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SEXUALLY DIMORPHIC BEHAVIORS

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Sexually Dimorphic Behaviors

Many behaviors exhibited by females differ from those characteristic of males. In extreme instances, one sex displays a behavioral pattern never exhibited by the other. More often, the form or frequency of the behavioral act differs between the sexes. These behaviors are often called "sexually dimorphic" by analogy with vertebrate secondary sex characteristics such as the combs of roosters or mammalian mammary glands. This review examines dimorphic behaviors, with emphasis on proximate neural and endocrine mechanisms governing sex-typical behaviors in vertebrates. A number of more comprehensive reviews have recently dealt with selected aspects of this topic (Clemens & Gladhue 1979, Goy & McEwen 1980, Pfaff 1980, Arnold & Gorski 1984, Konishi 1985, Kelley 1986b, Crews & Moore 1986). My aim here is to focus on a few sexually dimorphic behavioral systems for which extensive neural and endocrine data are available. These systems are exemplars of three sexually dimorphic characters: parental care, mating reflexes, and courtship. For parental care, the most extensive body of neural and endocrine research has been on mammalian maternal care. Relatively less attention has been paid to species with biparental (birds) or paternal (fish, frogs) systems. Our understanding of neural and endocrine control of copulatory postures (e.g. rat lordosis and penile reflexes, amplexus in frogs) has progressed recently due, in part, to the development of certain tractable experimental paradigms (reviewed in Kelley 1986b). Finally, I consider courtship—which includes both mate attraction and intrasexual competition—in several systems, including bird and frog song, duetting in tropical birds, and polyandry in the jacana and phalarope.

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Evolution of Sexually Dimorphic Behaviors

Why are some behaviors the exclusive province of one sex in a particular phyletic group, yet are monomorphic or even "sex reversed" in closely related species? Sex differences in behavior are the result of evolution (Darwin 1871, Williams 1966, Trivers 1972, Maynard Smith 1978). Both natural and sexual selection contribute to sexual dimorphism (e.g. Lande 1980, Partridge & Halliday 1984). Natural selection affects those characters directly connected with the propagation of species including fertilization, successful insemination or the rearing of offspring (Darwin 1871). Thus, sex differences in reproductive organs, in appendages used in copulation or in structures used in the nourishment of young are attributed to natural selection as are their behavioral counterparts, copulatory reflexes and parental behaviors. In some species, males and females differ markedly in structures not associated with reproduction, e.g. bill size and shape in birds (Selander 1972). Such structural dimorphisms also arise by natural selection and contribute indirectly to the propagation of the species (Darwin 1871).

Certain differences between the sexes are not so easily characterized as contributing to the propagation of species. Darwin (1871) considered the evolution of these characters to be driven by a separate process, sexual selection. These characters impart a competitive advantage in contests for mating opportunities. Either the character is particularly helpful in same sex competitions for mate access (intrasexual selection) or it imparts an advantage in attracting a member of the opposite sex (intersexual selection). In intrasexual selection, the selective driving force is predominantly on the behavior of the sex competing for access to mates whereas intersexual selection also acts on the choice behavior of the courted individual. In both intrasexual and intersexual selection, behaviors of both partners have consequences for reproductive success and for fitness. Current theoretical approaches to evolution of these interactions [notably the models of Lande (1980) and the notions of "evolutionary stable strategies" and of mathematical games theory analyses (Maynard Smith 1982)] appropriately emphasize these interactions among individuals.

Intersexual selection is commonly assumed to drive sexual dimorphism in two behavioral systems reviewed here, bird song and frog mate calling. Songs of male birds are believed to function in attracting females by advertising desirable attributes (Thorpe 1961, Searcy & Andersson 1986). For example, in species that add new song types with each season, repertoire size should be a good guide to age and thus survival ability—a useful attribute in a potential mate. In canaries, more complex songs are known to be more attractive to females and more stimulatory to reproductive

activity than their simpler counterparts (Kroodsma 1976). In frogs, advertisement calls attract females to the breeding site; in some species vocal courtship by individuals contributes to mate selection (Wells 1977). Bird song also functions in territorial defense; for example, in acoustic competition between neighboring males in temperate bird species. Whether females can assess overall song complexity (intersexual selection) or whether they are instead merely the prizes in an elaborate acoustic battle between males (intrasexual selection; see Kelley 1986b) is not yet clear. Recent studies suggest that other dimorphic characters such as elaborate male plumage in birds of paradise, function to intimidate rival males rather than attract females (Le Croy 1981).

In frogs, an advertisement call can convey two messages. In the Puerto Rican tree frog, *Eleutherodactylus coqui*, males utter a "coqui" call during the breeding season. The "co" note serves as a territorial or agonistic signal to adjacent males, the "qui" note is attractive to females (Narins & Capranica 1980). In some anuran species, of which *E. coqui* is an example, males breed over a relatively prolonged period and defend sites suitable for calling, oviposition and/or feeding (Wells 1977). In others, the "explosive breeders," conditions suitable for spawning are only briefly available; females do not choose individual males, but instead locate the entire calling chorus (Arak 1983). There is little evidence for ritualized male/male competition in explosive breeders (Wells 1977).

As the above examples suggest, males compete for females in most vertebrate species. Courtship is thus an example of a characteristic male behavior. The predominance of male courtship and competition follows, I believe, from two characteristics of vertebrate mating systems: anisogamy and parental care. One sex (female) puts effort into producing a large gamete (anisogamy), a resource for which the other sex (male) competes. In some species, effort directed at offspring extends beyond gamete production and fertilization. This "parental investment"—defined broadly by Trivers (1972) as all allocation of effort in rearing existing offspring at the expense of producing additional offspring—includes the parental care seen in some species. Maynard Smith (1978, 1984) has classified the most common patterns of parental care as follows: the most common "system" is no parental care; if there is care it is typically uniparental, if there is internal fertilization, care is usually maternal, and if there is external fertilization, care is usually paternal but may be biparental. Parental care, when it exists, is thus usually a sexually dimorphic behavioral characteristic. In frogs and fish, there are numerous examples of paternal care (nests, fanning the eggs, mouth brooding, and the like). In birds, the most common pattern is biparental; the second most common pattern is maternal care. Care includes feeding the young and even extends, in some

Columbiformes, to the production of crop "milk" by both sexes. In mammals, however, the most common pattern is maternal care. Male contributions, though important when present, are largely indirect, e.g. territory defense. Parental care in rats, then, is an example of a characteristic female behavior.

