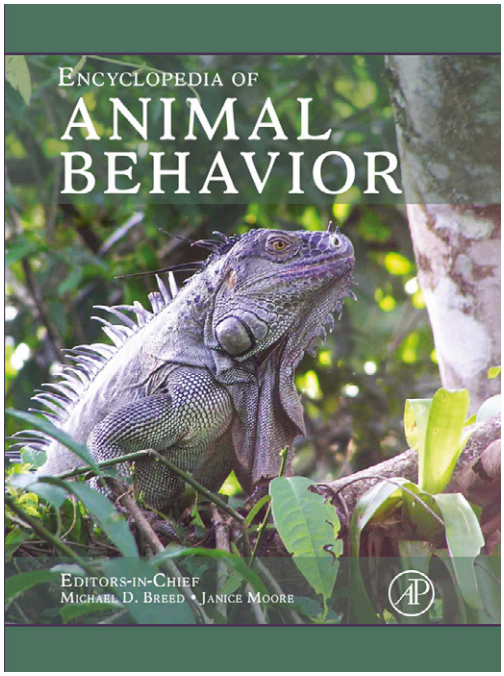

Crews D. (2010) Neural Control of Sexual Behavior. In: Breed M.D. and Moore J., (eds.) Encyclopedia of Animal Behavior, volume 2, pp. 541-548 Oxford: Academic Press.

Neural Control of Sexual Behavior

D. Crews, University of Texas, Austin, TX, USA

Introduction

The brain is a sexual organ, which like the gonad, is initially bipotential, differentiating into one of two types. More than a century of scientific research has established that the brain is the mediator and regulator of all aspects of reproduction. In this article, I trace the evolution of ideas related to brain organization and the control of sexual behavior. The question guiding investigators underwent a major paradigm shift 50 years ago: from the original emphasis on the bisexual nature of the brain to how the brain happens to differ in males and females. This may not seem to be an important distinction, but considering that, in the first instance, the emphasis is on the similarity of the sexes while in the second and current perspective, importance is placed on the differences between the sexes, there definitely is a shift in direction. In both psychology and biology, it is commonplace for investigators to not so much solve problems as to create new questions, without resolving the original question with the advent of newer techniques; in this instance, effort toward understanding the brain's inherent bisexuality was deflected to understanding the organ's sexual differentiation. Recently, a new paradigm has been introduced, which may reunite researchers as they address the two questions.

100 Years Ago

In the late 1800s, the focal question was why one sex would behave like the opposite sex, a phenomenon noticed more commonly in some species. The late 1800s and early 1900s marked the beginning of the realization that reproduction and sexuality differed in origin and consequence. In particular, Richard von Krafft-Ebing and Sigmund Freud speculated on the bisexual nature of the brain. It was during this period that 'bisexual' came to mean 'bipotential,' meaning that the same anlagen (the rudimentary beginnings of an organ, usually in the embryo) would give rise to one of two states, rather than the same structure housing two distinct states. Some time later, researchers such as Eugen Steinach (Austria) and Calvin Stone (USA) demonstrated that the interstitial (Leydig) cells (and not the Sertoli cells) of the testes produced the hormones (initially called 'incretions') responsible for seasonal as well as pubertal growth of secondary sex characters. These and other researchers (e.g., Carl Moore) suggested that hormones cause an 'eroticization' of the central nervous system, though there was considerable debate as to whether they were acting generally or at specific sites. What was resolved by 1940 was that hormones changed the individual's sensitivity to specific stimuli (e.g., tactile, visual, and odor cues). It was also accepted that while males and females exhibited characteristic behaviors, they had the capacity to exhibit the behavior of the opposite sex. Indeed, Frank Beach in his compendium *Hormones and Behavior* devoted its second chapter ('Reversal or Bisexuality of Mating Behavior') to this common observation. Like others before him, he stressed that such heterotypical behaviors were exhibited alternately, never coincidentally, and were elicited by the stimulus context, and not by specific hormones. It is important to note here that early ethologists such as Tinbergen also emphasized the role of tonic inhibition in switching between behaviors.

> The general impression that one gains from a survey of the literature tends to throw some doubt on any concept of sex reversal which depends upon complete sex-specificity both of the behavioral mechanisms and of the gonadal hormones A somewhat more reasonable hypothesis would seem to be that in many if not all vertebrate species both males and females are equipped by nature to perform at least some of the elements in the overt mating pattern of the opposite sex.' (Beach, 1948, p. 69)

50 Years Ago

In 1959, a single publication by William C. Young and his colleagues changed the paradigm of behavioral endocrinology so much so there has been little work on bisexuality of the brain since that time; this seminal study set the trajectory of research on the neuroendocrinology of sexual behavior to the present day (this review is cited as Phoenix et al., in the readings at the end of this article). Indeed, for the past 50 years, almost all research in this area has focused on why males (or females) behave the way they do.

