete male mating responses are part of normal female behavior in many species (Young, 1961). According to Beach (1971, p. 269) "a substantial proportion of untreated female rats from certain strains whose nervous systems have never been exposed to testicular androgens execute mounting and thrusting responses when tested with other females that are in estrus." The intromission response may be seen infrequently (Ward, 1974; Beach, 1971). Relevant figures are presented in Table 1.

Table 1.--Mounting behavior in virgin, adult, intact females untreated in infancy*

15 females aged 90-180 days in 390 tests	Percentage of tests responses appear	Percentage of group showing response
IR--Mount with clasp	76.1	100
IR--Mount with palpi- tation and thrust	34.2	95
CR--Mount with thrust and intromission	5.4	34

*From Beach and Rasquin, 1942.

Beach and Rasquin (1942) found that mounting frequency and percentage responding increased with repeated testing and is not correlated with vaginal cycles nor affected by endogenous ovarian hormones. Further studies established that the same responses may be seen in prepubertally spayed females, indicating that these behavioral patterns are not organized or activated by pubertal hormones (Beach, 1942).

When normal females are ovariectomized prepubertally and given TP in adulthood, the effect is to shift response frequencies away from the incomplete forms toward the complete forms. This shift is more pronounced with higher doses but never reaches male frequencies. Table 2 illustrates the effects.

Beach (1971) concluded from this data on the normal

Table 2.--Mounting behavior of prepubertally spayed female rats administered various doses of TP in adulthood*

Percentage of all mounts in each category				
	Mount with <u>clasp</u>	Mount with palpitation and thrust	Mouth with intromis- sion	Mount with ejacula- tion
10 prepubertally spayed rats				
Pretreatment	71	28	1	0
2-10 mg. TP	36	58	6	0
12-24 mg. TP	13	78	9	0
12 normal males	2	28	63	7

*From Beach, 1942.

Beach (1971) concluded from this data on the normal female that "without androgenic stimulation during development the female rat's nervous system is capable of mediating all of the masculine responses with the noteworthy exception of the ejaculatory pattern" (p. 270).

The dose-response relationship between TP and behavior in these females indicates that the normal female perinatal environment does not prevent adult responsiveness to TP. In fact, injections of TP in adulthood normally enlarge the clitoris.

The evidence to follow suggests that perinatal androgenization has its effect upon behavior by mediating changes at the periphery--reversing, in effect, the pattern seen in

males deprived of androgens.

Because the differences between normal males and females in respect to incomplete mounting responses are quantitative, interpretation of the behavioral results of androgenization is difficult. Thus, if androgenized females have been masculinized, they should show fewer IRs and more CRs and ejaculations characteristic of the male pattern. The problem is compounded by disparities and omissions in reporting quantitative data. For example, data may be reported in terms of percentage of animals responding, mean frequency of response elements, or of the 3 responses excluding ejaculation combined, percentage of tests in which response was seen, and so on. These problems of measurement will become apparent in the discussion to follow.

A number of studies found that females exposed to androgen postnatally mount more often than control females (Ward and Renz, 1972; Nadler, 1969; Gerall and Ward, 1966; Harris and Levine, 1965). Gerall and Ward characterized the mounting frequency of androgenized females as intermediate between normal females and normal males. Whelan et al (1969) in contrast, found no increase in mounting frequency as a function of perinatal androgenization. In respect to the percentage of females mounting, androgenized females do not differ from normal females when cross-data comparison is made. For example, 95 to 100% of Beach and Rasquin's (1942) normal females displayed IRs as compared to 90% of Nadler's androgenized

females.

Ward's (1969) data suggest that the differentiating factor may be the amount of circulating androgen in relation to E-P rather than an early masculinizing effect on the female brain. She studied male mating behavior in female offspring of mothers given 2 milligram TP during prenatal days 16-20 and either oil or .5 milligrams TP days 1-40 postnatally (pre and P-P groups); female offspring of untreated mothers given either oil or .5 mill. TP days 1-40 postnatally (Control and Post groups); and normal untreated males. In addition, one-half of each group was spayed at 55 days of age. All groups were tested for male behavior before and after 40 days of daily TP injections of 1 mg. beginning on day 60 (puberty). In respect to mean IRs and CRs combined, females did not differ from males, with the sole exception of normal intact females. That is, normal spayed females did not differ from any of the androgenized females or from the males.

These results suggest that TP administered in adulthood is as "masculinizing" as perinatal treatment, providing the ovaries are removed. Conversely, it may be said that the normal female's brain is functionally organized for incomplete and complete male mating responses and that the absence of ovarian hormones permits fuller expression of them.

The measurement of frequency of intromission (CRs) provides a more accurate reflection of masculinization. In Beach and Rasquin's (1942) study, 63% of all mounts by normal

males were of the intromission type compared to only 1% of mounts by normal (prepubertally spayed) females and 9% by the same females given 12-24 mill. TP in adulthood. Unfortunately, Beach and Rasquin did not record mean frequency of these responses, so that a direct comparison with other data is not possible.

Many studies report enhancement of mean intromission frequency in females exposed to androgens pre and/or postnatally (Whelan et al, 1969; Ward and Renz, 1972; Ward, 1974; Harris and Levine, 1965; Nadlder, 1969). When Ward (1969) compared the mean frequency of CRS among treatment groups, all female groups differed significantly from normal males and normal intact females. Again, the performance of normal, spayed females did not differentiate them from androgenized females. Cross-study comparison indicates, furthermore, that normal intact females cannot invariably be differentiated from other female groups. Finally, the magnitude of difference between normal males and all other groups is striking.

Table 3 indicates that the quantitative performance of androgenized females resembles more the performance of normal intact females and unandrogenized males than that of normal males.

In a later study, Ward and Renz (1972) reported significant differences in male mating behavior as a function of timing of perinatal androgen administration. Repeating the methodology of Ward's (1969) experiment, they found that CRS

Table 3.--Mean frequency of intromission, or complete, responses in androgenized and unandrogenized male and female rats*

Mean frequency of intromission responses	
Females	
Normal Intact	0.7; 1.3; 2.0
Normal Spayed	2.0; 2.4
Pre TP Intact	1.3
Pre TP Spayed	1.4
Post TP (day 1)	2.5
Post TP (day 4)	1.7
Post TP (day 1-40)	
Intact	2.3
Spayed	1.5
P-P TP Intact	1.2
Spayed	3.0
Males	
Normal Intact	11.5; 11.3
Day 1 Castrate	3.8
Pre CA	3.0

*From Ward, 1969; Beach and Rasquin, 1942; Nadler, 1969; Levine and Mullins, 1964; Whelan et al, 1969.

were increased by both Pre and Post treatment, while mean IRs and CRs combined were increased only under Pre treatment. The figures are found in Table IV below.

The difference in figures reported above and in Table 3 points up the problem of quantitative data comparison. Here, for example, control females show CRs close to those of normal

males.³ It is also of interest to note that percentage measures differ from mean frequency measures. This point was made also by Nadler (1969), who noted that among females, though the percentage of control versus androgenized females who mounted was significantly different, the frequency in those who mounted was not. Individual differences of genetic origin most likely account for the differential response to early androgen reflected in such observations.

Table 4.--Male mating behavior in androgenized and control female rats*

	Mean IR + CR	Mean CR	Percentage showing more than 2 CRs
Control	46.5	8.1	69
Pre TP	92.5	18.7	75
Post TP	42.6	18.3	38

*From Wald, 1974.

The ejaculatory response (ER) ought to illustrate most clearly the supposed masculinizing effects of early androgens. The ER appears to be associated with timing or perinatal androgen administration, for it has never been observed in untreated females (Beach and Rasquin, 1942; Beach,

³Ward's measure of CRs may not, in fact, represent intro-missions. She fails to include in her definition of intro-missions the "backward lunge" reported by Beach (1971) and Money and Ehrhardt (1972), emphasizing merely the increased "vigor" of the response (see Part 3).

1942) or in prenatally androgenized females (Gerall and Ward, 1966; Nadler, 1969; Ward, 1969). In contrast, the ER was observed at least once in 73% of females treated P-P and in 30% of those treated postnatally. These figures may be compared with the figure of 65% of males showing ERs (Ward, 1974) and 68% showing the "full male pattern" (Harris and Levine, 1965). Ward (1974) noted that most P-P females ejaculated more than once per test and on more than one test. One female, in an extended final test, ejaculated 7 times. All females showed the characteristic post-ejaculatory phase.

It should be noted that ERs are also obtained in females administered EB postnatally, indicating that androgen is not necessary for the supposed masculinizing effect. Early estrogens exert a "paradoxical" effect on infant females, namely, potentiating masculine behavior. Levine and Mullins (1964) administered nine daily injections of 100 micrograms TP in adulthood after postnatal injections of estrogen. Some augmentation of mounting responses as compared to oil-treated subjects was seen. Mean IRs increased by about half, mean CRs increased from 2 to 3 per test, and ERs were observed (a mean frequency of about 2 per test).

Undaunted by the complexity of the data, Ward has attempted to determine the specific sensitive periods for the "development of specific components of the reproductive patterns. . ." (1974, p. 8). Concluding that the "differentiation of tissues regulating adult sexual behavior is indeed

governed largely by the same mechanism as that determining reproductive morphology", she suggests that the potential for IRS is organized during "early perinatal ontogeny"; that "somewhat later interventions" influence CRS and ERs, but not IRS; and that both pre and postnatal androgenization is necessary for the greatest ejaculatory capacity (1974, p. 8).

Beach (1971), on the other hand, attributed the appearance of the ER to the genital stimulation facilitated by an adequate coital organ "rather than a more complete stage of organization in crucial central neural circuits" (p. 271). Money and Ehrhardt (1972) also questioned whether neonatal androgen affects brain circuits or "by potentiating clitoral enlargement and changing neural sensory organization at the periphery (p. 70). Luttge and Whelan (1970) and Nadler (1969) too noted the relationship between intensity of behavior and phallic development.

Indeed, when the morphological changes produced by early androgens are considered, it seems likely that such factors as size and position of the phallus and body weight may account for the increased masculine forms of behavior. Table 5 provides the necessary data.

It is evident that ejaculatory responses occur most in P-P females, whose phallic length and position are not significantly different from normal males. Ward (1969) described the phallus of these females as "indistinguishable from the normal male." The fact that they ejaculated fluid is not sur-

prising in view of the advanced development of the seminal vesicles, prostates, and coagulating gland. There is no quantitative data on the morphology of EB females, in whom the ejaculatory response has been observed. However, Levine and Mullins (1964) noted that TP in adulthood resulted in clitoral enlargement in both EB and control females, though more noticeably in the former. Direct measurement of the phalli of unandrogenized males is also not available, but Nadler (1969) reported that prenatally-treated CA males' phalli were like the normal female clitoris, while day 1 castrates' phalli were similar to those of prenatally androgenized females.

Table 5.--Morphology and body weight in normal males and females and androgenized females*

	Ano-genital distance (cm.)	Body weight	Phallic length (cm.)
Normal Males	4.3	292	3.4
TP Females			
Pre	2.8	215	2.2
Post	2.0	280	2.1
P-P	3.6	298	3.2
Normal Females	2.1	252	0.4

*From Ward (1969).

It seems premature to analyze the "fit" between morphological status and intromission or CRS because of the contradictory data reported above. Taken together, this data suggests that the potential for all elements of masculine mating

behavior is functionally organized in the female rat but limitations of phallic size, position, and structure prevent the display of sufficient intromissions to elicit ejaculation. When androgens are present during the pre and postnatal periods and also in adulthood, phallic growth is adequate to ensure the ejaculatory response.

7. Direct Evidence of Brain Organization

The organizational theory has yet to be supported by direct evidence of brain changes supposedly caused by the perinatal hormonal environment. Beach (1971) cited photographic evidence of a "disorganized" neural structure in the brain of a female of the Ramstergig species--a "straight" species whose members show exclusively male or female behaviors. However, Young's (1961) statement still applies to those species and strains most commonly studied: "the neural tissues mediating behavior have not been identified in the sense that attention can be directed to cells in which a change or changes in response to hormonal stimulation could be correlated with the expression of sexual behavior" (p. 1204).

Obviously, in any species in which heterotypical mating responses are displayed, regions of the brain governing those responses must be functionally organized. The evidence to date indicates that regions of the hypothalamus are implicated in the display of male and female mating behavior and maternal or parenting behavior. The sexes do not differ in this regard. The regulating mechanism of pituitary gonadotropin

secretion is found in the median eminence; its destruction or implants of estradiol result in gonadal atrophy (Lisk, 1962; 1967). Lesions of the preoptic area of the anterior hypothalamus prevent the display of Lordosis (Lisk, 1967), while implants of estradiol or diethylstilbestrol elicit Lordosis in ovariectomized rats (Lisk, 1962; Pfaff, 1965). The same region of the hypothalamus, particularly its midline structures, must be intact for male mating behavior patterns to be displayed (Lisk, 1967). Electrical stimulation of the lateral preoptic and lateral anterior hypothalamus elicited male behavior in males and ovariectomized females (Vaughn and Fisher, 1962). Implants of sodium testosterone sulphate were placed in the medial regions of the preoptic area (Fisher, 1956; 1966).

The failure to test for female mating behavior in males is a significant omission and most likely reflects the belief that females remain more bisexual, while males become thoroughly masculine under the influence of early androgen (Money and Ehrhardt, 1972; Young, 1961). However, it is likely that Lordosis capacity is organized in males. For one, males lordose to high levels of estrogen in adulthood. Secondly, full female patterns of maternal behavior may be elicited by implants of androgen. Thus, the evidence supports the behavioral findings that both male and female potentials are functionally organized in the same brain regardless of the early hormonal environment.

One possible source of brain dimorphism may be found in differential uptake and retention of sex hormones in various brain loci. However, this method has yielded inconclusive results (Money and Ehrhardt, 1972). Whelan (1974) reviewed the evidence regarding estrogen. Of 18 studies comparing the presence of radiolabeled estrogen in the brains of males, females, and androgenized females, 10 found that neonatal androgen reduced estrogen uptake in the anterior hypothalamic-preoptic area while 9 found no striking sex differences. All studies noted that males and androgenized females are capable of accumulating estrogens. Whelan (1974) found no difference in uptake or in regional distribution of metabolites after estradiol administration. He concluded that "the evidence is not convincing that males and females differ in any meaningful way in their ability to concentrate estrogen in brain tissue" (p. 477).

A second possibility is that cellular response to sex hormones may differ in the sexes. It is now believed that steroid hormones act by binding to a cytoplasmic protein receptor and entering the nucleus, where they activate DNA replication (which regulates cell function) (Wilson, 1972).⁴ Such a difference, as Whelan (1974) points out, would be masked by the study of whole tissues. As regards estrogen, cytoplasmic

⁴Rubin has recently advanced the theory that ions of magnesium and other elements, rather than hormones, control cell function (Blakeslee, 1974).

binding of estradiol was found in the anteriorpreoptic and median eminence, but not in the cortex, of male, female, and androgenized female rats. However, sex differences were not found. The sexes did differ in hypothalamic nuclear retention of estrogen in a study by Whelan, but he considered the difference too small to account for differences in mating behavior (1974).

Thirdly, hormones may not alter biochemical mechanisms but may act to alter "growth and thereby neural connectivity" (Whelan, 1974, p. 478). Raisman and Field (cited in Whelan, 1974) found that females have more dendritic spine connections than males in the preoptic area and that these connections are at female levels in males castrated within 12 hours and in females androgenized on day 16; and at male levels in males castrated day 7 and in females androgenized day 4. Thus, capacity for lordosis is positively associated with the number of synaptic connections. In view of the behavioral findings, it is clear that the sexes differ relatively in this respect. This interpretation respects the wide individual differences found in animals and is also compatible with the finding that high, repeated doses of heterotypical hormones elicit heterotypical responses in most animals.