Mate attraction and parental care are closely connected. There are two predominant patterns in birds: monogamy with biparental care and polygyny with maternal care. A rare and fascinating exception is the polyandry with paternal care found in some shore birds, jacanas and phalaropes (Jenni & Collier 1972, Jenni & Betts 1978). What accounts for this role reversal? Mating systems can be considered as evolutionary stable strategies (Maynard Smith 1982). The goal is to maximize the number of surviving offspring. Each sex has two available strategies: guarding or deserting the young (Maynard Smith 1984). Which approach is taken depends both on the probability that the young will survive to reproduce without further care and on the strategy assumed by the mate. Maynard Smith (1984) considers the reversal seen in jacanas to have arisen from the more generalized "double clutching" tendency seen in other shorebirds—females lay one clutch cared for by the male and another by herself. Polyandry in jacanas and phalaropes could have arisen if a female instead obtained a new partner for the second clutch, thus freeing her to lay a third clutch and so on. These examples illustrate the phyletic specificity of the way sexual dimorphisms in mating strategy evolve. While the evolutionary factors shown to operate in birds, for example, are among those candidates for effects in other groups (e.g. primates), they by no means can be assumed to operate in the same way, no matter how appealing the analogy. Polyandry in human societies (Daly & Wilson 1978) usually does not involve role reversal in courtship or parental care.

The elements of competition and response in sexual selection lead to the powerful and rapid evolution of sexually dimorphic characters. Expression, however, is also subject to natural selection, which can either enhance or subdue sexual dimorphism (Lande & Arnold 1985). Sexually dimorphic traits may enable males and females to optimally exploit different resources and minimize competition. Dimorphism in bill size or shape may permit dimorphic foraging strategies (Selander 1972). Natural selection should also serve to check deleterious excesses attendant on sexual selection (e.g. the peacock's tail, Darwin 1871). The additional burden of parental care should attenuate extremes of sexual dimorphism; feeding or defending chicks can interfere with survival of the parent. Conversely, bright coloration of the male could increase the chances of attracting a predator to the nest. Thus one might invoke natural selection to explain why biparental, monogamous species (especially those living under strin-

gent but stable environmental circumstances) are less sexually dimorphic in morphology and behavior than are closely related maternal, polygynous species.

Finally, with regard to the interpretation of proximal control of sexually dimorphic behaviors, we must bear in mind that most physiological studies of animal behavior are conducted on a limited number of species under laboratory conditions that may not match those encountered by the animal in its native habitat (Crews & Moore 1986). For animals living in groups, for example, we cannot neglect the social context in which behaviors are observed (Goldfoot & Neff 1985). When tested alone, male and female Rhesus monkeys respond to distress signals from an isolated infant with equivalent frequencies and latencies (Gibber 1981). However, if the same male and female are housed together, only the female responds to the infant. Thus the unwary observer might conclude from the first testing situation that the sexes share parental care or from the second that care is the exclusive province of the female.

The Design of Sexually Dimorphic Systems

In principle, sex differences in behavior are produced in two ways (Figure 1). One is to provide adults of both sexes with all the requisite sensory, neural, and motor components necessary to produce the behavior but to arrange that the sexes operate in different environments. External environmental duality is achieved only in very rare instances of extreme dimorphism: for example, when the male is a parasite living on the female (Darwin 1871). Within the context of a social group, environmental duality can be achieved if certain social stimuli are only given to one sex. In the example of Rhesus monkeys and the stranded infant above, a male might never be alone with the infant and care-giving behavior would not be exhibited. A sex difference in environment could also result from differences in the internal milieu; for example, sex differences in gonadal and placental hormones. Many courtship and parental behaviors are controlled by hormones circulating in adults (Kelley & Pfaff 1978, Kelley 1978, Moore 1983, Crews & Silver 1985, Rosenblatt et al 1985). One way to produce female-only behaviors would be to arrange that one or more of the sensory, neural, and motor elements that produce sexually dimorphic behaviors function only in the presence of female-specific hormones.

Endocrine Control of Sex Differences

Circulating hormones exert very powerful control over reproductive behaviors in many species (for others, see Crews & Moore 1986). Sex differences in circulating titers of hormones (e.g. gonadal steroids) can suffice to account for sex differences in behavioral frequencies. The evi-

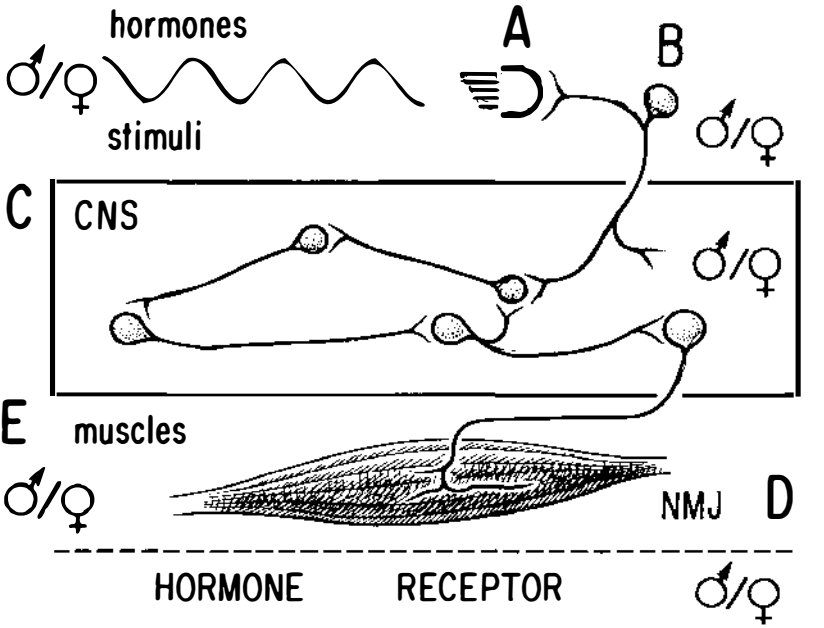

Figure 1 Possible mechanisms for generating sexually dimorphic behavior. In one, both sexes have all the necessary sensory (A), neural (B and C), and neuromuscular (D and E) components required, but one sex lacks access to the requisite stimuli or hormones (*above*). In the other, sex differences are due to sensory receptor (A), neural (B, C, D), or muscular dimorphisms (E), including sex differences in hormone receptor expression (*below*).

dence suggesting that androgens control male reproductive behaviors and that estrogens (and progestins) control female pro- and receptivity in a wide variety of species has been reviewed extensively elsewhere (Kelley & Pfaff 1978, Clemens & Gladhue 1979, Goy & McEwen 1980, Crews & Silver 1985). An example of an androgen-mediated behavior is clasping, a male-typical sex behavior in *Xenopus laevis* (Kelley & Pfaff 1976). A sexually active male initiates amplexus by swimming toward a female and grasping her with his forelegs. Castrated males cease clasping within one to two weeks; the behavior can be reinstated by treatment with exogenous androgens, but not estrogen. Completely male-like behavior can be induced in adult, ovariectomized females by androgen treatment. Brain regions that participate in the control of clasping appear to be capable of behavioral control in both sexes (Hutchison & Poynton 1963). Neurons that contain intracellular receptors for androgen are found in spinal cord motor pools that innervate arm muscles used in clasping in both sexes. Androgen induces increased excitability of these cells (Erulkar et al 1981). Thus, the sensory, neural, and motor elements necessary for clasping are

present in both sexes in *X. laevis* and females do not normally exhibit the behavior because of insufficient titers of androgen (Lambdin & Kelley 1986). Still not clear is whether androgen acts at one or more sites (sensory, neural, motor) to permit behavioral expression or whether one particular site (for example, arm muscle motor neurons) is limiting for behavioral expression.