Drawing the analogy with the differential development of the accessory sex structures during embryogenesis as described by Alfred Jost a few years previously, Young and colleagues suggested that a similar dual anatomy exists in the brain, proposing that just as the early hormonal environment determined the fate of the ducts that transport eggs (Müllerian ducts) or sperm (Wolffian), these hormones also acted on the developing brain, specifically on the

neural circuits subserving female- and male-typical sexual behaviors. In addition to its embryological foundation, the new perspective also built on the foundation laid earlier demonstrating that sexual behaviors were not simply dictated by sex steroid hormones, but reflected mechanisms intrinsic to the state of the brain itself. Although it was recognized that in some way the hormones were acting on the brain, the mechanism of this action was a mystery. It should be pointed out, however, that this new perspective in itself did not explain (nor did it seek to) the observation that individuals of either sex retain the capacity to, and commonly display, the behaviors typical of the opposite sex. The Organizational/Activational concept of Young and colleagues was further refined a few years later by Richard Whalen with the concept that the development of sex-typical behaviors resulted from two independent processes, namely, masculinization–demasculinization and feminization–defeminization (see section 'Are There Dual Circuits or a Single Circuit with Alternative Outputs?'). Put simply, 1959 marked a time when the salient question transitioned from 'why do males and females sometimes behave as the opposite sex,' to 'why do males behave like males and females like females.'

The Origin of Sexual Behavior

Before proceeding further, it is first necessary to raise the issue of the origin of sex itself. I am not referring to the evolution of sexual reproduction, or even why the preponderance of life forms exhibit two sexes. Instead of asking why sex evolved, it might be informative to ask who came first, male or female. The scientific view is that the 'female' was the first sex. (I would like to avoid the semantics for a moment as male and female are defined in terms of the opposite sex.)

There is little question among researchers that the first organisms simply cloned themselves. In each new generation, the complete genetic material of the parent and the siblings was identical. This same process occurs today in organisms that reproduce by parthenogenesis. In the process of evolution, the gametes were initially uniform in size (isogamy); but with time, they became different in size (anisogamy) and contained only one half of the genetic material that produced a new individual when the complementary types were fused (fertilization). This suggests – and evidence supports it – the supposition then that the first 'sex' was an egg producer. Put simply, what is called 'female' today was in fact the ancestral sex with males (sperm producer) relatively late entrants in the game of life.

Originally then, the brain was required only to coordinate and stimulate the production of eggs. With the development of two types of gametes came the need for behaviors that would be complementary, thereby ensuring fertilization. If one considers that the first sex was female, and males were derived much later in evolution, it stands to reason that behavior associated with ovulation (i.e., female-like receptivity) is the ancestral state and behavior associated with the delivery of sperm (i.e., male-like mounting) is a derived state. This more recent origin may account for 'male sexual behaviors' to be more plastic than are 'female sexual behaviors.'

Switching Between the Sex Roles

Early in development (the when and how varies between species), genes and hormones interact to organize the functional neuroanatomy such that later, as adults, males and females will exhibit complementary behaviors necessary for successful reproduction. This concept was originally built on an analogy with the sexual differentiation of the genital tract, and its characteristics were (i) completion during a limited sensitive window of embryonic development or shortly after birth, (ii) irreversibility, and (iii) the presumed existence of separate neural structures mediating male and female sexual behaviors. Although these particulars have been modified since to account for species differences, extensive research with rodents revealed a male-specific testosterone surge toward the end of in utero development, enabling later expression of male behavior (masculinization) and disabling later expression of female behavior (defeminization).

In formulating the Organizational/Activational Concept, Young and colleagues did not ignore, but did give rather short shrift to the observations available at the time that sexual behaviors characteristic of the opposite sex were displayed by individuals of most species studied, and particularly common in some. In view of this inherent and persistent bisexuality of the vertebrate brain, I believe that the analogy to the dual duct system was unfortunate and misleading. Rather, a more accurate perspective is to consider the network of limbic and hypothalamic nuclei involved in the control of sexual behavior to be a single entity, whose entirety is organized in a male- or female-typical way. How the implications of this perspective for the way research is conducted differ from those of a model based on the independent existence of separate 'centers' for male and female behavior in several ways is discussed further below.

