I. SEXUALLY DIMORPHIC BEHAVIOR: DEFINITION AND THE ORGANIZATIONAL HYPOTHESIS

Introduction

Sex differences in behavior, like sex differences in body structure, are determined by a combination of genetic, hormonal, and external environmental factors. Dissection of the relative importance of these factors has occupied a major part of the research and writing on the subject, and a discussion of these issues took much of the time during the Work Session. First, the term *sexually dimorphic* was discussed, after which the organizational hypothesis concerning the development of these behaviors in mammals and birds was outlined.

The term *dimorphism* refers to the existence of two distinct *forms* within a single species. The term sexually dimorphic behavior, by extension, implies two different forms of behavior exhibited by the male and the female. Some workers have accepted usage of the term to mean not two different forms of behavior, but a response shown exclusively by one sex and not by the other. While some behaviors observed under natural circumstances, e.g., the ejaculatory pattern, are present in one sex but not in the other of most species, few other kinds of behavior could be classified as sexually dimorphic by this strict definition. Under appropriate conditions of hormonal treatment and testing, for example, male rats will display receptive and lordosis behaviors, and female rats will exhibit circling and mounting behaviors. In the discussion and presentations that follow, the term sexually dimorphic does not usually indicate the presence of a given response in one sex exclusively and its absence in the other, but, rather, any measurable difference in the parameters of the response for the two sexes. Thus, for example, both males and females eat the same food, but one sex may ingest measurably more than the other, or one may eat more often than the other without actually eating more food. The running activity of rats provides a different kind of example. Both sexes run, but the temporal patterning of their locomotor activity, measured daily, is distinctly different. Again, in a given species, hypothetically at least, males and females may not differ in the amount of aggression shown, but may differ markedly with respect to the occasions or stimulus situations that evoke aggression. For example, in territorial species, males may attack

primarily intruder males, and females may attack primarily intruder females. Finally, for hormonally mediated behavior, the sexes may differ only in the amount or kind of hormone normally involved in regulating the display of the behavior. Examples of such differences are to be found in the experimental induction of lordosis responses in male and female rodents when the male requires more estrogen than the female, or in the regulation of spontaneous mounting behavior, which, in some species, is facilitated by sequential estrogen and progesterone in the female and by testosterone in the male.

To describe these behavioral differences between the sexes as sexual dimorphisms does not violate common usage of the term by the morphological sciences. Among mammalian forms, both sexes have a pelvis. The difference between the sexes is not in the presence or absence of a pelvis or pelvic outlet, but in its size, girth, or other quantitative measure. Or there may be dynamic changes in the shape of the pelvis restricted to one sex and occasioned by pregnancy and parturition. Countless examples exist in morphology of sexual dimorphisms based only on quantitative differences, differences in intensity (e.g., coloration), or in response of a specific structure to hormonal stimulation. Nevertheless, despite the reasonableness of extending the term sexual dimorphism to behavioral differences, no implication is intended by the contributors concerning a structural or morphological basis for the measured dimorphism. The reader, moreover, is cautioned against drawing any such inference. Furthermore, the mere demonstration of sexual dimorphism cannot specify either its causation or its biological function. It was, in fact, the very task of the Work Session to determine, to the extent possible, the causes of a variety of described sexual dimorphisms in behavior, and for this purpose we have drawn heavily on the principles of sexual differentiation provided by embryologists, geneticists, and anatomists working with morphological sexual characteristics.

Current concepts of morphogenesis hold that the genetic sex of all vertebrates determines whether the embryonic genital ridge develops into a testis or an ovary. The means of action by which chromosomes direct the differentiation of the embryonic gonad are unknown; but it is known that the type of gonad differentiated determines by its secretory products whether male or female secondary reproductive organs develop. According to the organizational hypothesis (Phoenix et al., 1959), not only the reproductive organs but also the neural processes

mediating sexual behavior in mammals have the intrinsic tendency to develop according to a female pattern of body structure and behavior. However, they pass through a restricted period when a bisexual potentiality exists in both sexes. Circulating androgens from the testes are both morphogenetic and psychogenetic. They enhance the development of male behavior, as well as the differentiation of male reproductive organs, and in both instances the modifications induced by the hormone are enduring. Up to this time, ovarian secretions have not been assigned a significant role in the differentiation of the sexual behavior or reproductive tract characteristics in mammals, and female sexual characters develop even in the complete absence of ovaries. For female behavior, the critical organizing influence seems, in fact, to be either the absence of potent androgens, or, alternatively, if they are present, their concentrations must be too low to initiate the events that lead to a masculine organization. Moreover, estrogens circulating in higher than normal concentrations not only do not promote the development of female characteristics, but in some mammalian species they may act like the androgens to enhance masculine behavioral traits. This "organizational" hypothesis addressed itself to hormones in the bloodstream and not in the cell. Information as to whether a specific androgen (e.g., like testosterone) is a prohormone that needs to be converted into another substance in the cytoplasm or nucleus of cells comprising target organs before it produces its enduring modifications is viewed as clarifying the cellular mechanism of organization rather than as contradictory to the hypothesis.