Endocrine Control of Maternal Behavior in Rats

Steroid hormone treatment of adults may not produce an exact phenocopy of behavior in the opposite sex; the behavior seen may be less frequent, or it may differ in form or be less vigorous. One reason may be partial sexual differentiation— underlying sex differences in behavioral effector elements due to hormone action during development (discussed below). Another set of factors, however, are the combined actions of many hormones, levels of which differ between the sexes. One of the most intriguing systems in this regard is the control of maternal behavior in rats.

Under laboratory conditions, male rats usually exhibit little, if any, parental care when first exposed to newborn pups (paternal care is more frequent in other rodents, Elwood 1983). In contrast, parturient females—even those giving birth for the first time—immediately display the full array of parental activities: retrieving, grouping, licking, crouching, nursing, and nest building. Males (and virgin females) are fully capable of displaying parental behaviors and will do so if exposed to pups repeatedly (5–7 days; Rosenblatt 1967). Once parental behavior has been induced, it is always exhibited with short latency. The difference between primiparous parturient females and other adults is in the latency of the full response and the percentage of responsive animals upon *first* exposure to pups. All primiparous females are immediately fully responsive, while very few males or virgin females display "spontaneous" maternal behavior. The difference in latency between parturient females and others has been attributed to olfactory cues; latency to pup retrieval is reduced by anosmia (Fleming & Rosenblatt 1974). If newborn pups produce aversive olfactory cues, the increased "emotionality" of the virgin female may also contribute to pup avoidance (Fleming & Luebke 1981).

What accounts for the rapid onset of maternal behaviors after birth? A variety of evidence in many species implicates endocrine changes associated with pregnancy and birth (see Rosenblatt et al 1985 for a recent review). Four hormones have been implicated: estrogen, progesterone, prolactin and oxytocin (Moltz et al 1970, Zarrow et al 1971, Siegel & Rosenblatt 1975, Pedersen et al 1982). During pregnancy, estradiol rises gradually to maximum levels of approximately 155 pg/ml at day 15 of gestation; between days 20 and 22 (birth) levels fall abruptly to 10 pg/ml. Pro-

gesterone in circulation increases throughout pregnancy, reaching maximum maintained levels (approximately 70 pg/ml) two days before birth and then dropping precipitously (Bridges 1984). These endocrine changes result in very high P/E ratios around the time of parturition. Prolactin levels rise abruptly at the end of pregnancy (Morishige et al 1973). Oxytocin levels rise throughout gestation (Boer et al 1979).

Treatment with high doses of estradiol alone can reduce the latency to exhibit maternal behavior in virgin females or males (Bridges 1984). Progesterone by itself is ineffective, but acts synergistically with estrogen to reduce latencies to first display of full maternal responsiveness. Prior treatment with progesterone sensitizes females to the behavioral effects of estradiol (Bridges 1984). The onset of decreasing levels of progesterone appears to control the time of onset of behavioral responsiveness in primigravid or estradiol treated nulliparous rats (Siegel & Rosenblatt 1975, Bridges & Russell 1981). Progesterone and estradiol secretion during pregnancy appear to *prepare* the primigravid female to exhibit relatively short latency maternal behavior; neither hormone need be present when behavior is displayed. Effects on maternal behavior are proportional to the duration of treatment or of pregnancy (Bridges 1984).

Treatment with steroids alone, however, does not reproduce the very rapid, full behavioral responsiveness seen in parturient females (100% of animals tested are fully responsive to young within seconds or minutes of birth). This responsiveness is not necessarily related to parturition itself as full-term primigravidas and females delivered by caesarian section are also responsive to young (reviewed in Rosenblatt et al 1985). Two nonsteroid hormones have been extensively examined as possible mediators of short-latency maternal responsiveness: prolactin and oxytocin, both of which are elevated at parturition. Virgin females induced to show parental behavior by exposure to pups or steroid treatment also show increases in plasma prolactin levels whereas similarly treated males do not (Bridges 1983, Samuels & Bridges 1983, Tate-Ostroff & Bridges 1985). However, latencies to exhibit parental behavior (nest building, retrieval, crouching) are no different in parental females and males, even when steroid treated (Samuels & Bridges 1983). More powerful evidence for prolactin effects comes from studies showing that nulliparous female rats do not show steroid-induced decreases in responsive latency unless an intact pituitary is present (Bridges et al 1985). If an ectopic pituitary graft is given, some steroid-treated females (50%) display maternal responsiveness within 30 min of test initiation; only 10% of control females display such rapid responsiveness. Prolonged treatment with prolactin also significantly decreases maternal response latencies in steroid-treated females. Whether any prolactin-treated females showed the very rapid maternal respon-

siveness seen in animals with pituitary grafts remains unclear. Comparable results in males have not yet been reported.

Recent evidence, though still somewhat controversial, also implicates the hormone oxytocin in maternal behavior. Intracerebroventricular (ICV) injection of oxytocin into estrogen-primed ovariectomized rats induces immediate increases in the percentage of females displaying maternal behavior (Pedersen & Prange 1979, Pedersen et al 1982). Under optimal test conditions in a responsive strain (Sprague-Dawley rats obtained from Zivic-Miller), approximately 90% of ovariectomized estrogen-primed nulliparous females obtained high maternal behavior scores within one hour of contact with foster-pups (Fahrbach et al 1986). Some additional evidence that endogenous oxytocin participates in maternal responsiveness comes from results of studies in which oxytocin antagonists or antisera were applied ICV to pregnancy-terminated, estrogen-treated nulliparous females (Fahrbach et al 1985). These treatments significantly delay the onset of maternal responsiveness as well as the percentage of females showing maternal behavior within the first hour of testing. Maternal behavior of lactating females five days postpartum is not disrupted by treatment with an oxytocin antagonist, thus suggesting that oxytocin does not maintain maternal responsiveness. Unlike the effects of prolactin, short-term disruption of oxytocin (1 hr) was as effective as long-term treatment.