8. Conclusions

In summary, it is well-established that hypothalamic regulation of hypophyseal function is differentiated in the

sexes via the presence or absence of sensitive period androgens.⁵ It is also evident that differentiation of the external genitals is controlled by the same hormonal paradigm. However, the evidence does not support the secondary assumption that early androgens organize brain centers which regulate masculine behaviors or inhibit the organization of female behavior-controlling regions. Failure of complete genital differentiation in the sensitive period has sequelae on later behavior--an indirect effect most likely mediated by: (1) the stunting of growth and sensitivity of the phallus; and (2) the setting of response thresholds in target cells--perhaps through the growth of neural connections. Central nervous system regions which participate in sexual behavior appear to remain bipotential, or functionally organized in both sexes.

The organizational theory is based upon the assumption that differentiation of the external genitals, the hypothalamic gonadotropin regulating mechanism, and the brain mechanisms controlling sexual behavior occurs according to the same paradigm; namely, that androgens present during a brief critical period of ontogeny bring about male development and their relative or total absence permits female development. However, as Wheln (1968) and Beach (1971) have pointed out, ovulation and

⁵In rhesus (Phoenix, 1974) and in humans (Money and Ehrhardt, 1972) the cyclic pattern is not disrupted in androgenized females.

external genital morphology are mutually exclusive possibilities. Ovulation cannot occur in both the male and female pattern closely in time, nor can one possess both a clitoris and penis. However, mating behavior can and does occur in both male and female patterns within a brief period, and the display of one pattern does not affect reproductive or sexual capacity of the other pattern.

Moreover, it is not yet known which androgens are responsible for morphological and physiological differentiation (Wilson, 1972; Whelan, 1974). In fact, estrogens may be the hormones of sexual differentiation (Whelan, 1974). More likely, however, complex steroid metabolic processes are involved in differentiation rather than the simple androgen/no androgen principle.

Finally, the application of these principles to humans requires great caution. For one, species, strain, and individual differences among rodents and monkeys alone are striking, and there is no predictable progression along the phylogenetic scale (Hart, 1974). Experimental data on humans is limited to those syndromes which arise spontaneously or through medical error. While control of dosage and timing of hormone administration is not ethically possible, these studies have the advantage of providing data on gender identity formation in intersexes corrected by surgical and hormonal intervention.

CHAPTER IV

GENDER ROLE DEVELOPMENT IN NORMAL AND INTERSEX HUMANS

1. Introduction

In humans, sex is inferred at birth from the configuration of the external genitalia. In other respects, the sex of an infant cannot be distinguished reliably by adults (Lewis and Weinraub, 1974). Imposed cues of name, clothing, and hair length are needed to assure that others relate to the infant as a boy or girl. By the time language is established, however, a "core gender identity" is apparent--so much so that Money and Ehrhardt (1972) counsel against sex reassignment after 18-24 months. Sex typing is increasingly in evidence after age 2. By 6 or 7, awareness that gender cannot change is well-established--a nodal point of development like the earlier period of language acquisition between 18 to 24 months (Kagan, 1971).

The question whether sex differences are products primarily of subtle reinforcement or expressions of the infant's own "phyletic program" is most appropriately studied during infancy. Many researchers who are primarily interested in the development of object relations and self-object differ-

entiation are addressing themselves to this question (Lewis and Weinraub, 1974). In brief, research indicates that during the first two years of life minimal differences in mean and variance values on infant measures are found. Differences in patterning of behaviors vis-a-vis mother and father, and stability over time, are somewhat more in evidence (Kagan, 1971). Finally, differences in parental behavior toward infants--in the absence of differential behavior by the infant--are marked (Lewis and Weinraub, 1974; Korner, 1974; Moss, 1974; Lewis, 1972; Goldberg and Lewis, 1969). In Part 3, this research will be discussed in more detail. It appears, however, that the early psychological environment is of major importance in gender differentiation.

2. An Overview of Gender Role

Development in Intersexes

Nature provides a number of spontaneously occurring syndromes which result in intersex conditions.¹ All are ultimately of genetic origin, involving either sex chromosome abnormalities or autosome errors and resulting in gonadal dysgenesis or disorders of fetal endocrinology. A few intersex conditions

¹The term intersex refers to "a condition of prenatal origin in which embryonic and/or fetal differentiation of the reproductive system fails to reach completion as either entirely female or entirely male" (Money and Ehrhardt, 1972, p. 5). Conforming to Money and Ehrhardt's usage, the terms hermaphroditism and intersexuality are used interchangeably.

are the result of medical error. For example, Progestin-Induced Hermaphroditism (PIH), in which normal genetic females were born with masculinized genitalia, resulted from maternal exposure to synthetic progestins which were used to treat toxemia of pregnancy. Another example--technically not of an intersex syndrome--is that of a normal infant boy who was reassigned as a girl after ablation of the penis during a circumcision operation (Money and Ehrhardt, 1972). Table 6 provides descriptions of the common intersex syndromes.

As Federman (1967) has commented, the very harmony of development in normals obscures the origins of gender differentiation. The study of human intersexes allows one to isolate the various contributing factors and, if it does not permit conclusions as to which factors determine which characteristics, it does allow one to rule out many factors. For ethical reasons, of course, experiments analogous to those discussed in earlier chapters cannot be performed on humans.

Medical evidence suggests that sexual identity rests on at least 9 components, none of which invariably identifies the sex of a given person since any one or several may be discordant from the others (Federman, 1967). These components may be summarized as follows:

1. External genital appearance
2. Internal ductal differentiation
3. Identity of the gonads
4. Endocrinologic sex

Table 6.--Characteristics of common intersex syndromes*

SYNDROME	CAUSE	SEX CHROMOSOME CONSTITUTION	GONADS	INTERNAL ORGANS	EXTERNAL GENITALS
Adrenogenital Syndrome (AGS) in genetic females	Autosomal re- cessive; excess fetal and post- natal androgens	XX Chromatin Positive	*F	*F	All degrees of masculiniza- tion: enlarged hypospadiac phallus; labio- scrotal fusion common
AGS in genetic males	same	XY Chromatin Negative	*M	*M	*M
Progestin- Induced Herma- phroditism (PIH)	Synthetic progestins taken by mother	XX Chromatin Positive	*F	*F	Same as AGS females but commonly not as severe
Idiopathic Precocious Puberty (PP) in genetic males and females	Unknown	Normal XX or XY	*F or M	*F or M	*F or M

Key: *M and *F refer to normal male and female

Table 6 (continued)

SYNDROME	CAUSE	SEX CHROMOSOME CONSTITUTION	GONADS	INTERNAL ORGANS	EXTERNAL GENITALS
Testicular Feminizing Syndrome (TF)	Autosomal dominant or X-linked recessive or male-limited X-linked dominant	Apparently normal XY Chromatin Negative	*M	Undeveloped streak tissue	Grossly nor- mal but im- mature F: short vagina; small to nor- mal clitoris
Turner's Syndrome (TS)	Second sex chromosome missing; or mosaic and/ abnormal	If 45, XO Chrom. Neg. If mosaic (XO/XX) Chrom. Pos.	Streak tissue	Streak tissue or immature F	Grossly nor- mal but im- mature F
Klinefelter's Syndrome	Failure of chromosomes to separate normally during meiosis of germ cells or mitosis in embryo	XXY and variants: XXXY, XXYY, XXXXY	Small testes; aspermia	Immature M	Small penis

Key: *M and *F refer to normal male and female

Table 6 (continued)

FETAL HORMONES	ADULT HORMONES	PUBERTY	FERTIL- ITY	TREATMENT	ASSOCIATED CHARACTERISTICS
Lack of cortisol and excess adrenal androgens and other hormones	Normal male levels if untreated; otherwise near normal F	Precocious and virilizing if untreated; otherwise *F (no hormones needed)	*F	Cortisone always; clitoridectomy; vaginal exteriorization; hypospadiac repair as needed	Elevated I.Q.; high energy expenditure; short stature as sequel to early, rapid bone maturation
Same	Excess if untreated; otherwise *M	Same; otherwise *M	*M	Cortisone always; no surgical correction needed	Same
Excess androgenic progestins	*F	*F (spontaneous)	*F	Surgical correction same as AGS	Same except normal stature
*F or M		Precocious; concordant with genetic sex	*F or *M	School advancement if I.Q. permits	Elevated I.Q. in some samples; short stature as sequel to early rapid bone maturation

Key: *M and *F refer to normal male and female

Table 6 (continued)

FETAL HORMONES	ADULT HORMONES	PUBERTY	FERTIL- ITY	TREATMENT	ASSOCIATED CHARACTERISTICS
*M androgen production but target cells unresponsive	*M Possibly enhanced testicular estrogens	Spontaneous F secondary sex characteristics; amenorrhea	Nil	Removal of testes and Wolffian tis- sue (to prevent cancer) and replacement of estrogen	Tall stature
No gonadal hormones	None; estrogen replacement	None, unless estrogen given	Nil	Estrogen to induce puberty	Extreme short stature (under 5 feet); webbed neck, shield-like chest, receding mandible, and others (variable)
Increased Follicle Stimulating Hormone	Below normal male levels	Delayed and weakly virilizing	Nil	Androgens	Lack of spermatogenesis; breast development; small testes; tall stature; risk of mental retardation

Key: *M and *F refer to normal male and female

*From Federman, 1967; Mittwoch, 1967; Grumbach and Van Wyck, 1974.

5. Genetic sex
6. Nuclear sex
7. Chromosomal sex
8. Psychologic sex
9. Social sex

The predictive value of any of the biological indices for gender identity is extremely low. In contrast, gender identity is concordant with sex of assignment and rearing in roughly 95% of cases reported in the literature (Money and Ehrhardt, 1972; Hampson, Hampson, and Money, 1955; Diamond, 1965).² A successful outcome is assured when the following conditions are met (Money and Ehrhardt, 1972):

1. The parents must have no doubt or ambiguity as to the child they are raising
2. First stages of corrective genital surgery should be made as early as possible
3. At the usual age of puberty, sex-appropriate hormones should be administered, when necessary, in accordance with statural growth

²The following definitions of gender identity and role are used interchangeably in the present paper, according to the usage of Money and Ehrhardt (1972, p. 4): gender identity refers to "the sameness, unity, and persistence of one's individuality as male, female, or ambivalent, in greater or lesser degree, especially as it is experienced in self-awareness and behavior; gender identity is the private experience of gender role, . . ."; gender role denotes "everything a person says and does, to indicate to others or to the self the degree that one is either male, female, or ambivalent; [including] sexual arousal and response; gender role is the public expression of gender identity, . . ."

4. The child should be counseled regarding diagnosis and prognosis at a level appropriate for his or her age
5. Parental counseling is needed when reassignment is made after 12 months of age

The most important of the above conditions is the first. Sex reassignment requests are made by hermaphrodites who "typically have a biography of uncertainty as to their sex of assignment" (Money and Ehrhardt, 1972, p. 153). On the other hand, when rearing is unambiguous and consistent, even gross anatomical abnormalities--such as precocious masculinizing puberty in a genetic female or micropenis in a genetic male--may be overcome. Nevertheless, agreement of the external genitals with assigned sex is of primary importance in the formation of a stable gender identity (Grumbach and Van Wyck, 1974; Federman, 1967).

Modern clinical management of intersex syndromes concurs with the observation that the potentiality for gender identity formation is highly plastic and largely independent of biological factors. Sex is assigned according to fertility and adult coital capacity. Where the two conflict, as in Testicular Feminization Syndrome (TF) males who lack a penis and will never be capable of male puberty, coital performance as a female is given precedence over the presence of testes. In females with the Adrenogenital Syndrome (AGS), the decision favors surgical reconstruction of the external genitals to conform to female fertility--which is unimpaired by fetal

androgenization--even when the phallic organ is adequate for male coital performance. In fact, undiagnosed and untreated AGS females assigned as males develop normal male gender identities. One such patient described by Federman (1967) married, served in the army, and began menstruating in young adulthood (manifested as periodic hematuria through the penis).

The most striking examples of this principle are provided by the following vignettes of hermaphrodite pairs matched for clinical diagnosis and status of external genitals at birth, but discordant for sex of assignment and rearing (Money and Ehrhardt, 1972). In the first two pairs, the above conditions were met fairly well, and gender identity conformed to sex of assignment and rearing. The three matched pairs were genetic females with AGS whose genitals were sufficiently ambiguous at birth to permit diagnosis as a male with hypospadias and undescended testes or as a female with AGS. Thus they were alike in genetic (nuclear and chromosomal), gonadal, and fetal hormonal sex.

In the first pair, who were treated fairly early with cortisone, the girl was initially diagnosed as a male and reannounced at two months as a female.³ Genital feminization and cortisone treatment were begun at two years of age. Although tomboyish in childhood, "otherwise, everything about

³ Reassignment refers to a change of assigned sex after 18 months. Prior to that time, one speaks of a sex reannouncement (Money and Ehrhardt, 1972).

this girl was very attractively feminine to all who interviewed and knew her" (p. 155). The second member was diagnosed as a male with hypospadias and undescended testes. Three unsuccessful masculinizing operations (involving the urinary system) led to the correct diagnosis at 3-1/2 years. Because his gender identity was well established by this time, it was decided to permit the male diagnosis to stand. Cortisone treatment and appropriate surgery (hysterectomy, gonadectomy, and phallic repair) were carried out. At puberty, artificial testes were implanted and masculine pubertal changes induced by androgen therapy. This boy was accepted by all as a boy. Except for some "trepidation" about his coital performance because of his small penis and artificial testes, he was satisfied with his gender.

In the second pair, correct diagnosis was not made until the age of 12. The child raised as a girl had masculinized during childhood because cortisone had not been supplied. Cortisone treatment permitted a feminizing puberty. Surgical exteriorization of the vagina and removal of the enlarged clitoris was performed. The girl married at age 27. The other member of the pair, diagnosed as a male with AGS, was raised as a boy. Cortisone treatment begun at age 3-1/2 had prevented the precocious masculinization seen in the girl, but estrogen production by the ovaries at puberty induced breast development, which led to the correct diagnosis. Since his male gender identity was firmly established, corrective

surgery and testosterone treatment--not necessary for virilization, but ideal--were carried out to permit conformity of gender identity and body phenotype.

In the third matched pair, the original assignment and rearing had been ambiguous--though ostensibly one was called a girl and the other a boy. In the absence of cortisone treatment, precocious virilization was apparent in both children. Both, incidentally, showed elective mutism, which disappeared once a sex reassignment had been decided upon and carried out. The "girl" chose to become a boy and the "boy" to be a girl.

The evidence of gender identity formation in matched pairs of hermaphrodites suggests that none of the biological indices of sex identity (see p. 56) "preordains" the direction of gender identity. Once gender identity is established, furthermore, anomalies of the phenotype do not alter the sense of gender. The boy who grows breasts and discovers that he has a womb and ovaries remains a boy; the girl who becomes virilized or discovers she has testes (TF) remains a girl. The same is true of normal people who receive heterotypical sex hormones for treatment of cancers (Money, 1961; Money and Ehrhardt, 1972).

Leading textbooks on human intersex conditions have adopted the view that gender role is differentiated postnatally from a state of sexual neutrality at birth (Grumbach and Van Wyck, 1974; Federman, 1967). They view the early psychological environment and the conformity of the genitals

to assigned sex as major factors in gender differentiation. This view was held originally also by Money and his colleagues, but during the past twenty years it has undergone some modification (Hampson, Hampson, and Money, 1955; Money and Ehrhardt, 1972). Though Money and Ehrhardt (1972) hold that:

. . . the period of greatest risk for errors of gender identity formation, of long-lasting effect in the brain, is after birth, and at around the time of acquiring the native language. . . , (p. 245).

they also suggest that:

. . . the central nervous system, insofar as pre-natal hormonal factors made it sexually dimorphic, passes on its program in the form of behavioral traits which influence other people, and which are traditionally and culturally classified as predominantly boyish or girlish. (p. 4).

They make the inference of brain dimorphism on the basis of both the animal research reviewed in Chapter III of this paper and their clinical studies of hermaphrodites carried out over the past 10 years. The authors point out that the androgenized genetic female "has no difficulty" differentiating a male gender identity if raised as a boy, while the female gender identity developed by such persons typically includes a "tomboyish temperament." The hypoandrogenized genetic male, on the other hand, is said to display a temperament which facilitates a feminine gender identity. It is variously described as "not sissiness," but as "a quietism against which much effort is needed in order to match the behavioral demands that boys put on one another" (Money, 1972, p. 3). One such boy was anecdotally termed as "unassertive, noncompetitive

loner" (Money and Ehrhardt, 1972, p. 153). The same authors state, however, that:

Boys exposed to an insufficiency of prenatal androgens are possibly rather placid, but the chief effect in them would seem to be after the age of adolescence. Then there is a deficit of male orgasm and male sexual initiative leading to arousal, for replacement androgen is usually needed (p. 245).