Activation and Deactivation, Inhibition, and Disinhibition

A second kind of plasticity, observed in adulthood, is the activation and deactivation of behavior. Females display receptive behavior during the periovulatory phase of the ovarian cycle when estrogen levels are high, and at other

times reject courting males. Males display mounting and other copulatory behaviors toward receptive females throughout the breeding season when androgen levels are high, and at other times show no particular interest in females. Activation of copulatory behavior appears to depend on gonadal sex steroids, being eliminated by gonadectomy and activated by exogenous testosterone (males) or estrogen and progesterone (females). However, gonadectomy followed by administration of the sex steroid typical of the opposite sex generally is not thought to activate the behavior typical of the opposite sex, a failure that is attributed to the permanent effects of developmental organization.

If one accepts this Developmental Organization followed by Adult Activation paradigm, one tends to view sex differences in brain structure as likely candidates for being involved in the display of male-typical behavior by males and female-typical behavior by females. Experimentally one asks how these differences arise during development, and then how the sexually dimorphic circuits are activated in adulthood. This perspective is little changed in recent years as the use of genetically modified mice has entered mainstream research on hormone-brain-behavior research.

Are There Dual Circuits or a Single Circuit with Alternative Outputs?

An influential conceptualization of how the brain might differentiate in males versus females was the Orthogonal Model of Richard Whalen (**Figure 1**, top panel). Summarizing the evidence to date, Whalen concluded that sexuality was not a one-dimensional or linear continuum, with masculine and feminine at opposite ends as originally proposed by the early philosophers. Rather, Whalen suggested that sexuality comprises two distinct dimensions, one signifying the degree of masculinization and the other the degree of feminization. In the process of organization, these were affected differently to result in individuals typically displaying behaviors consistent with their gonadal sex.

The model was believed (and continues to be so by many) to reflect brain differentiation, along with an explicit identification of particular brain areas corresponding to masculine and feminine tendencies (e.g., see the work of McEwen listed in the readings at the end of this article). Early studies established that the medial preoptic area (mPOA) was the final integrative area necessary for the display of the male-typical mounting behaviors with the ventromedial nucleus of the hypothalamus (VMN) playing the comparable role in female-typical sexual receptivity (**Figure 1**, bottom panel). A common assumption was that when applied to the brain, the Orthogonal Model suggested that these particular nuclei were differentially influenced by early hormonal milieus and represented by two (dual) circuits, a view that is consistent with the canonical Organization-Activation paradigm outlined earlier. However, unlike the definitive work on the song system in birds, and despite the abundant work on sex differences on morphological and neurochemical aspects of mPOA and VMN in the mammalian brain, there is remarkably little evidence that the recorded differences are more than correlates of observed sexually differentiated behaviors. As Södersten put it, "the search for morphological sex differences in adult rat brains that are caused by the 'organizing effect of perinatal androgen' and that can be related to sex differences in behavior has not been fruitful and may continue unrewarded."

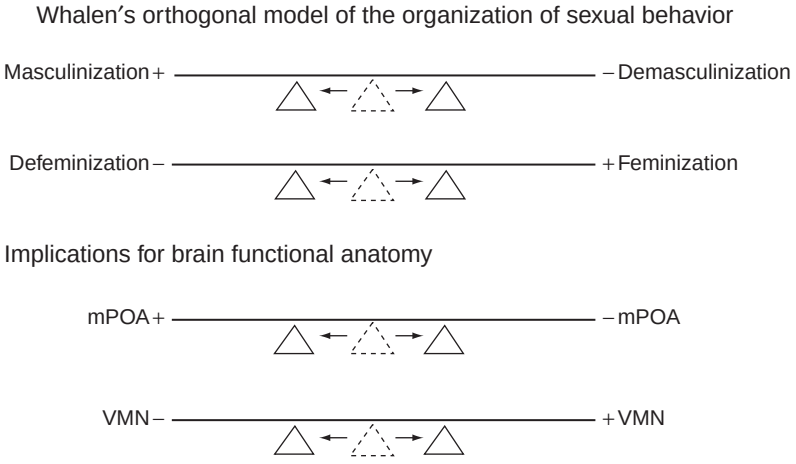

Figure 1 Whalen's (1974) Orthogonal Model for the Differentiation of Sexual Behavior (top panel). Masculinization and feminization are considered separate neuroendocrine organizational processes, such that during development in males masculine traits are enhanced and feminine traits are suppressed (=defeminization); the complement is postulated to occur in females. Extensive research indicated the final integrative area for mounting is the medial preoptic area (mPOA) and for receptivity the ventromedial hypothalamus (VMN). Thus, when the Orthogonal Model was applied to the brain (bottom panel), a parallel process of enhancement and suppression was believed to occur in the mPOA and VMN.