There are two probable endogenous sources of prolactin and oxytocin: the pituitary or CNS neurons. Studies by Bridges and colleagues (1985) on ectopic pituitaries strongly suggest that the behaviorally important source of prolactin is the hypophysis. The source of oxytocin is less clear since comparable studies on hypophysectomized animals have not been carried out. Oxytocin is synthesized by neurons in the paraventricular (PVN) and supraoptic nuclei of the brain. There are multiple CNS targets of oxytocinergic fibers including fibers that end in the posterior pituitary and supply circulating oxytocin. Based on ICV administration of the hormone, antagonists, and antisera, oxytocin appears to work on the CNS (Fahrbach et al 1985). Knife cuts severing the efferents of the PVN nucleus do not disrupt ongoing maternal behavior (Numan & Corodimas 1985). While it is also assumed that prolactin acts directly on the CNS (Bridges et al 1985), direct proof is not yet available.

Neural Control of Maternal Behavior in Rats

As described above, maternal behavior in the laboratory rat consists of a number of different behaviors (e.g. pup retrieval, licking, crouching), each quite complex and independent, in terms of ongoing motor output, of other components. Many of these activities, however, are eliminated by

lesions of the preoptic area (POA) of the hypothalamus (Numan et al 1985). In addition, implants of crystalline estradiol into the POA reduce the latency of primed, nulliparous females to respond to pups (Fahrbach & Pfaff 1986, Numan 1987).

Estrogen plays an important role in the production of short-latency maternal behavior (see above), and the POA contains many estradiol-concentrating neurons (Pfaff & Keiner 1973). The disruptive effects of POA lesions appear to be due to projections of medial POA neurons to the lateral POA (Numan et al 1985). These LPOA cells, in turn, are postulated to affect maternal responses by means of various efferent projections, notably those to the ventral tegmental area (VTA). How the VTA influences motor activity related to maternal behavior is unclear. One possibility is via projections to nucleus accumbens and subsequent pallidal outflow. Another is via efferents back to the POA (Numan 1985). It should be emphasized that results of comparable lesions in other POA targets (e.g. central gray) on maternal behavior have not yet been reported. In addition, there is no reason to believe that the POA to VTA to accumbens outflow is important only for maternal behavior; this pathway may function in a wide variety of "motivated" behaviors.

Sexual Dimorphism in Parental Behavior of Laboratory Rats

As described above, sex differences in parental responsiveness to pups consist primarily of the latency with which behaviors are exhibited on initial exposure: seconds or minutes for parturient females, days for males or nulliparous females. A hormonal regime, mimicking critical features of pregnancy, can produce short-latency parental responsiveness in males or nulliparous females (see above). Thus, I consider sex differences in parental behavior discussed here to result from sex differences in the endocrine environment of males and females (Figure 1). Both sexes can and do display full maternal responsiveness following prolonged exposure to pups, thus suggesting that all the requisite sensory, neural, and motor systems are present. The action of hormones can be seen as lowering a sensory barrier (aversive smell) to pup approach and facilitating, probably by means of hormone action on the POA, the motor components of parental care.

One of the most interesting features of the maternal experience is its permanence. Even in males or nulliparous females, sensitization by pup exposure produces a permanent decrease in latency of the maternal response. Parturition and maternal behavior have been reported (Hatton & Ellisman 1982) to be associated with a change in synaptic ultrastructure in the female PVN. Determining whether such changes also occur in

maternal males and nulliparous females as well as in other brain nuclei associated with the parental response would be of great interest.

A few reports indicate that males may be endogenously less responsive than females. First, mammary glands are not present in adult males, so actual suckling, milk ejection, etc does not occur. Second, neonatal administration of androgen to females reduces the percentage retrieving pups in a T-maze in adulthood (Bridges et al 1973). Exposure to pups induces increases in circulating prolactin in parturient females and steroid-primed females, but not in males (Bridges 1983, Samuels & Bridges 1983). Recall, however, that all three groups are equivalently responsive to young under these test conditions. Compared with the major sex differences in behavior associated with certain courtship systems (see below), sex differences in ability to display parental care are quite minor.

Sex-reversed Behaviors—Polyandry

One way to test the generality of exclusive endocrine control of behavioral dimorphism is to examine species that are sex reversed; for example, species in which females perform all courtship and males all parental care. As described above, biparental care is more common in birds than in mammals. In the American jacana (*Jacana semispinosa*), however, males provide almost all parental care; female contributions are limited to territorial defense (Jenni & Collier 1972, Jenni & Betts 1978). Females are polyandrous and defend territories that encompass those of up to four males. Do species exhibiting a reversal of the predominant behavioral pattern (males—courtship, females—parental care) also show a corresponding reversal in type of gonadal hormone secreted (males—estrogen, females—androgen)?

Rissman & Wingfield (1984) report that in another related polyandrous shorebird (*Actitis macularia*), androgen levels are greater in preincubating males than in females or incubating males; estrogen levels in males are lower than those of females. Thus despite some reversal in courtship and parental care, the usual pattern of steroid secretion (male—androgen; female—estrogen) persists. Interestingly, however, male levels of prolactin (associated with parental care in many species) are greater than levels in females (Oring et al 1986). Elevated male prolactin levels are also seen in another polyandrous species, Wilson's phalarope (*Phalaropus tricolor*). In this latter species, again, androgen levels in nonincubating males are greater than those in females or incubating males; estrogen levels are higher in females than in incubating males (Fivizzani et al 1986).

Thus a dichotomy in hormone/behavior associations exists in these sex-role reversed birds. Levels of gonadal steroids are similar to those seen in monogamous or polygynous species. Levels of prolactin—of pituitary

origin—are reversed: the incubating male has levels as high as those of females in species in which only females incubate. Gonadal steroids thus appear largely restricted by sex: estrogen to females, androgen to males. Prolactin, however, is not restricted; why? Estrogens and androgens have pleotrophic effects: testosterone is necessary for maturation of the male and contributes to fertility and estradiol plays an analogous role in females. The requirements of sex-specific gamete production and reproductive tract development may limit flexibility in secretion of gonadal steroids. If this is the case, "sex reversed" behaviors can be produced in at least two other ways in species in which the ancestral pattern was male-courtship-testosterone/female-incubation-estrogen. One is to emancipate behaviors from gonadal steroid control entirely (see Crews & Moore 1986); the other is to express receptors for sex-typical steroids in brain regions devoted to the "sex-reversed" behavior.