Even then, they note, the masculinizing effect is incomplete. A detailed discussion of this issue will be presented in Chapter V.

The attempt to ascribe a tomboyish temperament to androgenized children and a placid, unassertive, or uncompetitive cast to nonandrogenized children implies that the potential for these traits is organized in the brains of normal children. Though the authors believe that feminine potentials in animals are "inhibited" or "latent" in normal males and stress that in man this process "is probably not very complete, and is perhaps individually variable as well," they nevertheless lean toward the notion of greater bipotentiality in the genetic female and its resolution "in favor of unipolar masculinity" in the genetic male, through the agency of fetal androgens. This bias is particularly evident in the emphasis placed upon male differentiation as more difficult because something "special" must be added--that is, androgens.

In fact, the contrast of androgenized female to unandrogenized male is misleading, since all normal females are androgenized to a degree. Androgens are believed at present to be responsible for the growth of the external genitals and for the

sensitization of the perineal region in both sexes through the action of dihydrotestosterone (Wilson, 1972; Walsh, et al, 1974). They are thought also to be the "libido hormone" in both sexes (Money, 1961; Reichlin, 1971; Money and Ehrhardt, 1972; Waxenberg, 1959; Lunde and Hamburg, 1972). In Vitro studies suggest that both the adrenals and the ovaries produce androgens during the fetal period (Zaaijer and Price, 1971). Therefore, unandrogenized males--those who are insensitive to androgens or fail to form DHT in perineal tissues during the fetal period--really represent the "neutral sex" which develops in the absence of androgens (Federman, 1967). Turner's Syndrome (TS) females approximate the neutral sex since their failure to differentiate gonads deprives them of all fetal gonadal hormones--except for small amounts of androgens and estrogens secreted by the fetal adrenals.

Because of difficulties in measuring fetal androgen levels one cannot accurately place the various syndromes on a continuum in this respect. In humans, the parameters of timing, dosage, and type of hormone present during fetal life are not controlled. For example, masculinization of the genitals may be caused by excess adrenal androgens and other hormones or by synthetic progestagens. One judges the degree of androgenization by the degree of genital masculinization, but the animal data suggests that the two are not perfectly correlated--even when dosage and timing are controlled by injection (Ward, 1969).

Thus, though it is possible that the spontaneous development of the neutral female sex involves less risk than male development, it is not immediately apparent that the reproductive system of the normal female is "easier" to achieve, as Money and Ehrhardt (1972) suggest. In the neutral sexes, gonads are either lacking or are subject to rapid gonadal atresia. However, the half-million ova of the normal female complete the initial steps of oogenesis during the fetal period, after which they remain in a dormant stage for 12 to 40 years.⁴ In males, spermatogenesis begins at puberty (Mittwoch, 1973).

Furthermore, the traits typically seen in inter-sexes may represent adjustments to their genital abnormalities rather than sequelae to fetal hormonal effects on the brain. As was seen in the animal studies, the importance of an adequate male phallus to male mating behavior is primary. The evidence of hermaphrodite pairs, it will be recalled, indicates that fetal androgenization of the genetic female does not preclude gender identity formation as either male or female. Similarly, normal fetal androgenization of genetic males nevertheless permits a feminine gender identity--as in the case of the infant who suffered traumatic loss of the penis. However, the correlary of a genetic male, unandro-

⁴After puberty each oocyte proceeds through the meiotic divisions to form mature ova monthly over the 30 years of female fertility (Mittwoch, 1973).

genized fetally, and later assigned to either sex is rarely found. These patients are usually assigned as females for two reasons. First, the genitals are unambiguously female so tht diagnosis is often not made until late childhood or after, when inguinal hernia or failure to menstruate or conceive lead to the correct diagnosis.⁵ Secondly, it is not possible at present to construct a coitally adequate penis, though orgasmic capacity and desire are maintained in micropenis and amputation of the penis, as in clitoridectomy. Nevertheless, males with congenital micropenis or apenis and TF raised as males reportedly differentiate a male gender identity if assigned and reared as males, though it may be compromised (Stoller, 1968; Money, 1961; Money and Ehrhardt, 1972).

There does exist a syndrome of partial androgen insensitivity in genetic males in which the external genitalia are sufficiently ambiguous to permit assignment as males. Unfortunately, sufficient data on these males is lacking at present. Finally, there is no imaginable situation which would require the sex reassignment of a normal female. However, the available evidence provides no a priori basis for holding that

⁵When assignment of TF patients is made on the basis of palpable testes, the inability of the body to use androgens condemns such males to perpetual boyhood, since masculinizing puberty cannot be induced by present means (Money and Ehrhardt, 1972).

a normal female would be unable to establish a male gender identity within the normal range (with appropriate surgical and hormonal correction).

According to Phoenix, et al (1967) and Ward (1974), Nature's plan is to assure that a creature with the reproductive capacity of one sex possess the behavioral repertoire to reproduce. In humans, this repertoire is thought to include various traits and abilities which enhance sexual attraction and thereby survival. It is more clever, however, to assure enough flexibility of behavioral capacity to meet the vicissitudes of the developmental span from conception to sexual maturity, and the available evidence indicates that bodily dimensions remain bipotential until puberty is underway. For example, though there are minor mean differences in size and growth rate during childhood, no hormonal treatment is necessary until puberty in cases of sex reassignment. In fact, the sexes do not differ in the amount or type of sex hormones they produce until pre-puberty (Williams, 1956). The transformations in body build and contour achieved by pubertal hormone treatments are remarkable--even when puberty has already begun (Money and Ehrhardt, 1972). Even after pubertal hormones have arrested growth of the epiphyses and thus established the basic sexual cast of the body build, striking transformations are possible. It is unlikely that the brain would be organized or biased significantly toward masculine or feminine potentials during the fetal period

while the body remained totally plastic in its capacity for sex reversal (except, of course, reversal of genital morphology). Thus, Money and Ehrhardt (1972) state:

So much of gender-identity differentiation remains to take place postnatally, that prenatally determined traits or dispositions can be incorporated into the postnatally differentiated scheme, . . . (p. 103).

It remains to be seen whether such traits, in fact, exist.

3. Factors in Postnatal

Gender Role Differentiation

The organizational theory challenged the popular view of sex difference theorists that social learning models could account for the phenomenon of behavioral sex dimorphism. Maccoby and Jacklin (1974), in fact, present convincing evidence that imitation, identification, and reinforcement are not sufficient to account for the developmental changes in sex typing. They note, for example, that: (1) children's behavior is not similar to the behavior of the same-sex parent; (2) the choice of the same-sex model occurs after the appearance of sex typed behavior; and (3) children's display of sex typed behavior differs from that of adult models. Furthermore, they report that research on socialization shows few sex differences in parental behavior except in clothing, toys, and activities--with cross-sex behaviors and activities proscribed more often for boys. They believe that cognitive processes of the type described by Kohlberg must also play a role.

Kohlberg (1966; 1974) proposes that sexual attitudes (or gender identity) develop from the child's own cognitive organization of his experience rather than from the direct influence of social agents, instinctual wishes, or perceived genital differences. In his view, the attainment of gender constancy around age 6 or 7--parallel to the capacity for concrete operations and conservation--represents the true beginnings of gender identity. At this stage, the child assigns value to gender differences based upon deductions from physical attributes. In this scheme, males are more highly valued because of their size, strength, and aggression. Later, values are linked to social duties and finally to personal choice based upon values of mutuality and equality between the sexes.

There are a number of problems with this analysis. First, Kohlberg's emphasis on gender constancy draws attention from the events of the first years of life to those of middle childhood when the roots of gender differentiation are already obscured. His interest in gender constancy, of course, derives from his attempt to demonstrate the application of Piagetian principles to gender identity formation. Like Piaget, he stresses the logical, secondary process aspects of gender formation at the expense of the primary process fantasies and passions which accompany the development of self-other differentiation, attachment, and separation-individuation (Segal, 1964; Galensen and Norphe,

1974). Following the cognitive scheme, for example, one first begins to speak of gender awareness at age 2 or 3, when children label their gender correctly and others' with fair accuracy. Money and Ehrhardt (1972), however, stress that gender reassignment is best begun before 18 months of age, when gender identity formation is well underway. (Only when sex of assignment and rearing have been ambivalent do later reassignments have good prognosis.) They point out, furthermore, that parental counseling is required as early as 12 months to negotiate a successful reassignment because of the involvement in gender dimorphic rearing.

After the age of eighteen months, parents need still more frequent help. Even when this help is extended to the child, the problems of cognitive discordance may be incapable of resolution without some lasting residual signs of psychosexual impairment relative to reassigned sex (p. 179).

That sex reassignment is risky soon after the appearance of language is not coincidental, for thereafter the infant's own experience is filtered through the label of boy or girl with their associated cultural and familial values. Kohlberg (1974) himself speaks of a verbal child when he notes that gender--as the only basic category to which a child assigns itself--is of major importance in organizing its "social perceptions and actions" (p. 210).

Secondly, Kohlberg's scheme does not apply so well to girls. He argues that the young child's adoption of sexual attitudes derives from (1) his or her belief that there is little choice about sex typed roles; and (2) the "natural

tendency" to value the self and things like the self. There is ample evidence, however, that girls decline in self-valuation between the ages of 5 and 10. By the age of 4 or 5, the main role is already recognized as more prestigious and powerful and from then until pre-puberty, girls' value of their own sex decreases, they more often choose boys' toys and activities, say they are boys, and decrease in their identifications with their mothers (cited in Kohlberg, 1966, 1974 and Hetherington, 1970). During these same years, boys maintain preferred masculine interests and toys and reject girls' companionship and activities. Both sexes rate boys and men as "most worthwhile" at ages 8 and 15.

Kohlberg explains the failure of self-valuation in young girls as a correct deduction based upon the perceived physical attributes of men--that is, men's greater size, strength, and energy are thought to reflect psychological attributes of greater intelligence, aggressiveness, power, and dominance. It is not entirely apparent, however, that these are universal deductions and independent of transmitted cultural and familial values. When groups differ in actual power and prestige, their distinguishing physical characteristics are most likely "perceived" as the basis for the greater valuation of the powerful group. In fact, American children of both sexes appear to value dominance and power. Hetherington (1970), for example, found that both sexes imitated and were more similar to the more dominant parent--

whether male or female. Girls in father-dominant homes adopted the feminine sex roles more slowly and ambivalently than boys, who preferred the male role from the age of 4 or 5. The boys in mother-dominant homes--unlike girls with dominant mothers--were less likely to adopt the appropriate sex role. Hetherington concluded that the factors of parental behavior, social pressure to conform, and the cultural status of the sexes determines the rate and type of sex role development.

The foregoing discussion illustrates the problems with a purely cognitive theory of gender identity formation. Obviously emotional acceptance of the role to which one is assigned varies with the culturally-defined content of the role and may lag behind cognitive awareness of gender.

There is much evidence that gender awareness is present by the time language appears and perhaps earlier (Lewis and Weinraub, 1974). If so, sex is implicated in the process of self-object differentiation and in the development of object relations. Does the infant bring a uniquely male or female sensibility to its interactions with people and things, or do parents and others respond to the infant on the basis of sex as an attribute? Hermaphrodite evidence indicates that gender identity conforms to the sex of assignment and rearing, even when assignment is discordant with the biological indicators of sex. In these cases, there is no indication that biological factors preordain gender identity. The high

risk for ambiguous gender identity development associated with ambiguity of assignment and rearing suggests that parenting factors are crucial variables. Let us consider briefly, then, some aspects of parent-child interaction during the first years of life.

The research on mother-infant interaction during the early years has yielded three major findings: (1) there are few sex differences in mean frequency and variance of infant behaviors; (2) parents differ in their behaviors toward the infant as a function of sex; (3) there are different correlational trends in behavior for male and female infants; and (4) significant individual differences are found routinely in infant behaviors. In neonates, for example, significant individual, but not sex, differences are found in three behaviors which are thought to be central to the establishment of attachment: (1) arousal state, including the cry; (2) visual alertness, the precursor of eye-to-eye contact; and (3) soothability, the response to ministration (Korner, 1974). Also, no significant sex differences are found in activity or among a broad variety of sensory modalities. The only difference in infant behaviors found over the early months is greater irritability or fussing in males, which has not been consistently replicated--perhaps because it is a function of socioeconomic class (Moss, 1970; 1974; Lewis, 1972; Lewis and Weinraub, 1974). However, mothers are observed to treat their infants differentially by sex.

Moss (1970), for example, found that when fussing and crying was controlled, mothers of boys aroused and stimulated their infants more and mothers of girls imitated, or reinforced, vocalizations more. Analysis of the location and behavior of mothers during infant fussing showed that they more often "hover over" younger infants (3 weeks) of both sexes and boys of 3 months. Female infants appeared to protest for shorter periods and to quiet themselves without ministrations (Moss, 1970, 1974).

Moss' (1974) structured studies lent "moderate support" to the idea that visual regard and vocal conditioning at 3 months may be influenced by infant state in males and social experiences in females, but he viewed "stereotyped sex-role attitudes" as the major factor which "shaped the parental behavior" (p. 157). He emphasized that sex differences were not striking enough to "characterize the infant in a categorical way"; the major differences involved "merely different correlational trends. . ." He suggested, in addition, that the few differences found may be evident only when external conditions do not insulate males adequately (see p. 60) and are most likely magnified by the sex-role expectations of parents. This view represents a shift in Moss' thinking since 1970, when he argued that differential parental treatment is initially shaped by infant behaviors.

Lewis (1972), too, found that 3-month-old boys are held and girls are vocalized to significantly more often by

their mothers, even though the infant behaviors associated with their mothers' behaviors did not differ according to sex. Furthermore, when Lewis analyzed the direction of the interaction, he found that the 3 infant behaviors which elicit maternal responses--that is, the fret or cry, gross movement, and play--did not differentiate infants by sex. Mothers, however, responded with a wider variety of behaviors to boys and focused on distal, social behaviors with girls. Infant vocalization and smiling--which were more often responses to mothers' initiations--were also not differentiated by sex. However, when directed vocalizations were analyzed it was found that girls responded more often (63%) than they initiated dyadic vocalization, while boys responded and initiated vocalizations equally. This finding suggests that girls may be more vocally responsive in the first months of life than boys, perhaps because of earlier maturation of the left hemisphere of the brain (Kagan, 1971; Lewis, 1972). Kagan (1971) observed that girls tend to vocalize when excited or interested, while boys vocalize when distressed--another indication that girls may be more mature and receptive to social interaction. The same conclusion is suggested by Hittleman's (1975) finding that 2-day-old girls maintained longer eye-to-eye contact than boys, although the sexes do not differ in the number of latency of eye contacts. These subtle differences may lay the basis for earlier social interaction between girls and their mothers.

Indeed, Kagan (1971) argues that these subtle sex differences in patterning and stability may be expressions of biological dimorphisms. The source of these "predispositions" is thought to lie not in dimorphic brain organization of the type proposed by the organizational theory, however, but in the relative protection afforded females by the double X chromosome or in the supposed increased risk of error in male development because of the need for sensitive period androgens (Kagan, 1971; Money and Ehrhardt, 1972; Singer, et al, 1968; Sackett, 1974). According to this general point of view, the female is developmentally advanced and is "buffered" against environmental insult, while the male is relatively more vulnerably to environmental deprivation and less mature.