It is obvious that sexual behavior is the result of many brain nuclei acting in concert in addition to the external stimuli and the hormonal history of the participating individuals. An attractive formulation of this body of work is found in Sarah Newman's concept of a Social Behavior Network that underlies sexual behavior (**Figure 2**). By shifting the focus of study from single nuclei (nodes) in isolation to integrated networks, Newman predicted this would lead to new insights into brain-behavior relationships. Importantly, Newman focused on sex differences and did not consider the application of this model to address the question of the possible interactions within the network when animals display heterotypical sexual behaviors. Using this as a platform, I suggest that the sex-typical differences in behavior are the result of how the network activity varies as a result of the reciprocally inhibitory interaction of two root nodes (mPOA and VMN).

The Dual Circuits Model emphasizes how hormones act on dual neural circuits, one subserving male-typical mounting and the other female-typical receptivity, with each viewed as operating relatively independently of one another. Traditionally, research supporting this model is exemplified by study of a single sex with the dependent variable being the sex-typical behavior; c.f., mounting in male individuals, receptivity in female individuals. This has led to the development of models of the neural circuit of lordosis in female or mounting in males, but in isolation of its complement (**Figure 3**). On the other hand, the Common Network Model emphasizes how hormones act on a single neural network resulting in two mutually exclusive outputs. This model reflects the increasing appreciation of how brain nuclei are networked by neurochemical and molecular interactions and how these neural systems are fundamental (in an evolutionary sense), particularly when the brain must alternate between mutually exclusive behavioral outputs. The Common Network Model suggests then that sex-typical behavioral phenotypes are mirrored by specific neurotransmitter and molecular phenotypes in two functionally associated nuclei (as 'root nodes' of a larger network of nuclei). The whiptail lizard is instructive because it enables deconstructing the confounding properties of genotype-, sex hormone-, and developmental-specificity inherent in conventional mammalian model systems.

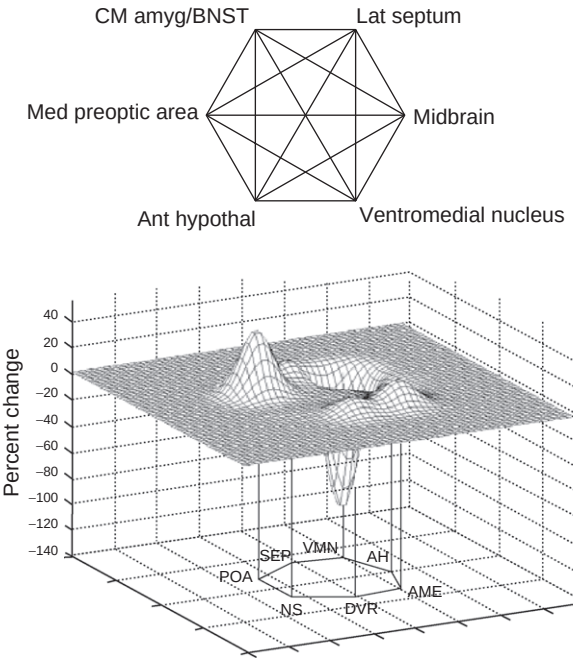

Figure 2 Newman's Social Behavior Network. Top panel illustrates how a limbic neural network consists of specific nuclei that are both hormone sensitive and reciprocally interconnected. The network of brain nuclei is similar in both sexes, but Newman proposed that the activity of the network is different in males and females when they display sex-typical (homotypical) behaviors. She did not speculate on the patterns of activity that may be reflected during the display of heterotypical sexual behaviors. Bottom panel depicts such a network as indicated by the pattern of metabolic activity (as measured by cytochrome oxidase histochemistry) in identified nuclei. The peaks and valleys indicate the *differences* in average abundance in each nucleus in sexually experienced male and female leopard geckos from the same incubation temperature; peaks indicate males greater than females and valleys indicate females greater than males. Geckos exhibit temperature-dependent sex determination and lack sex chromosomes, so differences are due to endocrine history and not genotype. Note the sex difference, particularly in the relationship between the preoptic area (POA) and the ventromedial nucleus of the hypothalamus (VMN). AH – anterior hypothalamus; AME – medial amygdala; DVR – dorsal ventricular ridge; NS – nucleus sphericus, homolog of the mediobasal amygdala; SEP – septum.