The hormonal induction of enduring effects on behaviors and morphology (i.e., the organizing action of the hormones) is restricted to a limited period of development, the so-called "critical period." Use of the "critical period" concept can be criticized on the grounds (1) that the changes induced by hormones are not identified physiologically and/or anatomically, and (2) that in the absence of such information the changes cannot be evaluated in terms of whether or not they are "critical." In preference to such loose usage of concepts, therefore, some investigators (Goy et al., 1964) have proposed the alternative term, "period of maximal susceptibility" (or sensitivity) to the actions of hormones on the tissues mediating behavior.

Regardless of the terminology, however, periods have been identified in early development when hormones can most readily effect enduring changes in the ways in which an individual is destined to

behave. In placental mammals, this period of sensitivity for the developing offspring does not bear a constant relationship to the event of parturition or birth. Based strictly on empirical studies, the most effective period for modification is shortly after birth for the rat (Grady et al., 1965; Harris and Levine, 1965), the mouse (Campbell and McGill, 1970; Edwards, 1971), the hamster (Swanson, 1971), and the ferret (Baum, 1976); it is prior to birth for the guinea pig (Goy et al., 1964), the sheep (Short, 1974), and the rhesus monkey (Goy, 1966, 1968). In certain species, e.g., the dog (Beach and Kuehn, 1970; Beach et al., 1972), the relevant hormones may have to be present for some time both prior to and shortly following birth. In the few scattered studies that have been done with the rabbit, efforts to identify a maximally sensitive period have been unsuccessful. Campbell (1965) reported that a variety of steroids injected into female rabbits for a few days following birth failed to modify sexual behavior. Anderson (1970) reported that prenatal injections of androgen abolished maternal behavior of female rabbits but were without effect on adult sexual behavior, ovulation, or ability to maintain pregnancy. In view of demonstrated empirical precedents (e.g., dogs), it seems that the rabbit may also require exposure to appropriate steroids both pre- and post-natally in order to induce marked masculinization of genetic females. This problem needs to be reinvestigated.

For the limited number of species studied, there is an apparent relationship between fetal or larval opening of the eyes and the time of effective hormonal influence. In all cases, the developing organism has to be exposed to the hormone prior to eye-opening for enduring modifications to be induced. Since opening of the eye is correlated with neural maturation and development, it is relatively likely that hormones have to act on a nervous system at a specifiable stage of incomplete development in order to induce the changes of interest. While eye opening may, in a very general way, mark the end of the period of hormonal sensitivity, no specific event has been identified that serves as a marker for the beginning of this period. Considering, moreover, the extensive array of psychological functions, processes, and behavioral patterns now known to be influenced by hormones present during early stages of development, the utility of the notion of a single "period" should be questioned. Perhaps, rather, a sequence of "periods" exists, such that each step in the sequence is sensitive to organizational actions of the hormones on only one or a few specific traits. Some evi-

dence supporting this possibility is presented in later sections of this report and, therefore, is not repeated here; but it may be of value to point out that the basic notion is consonant with what is known regarding the differentiation of separate portions of the mammalian male reproductive tract (Burns, 1961).

The original empirical studies on guinea pigs led to the hypothesis that androgens present before birth organized the pattern of sexual behavior into the male type (Phoenix et al., 1959). In those studies, evidence was presented demonstrating not only the enhancement of behavior normally typical of the male (mounting) but also suppression of behavior normally typical of the female (estrogen-progesterone induction of lordosis). Inasmuch as the male guinea pigs studied at the time showed only weak and irregular lordosis responses to induction procedures, such fragmentary responses were considered male-typical. Therefore, when the same kind of fragmentary and weak lordosis responses were found to be characteristic of genetic females exposed to androgen prenatally, this "suppressed" form of lordosis was conceptualized as one aspect of the masculinizing action of prenatal androgen. This suppressive action of lordosis was thought to be as significant to the organization of behavioral maleness as the enhancement of male behavior itself. In this respect, then, both suppression of female characters and enhancement of male characters were subsumed under "masculinization." In the time since the original statement of the organizational hypothesis, additional studies, some involving different species, have shown that the suppression of female-typical behavior can be accomplished independently of the enhancement of male-typical behavior. These discoveries have led to the adoption of a new terminology. The term "defeminization" has been adopted and widely used to refer to hormonal effects involving the suppression of female-typical behavior in genetic females only. The term "masculinization" is now generally reserved only for hormonal effects involving the enhancement of male-typical behaviors in genetic females. For the genetic male, complementary terms of "feminization" and "demasculinization" have been brought into usage. More complete discussion of these terminological problems can be found in Beach (1971) and Goy and Goldfoot (1973).