The relative vulnerability of the male is well documented by human and animal data. Girls are more advanced skeletally and neurologically at birth by at least "2 weeks" and more so as development progresses (Tanner, 1974). Boys account for more conceptions, abortions, still births, infant mortality, susceptibility to disease, mental defects, and learning and behavioral disorders (Singer, et al, 1968). Apgar levels below 6 were more common in boys in a study of 15,000 children reported by Singer, et al, (1968), and boys accounted for three quarters of the almost 200 abnormalities examined. At 8 months, the boys were significantly poorer in mental, motor, and final diagnostic indicators--although

they were better on the social-emotional scale--and at 12 months, again, boys were less advanced neurologically. Small samples of 71 to 100 children revealed significantly poorer speech and hearing development in 3-year-old boys and psychological development in 4-year-old boys. However, no sex difference was found on the Stanford-Binet I.Q. at 4 years. The authors attributed these differences to the relative biological integrity of females conferred by their sex chromosome constitution, though they allowed that incompatibilities between the male fetus and its mother may play a role.⁶

Primate research documents profound sex differences in infants' responses to deprivation beginning in the neonatal period. Sackett (1974) found that male rhesus monkeys are significantly more affected by maternal and social deprivation than females. Partial and total isolate males showed significantly more self-aggression, less exploration in a novel environment, more aggressive and fearful behavior, and a lower survival rate in a wild environment (33% to 78% of females) although socially-reared animals displayed only minor sex differences on these variables. Moreover, the females were

⁶It is presently believed that one X chromosome in the female and the Y chromosome in the male are genetically inactive, except for a small part which may govern fertility. According to the Lyon hypothesis, inactivity of the second X chromosome of the female is random in any given cell except that a faulty X chromosome is always inactivated. Thus, the female needs 2 recessive alleles to manifest the trait, while boys need only 1, having no dominant allele to suppress it (Mittwoch, 1967).

not merely less devastated than males--they approached normal behavioral levels.⁷ The author hypothesized that males were deficient in the ability to inhibit inappropriate responses rather than in perceptual or learning ability. He suggests that females may develop the physiological basis for response inhibition prenatally or may not need postnatal sensory input to develop inhibition because of biochemical differences pre or postnatally.

In rats, the adult mating behavior of male offspring of mothers stressed during pregnancy and of male neonates stressed postnatally was adversely affected (Ward, 1969). Female mating behavior of females was not affected, but they did not show the mounting behavior typical of females. The males showed a decline in ejaculation and an increase in lordotic responses to E and P injections, suggesting that the increased maternal production of adrenal hormones in response to stress interfered with the production of androgens necessary for masculinization. Bodily morphology appeared to be that of normal males, though Ward failed to measure the more sensitive indicators of androgenization. The behavioral effect, moreover, was not permanent, since TP and mild electric

⁷Since females living in laboratory conditions attack other females more often than males attack males or females, the increased aggression of deprived males is most likely not due to a sex difference in aggression (Sackett, 1974).

shock in combination induced ejaculatory levels equal to those of control males in 60 to 90% of the experimental animals. However, a few of the males remained responsive also to E and P and were behaviorally bisexual, depending upon the eliciting hormones.

Finally, Money and Ehrhardt (1972) believe that the greater incidence among males of gender identity disorders--obligative homosexuality, transexuality, transvestism, and paraphilias--may reflect yet another manifestation of male vulnerability, based possibly in errors of fetal hormonal balance.

There is thus considerable evidence suggesting that the precocity-vulnerability thesis may have some bearing on early development. At present, its implications are a matter of speculation, however. Kagan (1971), for example, proposes that girls' presumed cognitive precocity and earlier language acquisition may cause them to: (1) show fear earlier and more intensely; (2) be less variable on psychological dimensions, so that they are more easily socialized and thus form greater and longer maternal attachment; and (3) be less active motorically. However, none of these hypotheses is supported by data (Maccoby and Jacklin, 1974; Kagan, 1971; Lewis and Weinraub, 1974). It is an odd twist of logic, moreover, that precocity should lead to a relative weakness. This type of thinking is most evident in Kagan's (1971) notion that because left hemisphere dominance--and hence, language

skills--is initially more advanced in girls, boys develop a "more even development of mental talents" during early childhood, with spatial skills, elaborated in the right hemisphere, developing to higher levels in them.

It should be added that even the few sex differences which do appear seem rather at odds with what one might predict on the basis of the precocity-vulnerability scheme. Let us consider attachment behavior. Lewis and his colleagues have gathered some evidence that the mode of interaction between parents and infants may follow a different course in males and females--though the findings are mainly non-significant trends. At 3 weeks and 3 months, the mother-daughter dyad tends toward distal modes (looking, vocalizing), while mother and son interact in proximal and distal modes equally. Boys, however, receive significantly more proximal stimulation than girls. By 6 months, girls receive more stimulation of both kinds, while the amount of proximal contact received by boys declines. By ages 1 and 2, the sexes do not differ in the mean frequency of proximal and distal behaviors directed towards the parents. Both show a non-significant decline in proximal behaviors toward mother and a significant increase in attempts to be close to father between the first and second years. Both also show a significant shift to more distal modes of interaction with both parents; increases in vocalization to father increase most markedly. However, a sex difference is evident within this matrix. At 1

year, girls focus more attention on mother, while boys direct proximal behaviors toward mother but look more at father. By age 2, both sexes show a non-significant shift from touching to looking at father, but only boys make the same shift--and at statistically significant levels--towards mother. Lewis and Weinraub (1974) propose that this pattern occurs because of parental reinforcement. Boys are discouraged from maintaining proximal contact with mother and father because of the cultural values of male independence and negative sexual-homosexual implications of touching. Girls are discouraged from such contact with father for sexual reasons, but are permitted to maintain such contact with their mothers. This interpretation concurs with Goldberg and Lewis' (1969) finding that the amount of proximal behaviors initiated by mother at 6 months is related to the amount of such behaviors sought by the infant at 13 months in a free play situation. Girls, who received more such handling at 6 months, sought it more in the later situation. However, the same pattern was found also among the boys.

Another implication of the precocity-vulnerability notion is that one should see stronger social class differences among boys if vulnerability is exposed in a more deprived environment. However, such differences are found more often in girls. For example, middle class girls are significantly advanced in language, reading skills, and the Embedded Figures Test performance compared to lower class girls (Kagan, 1971). He states:

Lower class mothers of daughters project their greater sense of impotence on to them and are less likely to stimulate, encourage, or reward their daughters' simple accomplishments (p. 185).

At 4, 10, 27, and 48 months, middle class mothers are observed to talk to, entreat, and chide their daughters when teaching them a new task more than lower class mothers. Class differences, furthermore, increase with age and are related to such variables as language, attention span, and problem solving but not to smiling, activity level, spontaneous verbalization and other personality variables.

Some have suggested that male vulnerability is not a major determinant of behavior under ordinary conditions, but is exposed only in a deprived environment (Maccoby and Jacklin, 1974; Moss, 1974). However, the absence of marked infant sex differences suggest that the deprivation must be profound to significantly affect males.

Infant differences are not found in average frequency or distribution of behavior, but in correlates of behavior, stability over time, and parental behavior. This raises the question whether the sexes are predisposed to respond differently to same-sex and opposite-sex persons. This intriguing hypothesis is suggested by several findings.

A number of studies, for example, suggest that eye regard is a function of the sex of the infant and the social object. Lewis and Weinraub (1974) report that infants of 10 to 18 months looked longer at slides of same-sex infants. Girls show this effect 3 months before boys and more strongly.

Infants were also able to differentiate opposite-sex--but not same-sex--pictures from pictures of themselves. Other data reviewed by the authors indicates that infants vocalize more at 1 year to the same-sex parent and look more at same-sex peers during play at 1 to 1-1/2 years. At 2 years, boys look significantly more at their fathers and girls at their mothers in a play situation.

Until these cues used for differentiation have been understood, the meaning of this behavior cannot be known. However, the possibility that pre-verbal infants perceive gender differences cannot be discounted. It is increasingly evident that infants are more advanced than previously thought. Hittleman (1975), for example, has found that 95% of eye-to-eye contact is maintained by the neonate. A biological predisposition to respond to same-sex objects would greatly enhance the survival of females, and since our primate and hominid heritage rests on a same-sex social order, early gender perception may represent an evolutionary adaptation.

Alternately, differential eye regard may represent a self-other differentiation (Kohlberg, 1974). Girls' relative precocity in interaction with mothers' differential behaviors may enhance earlier self-other perception in girls. That self and gender may be associated as early as 10 months is not surprising, for genital awareness is present between 7 to 10 months and is responded to differently by mothers (Galenson and Roiphe, 1974; 1975). In boys, genital play begins at 7 months

and continues until and after the onset of true masturbation at 15 months. Girls begin genital play at 10 months, after which their play declines or continues indirectly until the onset of true masturbation at 15 months. Following the discovery of genital difference at 15 months, girls have more severe reactions than boys and either stop masturbation, continue without pleasure, or continue indirectly. It is probably not incidental that most girls in this study had not been supplied a name for the genital or one which did not differentiate between urinary, anal, and genital functions. In any case, even before the appearance of language, infants have experienced different genital awareness and maternal responses to it on the basis of sex.

In summary, the identification and significance of important factors in postnatal gender role differentiation remains a matter for speculation. Taken as a whole, the data points to parental behaviors as the major catalysts of sex-differentiated behavior in infants. The relative cognitive and verbal precocity of the female may contribute to the development of sexual dimorphism, but its impact appears to be minor.

CHAPTER V

SEQUELAE TO ANDROGENIZATION OF THE GENETIC FEMALE: A COMPARISON OF GENDER ROLE IN ANDROGENIZED AND NON-ANDROGENIZED INTERSEXES

1. Introduction

In the present chapter, we shall look more closely at the quality of gender identity formation in various androgenized and non-androgenized intersexed patients and their controls. The data discussed in the following pages comes from the research of Money and his colleagues. The reader may refer to the Glossary and Table 6 for details of the syndromes. Table 7 describes characteristics of the samples discussed here.

2. Tomboyism

Intense Energy Expenditure

The most ubiquitous feature of fetally androgenized genetic females is "vigorous outdoor activity" of "intense energy expenditure" in sports and games. Roughly 70 to 90% of androgenized girls are reported to have long-term interest in such play as compared to about 33% of matched controls (.10) and 10% of unaffected female siblings of AGS boys and girls (.001). This is not a general elevation of activity level--

Table 7.--Some characteristics of intersex samples

Name of Sample	Sample Size		Approximate Mean Age (and range)	Percentage Treated within 1st year
	Patients	Controls		
Fetally Androgenized Genetic Females				
EAS	15	15	10.6 (5.2-16.1) 10.8 (5.9-15.2)	86 (cortisone)
AGS Sibling study	17	11	10.8 (4.3-19.9) 12.9 (6.8-24.7)	83 (cortisone) 35 (surgery)
LAS	23	--	26 (8-47)	0 cortisone after age 8; surgery 2-47 years; 96% diagnosed at birth
PIH	10	--	unknown (3.9-14.3)	60 diagnosed (40 diagnosed between 1-1/2- 3 years)
Fetally Androgenized Genetic Males				
AGS Sibling study	10	16	11.8 (4.8-26.3) 13.2 (6.8-23.7)	60 (cortisone)
Preco- cious Puberty	14 AGS 4 IPP	--	unknown	0 All were seen at least 4 years after onset of puberty (as early as 3 years)

Table 7 continued

Name of Sample	Sample Size Patients	Size Controls	Approximate Mean Age (and range)	Percentage Treated within 1st year
Fetally Unandrogenized Genetic Males				
TF	10	--	16 (7-27)	0 40 between 5 and 13 years; 60 after 15 years; Gonadec- tomy--15.6 (6.3-21.1) Estrogen--16.0 (11-21); 80% required vagina repair
45 X,O Genotypes				
TS	a) 73 b) 15	15	15.2 (12-20) 12.6 (8-16.5)	Unknown

*See Glossary for definition of samples.

which, in any case, does not significantly distinguish between normal boys and girls (Maccoby and Jacklin, 1974; Kagan, 1971)--nor hyperactivity, which is found more often in boys (Maccoby and Jacklin, 1974).

The energy-enhancing effect is not confined to genetic females with AGS; AGS males were also significantly different from unaffected male siblings. Some 80% of male patients showed long-term intense activity as compared to 18% of the male siblings (.05).

Unfortunately there are no normative samples of the frequency of this pattern in normals. However, some 60% of the matched controls of EAS girls showed vigorous outdoor play for periods of two to three years during childhood, so that 93% of this control group were highly active during part or all of childhood. Thus, the distinguishing characteristic is the long-term aspect. Furthermore, only a minority of the sibling samples report long-term vigorous play and mothers of AGS patients did not differ from the EAS matched controls, with 40%. Since sample sizes were too small to permit comparisons of AGS with their siblings by sex, it is not known what effect highly active patients might have on male or female siblings.

Data on non-androgenized intersexes on this variable are not complete. TS females, whose only source of androgens is via the adrenals, show significantly less vigorous activity than normals. This question was not studied in TF women, who

are insensitive to androgens. Though they tend to be "ultra-feminine" in gender identity, 20% of the sample were rather tomboyish. Hypogonadal males were also not studied as a group.

Since both males and females with AGS are similarly distinguished from their same sexed peers on this variable, the effect is properly not a "masculinizing" one implying dimorphic brain organization, but may reflect an alteration of metabolism. One might postulate an effect on oxygen consumption or nitrogen metabolism (which contributes to muscular strength and energy). In support of this hypothesis, Money (1961) noted that both hypogonadal males and AGS females after cortisone treatment report a loss of strength, reflecting perhaps a decline in nitrogen metabolism. This has not been tested systematically, however.¹

Furthermore, it has not been established that this trait is potentiated during the prenatal period--though the fact that the same pattern is reported in PIH girls, in whom exposure to progestins ends at birth, suggests that prenatal factors are primary. However, the PIH sample was of high socio-economic level and a significant proportion of the mothers and siblings of these girls were tomboyish, leading the authors to suggest that tomboyism was:

¹Personal Communication from M. Schwartz, Research Associate, Johns Hopkins Hospital, 1974, 1975.

. . . a frequent characteristic in the development of middle-class suburban and rural girls who have both the space and the tradition of the outdoor life (Ehrhardt and Money, 1967, p. 96).

Furthermore, boys and girls who were exposed to progestagens during fetal life--without a masculinizing effect on the female genitals--were not advanced in physical education skills, though they did stand and walk earlier than their controls (Dalton, 1968).

In the AGS samples reported in this paper, "early treatment" was considerably delayed.² According to Van de Weile (1974) cortisone replacement is "frequently not adequate" in children with AGS. He estimates that 10-20% are difficult to regulate and thus are allowed slightly higher androgen levels since too much cortisone would prematurely arrest growth. Since androgen levels are almost undetectable throughout childhood and are at equal levels in the sexes (Williams, 1956; Donovan and Van der Werff Ten Bosch, 1965), higher levels might contribute to precocious growth, masculine body build, and muscular strength and energy sufficient to account for the "vigorous outdoor activity" seen in AGS children.³ Moreover, the assumption that this effect is

²Early-treated girls: 83-86% treated within first year; Early-treated boys: 60% treated within first year (Ehrhardt, Epstein, and Money, 1968; Ehrhardt and Baker, 1974).

³M. Schwartz suspects there are body build differences in AGS girls as a result of wrong dosage or failure to take the medication (personal communication, 1975).

caused by androgens may be premature. In AGS, androstenedione--not testosterone--is the major steroid produced. Large amounts of 17-hydroxyprogesterone and estrogens are excreted along with other steroids (Vande Weile, 1974).

It is possible, of course, that the same objective level of activity is rated--according to cultural stereotypes--as more intense in girls and less so in boys. The energy level of boys with precocious puberty (of either idiopathic or AGS origin) was characterized as "commensurate with physique, not age" (Money and Alexander, 1967). The descriptions by mothers of PIH of their daughters' "most tomboyish traits" suggest that sex-role expectations do affect the ratings of this variable. They include interest in baseball, liking to run and jump, playing with boys, playing with guns and soldiers, getting so dirty outside and not caring, and not being charming (Ehrhardt and Money, 1967).

It is not clear, then, whether vigorous outdoor activity is a sequel to fetal androgenic effects on the brain, on metabolic processes fetally or postnatally, and/or a sequel to rearing factors. Among the latter, one might postulate an unconscious doubt concerning the unequivocal femininity of a girl born with ambiguous genitals which is manifested in greater permissiveness to pursue athletic pursuits--or in proscipition and counter-rebellion. Further studies of PIH girls of lower socioeconomic levels would aid in resolving this question. Observational studies of PIH and AGS girls would be ideal.