Duetting in Tropical Song Birds

In temperate birds, song is the exclusive province of the male. In the tropics, however, both sexes sing in many species; often a close temporal relationship exists between songs of a mated pair. Such synchronized songs are termed duets (Thorpe 1972, Farabaugh 1982). In many song birds, song production in adulthood is controlled by androgen secretion (Prove 1983). Brain regions implicated in song control in finches (Nottebohm et al 1976) contain cells that concentrate androgenic steroids (Arnold et al 1976). The absolute number of hormone-concentrating cells, as well as the percentage of such cells, is greater in male than in female zebra finches (Arnold & Saltiel 1979, Nordeen et al 1986). Brenowitz & Arnold (1985) examined androgen accumulation in bay wrens (*Thyrothorus nigricapillus*), a species in which females always sing the leading song in duets (Levin 1983, 1985, Brenowitz et al 1985). After administration of radioactive testosterone, males and females exhibited the same proportion of steroid-accumulating cells in two telencephalic song nuclei, hyperstriatum ventrale, pars caudalis (HVc) and magnocellular nucleus of the anterior neostriatum (MAN) (Brenowitz & Arnold 1985). Whether testosterone itself or one of its major metabolites (estradiol or dihydrotestosterone) is the active form of the hormone accumulated in these brain regions remains unclear. In zebra finches, cells in HVc accumulate testosterone or dihydrotestosterone but very little, if any, estradiol (Arnold et al 1976). If we assume that hormone activation of HVc is essential for song production, it may be that female bay wrens achieve song by androgen secretion. Alternatively, cells in HVc of bay wrens might express estrogen receptors, and be activated by estrogen in females and by testosterone aromatized to

estradiol in situ in males. Considering the conservative nature of hormone secretion in birds (see above), the latter scenario seems more likely.

The accumulation of androgens in cells of HVc in male zebra finches is regulated by male-specific secretion of estradiol around the time of hatching (Nordeen et al 1986, Hutchison et al 1984). Thus, in bay wrens, either HVc cells are emancipated from the requirement for neonatal estradiol in both sexes or both sexes secrete estradiol (Brenowitz & Arnold 1985). Unlike jacanas and phalaropes, however, bay wrens are not sex-reversed. Both sexes contribute to parental care; certain aspects of courtship are male specific; females contribute most nest building (R. Levin, personal communication). If early estrogen secretion is responsible for "masculinization" of song nuclei in bay wrens, alternative mechanisms must operate to preserve other behavioral dimorphisms.

Sensory Sexual Dimorphism

Sensory structures are frequently sexually dimorphic in invertebrates. An example is antennal olfactory receptive organs of moths (Hildebrand 1985). In *Manduca sexta*, the female releases pheromones by extrusion of an abdominal gland. Both sexes have antennal receptors that respond to plant odors, but only the male antennae exhibit sensilla with receptors for the mate attraction pheromone (Schneiderman & Hildebrand 1985). In both sexes, antennal olfactory receptors project to glomeruli in the olfactory lobe (Schneiderman et al 1982). Only males have specialized olfactory glomeruli that contain processes of interneurons responsive to female-produced pheromones (Schneiderman et al 1982).

The neural system that drives male approach to the signalling female is governed by sex differences in the sensory periphery. If the imaginal disc containing antennal precursor cells is transplanted from a male to a female larva, a male antenna will develop as will a male-like olfactory glomerulus with pheromonal responsive interneurons (Schneiderman et al 1982). The available data support the hypothesis that male afferents arriving at the antennal lobe are responsible for organizing a masculine neuropil. Still unclear is whether the response characteristics of the glomerular interneurons are changed by exposure to male antennal afferents or whether afferents affect the survival of a preexisting class of responsive cells. In either case, the possession of a male antenna has a profound influence on the behavior of the moth. Schneiderman et al (1986) have recently shown that a female receiving transsexual grafts will display male-like zigzag flight approaches when placed within a pheromone odor plume. In the most extreme case, the implanted female attempted to mate with the odor source. Zigzag flight patterns were shown even by females receiving a unilateral

antennal graft. Masculine sensory input could be responsible for neural reorganization, including the production of male-like motor patterns. However, genetic females will sometimes display zigzag flight patterns toward a food source, thus suggesting that input from grafted male antennae may access an existing flight pattern generator. Some additional evidence in support of this interpretation is that the behavior of the transsexually grafted female described above culminated in oviposition.

Sexual Differentiation Within the Nervous System

For some sexually dimorphic behaviors, central nervous system participants have been described. These behaviors include the lordosis reflex of female rats (Pfaff 1980), copulatory reflexes in male rats (Hart 1980, Sachs 1981), song in birds (Nottebohm et al 1976) and calling in frogs (Schmidt 1976, Wetzel et al 1985, Kelley 1986a). Many of these systems show marked sex differences in brain nuclei that control dimorphic behaviors. For example, the volume of the CNS nucleus, number of cells, dendritic extent, synaptic input, axonal arborizations, post-synaptic targets, and number of hormone-accumulating cells have all been shown to be sexually dimorphic (Nottebohm & Arnold 1976, Greenough et al 1977, Gorski et al 1978, Gurney 1981, DeVoogd & Nottebohm 1981, Konishi & Akutagawa 1985, Wetzel et al 1985, Nordeen et al 1986). That these sex differences contribute to differences in behavioral expression seems reasonable (Gurney 1982, Kelley 1986b). For example, sex differences in the volume of telencephalic song nuclei in finches (HVc, hyperstriatum ventrale, pars caudalis; RA, nucleus robustus archistriatalis) correlate closely with behavioral differences. In zebra finches, song in females is exceedingly rare, even under appropriate endocrine conditions (see below). The male/female ratio in volume of telencephalic song control nuclei is approximately 5/1, depending on the region; one nucleus (X) is not even discernable in females (Nottebohm & Arnold 1976). The volume of telencephalic song control regions in adult zebra finches and white crowned sparrows does not vary seasonally nor can the nuclei be induced to grow by hormone treatment in adulthood (Gurney & Konishi 1980, Baker et al 1984). In canaries, however, some androgen-treated females sing. Song frequency and size of vocal control nuclei can be increased by hormone treatment. Brain nuclei, while dimorphic, are normally less so than in zebra finches (Nottebohm & Arnold 1976, Nottebohm 1980, 1981). In some tropical species, both males and females sing (see above). Song control nuclei in several duetting species have been examined (Brenowitz et al 1985, Brenowitz & Arnold 1986). In species such as the bay wren, where females sing as frequently as males, there is no sex difference in volume of song control nuclei (Brenowitz & Arnold 1986). In duetting

species with greater male song complexity (e.g. the rufous and white wren), song nuclei are dimorphic though less so than in canaries or zebra finches (Brenowitz & Arnold 1986). Thus, behavioral and brain dimorphisms are well correlated.