The real advantage of the use of terms like masculinization and defeminization (or feminization and demasculinization) lies not alone in the conceptualization of these as independent processes. The use of

these terms encourages questions about spontaneous bisexuality that might be overlooked with a different theoretical framework. For example, some female guinea pigs show frequent mounting behavior as well as vigorous lordosis at the time of spontaneous or induced estrus, and these characteristics are genetically influenced (Goy and Young, 1957; Goy and Jakway, 1959). Adoption of the newer terminology readily facilitates the question: "What is the agent that masculinizes without defeminizing these females?" An alternative restatement of this question might be: "What are the conditions that impose a bisexual organization on females within a defined genome?" The summaries of discussions from the Work Session only hint at possible answers to such questions, but, at the very least, they show that the contemporary form of question avoids more of the purely semantic problems than was previously possible. While there is still reasonable and serious dispute regarding the biological cause of different organizations of sexuality and sexual behavior, hormonal hypotheses have earned a respectability that allows their inspection even for problems of human sexual behavior, a permission that was not readily granted by clinical workers a few decades ago.

As far as can be ascertained, behavioral traits that exhibit sexual dimorphism are influenced only by the gonadal hormones, regardless of whether these hormones are secreted by the gonads, the adrenals, or both. Restriction of behavioral sexual dimorphisms to gonadal hormone influence may reflect the fact that many or all of these traits are directly or indirectly related to reproductive fitness. As Nottebohm emphasizes in his discussion of avian sexual dimorphisms in a later section of this report, the functions of behavioral dimorphisms in attraction and arousal of mating partners are adaptive when their display reflects full reproductive competence (i.e., fully functional gonads).

Studies of sexually dimorphic behavior are numerous for mammalian species and much less well represented for other forms. It will not be surprising, therefore, if what has been learned about hormonal influences on such characters requires revision and extension as more data become available. Even for mammals, however, evidence clearly supports the classification of male-typical characters into three basic types in terms of their relations to the gonadal hormones. Type I encompasses those behavioral characteristics that cannot be brought to full expression unless the relevant hormone(s) is (are) present in ade-

quate amounts in the circulation during both the critical period of early development and *also* during a later life stage. Some behavioral traits, in other words, require both early organizational actions and later activational actions of the hormones. It is not without interest, moreover, that the hormones that accomplish the organizational effects and those that activate the behavior at a later age are most often the same. Examples of behavioral traits that require hormonal actions both early and late in development are male intromissive and ejaculatory behavior (see Gerall's section) and the male fighting behavior of some strains of mice (see Beatty's section). These behavioral traits find a distinct parallel in the actions of androgens on male accessory reproductive organs like the prostate and seminal vesicles. For these structures, organizational actions during an early critical period first effect structural differentiation, and later in life activational actions induce secretory function.

A second type of relationship (Type II) characterizes those behaviors that seemingly require only activation at later ages by the appropriate hormone. For these behavioral traits either androgen is not necessary during the early critical period, or the amount of androgen normally present at that time in both sexes is sufficient for their organization. An example of this kind of trait is the yawning behavior of rhesus monkeys (Goy and Resko, 1972). This response is ordinarily displayed much more frequently by adult males than by females or juveniles of either sex (Bielert, 1978). However, its frequency of display by females or juveniles can be augmented to a level equal to or exceeding that of the normal male by administration of exogenous testosterone. The mounting behavior of some strains of rats may be another example (Whalen et al., 1969), although females that mount as adults due to exogenous testosterone may have undergone some in utero virilization by exposure to their brothers' androgens (Clemens and Coniglio, 1971). Morphological parallels exist for this type of trait as well, and the induction of the growth of facial hair and balding response of human beings are well-known examples. These traits, more common in adult males than females, can be induced in the latter by exogenous testosterone given only in adulthood.