Dominance, Competitiveness, and Aggression

The psychological research is unclear as to whether the sexes differ in competitiveness and dominance but it is well-established that boys are more aggressive beginning in early childhood and declining with age (Maccoby and Jacklin, 1974). The findings of androgenized children go counter to the hypothesis that these traits are the result of a fetal androgen effect upon the brain. As compared with their controls, fewer EAS girls fight back if attacked (45 to 73%)-- though fewer also withdraw when attacked (6 to 20%) (Ehrhardt, Epstein, and Money, 1968). Only 6% of each group was said to start fights. Since no information was available on 39% of EAS girls, however, these findings are inconclusive. It is probably significant that both PIG and AGS boys and girls did not differ from their controls on the variable of "starts fights." Ehrhardt and Baker (1974) are studying this question in more depth.

The strongest evidence against this hypothesis comes from studies of boys with precocious puberty (of AGS or idiopathic origin) who were untreated until puberty had been underway for at least 4 years. The boys show a:

. . . relative infrequency of aggression in all the patients as a group . . . the significant lesson from these precocious boys is their lack of aggressive attack and their outstanding gentleness with other smaller children whom they could easily have battered when provoked by them . . . instead of attack, one saw almost a parental concern for smaller members of the species and a setting aside of combat with larger ones (Money and Alexander, 1969, p. 115).

In explanation of this seeming paradox, the authors point to the social adaptation required of these boys, who face the problem of being years ahead of age mates in physical growth yet retarded in social age relative to older boys who are their physical peers.

As regards competition and dominance assertion, again no differences have been found between androgenized girls and their controls. Though Money (1972) wrote of a "capacity for competitive rivalry and dominance assertion in boy's play groups," he contradicts this in his book (Money and Ehrhardt, 1972):

It appears that the girls of the two diagnostic groups did not strive for dominance in competition with boys, and they were not interested in the rivalries of other girls (p. 100).

The authors suggest that rivalry was inhibited because the girls were "socially identified as girls" and might be rejected from boys' play groups if they attempted to assert dominance. Again, an explanation based upon adaptation to prescribed gender role behavior is offered to account for the lack of "masculine" behaviors in androgenized girls.

Clothing and Personal Adornment

Assuming a great interest in outdoor play, one would expect other aspects of tomboyism to follow naturally. Thus, one is not surprised that such girls prefer "masculine-

derived clothing"⁴ and an emphasis on the functional--especially during middle childhood. A majority of EAS (58%) and PIH (80%) girls preferred boys clothes but were not averse to dresses and frills. About one-third of EAS girls wore mostly girls' clothing as compared to about three-fourths of their controls. Approximately the same proportion of PIH girls and EAS controls preferred girls' clothing (20-25%) compared to only 6% of EAS girls. In the study of AGS versus their siblings, the question was put differently--namely, in terms of their preference for the functional over the attractive. Here, AGS females differed significantly from their female siblings in that 78% preferred the functional compared to 20% of siblings and 47% of mothers. Among the boys, surprisingly, both AGS and their siblings were alike in that half preferred the functional and half the attractive--an indication that concerns with attractiveness are not dimorphic (Ehrhardt and Baker, 1974). PIH were not unlike their siblings and mothers in their preference for pants--reflecting perhaps the upper middle class bias of the sample. Finally, Allen's (1975) data on college women found figures in clothes preference closer to that of androgenized girls than to normal controls.⁵ Jeans and functional clothes were preferred by 64%, dresses by

⁴The term refers to pants and shirts made for girls, not to actual boys' clothing.

⁵Personal communication from J. Allen, unpublished doctoral dissertation, 1975.

23%, dresses exclusively by 2%, and pants exclusively by 4%. Many commented that they switched to masculine clothing only after school rules relaxed, permitting them to wear pants.

Interest in jewelry, make-up, and hair styling did not differentiate EAS or PIH girls from their controls. However, both AGS boys and girls differed significantly from their siblings in that 55% had no interest in adornment compared to 10% of the sibling samples. TS females also expressed significantly less interest in adornment than their controls--a reflection, perhaps, of their feelings of unattractiveness (see p. 82).

We can conclude that the preference for pants and comfortable clothing is a function both of a life style of vigorous activity and of cultural rules regarding dress.

Preference of Boys as Playmates

One might also expect that girls with strong interests in sports and games would seek out like-minded peers. That these tend to be boys is not surprising, considering the restricted opportunities for girls to pursue athletic interests. Perhaps with the new emphasis on girls' sports in the past five years, this "trait" will not be apparent in the future. In any case, one-half of AGS girls preferred boys as playmates and EAS and PIH girls were significantly different from their controls in this respect.

Satisfaction with Gender Role

Finally, 35 to 52% of the AGS samples were ambivalent or thought they would have preferred to be boys in response to the question "is it better to be a boy or a girl"? compared to 6% of controls and 10% of AGS mothers.⁶ PIH, however, were not different from controls in this respect. None of the AGS girls, moreover, was unhappy to be a girl, believed she had been assigned to the wrong sex, or wished to change her sex. AGS boys, AGS siblings, TF, TS, and PIH were not different from their normal controls in their rated satisfaction with gender role.

If one assumes that AGS and PIH are similarly affected by the androgenic fetal environment, how can the differential satisfaction with the female gender role be explained? Firstly, dissatisfaction may stem from bodily concerns related to reproductive function or sexual attractiveness or from a more generalized concern about freakishness. The PIH condition, in these respects, may be more benign. Federman (1967), for example, states that genital masculinization is less pronounced in PIH--although all degrees of masculinization are possible. Diagnosis of the present sample was somewhat earlier on the average, but the samples do not appear to differ on the impor-

⁶Of the EAS sample, 33% were ambivalent and 20% wished to be boys. These responses were combined in the AGS sibling study (35%). Of the AGS mothers and TF samples, 10% were in the combined category.

tant factors of age of clitoridectomy, hypospadiac repair, or vaginal exteriorization. A major difference between PIH and AGS is that PIH require no further follow-up (except when vaginal exteriorization is required) after genital repair, while AGS must be regulated on cortisone and take daily medication. Furthermore, though PIH and AGS girls are fertile, AGS, as a genetically inherited condition, may arouse more concerns about normalcy. This is supported by the fact that AGS boys appear to be more concerned than male siblings with issues of sex, marriage, and fathering (see Part III). Secondly, the extreme elevation in I.Q. of the PIH sample, relative to both the population norm and other AGS samples, and the upper middle class bias of the sample may have contributed to a better adjustment to their condition. Most of the PIH girls (80%) were considered by parents and examiner as "socially mature, . . . easy to get along with, flexible, and reasonable . . . self-assertive and independent . . . and self-reliant . . ." (pp. 94-95). Thirdly, the fact that the mothers and siblings of at least half the sample were themselves tomboyish would be expected to enhance satisfaction with gender role.

In conclusion, it may be noted that the tomboyism characteristic of androgenized girls neither forms a coherent unity within the two syndromes, nor is consistent between them. The only truly distinguishing trait is that of increased vigorous activity--which is characteristic of AGS boys as

well. The more superficial aspects either do not distinguish androgenized girls from normal girls or may be viewed as normal concomitants of outdoor interests. Finally, the traits of aggression, competition, and dominance--which are the hallmarks of the masculine stereotype--appear in neither hyperandrogenized girls nor boys. If failures of prediction are excused by appeal to the argument that gender role demands override sexually dimorphic predispositions (Money and Ehrhardt, 1972), then one cannot hold that such predispositions are responsible for gender role behavior in normals (Diamond, 1968). In fact, on the basis of cultural sex-typing, one would expect to see less cross-sex-typing in hypoandrogenized genetic males raised as boys than in androgenized genetic females raised as girls. Although the data on males is sparse, it appears to support this prediction (Stoller, 1968; Money, 1972; Money and Ehrhardt, 1972).

3. Maternalism

Tomboyism in androgenized girls may represent a masculinizing effect of androgens on the fetal brain, according to Money and Ehrhardt (1972). This same concept, they suggest:

. . . may apply also to the inhibition of pathways that should eventually subserve maternal behavior. More correctly, one might say partial inhibition of these pathways, for normal males are capable of paternalism, much of which is identical with maternalism, both being manifestations of paternalism or caretaking (p. 103).

Before considering the hermaphrodite evidence, let us briefly review aspects of maternal behavior in non-human mammals. The capacity for maternal behavior is functionally organized in both male and female brains in regions of the medial preoptic area. Fisher (1956) elicited all elements of maternal behavior in normal male rats by stimulating loci in the medial preoptic with minute amounts of sodium testosterone sulphate. Behavioral elements occurred in the absence of appropriate stimuli, but the presence of pups permitted the integration of behavioral elements into normal sequences. Controls tested with neural excitants, saline, and electrical stimulation failed to produce similar responses.

Research on the effect of perinatal hormone environment on later maternal behavior is lacking. Quadagno and Rockwell (1972) tested adult maternal behavior in normal males and females, neonatally androgenized female and neonatally castrated male rats. Though control males and females differed in the display of licking, crouching, nest building, and retrieving, the sole effect of androgen was to reduce retrieving behavior. On other variables, androgenized females did not differ from normal females nor castrate from normal males. Phoenix and McGee (1974, cited in Maccoby and Jackson, 1974) note that immature monkeys show all components of maternal behavior except retrieval, seen only in females.

In many species, male aggression towards infant precludes caretaking responses. Rosenberg, et al (1971), how-

ever, found that aggression--causing death--gave way to nurturant behavior when adult males were exposed to several litters. Goodall (1971) noted strong interest in infants in some adult male chimps and hypothesized that the occasional aggression of these males had led mothers to adopt general rules against infant handling by grown males. One may conjecture that in humans this "instinctual" aggression has all but disappeared under the impact of the cultural elaboration of the paternal role. It persists, of course, in parents who as children were battered.

In animals, the sex differentiated responses are elicited, in part, by hormones present during the perinatal period. In both rats and macaques, for example, mother-infant separation shortly after birth greatly attenuates maternal responses (Sackett and Ruppenthal, 1974). There are also indications that maternal behavior is not automatically released by parturition hormones, but requires repeated experienced. Moltz, et al (1971, cited in Maccoby and Jacklin, 1974) ovariectomized primiparous and multiparous females and delivered the infants by caesarian section. Maternal behaviors were disrupted only in the primiparous, suggesting that prior mothering experience was more important than hormones in releasing and maintaining maternal behaviors.

Money and Ehrhardt (1972) hypothesize that the trait of maternalism is first manifested in doll play and later in interest in infants and their care. If this trait appears

as a sequel to effect of fetal hormonal environment, one ought to see it clearly in non-human mammals. Sackett and Ruppenthal (1974) studied the attraction of adult female macaques to neonates. Given a choice of adult, 3-year-old, 1-year-old, or neonate females, only multiparous, feral-reared subjects preferred the neonate. Nulli and primiparous, feral-reared subjects had no significant preferences, and laboratory-reared subjects preferred the adult females, regardless of parity. Thus, it would seem that: (1) no "natural" preference for neonates exists, even after one birth and mothering experience, despite a "feminine" fetal environment; (2) the experience of mothering plays a greater role than the hormones of fetal life, puberty, or parturition (supported by the lack of preference among nulli and primiparous females); and (3) laboratory-rearing may negate the effects of parity and thus raises questions about the application of research results on laboratory-reared to feral animals in general.

Rearing conditions also affected the attractiveness of the mothers' own infants. Feral-born multiple mothers, natural feral-born mothers, and motherless mothers lived with their infants for 8 months and then were separated for 2 weeks. When provided a choice between their own or other infants, only natural mothers preferred their own. However, these mothers preferred adult females to infants when given the choice. Furthermore, when mothers' living quarters were

restricted so that interaction with other adult females was not possible, there was a "marked reduction" in infant attractiveness, which did not occur if adult female interaction occurred.

Returning now to the human evidence, let us examine the hypothesis that maternal behavior-mediating pathways are partially inhibited when fetal androgens are present. Money and Ehrhardt (1972) hold that "girishness" is expressed in childhood play and fantasies of romance, marriage, and maternal role. "Developmentally, maternalism appears first, in connection with its rehearsal in doll play" and in interests in infants and their care in later childhood (p. 101). The three groups of Androgenized girls differ from controls and female siblings in their relative lack of interest in dolls and other girls' toys.⁷ Some 20 to 40% of androgenized girls played with girls' toys primarily as compared to 80 to 90% of controls. TS and TF girls did not differ from their controls.

On other measures of maternalism, however, three of the diagnostic groups contradict the authors' predictions. These are TF and PIH females and AGS boys. It will be recalled that TF are genetic males who, because of a genetically-caused inability to use androgens, fail to develop masculine

⁷S. Coates (1974) discusses evidence that a degree of cross-sex-typing may be related to the development of field independence in young children. She notes that girls rated as primarily interested in dolls and house play and boys rates as tough and aggressive tended to be more field dependent.

internal and external genitals despite the presence of testes. In the sample represented here, 60% were diagnosed after puberty and learned of their sterility between the ages of 15 and 19; the others were between the ages of 5 and 13. These females are characterized as "ultrafeminine" in gender identity, with most wanting marriage and children exclusively (80%) and all fantasizing the "maternal role" . Of the adults, 60% were married and one was engaged. Two had adopted children and two were planning adoption. However, their attitudes toward pregnancy, newborns, and breastfeeding were oddly discordant with their otherwise "feminine" qualities. For example, 70% did not fantasize pregnancy and delivery; 60% felt negatively towards pregnancy, 10% were noncommittal; and 80% were against breastfeeding. Although 60% thought older infants or toddlers were "cutest", the same percentage expressed positive feelings about caring for newborns. The most likely explanation for these findings is that knowledge of sterility dampened their enthusiasm for functions of which they were not capable (Money, Ehrhardt, and Masica, 1968).

As regards the various measures of maternalism, furthermore, the data do not consistently distinguish androgenized girls from nonandrogenized girls or from their controls. For example, the wish for marriage and children exclusively is present in a minority of androgenized girls (6 to 30%), but

there is evidence that normal females⁸ are more similar to androgenized girls. A recent Roper poll (Klemesrud, 1974) reported that 38% of American women preferred marriage and children exclusively, and Allen's (1975) study of college women found that 48% were questioning whether to have children, 14% did not want children, and 38% either assumed they would or gave no response. Of those wishing to combine marriage with a career, again, normal women (Roper poll, 52%) are more similar to PIH (50%) and EAS (50%) than to EAS controls (26%) and TF (20%). In the AGS sibling study, the question was put in terms of whether a career was equal to or more important than marriage. Here, no differences were found among AGS girls (70%), their mothers (70%), and their female siblings (63%), nor between AGS males (65%) and their male siblings (90%). It is not known if AGS girls emphasize career over marriage; 20% of PIH and 33% of EAS do so, as compared to 6% of EAS controls and 10%-14% of normal women. It is of interest to note that the emphasis on marriage and career is found in those groups studied during the mid-1970's, regardless of fetal hormonal status. Thus, this aspect of "maternalism" appears to reflect primarily changing cultural mores.

A second factor which may be hypothesized to interfere

⁸ Aged 18 and over and representative of all economic and educational levels.

with full acceptance of the cultural gender role is the concern for bodily integrity. Patients variously face sterility, failure to menstruate, surgical repair of the external genitals, vaginoplasty (often "elective for coitus"), prosthetic testes, delayed puberty, extreme short stature, and other somatic disfigurement (in TS). The reader is referred to the Glossary for further details. One result may be that adult sexual roles are approached warily. For example, on the criterion of percentage of adults married, TF (60%) did not differ from Late-treated AGS (LAS) (56%). Furthermore, 85% of LAS women who married did so after cortisone treatment. Among TS patients, only 32% of the adults were married. Other samples are not yet adult. Another example is found in AGS boys, of whom 55% report daydreams about wedding and marriage compared to 19% of the male siblings. Also, 65% of AGS boys express interest in infant care compared to 20% of male siblings. Among EAS girls, on the other hand, some 59% are noncommittal and among AGS girls, 55% are negative or noncommittal. Nearly all control girls express positive interest in infants. These findings suggest that AGS boys' greater concern than normal boys with issues of love, sex, and procreation and AGS girls' apparent diffidence and lack of commitment are expressions of their underlying concerns with normality and acceptability. Money and Ehrhardt (1972) describe the following as "characteristic" of LAS girls"

Even after good surgical and hormonal feminization of appearance has been achieved, such a girl is diffident and fearful of rejection--as though the old body image will not fade. . . . Inchoately, the girl acts as if her first lover will magically decipher her old body image and know that she used to be some sort of freak (p. 158).