As described above, many sex differences in behavior are attributable to sex-specific hormone secretion patterns. Some effects of hormones are reversible. An example is the induction of clasping by androgen in adult clawed frogs (Kelley & Pfaff 1976). A classic example of an irreversible effect of hormones is the masculinization and defeminization of neonatal female rats given testosterone (Goy & McEwen 1980). Androgen administration produces permanent changes in behavioral capabilities, including sensitivity to the behavioral effects of steroids. Permanent effects usually, though not necessarily, occur during a circumscribed period early in development (Goy & McEwen 1980). Reversible effects are usually (though again not necessarily) characteristic of sexually mature adults. Both sorts of effects require steroid hormone receptors; mutants defective in receptor expression are behaviorally insensitive to these hormones (Olsen 1979).

Sex differences in dimorphic brain nuclei are also controlled by secretion of gonadal steroids (Gurney & Konishi 1980, Arnold & Gorski 1984). Some dimorphisms are due to the action of circulating hormones on the adult brain and are reversible. Song control nuclei in canaries wax and wane in volume seasonally in response to changes in circulating androgen (Nottebohm 1981). Other sex differences in the brain appear to result from the irreversible action of steroids during development of the nervous system (Raisman & Field 1973, Gurney 1981, Jacobson et al 1981). The volume of telencephalic song control nuclei in zebra finches is irreversibly established by hormone-stimulated growth at hatching and prior to the first singing season (Gurney & Konishi 1980, Hutchison et al 1984). Early hormone secretion regulates the number of androgen-responsive cells in these areas (Arnold & Saltiel 1979, Nordeen et al 1986). Organizational and activational effects of steroids on behavior and on responsible brain nuclei are thus closely correlated.

Cellular Contributions to CNS Sexual Dimorphism—Cell Number

At the cellular level, there are many kinds of sex differences in behavioral effector brain nuclei: volume, cell number, somal size, dendritic branching, afferent volume, percentage of hormone-target cells (reviewed in Arnold & Gorski 1984, Konishi 1985, Kelley 1986b). Behaviorally relevant brain dimorphisms must ultimately contribute to differences in synaptic connectivity. Thus we might expect to find certain axonal projections in one sex and not in another or to find sex differences in the robustness or efficacy

of synaptic connections. This last mechanism has been considered in detail at certain sexually dimorphic neuromuscular synapses (see below). In CNS nuclei that contribute to vocalization in frogs and birds, some connections are reduced or absent in females (Wetzel et al 1985, Konishi & Akutagawa 1985). The number and placement of synapses also can differ in telencephalic and diencephalic nuclei (DeVoogd & Nottebohm 1981, Raisman & Field 1973). Whether differences in synaptic connectivity between the sexes arise from different projections and/or from different frequencies of homologous neurons remains to be clarified.

Many sexually dimorphic brain nuclei exhibit differences in cell number. In song bird nuclei HVc and RA, the rat spinal nucleus of the bulbocavernosus (SNB), and motor nucleus N.IX-X of clawed frogs, the number of neurons is greater in males than in females (Gurney 1981, Breedlove & Arnold 1981, Kelley 1986a). The number and efficacy of contacts between neurons contribute to cell number—afferent input, connection to appropriate targets, and synaptic competition are all important (Hamburger & Oppenheim 1982). Thus manipulation of cell number in a key brain nucleus can have dramatic effects on cells synaptically connected to that nucleus and on cells in competition for afferents and efferents.

The effects of changes in cell number during development on synaptic connectivity are diagrammed in Figures 2 and 3. Consider the consequences of adding additional cells to brain nucleus B (Figure 2). Neurons in nucleus A, which otherwise would have died (x) due to an insufficient target in B, now survive. The additional neurons in B now send axons to C and effectively compete with axons from D; this leads to the death (X) of the latter cells. The end result is a robust connection from A to B to C and no connection from D to C. If, instead of cell addition, nucleus B loses neurons, a very different scenario ensues (Figure 3). Cells in A do die (x) due to an insufficient target in nucleus B. Cells in B die due to lack of afferents. Cells in D that project to C now survive due to lack of competition. In the first case, the A to B to C connection is established and D to C lost. In the second, the A to B to C connection is lost, but D to C survives. These alternative cases illustrate how cell number in a key brain nucleus can make dramatic contributions to synaptic connectivity. If, for example, androgen maintains cell number in nucleus B, its secretion in males (Figure 2) and not in females (Figure 3) would generate sex-specific patterns of CNS circuitry. There is quite good evidence that a series of events, similar to that outlined here, contributes to sexual dimorphism in connectivity in zebra finch brain (Gurney 1981, Arnold & Gorski 1984, Konishi & Akutagawa 1985, Nordeen et al 1987b).

Neuron number in a CNS nucleus is governed by proliferation, migration, and death. Administration of steroid hormones can affect cell