Type II behavioral traits, which can be activated at will independently of organizational influences, are not enduring features of the individual. Such traits are manifested only during the time the activational hormone is present. When the hormone is removed by castration

or declines spontaneously, the manifestation of the behavior is measurably altered, usually lessened in frequency or intensity. These traits depend entirely on concurrent hormonal levels during later ages when the behavior is normally displayed, and they differ from Type I traits not in their activational requirements but in their independence from organizational influences of the hormones during the critical period.

Type III traits, perhaps because of their more recent discovery, or perhaps because definitive evidence for them is more difficult to obtain, occur less frequently than Types I and II. These behavioral traits require only organizational actions of androgens, and no activational influence is required for their full expression by the individual. Such traits are manifested as well, or nearly as well, in males castrated prior to puberty (but after the end of the early critical period) as in intact males. They cannot be "activated" in spayed females by administration of exogenous androgen during the postcritical period stage of development; but they can be easily induced in females by appropriate treatment with androgens during the critical period. Examples of such types of behavior are the juvenile play and mounting behavior of rhesus monkeys (see later discussion by Goy in this section) and the micturitional patterns of the dog (Martins and Valle, 1948; Beach, 1974). No parallel morphological systems come readily to mind beyond the basic sexual differentiation of the reproductive tract tissues.

Recognition of the general and usual existence of these three types of relationships between hormones and sexually dimorphic behavior provides a perspective that renders a common mechanism of hormonal action unlikely. The complete contrast between Type II and Type III, the former operating entirely through activational mechanisms and the latter entirely through organizational mechanisms, suggests, at the very least, that the nature of the hormonal interactions with cellular machinery might contrast correspondingly.

This introduction to the problems of hormonal regulation of behavioral sexual dimorphism would be incomplete without some added information on other vertebrate classes. Birds, reptiles, amphibians, and fishes are clearly more diversified and less completely studied than mammals. In one precocial avian species, the Japanese quail, Adkins (1975) has shown that injection of fertile eggs on day 10 of incubation with either testosterone propionate or estradiol benzoate produced feminized males and normal females. Such treated males showed suppression of male sexual responses as adults and augmentation of

feminine receptivity, exactly the opposite of the general effects of steroid treatment in mammals. Estradiol was clearly more potent in demasculinization and feminization than testosterone, and the latter hormone probably accomplishes its organizational effects through aromatization to estradiol or estrone. Thus, although organizational influences of hormones are demonstrable in birds, both the effective hormone and the sex affected are different from the mammalian case, and resolution of this difference has been sought in terms of the influence of heterogamety on hormonal organization. Paralleling the mammalian story, however, the period for organizing actions of estrogens in birds is prenatal in precocial and postnatal in altricial forms. Orcutt (1971), using altricial pigeons, obtained evidence for demasculinization and feminization of males treated with implants of estradiol for varying periods of time post-hatching.

Studies appropriate to the concerns of the Work Session have not been carried out in reptiles, and the information on amphibians and fishes is incomplete for present purposes. Nevertheless, frogs, toads, newts, and salamanders can be completely sex-reversed by incubating fertilized eggs in water containing small amounts of hormone (Burns, 1961; Foote, 1964; Gallien, 1965, 1967). These sex reversals are so complete that genetic females grown in water containing testosterone develop fully functional testes, produce sperm, and mate with normal females to produce only female offspring. Conversely, in other species, males grown in water containing small amounts of estradiol are comparably reversed and will mate with normal males. Unfortunately, for these species information is lacking on specific hormone-behavior relationships in adults and on the reversibility of sexually dimorphic behavior at later ages. In short, it is not known whether critical periods exist for the organization of sexually dimorphic behaviors of any sort.

Of all the vertebrate phyla, fishes are the most diversified and least understood. Among teleosts, hermaphroditism is an extremely common occurrence. However, our search for examples that might provide evidence for organizational influences that conform to those of mammals has to exclude these spontaneously hermaphroditic forms. Complete transformation of all female offspring into males has been accomplished in *Tilapia mossambica* by treating fry with methyltestosterone for about 2 months after hatching (Clemens and Inslee, 1968). Newly hatched goldfish, treated for about the same period of time post-hatching, were transformed either to all-female broods when estrogens

were used or to all-male broods when androgens were used (Yamamoto and Kajishima, 1968). The addition of either testosterone or estradiol to the aquarium water of young cichlids resulted in feminization of males, and such feminized males could be bred to normal males (Hackmann and Reinboth, 1974). When sexual differentiation of the gonad normally occurs before birth (or hatching), treatment of fry is ineffective. For the viviparous guppy, treatment of gravid females with methyltestosterone for only 24 hours resulted in all-male broods (Dzwillo, 1962).