According to the organizational theory, TF ought to present as "ultrafeminine" women since they are totally insensitive to androgens. This a priori bias appears to have colored the interpretation of the data in several ways. First, questions asked of this group focused on pregnancy and infant care and on such questionable "traits" as "affectionateness toward the people they love" and attitudes toward housework. Secondly, the tomboyish responses of 2 of the 10 patients were dismissed because they were still adolescent with no dating experience.⁹ In the younger samples of androgenized girls, such interests were considered tomboyish and no caveat was made against waiting until dating was underway to judge the femininity of androgenized girls. Thirdly, the authors belabored the point that these genetic males were really good mothers--perhaps an inadvertant remnant of the American's intolerance for girlish behavior in boys:

An overpowering desire to hold, cuddle, and care for the totally helpless newborn is perhaps the epitome of motherhood. . . . Six [of ten] patients strongly expressed such maternal desires. . . .

⁹The 2 rated marriage and career as equally important. Their choice of career--veterinarian and riding instructor--were seen as "related to their girlish love of animals" (p. 110).

Theirs was motherly love for babies and not simply a desire for a manifest symbol of their female role. (p. 111).

The authors underlined their concern in comparing "very feminine" TF with male transexuals:

The care of the tiny newborn, however, is for the most part less to their liking than the care of toddlers. By contrast, the women with the Androgen Insensitivity Syndrome typically like to cuddle and nurse tiny, helpless babies--and do very well, maternally (p. 112).

The maternalism of TF women may be interpreted on the one hand as evidence that neither the presence of male sex chromosomes or gonads, nor the absence of uterus and ovaries affects the capacity to be an adequate mother. On the other hand, it may be taken to support the notion that the absence of fetal androgens permits the differentiation of maternal traits along with a female phenotype. This notion is not, in any case, supported by the evidence discussed above. Additionally, PIH data reveal that they not different from controls on interest in infant care, fantasies of marriage and maternalism, or satisfaction with gender role--though they are tomboyish in other respects. Aside from the unlikely hypothesis that the fetal progestagens to which they were exposed selectively affected only the external genitals and the brain pathways involved in vigorous activity, it seems reasonable to assume that factors unique to the sample or to the syndrome account for these minor differences. These were discussed in Part II (see pp. 100-101).

On the basis of the data presented, one might question whether the concept of a maternal trait is valid. Money and Ehrhardt (1972) stress 2 characteristics: (1) preference for infants over toddlers and older children; and (2) preference for the maternal role over marriage alone or marriage with or without a career. They hold that this trait is "suberved" by pathways in the brain which develop in the relative absence of androgens.

It is well-established that mothering behaviors do not develop in most "motherless mothers" (Harlow and Harlow, 1966). Despite normal feminine fetal environments and normal endocrine processes associated with menstruation or estrus, pregnancy, parturition, and lactation, rhesus monkey females deprived of mothering in infancy are mostly either indifferent or brutal to their offspring.¹⁰ Both peer and maternal contact are necessary for full development of social behavior. Additionally, the foregoing discussion (see pp. 103-106) indicates that repeated mothering experience is needed to establish preference for infants. The lack of preference for infants found in nulli and primiparous females, finally, does not provide support for the notion that human females possess an

¹⁰Motherless mothers are proportionately more brutal to male infants. Ruppenthal, et al (1974, cited in Sackett, 1974), found that while female and male infants were treated indifferently in about equal proportions, 46% of males were abused compared to 11% of females, and 12% of males were treated adequately compared to 39% of females.

inherent attraction for infants or children in general.

On the other hand, considering the crucial importance to infant survival and successful procreation of mother and peer contact in infancy, it is in the interest of human and animal primate groups to produce good mothers. Primate females may indeed socialize their offspring. Mothers of monkeys are observed to inspect the genitals of their neonates. In fact, identification of sex is made by observing mothers' behaviors, since males tend to be examined repeatedly during the first days of life (Rosenblum, 1974). Human cultures also socialize their females to become mothers. Unandrogenized males raised as males do not prefer marriage and infant care, but if raised as females, they do.

Furthermore, motives for bearing children are not based on such a maternal trait cross-culturally. A study of unmarried White American, Indian Muslim, and higher Varnas of Indian Hindu college students found that the primary motives in Hindu and Muslim women were elevation of status and financial and personal security (80 and 70%, respectively). Those citing the motive of mutual love comprised 2 to 12% of the samples. In contrast, in American White women mutual love and interest in development accounted for 92% of the responses (Meade and Singh, 1973).

In summary, the evidence for a maternal trait independent of cultural learning is not supported by the data.

4. Sexual Response and Eroticism

Few hypotheses have been generated from the study of human intersexes regarding fatal hormonal effects on sexual response, eroticism, and object choice--perhaps because they conform so consistently to sex of assignment and rearing (Money and Ehrhardt, 1972).

Cross-cultural research suggests that males are more sexually active than females; however, this difference is not found in pre-agricultural societies (Ford and Beach, 1951). D'Andrade (1966) notes that sexual restrictiveness--especially of females--is associated with male-dominant social structures, as is extreme sex-typing (see Chapter I). Primate studies, on the other hand, report that females are promiscuous and highly active during estrus--Primate females may mate 20 to 50 times daily during their estrus peak. Van Lawick-Goodall (1971) noted that Flo, the most popular chimp of her troop, bore many wounds from exhausted partners she had continued to solicit.

Until recently, it had been assumed that non-human females were not orgasmic. Hamburg (1974), for example, proposes that female orgasm is a relatively recent evolutionary adaptation which may have strengthened interpersonal bonds. No cross-cultural data suggests that females are not orgasmic, though the use of male researchers precludes much information of sexual practices of women (D'Andrade, 1966). Orgasm

in sub-primate females is difficult to detect, except in the cat, whose characteristic call and post-coital spasms and genital licking suggest that it does occur (Rosenblatt and Aronson, 1966). Recently, field studies of stump-tail macaques have reported female orgasm marked by characteristic facial expressions and body movements in both heterosexual and homosexual encounters (Chevalier-Skolnikoff, 1974). Michael, et al (1974) have reported male-like mounting behavior in many primate species, including evidence of orgasm.

Since primate morphological differentiation involves the development of both glans clitoris and penis from the identical genital tubercle and since both the number of nerve endings in the glans is identical, and perineal sensitivity and growth are governed by the same androgen (DHT) prior to tubercle differentiation, it is unlikely that males and females differ in the physiological capacity for orgasm (Masters and Johnson, 1966; Wilson, 1972, Walsh, et al, 1974). Masters and Johnson (1966) showed conclusively that the glans clitoris is the center of sexual sensation in the female--as the glans penis is in the male. They stated:

Aside from obvious anatomical variants, men and women are homogeneous in their physiological responses to sexual stimuli (p. 285).

For example, the sexes do not differ in speed of arousal or time to orgasm when the glans is directly stimulated; the slower response of the female in coitus occurs because glans stimulation is indirect. However, females are capable of multiple

orgasm, are more variable in orgasmic responses, and have slightly more orgasmic contractions than males. Despite the bipotential origins and preserved similarity of physiological response, the subjective experience of sexual response differs in the sexes. It is not clear to what extent these differences are due to sex-role attitudes.

Sex play and masturbation in early childhood

All intersexes raised as girls report a typical feminine pattern of genital play in childhood--that is, they do not differ from controls in sexual curiosity or play (Ehrhardt, Epstein, and Money, 1968; Ehrhardt, Evers, and Money, 1968; Ehrhardt and Money, 1967; Money and Mittenenthal, 1970; Money, Ehrhardt, and Masica, 1968; Money and Ehrhardt, 1972; Money, and Alexander, 1969). Indeed, reports of masturbation and interest in genital morphology are absent in at least three-quarters of girls. Boys and girls with precocious puberty are also not different from controls in this respect (Money and Walker, 1971; Money and Alexander, 1969). Therefore, it appears unlikely that fetal androgen levels or relative phallic size contribute to sex differences in genital play independent of sex of rearing.

There is evidence that normal boys begin genital play earlier than girls--at 7 and 10 months, respectively (Galenson and Roiphe, 1974). This may be due to the relatively greater accessibility of the penis, but it is not

inconceivable that the earlier onset is related to differential maternal behavior. For example, several children (mostly boys) who began genital play at 4 to 5 months had experienced "undue parental physical intimacy during the first 6 months of life" (p. 227). It is possible that the greater proximal stimulation directed to boys between birth and 6 months accelerates genital awareness.

Despite the reported absence of masturbation in girls, it is known that both sexes masturbate from the age of 15 months. However, both the reaction of the infant to awareness of genital difference and the subsequent course of masturbatory stimulation diverges soon after (see p. 66). It is difficult to explain the course of female development--the depressed mood, the suppression of overt masturbation, and the intense anal exploration--in terms of either biological patterns or castration anxiety. Maternal responses to genital curiosity in girls appear to be of 3 types: (1) no name for the girl's genitals is supplied; (2) an undifferentiated reference to genital, urinary, and anal functions is supplied--such as "tushy"; or (3) an equivalence is drawn between the male's penis and the female's future baby (Galen-son and Roiphe, 1974, 1975; Money and Ehrhardt, 1972). These cultural practices may well create castration reactions, for they communicate the notion that: (1) the source of her genital pleasure is not a specific, differentiated organ like the male's; and (2) her special nature is not to have such an

organ (nor such pleasure) but to have a baby instead. Presumably, the change in masturbatory pattern which follows represents the girl's attempt to repress her subjective feelings in accordance with "reality."

Adolescent dating and eroticism

The majority of the samples of androgenized females were not yet adolescent, so that definitive statements concerning erotic choice and response cannot be made. However, the evidence thus far suggests that erotic choice conforms to assignment and rearing (Ehrhardt and Baker, 1974; Money and Ehrhardt, 1972). Dating tends to be delayed in several syndromes, however, which appears to reflect concerns about sexual attractiveness in both sexes and potency in males. Turner's Syndrome (TS) females, for example, face unique problems of extreme short stature, somatic disfigurement, and pubertal failure. In order to gain a few inches, estrogen treatment may be delayed--thus delaying puberty well into teen-age. In consequence, TS are juvenilized by others. This is less true of those who have achieved hormonal puberty; nevertheless, Money and Mittenenthal (1970) found that regardless of age or pubertal status, only 60% are treated age-appropriately. Furthermore, the incidence of psychopathology in TS is quite high, and it is correlated not with the stigmata of the condition but with parental behavior. More than one-third of TS girls studied by Money and Mittenenthal (1970) were found

to be overprotected, rejected, or subjected to overt parental pathology. Secondly, androgenized girls--even when treated early with cortisone--may require vaginal exteriorization in teen-age, when the vagina is more likely to be kept open by coitus. Since puberty is rarely delayed in AGS and PIH girls, it is likely that reticence in initiating dating is related to issues of attractiveness and acceptability. Late-treated AGS, of course, face many additional problems. Untreated in the early years, they grow faster than age peers and develop the musculature and hirsutism of early male adolescence. Later, their voices deepen, the clitoris enlarges, and acne and facial hair may appear. The epiphyses close early so that they are shorter than normal--often five feet.¹¹ At puberty breast development and menstruation do not occur. That such virilized females maintain a female gender identity if raised as females is indeed remarkable. Equally remarkable, however, is the transformation in body contour that is possible if cortisone treatment is begun before statural growth is completed.

Although intersexes raised as males have not been studied systematically, there is ample anecdotal evidence that similar anxieties contribute to reticence about dating and attempting coitus. The concerns of AGS males raised as

¹¹The same course of development occurs in untreated AGS males.

males have already been mentioned (see p. 81). AGS females raised as males are said to view petting and coitus with "trepidation" because of their synthetic testes and small penis, though sexual arousal and object choice are concordant with sex of rearing (Money and Ehrhardt, 1972). Males with micro-penis and TF raised as males must make adjustment for their lack of a normal penis, but arousal and orgasmic capacity are not lost (Money, 1965).

Cryptorchid males are noted to have "later than normal onset and lower frequency of masturbation and other sexual outlets. . . ." (Meyer-Bahlberg, et al, 1974, p. 295). Since this condition is not normally related to fetal hormonal abnormalities, no relation to brain organization is assumed; however, adult endocrine pathology may be indicated. Intersexes raised as males have defects related to penile size or structure (hypospadias); testicular absence, degeneration, or cryptorchidism; sterility, short stature; or abnormal levels of testicular or pituitary gonadotropins. The literature suggests that, while they normally develop a normal male gender identity, they are subject to periods of anger or depression and may be "loners" (Money and Ehrhardt, 1972).

The incidence of pronounced psychopathology among intersexes--according to present evidence--is low. TS is the only syndrome to be studied in this respect and there, pathology was related to parental treatment (see p. 118). In the TF sample, 2 of the 3 girls who were "psychologically

disturbed to any significant degree" were also diagnosed late (at 5 or 6). Their mothers:

. . . reported that thenceforth they found themselves explicitly looking for signs of masculinity in the comportment and interests of the child (Money, Ehrhardt, and Masica, 1968, p. 113).

Elective mutism was mentioned in regard to 2 AGS females raised ambivalently (see pp. 64-65) and 1 PIH girl (Ehrhardt and Money, 1967). Mild symptomatic habits--such as enuresis and nail biting--were reported in 30 to 60% of PIH girls. Three-quarters of PIH mothers were worried about physical or behavioral abnormality in their daughters: their fantasies included the fear that "she might become another Christine Jorgensen," might not be normal or able to have children.¹²

The clearest evidence that parental expectations play a major role in gender role development--and perhaps in psychological disturbance in intersexes--comes from cases of sex reassignment. Money and Ehrhardt (1972) describe the changes in parental behavior which accompanied the reassignment of 2 genetic males as females at 17 months. Hair, clothing, toys, activities, parental role playing, genital play, and sex education underwent complete reversal. In fact, the mother of the boy whose penis was ablated reassured the girl that instead of a penis she would one day menstruate and have babies--

¹²PIH parents--three-quarters of whom were highly educated--had been informed as to the normality and fertility of their daughters.

which of course she never would, lacking a uterus and ovaries.

Adult sexuality and object choice

The only intersexes studied as adults are LAS and TF women. In a comparison of gender identity in 10 TF and 23 LAS women, Masica, et al (1971) found a tendency among TF women for lower sex drive, lower awareness of sex drive, less assertiveness in heterosexual relationships, and less versatility in coitus. They also reported a significantly lower frequency of sexual arousal in response to visual stimuli (.05). The organizational theory would predict that excess fetal androgens potentiated a more masculine type of sexual response.

According to Money and Ehrhardt (1972), sexual response to visual and narrative stimuli is "more frequently associated with the male, insofar as threshold of arousal is concerned." Ehrhardt, et al (1968) characterized the response of LAS women as follows:

The arousal was in the women's own genitals and was such as might lead to masturbation in the absence of a partner. It was not the sentimental arousal, more typical of the normal female, which leads to romantic longing for the loved one alone and which will, in his absence, require waiting for his return (p. 120).

Experimental studies of genital arousal in response to various stimuli suggest otherwise. Sigusch, et al (1970) showed slides of nudes, petting and coitus to 50 male and 50 female German college students. The students rated their sexual arousal,

bodily reactions, and sexual behavior for the 24 hour period before and after the experiment. Reported genital arousal did not differ between the sexes, nor did sexual behavior following the experiment. There was a trend for males to rate themselves as more sexually aroused but the means were similar and there was a large overlap--for example, one-third of females rated the petting and coitus sequences as more arousing than the average male did. A similar study by the same authors suggested that females respond as males do but are less prone to express it because of sex-role stereotypes (Schmidt and Sigush, 1970). Fisher (1973) has reviewed other similar studies and reports no differences in response to narrative stimuli.