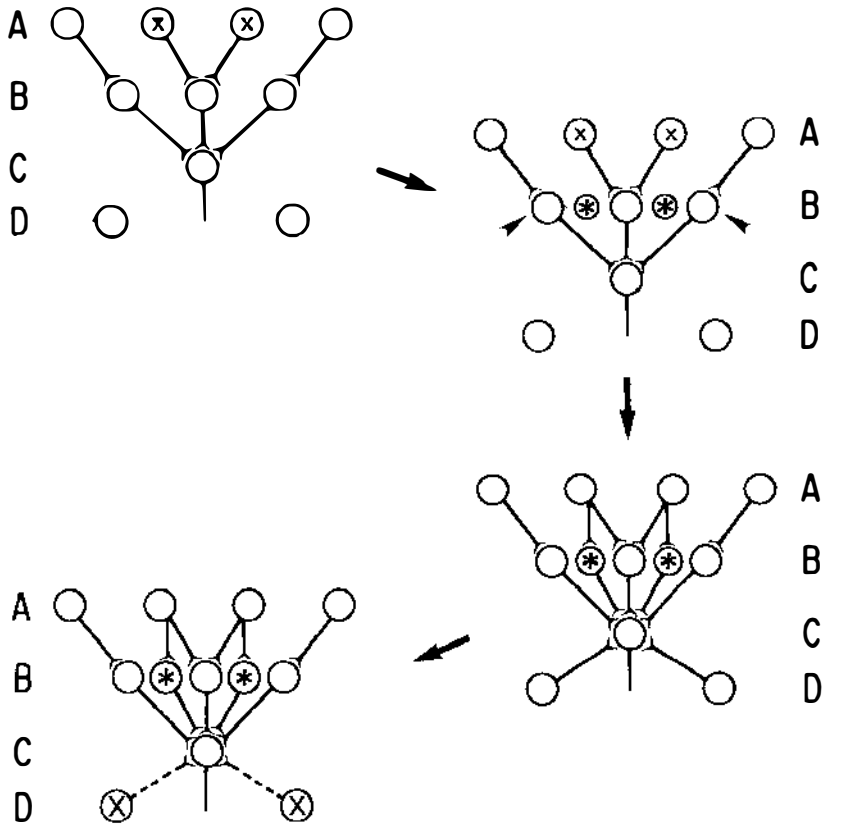

Figure 2 Generation of sex dimorphisms in neuronal circuitry by differences in cell number. *Ab initio* (*upper left*), some neurons (x) in nucleus A are destined to die because of the limited target availability in nucleus B. The addition of neurons (*) to nucleus B rescues these neurons in A, preserving and strengthening the A-to-B connection. However, afferents arriving at nucleus C from D now (*third panel*) face stiff competition and ultimately die (X, *lower left*).

number in dimorphic, behavioral effector nuclei, thus suggesting that steroids control one of the above processes (Gurney 1981, Arnold & Gorski 1984, Arnold 1984, Konishi & Akutagawa 1985, Sengelaub & Arnold 1986). For the most part, neurogenesis is complete before steroids are secreted or affect cell number, and before steroid receptors appear (Jacobson & Gorski 1981, Breedlove et al 1983, Gorlick & Kelley 1986, 1987). However, one song nucleus in the brain of canaries and zebra finches, HVc, continues to add new neurons in adulthood by migration from an overlying stem cell population (Goldman & Nottebohm 1983). Some new

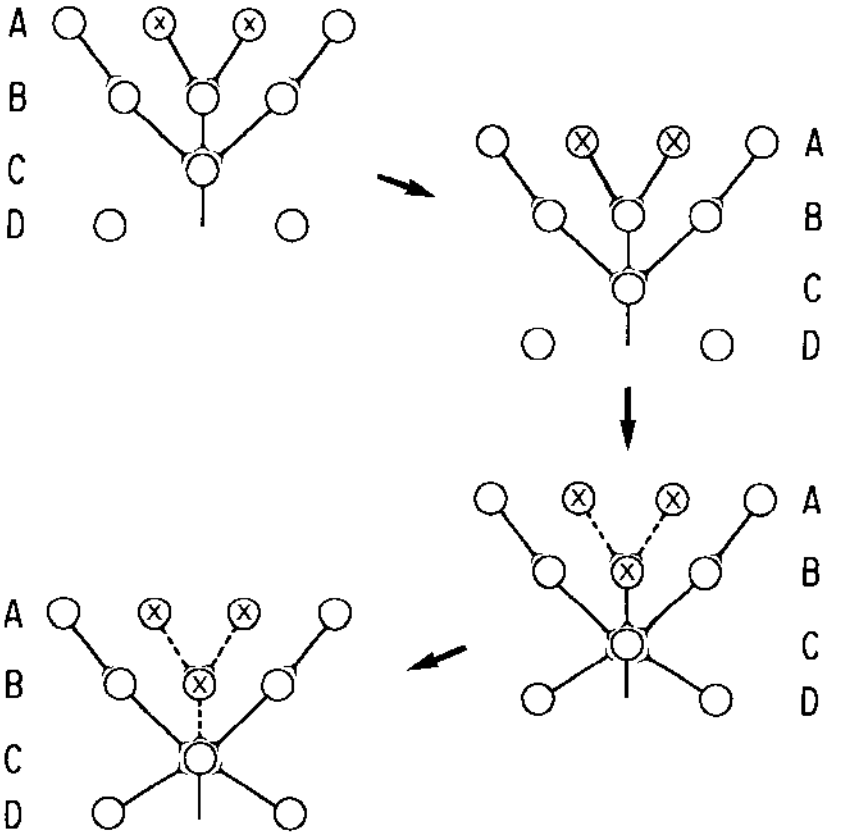

Figure 3 Consider instead the case of cell loss in nucleus B. Consequences include loss of neurons (x) in A, loss of targets in B, and ultimate degeneration of the A-to-B connection. The D-to-C connection, however, is preserved in this case, resulting in a set of connections different from those established by cell addition to B. Sex-specific hormone regulation of cell number in one, strategically placed brain nucleus (by proliferation, migration or death) could, in this fashion, create sex differences in connectivity.

neurons in HVc are responsive to acoustic stimuli, thus indicating their incorporation into functional circuitry (Paton & Nottebohm 1984). New neurons in HVc thus might contribute to sex differences in cell number (Nordeen et al 1987b).

In rat spinal cord, the sexually dimorphic SNB neurons arrive at their characteristic location by secondary migration from an adjacent motor nucleus (Sengelaub & Arnold 1986). Sex differences in neuron number, however, seem to be primarily due to prevention of ontogenetic cell death (Nordeen et al 1985). Cell death has also been postulated to account for

male/female differences in the song nucleus, RA, of zebra finches (Gurney 1981, Konishi & Akutagawa 1985). Steroids could prevent ontogenetic cell death by affecting synaptic connectivity (more cells, larger somata and dendritic trees, more numerous axonal projections) or by exerting direct or indirect trophic control of developing targets in a manner analogous to developmental actions of nerve growth factor. One very puzzling finding has been that, although estradiol increases somal size and cell number in HVc and RA of nestling zebra finches, these regions have few, if any, intracellular receptors for estradiol either in adults (Arnold et al 1976) or neonates (Nordeen et al 1987a). Estradiol may be acting via membrane receptors or afferents from estrogen target neurons. Alternatively, estradiol may be promoting the secretion of a growth factor or synthesis of growth factor receptors in dimorphic target neurons.

One contribution of steroid hormones to regulation of sexual dimorphism may be control of expression of cell types. This has been a difficult question to address experimentally because of the paucity of cell type markers. Male and female brain nuclei appear to contain the same morphological cell types (Gurney 1981, DeVoogd & Nottebohm 1981, Kelley 1986b). However, output cells in female zebra finch HVc project only to a rim of tissue surrounding RA and not into RA itself (Konishi & Akutagawa 1985). It is not clear whether male and female RA/HVc nuclei contain different output neuron types or different projections of homologous neurons. Perhaps the best evidence on cell type comes from studies of androgen-concentrating cells in the zebra finch telencephalon; females have many fewer such cells in MAN and HVc (Arnold & Saltiel 1979). During development, estrogen acts to preserve androgen-concentrating MAN cells in the face of massive ontogenetic death of other MAN cell types (Nordeen et al 1987b). Thus some sex differences in song nuclei are likely to be due to differences in cell type. Estrogen may rescue existing androgen-concentrating cells or else increase receptor expression in previously insensitive neurons.