In general these studies of experimental sex reversal have not investigated hormonal influences at later ages, and the extent to which these same species could be sex-reversed as adults has not been carefully worked out. A loss of plasticity with maturity is certain for hormonal reversal of the gonad in some forms (Hackmann and Reinboth, 1974) and is suggested for behavior by the finding that adult female *Platypoecilus variatus* treated with methyltestosterone showed only weak and preliminary male courtship patterns (Laskowski (1953). Clearly, much more work is needed before concepts like the critical period can be meaningfully applied to fishes, and for spontaneously reversing forms such a concept is not likely ever to be applicable without modification.

Spontaneous sex reversal from functional female to functional male is well known and occurs among such diverse forms as zooplanktivores (Popper and Fishelson, 1973), cleaner wrasses (Robertson, 1972), gobies (Lassig, 1977), and parrot fish (Choat and Robertson, 1975). Though "spontaneous," once reversal has occurred, no reversal or regression to the original type has been documented for any species. Recently, Shapiro (1977) has completed an elegantly detailed study of sex reversal in the protogynous coral reef fish, *Anthias squamipinnis.* In this species all juveniles mature as females, and only some transform later into males. This social species lives in heterosexual groups, and the loss of a single male from the group is followed by a surprisingly rapid sex reversal (requiring only 1 week or so) in one of the females. The changes include transformation to the color pattern, gonadal histology, and behavior of the normal male. For each group, the transformation is limited to a single female, and the factors determining which female will undergo tranformation are not entirely clear. In part, however, the transforming female is suddenly treated (i.e., behaved toward) quite differently by the other female members of the group. Well in advance of any outward physical signs of change, the

nonreversing females behave toward the reversing female as though she were male. Shapiro (1977) argues that, since all-female groups occur in nature, it is the change in social behavioral patterns that are more causal to the females' sex reversal than the removal or loss of a male. This fascinating model for social environmental control of hormonal functions deserves detailed future study. The phenomenon may have parallels or even partial homologies at other phyletic levels. The opposite type of spontaneous sex reversal (from male to female) also occurs among fishes. In the anemone fish *Amphiprion,* a monogamous but group-living form, the single female (always the largest and oldest in the group) suppresses the transformation from male to female by aggressive dominance over the smaller and subordinate males (Fricke and Fricke, 1977). Only one of the males in the group, the most dominant, has fully functional testes, and in all other males testicular development is correlated with dominance status.

This brief survey of vertebrate sexuality serves only to show that no fundamental uniformity exists that is readily apparent. Phyla differ, as do species within phyla, with regard (1) to the hormone that has morphogenetic and psychogenetic potential, (2) to the genetic sex that can be more easily reversed, (3) to the state of maturation at which reversal can occur, (4) to the extent to which sex can be hormonally reversed, and (5) with regard (probably) to the role of hormones in the organization and activation of specific behaviors. Nevertheless, despite these differences, there is, as yet, no compelling evidence against the most abstract level of generalization that would assert the possibility that both organizational and activational influences of the gonadal hormones are represented among all vertebrate phyla. The fact that both kinds of hormonal influence may not be demonstrable in every species is not, after all, a more difficult conceptual flaw than the circumstance that both influences are not always demonstrable for every type of sexually dimorphic behavior shown by a single species. Nor is it any more disconcerting, logically, than the fact that a sexual dimorphism found in one species may not be present in another, or may be present but totally reversed in a third. On the contrary, the boundless variation of behavioral and morphological sexual dimorphisms is one of the richest challenges to empirical science in general and to endocrinology and neurology in particular. In searching out mechanisms of proximate causation, we cannot afford to ignore the adaptive functions of these dimorphisms; we must be willing to

entertain the possibility that some dimorphisms have neither a genetic nor a hormonal basis. The notion that selection strongly favors the complete environmental determination of sex in some species (Charnov and Bull, 1977) obliges us to tolerate a possible like determination of behavioral characters typical to each sex. For some highly social species, like the human being, culture may define the types and limits of sexual dimorphisms. Worse luck yet, the individual human being may be forced to learn or acquire those dimorphisms that, like the sex-reversing *Anthias squamipinnis,* the behavior of his or her peers thrusts upon him/her.