Furthermore, it may be questioned whether the 2 groups differ significantly from each other and from normal women. Normal women of comparable age and class (to LAS women) rated themselves as slightly above average in libido, while 47% of LAS stated they were above average and 36% average in this respect (Fisher, 1973). TF women rated themselves similarly: 22% above and 66% average. The fact that the TF sample was of a lower socio-economic class could account for the differences in ratings, since female libido and orgasm are correlated with social class (Fisher, 1973). The groups, moreover, did not differ greatly in frequency of orgasm--though different scales make comparison difficult. Among normal women, 39% reported having orgasm "always or

almost always" and 55% reported it "more irregularly"; among LAS, the figures were 9% "always" and 60% "most of the time"; and among TF, 22% said they "always" had orgasm and 66% "most of the time."

The enlarged, erectile clitoris of untreated AGS women does not appear to contribute markedly to erotic response, since TF--who tend to have slightly smaller than normal clitori and underdeveloped labia minora, reflecting androgen insensitivity (Federman, 1967)--are not less capable of orgasm.¹³ Though a minority of LAS women considered themselves "hypererotic" before clitoridectomy and cortisone treatment, 81% rated themselves normal before and after treatment (Money and Ehrhardt, 1972). However, they did report less masturbation because the clitoris was no longer erectile, and increased sensation in the breasts.¹⁴

Present evidence suggests that sexual object choice conforms to sex of assignment and rearing (Money and Ehrhardt, 1972). Although most samples are not yet adult, none of the pre-pubertal and pubertal children show signs of bisexual or homosexual aim. The exception to this rule are LAS women, of whom 52% are exclusively heterosexual, 9% are heterosexual with "occasional homosexual experience," 3 to 9% are

¹³Masters and Johnson (1966) state that clitoral size is not related to sexual response.

¹⁴The clitoris of normal females is not erectile (Masters and Johnson, 1966).

bisexual, and 30% are sexually inexperienced. None are exclusively homosexual. The incidence of homosexual or bisexual erotic imagery and dreams is 44% (Masica, et al, 1971; Money and Ehrhardt, 1972). In contrast, EAS, PIH, TS, and TF are like their controls on these variables.¹⁵ Rather than ascribe the evidence of homosexuality to postnatal androgenic effects on the brain, one might consider the fact that LAS women were severely virilized from early childhood, with enlarged, erectile clitori, short stature, lack of feminine puberty, male body contours, beard growth, and voices (see p. 118). That 85% of those who married did so after cortisone treatment indicates the barriers to heterosexual involvement posed by their virilization. Studies of adult EAS women will resolve this question, since many of the EAS women were also subjected to postnatal androgens. The fact that one-third of LAS women have no sexual involvements (except masturbation in 17% of the women) lends further support to the hypothesis that problems in sexual functioning relate to feelings of unattractiveness rather than to fetal brain organization.

Males with Klinefelter's Syndrome (XXY) are also at risk for homosexuality as well as mental retardation, psychopathology, and various vascular and heart diseases. These males have a weak, virilizing puberty, with sterility, breast

¹⁵In the TF sample, 1 woman had homosexual experiences and fantasies during adolescence (10%).

development, and "minute testes." (Federman, 1967). Whether they are predisposed to "errors of gender identity formation"--as Money and Ehrhardt (1972) suggest--is unclear, since they face so many problems socially. Though compared to other hypogonadotropic males their social adjustment is poorer--indicating that depressed plasma testosterone levels are not the cause--a social contribution cannot be discounted.

In general, then, intersexes raised as females appear to develop as females in regard to sexual arousal and object choice. The finding that TF are much like normal women in these respects is particularly interesting in view of the fact that androgens--probably testosterone--are thought to be the "libido hormones" in both men and women (Sawin, 1969; Money, 1961; Reichlin, 1971; Davidson, 1972; Money and Ehrhardt, 1972). Reichlin (1971) reviewed the evidence on sexual desire and response in women after oophorectomy and adrenalectomy for treatment of breast cancer. No change in sexuality accompanied the loss of ovaries in 66 to 90% of the women, whereas none of the women undergoing adrenalectomy retained desire and only 15% retained sexual gratification (Reichlin, 1971; Waxenberg, 1963, cited in Money and Ehrhardt, 1972).¹⁶ On the other hand, administration of androgens to adult women "dramatically increases sexual drive and activity" (Lunde and Hamburg, 1972).

¹⁶ The loss of desire was not due to the trauma of breast cancer or mastectomy for a comparable sample of women who underwent radical mastectomy alone showed no change in desire, activity, or gratification (Reichlin, 1971).

The adrenal cortex is the major source of androgens in women. Kirschner and Bardin (1972) estimate that 65% of testosterone in women is synthesized from precursors in peripheral tissues and 35% is secreted by the adrenals and ovaries. Recent studies by Abraham (1974) utilizing dexamethasone to suppress adrenal hormones indicate that the adrenal contribution of androgens is constant, rather than cyclic.

Since TF women are insensitive to androgens and do not produce progesterone, their capacity for sexual arousal and orgasm must derive from estrogens--which appears unlikely. The answer to this riddle must await further research.

In summary, sexual response and object choice--like most other facets of personality development in intersexes--appear to conform to sex of assignment and rearing. Problems of body image and conformity to sex stereotypes affect these aspects of gender identity but not in ways that suggest lasting effects of fetal brain organization.

5. Intellectual Abilities

Sex-role stereotypes have always included the notion that men think differently from women. The early work on individual differences, for example, considered--and rejected--the ideas that extremes of intellectual ability were found more frequently in males and that males were influenced more by environment and females by heredity (Tyler, 1958). Maccoby and Jacklin's (1974) review of sex difference research dispelled

the beliefs that creativity and higher level cognition are attributes of the male while repetitive, rote skills are the province of females (see pp. 1-2). Even the research on field dependence-independence--which is commonly interpreted as evidence for sexually dimorphic cognitive styles--has been undermined by the findings that sex differences favoring males become significant only after age 17 and are reversed in preschool children (Witkin, et al, 1962; Coates, 1974). It is of interest, then, that of the 4 "fairly well established" sex differences cited by Maccoby and Jacklin (1974), 3 concern intellectual abilities--that is, verbal ability, favoring girls; and visual-spatial and mathematical abilities, favoring boys.

That these differences appear only around puberty raises the question whether they are "activated" by the pubertal hormones as a sequel to fetal brain organization--an hypothesis originally applied to mating behavior by Young (1961).¹⁷ Similarly, Diamond (1968) argues that "feminoid cognitive abilities" may be ultimately coded in the sex chromosomes and mediate by the sex hormones. Alternately, cultural sex-typing may give rise to these differences. Both motivational factors and experiential factors are viewed as mediators of the development of field independence at adolescence

¹⁷It may be recalled, however, that ovariectomy of female rats pre or post-pubertally did not result in differences in the display of mating behavior (Beach, 1942; Beach and Rasquin, 1942).

(Witkin, 1966; Maccoby, 1966; Sherman, 1967, cited in Coates, 1974).¹⁸ Coates suggests that the polarization of sex roles and of motivational and experiential factors after puberty may permit sufficient divergence in personality functioning for sex differences to become statistically significant. The same analysis may apply to the abilities discussed in this section. By investigating these skills in the relevant hermaphrodite syndromes, one may assess whether hormonal or chromosomal factors are implicated in dimorphic intellectual functioning.

General Intelligence and Fetal Androgen Environment

During the 1960's a number of papers reported that mean Full Scale I.Q.s were significantly elevated and skewed toward the upper end of the distribution in both EAS and LAS females and males, in females with PIH, and in male and females with idiopathic precocious puberty (IPP) (Money and Alexander, 1969; Money and Lewis, 1966; Ehrhardt, Epstein, and Money, 1968; Money, et al, 1967; Ehrhardt and Money, 1967). The original finding on AGS suggested that excess fetal androgens might "favor advanced intellectual development" of the central nervous system (Baker and Ehrhardt, 1974). Although high levels of fetal androgens seemed implicated in AGS and PIH, the

¹⁸ Maccoby (1966, 1970) stresses motivational factors of active independence versus passive dependence. Sherman (1967) emphasizes issues of experience with objects and activities.

fact that both genetic males and females were equally affected argued against the assumption that normal males benefit in this respect relative to females. Further evidence against a fetal androgenic etiology lay in the fact that IPP--which is rarely congenital or neonatal in onset--was also associated with elevated I.Q. Moreover, since the sexes were alike in this regard, the possibility that estrogens or progesterones were responsible could not be discounted (Money, et al, 1967).

Indeed, the role of progestins is not clear. The pathway of steroid biosynthesis is from cholesterol to progesterones to androgens to estrogens (Williams, 1956). Synthetic progesterone which did not masculinize female genitalia was associated with earlier development and accelerated school achievement in affected boys and girls, though not with accelerated I.Q. (Dalton, 1968).¹⁹ Furthermore, these effects were positively correlated with dosage, and achievement was associated with exposure before the 16th week.²⁰

The possibility that I.Q. levels were a function of socioeconomic class was suggested by a significant correlation in these 2 variables in the PIH sample (Ehrhardt, Epstein,

¹⁹ Mothers were given synthetic progesterone to treat symptoms of toxemia of pregnancy.

²⁰ It was not possible to analyze the relationship between timing of exposure and attainment of developmental milestones.

and Money, 1968). In the 3 studies for which social class data was provided, all were weighted toward the upper middle class.

Recently, however, Baker and Ehrhardt (1974) have found evidence suggestive of a genetic basis for accelerated I.Q. in the AGS syndrome. They compared AGS boys and girls with their unaffected siblings and parents on a variety of intellectual measures. The elevated I.Q. was characteristic of the entire sample, suggesting that the genetic trait for AGS is linked to one favoring intellectual development. Supporting this conclusion were the following: (1) patients, siblings, and parents did not differ from each other in mean I.Q. or distribution, but were significantly different from the population norm; (2) siblings were the only group to show a significant correlation between I.Q. and socioeconomic level; and (3) the sibling distribution did not differ from the population norm. Because AGS is caused by an autosomal recessive trait, both parents must be carriers for it to be expressed. Of 4 children born to heterozygous parents, 1 would be expected to have AGS, 2 to be heterozygote carriers, and 1 to be genetically unaffected. Thus, the sibling sample would be expected to be more variable than their parents and AGS siblings--all of whom are either homozygous or heterozygous carriers of the trait (Baker and Ehrhardt, 1974).

The authors noted that other hormonal processes associ-

ated with AGS--and perhaps with heterozygote carriers of the AGS trait--may account for their elevated I.Q. levels. However, the data do not support the theory that excess fetal androgens enhance intellectual development.

Specific Abilities

The AGS sibling study also permitted a test of several hypotheses relating fetal androgens with sexually dimorphic cognitive abilities. Employing the Cohen Verbal comprehension and Perceptual factors and the Verbal, Numerical, and Spatial factors of the Primary Mental Abilities tests, the authors investigate the hypotheses that fetal androgens would be positively correlated with VSA and MA and negatively correlated with VA.²¹

Patients, parents, and siblings did not differ significantly from each other on either the Verbal and Performance scales of the Wechsler scales or the Cohen factors. The Primary Mental Abilities tests yielded the only significant finding. AGS patients of both sexes declined significantly in the numerical subtest relative to their siblings and to their performance on the Verbal and Spatial subtests. No significant differences were found between the group on the Verbal

²¹ Cohen factors: (1) Verbal: Information, Comprehension, Similarities, and Vocabulary; (2) Perceptual: Block Design, Object Assembly. The authors also administered the Embedded Figures Test, Conservation Kit, and Differential Abilities Tests but the data has not yet been analyzed.

and Spatial subtests. Some trends in the data supported the hypotheses, but often did not. For example, though AGS males had the highest Performance I.Q. score and female siblings, the highest Verbal I.Q. score, male siblings and mothers were lowest in both.

It is possible that differences did not appear because of the small sample size and wide age range studied. Maccoby and Jacklin (1974) have noted that sex differences in these abilities do not become significant until age 11 or after. However, the similarity in numerical ability in AGS regardless of sex suggests a genetic origin.

A genetic origin for the specific deficit in aspects of VSA and MA found in TS is also likely. TS typically are found to have difficulty in space-form recognition, directional sense, and numerical skills (Money and Granoff, 1965). Because these contribute to performance scale deficits (Cohen perceptual factor), mean I.Q. tends to be lower than normal. Verbal ability is unimpaired. However, neither mean I.Q. nor space-form blindness were correlated with the 3 major somatic stigmata or the number of stigmata (Money and Granoff, 1965). The authors did not investigate the relationship of these deficits to visual and auditory impairment which are also found in TS (Hirschorn, 1974, personal communication).

Diamond (1968) postulates that cognitive deficits in TS represent the "female-biased" nervous system which results from the absence of fetal androgens. It appears that pro-

ponents of the organizational theory confuse the notion of the neutral sex--which develops in the total absence of gonadal hormones, as in TF and TS--with the normal female, in whom gonadal hormones are not lacking. Secondly, adrenal and maternal hormonal influence during fetal life are not lacking in TS. Thirdly, TS are characterized by a variety of sex chromosome constitutions--which may include Y chromosomal and hormonal influence during fetal development. In about 50 to 80% of TS the second chromosome is missing--45, XO (Federman, 1967; Mittwoch, 1967; Money and Granoff, 1965). The second chromosome is mosaic in another third of TS (Mittwoch, 1967). In the remaining cases, it is abnormal (fragmented or isochromosome) and/or mosaic (Federman, 1967). Mosaicism refers to the presence of different cell lines with different sex chromosome constitutions. For example, the presence of leydig cells and clitoral enlargement of some TS women is thought to represent some testicular development caused by the presence of the Y chromosome in some cell lines (Nickolas, 1974, personal communication). Both XO/XX and XO/XY mosaics have been identified in TS. Unfortunately, the possible genetic variables contributing to the cognitive deficits of TS are too complex to be determined by present methods of analysis.

Finally, the possibility of an experiential deficit related to the genetic abnormality of TS is not inconceivable. Money and Mittenhal (1970) believe that the "inertia of emotional arousal" characteristic of most TS may be generally

determined. This trait includes:

. . . compliancy, phlegmatism, stolidity, equability, acceptance, resignedness, slowness in asserting initiative, and tolerance of personal adversity (p. 54).

It is this trait which may account, as well, for TS's lower physical activity and aggression relative to controls (Money and Ehrhardt, 1972). We shall consider this issue in more detail below.

There are essentially 2 views of the origin of cognitive sex differences. The first proposes that motivational and experiential factors related to sex role development account for the emergence of sexual dimorphisms around puberty (Maccoby, 1974; Coates, 1974). The second proposes that sex chromosomal and fetal and postnatal sex hormone differences provide a predisposition for masculine or feminine modes of cognition. Hamburg (1974), for example, has suggested that verbal skills favoring females evolved out of the mother-infant dyad and that visual-spatial and math abilities evolved in males through the development of hunting, warfare, and territorial expansion.

Genetic factors in dimorphic special abilities would most likely be found on the X sex chromosome, for the phenomenon of X-linked transmission of many diseases and abnormalities is well-established while no evidence of Y-borne genes has been found (Mittwoch, 1967).²² The female receives an X chromosome

²²The male manifests the recessive trait because he has no dominant X-linked character to suppress it; the female needs 2 recessive alleles to show the trait (Mittwoch, 1967).

from both father and mother while the male's X chromosome is of maternal origin. One X chromosome of the female and the Y chromosome of the male are believed to become genetically inactive soon after conception. In the female, the pattern of inactivation is thought to occur randomly, so that half of her cells bear the maternal X and half bear the paternal X. The paternal and maternal X chromosomes are believed to be identical, according to present evidence, and are responsible for metabolic processes necessary for life (Mittwoch, 1973; Hirschhorn, 1975, personal communication).²³

Maccoby and Jacklin (1974) have reviewed the evidence for genetic inheritance of specific abilities. They cite evidence that one element contributing to VSA may be transmitted by an X-linked recessive gene--an hypothesis which is being tested in XXY and XYY males by Witkin and Hirschhorn (personal communication, 1974). Another genetic element for VSA is not X-linked (Stafford, 1961, cited in Maccoby and Jacklin, 1974). An X-linked recessive element would account for the appearance of VSA in 50% of males and 25% of females. The authors of the review found little evidence that VA and MA are X-linked.