Control of Sexually Dimorphic Behaviors at the Neuromuscular Junction

Finally, sex differences in behavioral expression can also be controlled by differences in motor neurons and muscles (Figure 1). In the extreme, muscles may be completely absent in one sex, as in the levator ani/bulbocavernosus muscles (LA/BC) in rats. These muscles control copulatory reflexes in males that are essential for successful insemination (Hart 1980). At birth, LA/BC muscles are present in both sexes but involute in females unless supplied with androgen (Breedlove & Arnold 1983b). The motor neurons supplying these muscles (the SNB nucleus) are approxi-

mately five times more numerous in adult males (Breedlove & Arnold 1983a,b). SNB motor neurons express androgen receptor in adults (Breedlove & Arnold 1981), as do their targets, the LA/BC muscles (Jung & Baulieu 1972). Administration of androgen during the perinatal period prevents ontogenetic cell death in female SNB cells (Nordeen et al 1985). Androgens rescue male SNB neurons from cell death by acting on the LA/BC target muscles. Autoradiographic studies show that there is no androgen accumulation in SNB cells at developmental times when rescue occurs (Breedlove 1986a). SNB cells that do not express functional androgen receptor can be rescued from ontogenetic cell death provided that both androgen and androgen responsive LA/BC muscles are present (Breedlove 1986b; see also Kelley 1986b). Cells of the SNB in males remain responsive to androgen into adulthood; testosterone can induce extensive dendritic growth in castrates (Kurz et al 1986).

Even when homologous muscles are present in both sexes, sex differences in muscle fibers or at the neuromuscular junction may constrain behavioral expression. The most dramatic example is the larynx of clawed frogs, *Xenopus laevis* (see Kelley 1986a for a more extensive review). Male frogs use mate calls, trills with alternating fast and slow phases, to attract and excite females (Wetzel & Kelley 1983). Females use a slower, monotonous trill-ticking to avert or terminate male clasp attempts (Weintraub et al 1985). While males also tick when clasped, females do not mate call even when given the appropriate male-typical hormones in adulthood (Hannigan & Kelley 1986). Brain regions that participate in vocal production have been identified (Schmidt 1976, Wetzel et al 1985) and some are sexually dimorphic in cell number, dendritic extent, somal size, and connectivity (Kelley 1986a). The male central nervous system generates the mate call temporal pattern and rate of activity in laryngeal motor neurons (Tobias & Kelley 1987).

Laryngeal muscles and neuromuscular junctions are responsible for sex-typical constraints on the rate at which calls can be produced (Sassoon et al 1987, Tobias & Kelley 1987, 1988). The isolated male larynx can be induced to mate call by providing the mate call pattern of stimulation to the laryngeal nerves; the female larynx cannot call at mate call rates under these conditions (Tobias & Kelley 1987). Adult males have eight times the female number of muscle fibers, and cells are of the fast-twitch, fatigue resistant type (Sassoon & Kelley 1986, Sassoon et al 1987). Levels of androgen receptor in adult male muscle are four times that of females (Segil et al 1987). Male muscle fibers require repetitive stimulation to produce action potentials, probably because most synaptic terminals release only small amounts of neurotransmitter in response to a single shock to the laryngeal nerve (Tobias & Kelley 1988). Adult female la-

ryngeal muscles are heterogeneous in fiber type; most are slow twitch (Sassoon et al 1987). All female fibers produce action potentials when the nerve is stimulated. Further, muscle fibers are extensively dye coupled in females, thus suggesting that they function syncytially (Tobias & Kelley 1988).

Characteristics of the muscle fibers themselves and of their synaptic innervation are well matched to the demands of vocal production in each sex. The slow rate of ticking permits relaxation of female, slower twitch fibers between each contraction. The high safety factor at female neuromuscular junctions and coupling of fibers ensures that the small number of fibers will contract together effectively enough to produce a click. The large number of fibers in the male provides sufficient cells for recruitment; fiber characteristics permit fast twitches and fatigue resistance. Low safety factor synapses with attendant requirements for facilitation in males ensure that some fibers will not contract during every nerve volley; this mitigates fatigue and facilitates recruitment.

Certain developmental events contributing to laryngeal sex differences have been identified. As is the case in the central nervous system, control of cell number and type in the neuromuscular periphery is a key component. At metamorphosis, laryngeal muscle fiber number in both sexes is the same as in adult females (Sassoon & Kelley 1986). In response to rising androgen levels, males add new fibers for the next ten months; no net addition of muscle fibers occurs in females (Sassoon et al 1986, Sassoon & Kelley 1986). In addition to controlling fiber number, androgen also influences fiber type expression. In juvenile females, exogenous androgen converts the heterogeneous pattern of female fiber types (predominantly slow) to a homogenous, all-fast profile (Sassoon et al 1987). Androgen treatment does not similarly convert fiber types in adults.

Regulation of synaptic input to laryngeal muscle fibers during development has not yet been examined. In adults, some male muscle fibers are multiply innervated (Tobias & Kelley 1988). One possibility is that the multiple innervation of single fibers seen in males reflects androgen-induced prevention of synapse elimination. This mechanism has been proposed in the androgen-sensitive LA/BC muscle of male rats, which is retarded, relative to other muscles, in withdrawal of supernumerary axon terminals (Jordan et al 1986).

Summary and Conclusions

Sex differences in behavior are the result of natural and sexual selection. The dimorphic classes of behavior described here, courtship, copulatory, and parental behaviors, reflect both kinds of evolutionary selective pressures. We can further distinguish two kinds of mechanisms that produce

differences in male and female behaviors. In one, both sexes can perform a behavior but one does not because of sex differences in the external stimuli or the endocrine milieu. Maternal behavior in rodents falls into this category, as do certain other reproductive behaviors. In the other, the sensory, CNS, or motor components that produce behaviors are different in males and females. Many courtship and copulatory behaviors are in this category. I have considered some cellular mechanisms that generate sex differences in behavioral effector neurons, including sensitivity to hormones, cell number, and synaptic connectivity. A common feature of many such systems is a degree of developmental arrest: sexually dimorphic, hormone-sensitive neurons or muscles are immature at stages when other cells have completed differentiation. The cellular and molecular processes whereby hormones harness the developmental programs of behavioral effector cells remain largely unknown and are the focus of active investigation.

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