As regards the "activational" role of pubertal hormones, the only evidence argues against the view that androgens are responsible for VSA skills. Broverman, et al (1968,

²³The 45,XO fetus (TS) is viable, while the 45,OY fetus is not (Mittwoch, 1967).

cited in Maccoby and Jaclin, 1974) hypothesized that androgens would facilitate set-breaking and reorganizational (Type B) tasks and estrogens would enhance simple, repetitive, over-learned (Type A) tasks. Instead, he found that injections of testosterone to male subjects resulted in a decline in Type A tasks--though the control group, injected with saline, declined even more. In a second study, Broverman found that masculine physical characteristics and measures of androgenic metabolites were negatively correlated with Type B tasks and positively correlated with Type A tasks.²⁴ Finally, Type B tasks (PMA Spatial test and WAIS Block Design) were negatively correlated at age 18 with masculine physical characteristics in boys but positively with these characteristics in girls. These findings--while they certainly argue against a hormonally-related enhancement of dimorphic skills--may be related to the hypotheses about rigidity of sex typing discussed by Coates (see pp. 79, 97). That is, hypermasculinity in boys appears to be associated with field dependence and poorer VSA while some cross-sex-typing in girls enhances these skills.

The motivational and experiential theories stress the importance of personality variables in the expression of intellect. Maccoby states:

Certain modes of thought may depend on the development of certain aspects of personality we have previously

²⁴Here, the measures were WAIS Block Design and the Embedded Figures Test.

thought of as "personality" rather than as qualities of intellect (Maccoby, 1970, p. 22).

Sontag, et al (1958) found that such traits as competitiveness, assertion, and independence--the "achievement motive"--at age 6 were associated with an increase in I.Q. over the following years, whereas passivity, dependency, and shyness were predictive of decreases in I.Q. Maternal behaviors which contribute to assertion and independence appear to be associated with high MA relative to VA and Field Independence, while maternal overprotection, restrictiveness, and lack of tolerance of self-assertion are more characteristic of the field dependent child, and with VA superior to MA (Maccoby, 1970; Sontag, et al, 1958).

Carlsmith (1964) studied Harvard students whose fathers had been absent during World War II. He found that the father-absent men were superior in VA relative to MA and that this ratio increased as a function of age at absence and length of absence. The younger the child and the longer the absence, the greater was the superiority of VA over MA. Carlsmith suggested that a close, positive father-son relationship is a "prerequisite for the masculine conceptual approach." It is interesting that women mathematicians have in common a close childhood relationship with their father (Bieri, cited in Maccoby, 1970).

Sanders, et al (1960) found characteristic differences in personality and metabolism in college males with

high VA-low MA, low VA-high MA, and high VA and MA scores on the college admission test. However, it should be noted that only 3% of the freshmen were superior in MA skills, indicating that this is not the "typical" male pattern, as suggested by Carlsmith (1964).²⁵ High VA males were immature, counter-dependent, physically inactive, idealistic, subjective, intuitive, and imaginative. High MA males were objective, systematic, physically active, ambivalently dependent on authority and group affiliation, and introspective. High VA-MA males were high achievers, leaders, orderly and persevering, conformist, and--like high MA males--they had higher metabolism. These studies are suggestive of a significant parental effect upon the development of cognitive styles, and point to issues of dependency and separation as the hub of this development.

Finally, one must consider the possibility that even if no sex differences exist biologically, the demands of sex role stereotypes will render girls' interests and abilities in the scientific and mathematical fields lethal to their sense of femininity. Wahlberg (1969) proposes that such considerations play a major role in the intellectual and career development of women. In a national sample of high school physics students, girls excelled in I.Q. and scholastic aptitude for physics, while boys excelled in physics achievement. Though the students were more masculine in values than

²⁵Harvard may select high MA males.

the norm, girls embraced more of the typical feminine values and expressed more concerns about becoming physicists.

At present, then, there is little evidence that fetal or postnatal hormones contribute to the development of sexually dimorphic cognitive skills. Further research on adult inter-sexes would aid in clarifying these issues. As regards genetic effects, current research on an X-linked VSA factor may establish the existence of such a trait in males. However, the X-linked mechanism predicts that the trait would be found in 25% of females--more than enough to fill our universities and research centers.

CHAPTER VI

SPECULATIONS ON THE ROLE OF THE SEX CHROMOSOMES

Finally, we come full-circle to reconsider the role of the sex chromosomes in sex determination and differentiation. It was stated earlier that the sole function of the sex chromosomes is to determine the sex of the gonads, after which fetal hormones assume further responsibility for sex differentiation (see Chapter II). However, all characteristics of living beings are ultimately determined by the chromosomes, among which the sex chromosomes stand out as unique in several aspects.

Both male and female gametes contain an X sex chromosome which is identical and which is thought to regulate crucial metabolic process necessary for life. This is indicated by the fact that 1 X chromosome is viable (TS), whereas 1 Y chromosome is not (Mittwoch, 1967). While many genes have been found on the X chromosome, the Y chromosome appears to have few or no genes (Mittwoch, 1967). Therefore, it seems likely that: (1) sex determination and differentiation are the province of the X chromosome genes; and (2) since normal gonadal development requires the presence of a second X or Y chromosome (as is evident by the lack of gonadal development in 45, XO TS),

the presence of the second chromosome must be necessary.

Hamerton (1971) has proposed that sex determination occurs via a "male controlling center" on the Y chromosome (or the short arm of the Y) which activates the gene on the X chromosome regulating testicular development and inhibits the gene for cortical development.¹ An active female determinant of gonadal development may not be necessary. However, it is clear that both X chromosomes are necessary for ovarian development and fertility, for in TS, the germ cells undergo "rapid and uncontrolled" atresia in the absence of the second X. Controlling centers for statural growth may be on the short arms of the heterochromatic X and Y chromosomes, as is indicated by studies of isochromosomes.²

In Hamerton's view, controlling centers are functional only when the second chromosome is in a heterochromatic state. Heterochromatin refers to a state of condensation observed in the second X and Y chromosomes during interphase.³ Heterochromatin possesses the genetic characteristics of

¹It will be recalled that the cortex of the primordial gonad develops into an ovary and the medulla into a testis (see p. 15).

²Isochromosomes are duplications of the short or long arms of a chromosome--(the corresponding arm(s) being missing).

³The doubling of DNA occurs during interphase, which denotes nuclei which are not in mitosis, or cell division (Mittwoch, 1967).

genetic inertness or inactivity;⁴ synthesis of DNA later than the autosomes and the other X chromosome (which are genetically active, or euchromatic); and preferential association with the nuclear membrane and nucleolus (Mittwoch, 1967). Its genetic effects are a matter of speculation, but at present it is believed that the presence of the second heterochromatic X or Y is necessary for fertility and may be responsible for regulation of metabolic processes (Mittwoch, 1967; 1973) and "Psychosexual development" (Hamerton, 1971).

Hamerton's view of sex determination and differentiation, then, allows for specific biochemical differences between the sexes and for the possibility of sex genes or parts of chromosomes determining sexually dimorphic behavioral characteristics.

Mittwoch (1967, 1973), on the other hand, believes that heterochromatin regulates the rate and timing of growth, or "minor differences," rather than major differences--which would be regulated by Mendelian-type inheritance which is due to specific biochemical differences. Biochemical differences refer to differences in, for example, hormone receptors specific to testosterone or estradio, or in pathways of biosynthesis which are assumed to be due to the manufacture of specific proteins in accordance with the genetic code. Mittwoch (1967) argues that the production and effects of hormones are regulated

⁴The inactive chromosome stops transcribing but still replicates itself. Replication is delayed in heterochromatic X chromosomes (Swanson, et al, 1967).

by quantitative variables.

Wilson and King (Rensberger, 1975) have recently advanced the hypothesis that major evolutionary changes:

. . . are caused by new arrangements in the sequence of old genes that simply change the timing and duration of a gene's activity. . . .

rather than by mutations that alter the structure of individual genes. Such modification:

. . . . mean that bones grow longer or change their shape, that fur grows or does not, . . . Bone remains bone, hair remains hair, and brain cells remain brain cells, but the size or number of any of these may change, and these are the differences that distinguish one species from another.

Mittwoch suggests, similarly, that secondary sex characteristics develop according to the growth rate of the target organs, rather than because of protein-regulated processes specific to one or the other sex.

Unfortunately, this writer is not sufficiently versed in genetic theory to assess the validity of these opposing views. It may be said, however, that if Hamerton's view of psychosexual development is correct, then a mechanism for reactivation of repressed genes must exist. How else is one to explain the normal female development of a genetic male re-assigned as a female after traumatic loss of the penis (Money and Ehrhardt, 1972)?

Perhaps when the question of the mechanism of action of the sex chromosomes and sex hormones is answered, we shall be one step further in our understanding of the biological bases of sex differences.

CHAPTER VII

CONCLUSIONS

The organizational view that the brain becomes sexually differentiated and fixed in its function early in development is not supported by the data reviewed in this paper. In rats, although normal sensitivity to sex hormones may be reduced or lost in experimental animals--exemplified by the reduced sensitivity to progesterone characteristics of androgenized females and normal males--the sexual behavior repertoire appears to remain bipotential. In those cases wherein certain behavior elements cannot be elicited under any regimen--such as the loss of ejaculatory capacity in unandrogenized male rats--permanent defects in growth and sensitivity of the phallus offer a satisfactory explanation. More important is the finding that normal males can be induced to display female lodotic patterns when given the proper hormonal regimen, and that androgenized females remain receptive, though on a different temporal basis.

Certain factors of design and method serve, unfortunately, to confound the data and to retard a resolution of the many paradoxical findings on rats. Such problems as variations in the definition of normal behavior, in methods of

measurement, and in administration and choice of hormone, added to the complexities of wide individual, strain, and species differences serve as examples. Certainly, however, the contradictory findings in the use of the various forms of androgens and estrogens as masculinizing agents raise fundamental questions about the adequacy of the androgen/no androgen hypothesis of masculinization and about the role of fetal androgens in normal female development. Despite these unresolved hormonal and methodological issues, however, the data strongly support the view that the brain remains sexually undifferentiated, or bipotential, in function.

The human data, though undermined also by many methodological problems, establishes clearly that gender role is neither fixed nor potentiated during fetal life but like the body--with the exception of the genitals--remains plastic or bipotential well into puberty. More precisely, a consistently and unambiguous gender identity appears to be necessary for psychic stability, but one need not possess the various biological indicators of sex--either structural or functional--to achieve normal psychosexual differentiation. A fuller understanding of the biological mechanisms of differentiation will not alter this fact.

The related hypothesis--that fetal hormones predispose the sexes to develop certain dimorphic traits--as suggested by Money and Ehrhardt (1972)--is given very limited support by the data. Problems of method and design, which are partly

inherent in the use of natural subjects, unfortunately preclude firm conclusions. Among these problems are: (1) the uncontrolled type, dosage, and timing of hormonal variables; (2) the use of small samples uncontrolled for age, socioeconomic and educational status, and race; (3) the psychological effects of illness and abnormality on self and others; (4) the lack of adequate research on parents of intersexes; and (5) the lack of clinical data of greater psychological depth on intersex subjects. With these limitations, it is indeed striking that among the various traits studied, only the trait of "vigorous energy expenditure" was suggestive of a fetal hormonal effect. Further clinical research on normal tomboyish and athletic girls and on partially or completely unandrogenized boys and girls should clarify the relative contributions of fetal hormones and parental expectations to active and placid temperaments in children.

Alternately, one cannot discount the possibility that subtle differences in the amount and type of sex hormones act at certain stages of postnatal development to foster differences in perception, metabolism or temperament. However, present evidence indicates that the sexes do not differ in sex hormone make-up until approximately 18 months prior to puberty; and no associations between postnatal sex hormone levels and the appearance of statistically significant sex differences are apparent. Several additional lines of evidence argue against this possibility. First, the condition of precocious puberty is not associated with precocious emotional and social development.

Second, children whose sex is reassigned during childhood require no additional sex hormones until puberty, yet they develop gender roles concordant with assigned sex if reared unambiguously.

Another possibility is that postnatal maturational processes may operate quite independently of the sex hormones, for example, through the agency of sex chromosomal mechanisms. Male vulnerability and female precocity and sex differences in maturation of the lateral hemisphere of the brain--if valid phenomenon--may be influenced by such mechanisms. Until genetic mechanisms are understood and subject to empirical control, however, this question must remain unexplored. At present, no known intersex syndrome would permit an adequate test of this hypothesis because of confounding hormonal or other variables, even if an unequivocal dimorphic characteristic could be identified.

In summary, biology lends an orientation to human behavior through the differentiation of a male or female somatotype and reproductive capacity. This orientation is not fixed or determined by biological factors, except insofar as biology creates all potential characteristics as individual differences. Sharing these characteristics across sex lines, individuals are socialized into the particular range of behaviors, attitudes, and skills deemed appropriate to the continuance of their particular culture. Until recently, cultures have based sex roles upon the inescapable fact of reproductive difference. Whether the widespread use of birth control will permit significant modifications in sex roles must await the further elaboration of cultures in which eroticism and reproduction are independent.

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GLOSSARY

AGS: Adrenogenital Syndrome (Congenital Adrenal Hyperplasia)

AGS Sibling Study: 2 samples of AGS girls and boys treated early with cortisone and matched with unaffected sibling and parents

amenorrhea: absence of menstrual periods

androgen: Male sex hormones produced by the testis, adreanal cortex, and ovary. There are several androgens, which differ in biological strength and activity.

anorchia: Degeneration of the male testes after initial differentiation. Synthetic testes and androgen replacement at puberty are required.

corpora cavernosa: the 2 columns of erectile tissue which support the body of the clitoris and the penis

cryptorchidism: the condition in which one or both testes are undescended

dysgenesis: abnormal or incomplete differentiation of an organ or tissue resulting in deformity or malfunction

EAS: sample of AGS girls treated early with cortisone, matched with normal controls

epiphysis: A piece of bone separated from a long bone by cartilage in childhood, but fusing at puberty with the larger bone. Fusion stops further statural growth.

gynecomastia: Development of breasts on a male. It may occur spontaneously or in response to hormone treatment.

hirsutism: hairiness, as in virilized women

hypogonadal males: refers to genetic males in whom fetal and/or postnatal androgens are insufficient for reasons not understood

hypopituitarism: A generalized deficiency of hormones. Incomplete failure may involve the gonadotropins--which stimulate the gonads to produce their hormones.

hyporchia: Partial degeneration of gonads which have differentiated as testes. Treatment with testosterone is sometimes required.

hypospadias: A birth defect in the position of the urinary opening on the penis. Mild hypospadias: opening is near the tip of the penis. Severe hypospadias: opening is in female position and the penis has an "open gutter" on its underside, instead of a covered urinary canal. The penis may be normal or small in size. A small hypospadiac penis is identical to an enlarged clitoris below which is a single opening or urogenital sinus leading to both the urethra and the vagina.

LAS: sample of AGS girls treated late with cortisone

microphallus or micropenis: Resembling the clitoris in size. It may be hypospadiac or carry the urethral tube.

PIH: sample of girls androgenized by fetal exposure to progestins (Progestin-Induced Hermaphroditism)

Precocious Puberty: sample of 14 late treated AGS boys and 4 boys with Idiopathic Precocious Puberty (IPP)

progesterone: female sex hormone produced by the ovary in the corpus luteum following ovulation, by the placenta during pregnancy, and by the testis.

TF: sample of genetic males with the Testicular Feminizing Syndrome (an Androgen Insensitivity Syndrome)

TS: sample of genetic 45 X,0 (and other) phenotypes with Turner's Syndrome, who lack gonads and therefore are not exposed to fetal gonadal hormones