Reciprocal Inhibition Between the POA and the VMN

Certainly there is ample evidence that the mPOA and VMN are crucially involved in the control of male- and female-typical sex behaviors, respectively. What is less part of the current orthodoxy is the possibility that the two centers work in concert, albeit in a mutually antagonistic manner. However, the involvement of each brain area in behaviors typical of the 'other sex' is not lacking. Two examples are that implantation of testosterone into the VMN restores sexual motivation, but not copulatory behavior itself, in castrated male rats; administration of either androgen receptor antagonists or microlesions within the dorsomedial VMN impairs sexual motivation and copulatory behavior in male rats. Further, multiple lines of evidence indicate the mPOA and VMN are functionally related in an opposing fashion; the mPOA projects to, and receives, projections from the VMN. The mPOA and VMN also have opposing roles in the control of autonomic function and female reproductive behavior characterized by Pfaff and colleagues: 'net effect of the outputs

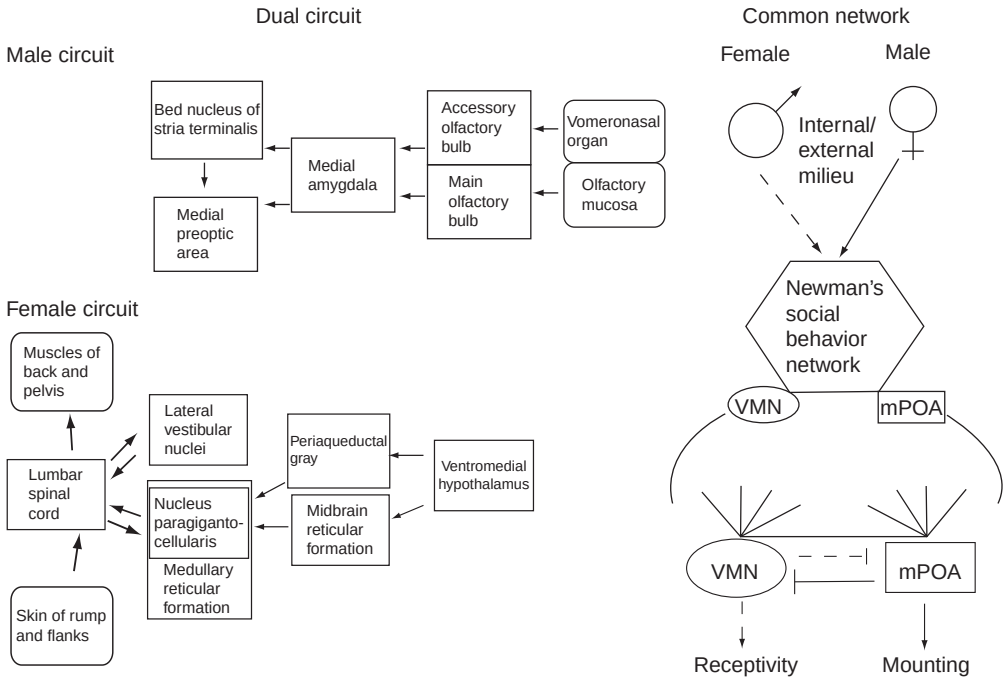

Figure 3 Two contrasting models of the neural mechanisms underlying sexual behavior. The Dual Circuits Model (left panel) suggests separate neural circuits (circles and squares represent brain nuclei/areas) ending with the medial preoptic area (mPOA) or ventromedial nucleus of the hypothalamus (VMN) as final integrative areas for male- and female-typical behaviors. The Common Network Model (right panel) posits that a common network of nuclei (Newman's Social Behavior Network) is involved in sexual behavior of both males and females, and it is the reciprocal interaction between two root nodes of this network (mPOA and VMN) as well as the hormonal history of the individual and the nature of the stimulus context that determines the type of behavior exhibited. Neuroanatomical pathways mediating copulatory behavior. In the Dual Circuits Model, the brain nuclei known to be important in the expression of the various behaviors are depicted as square boxes, and the projections between them (of which some are excitatory and some inhibitory) are shown as arrows. Sensory and effector organs are shown as rounded boxes. Brain nuclei critical for hormonal control over behavior are shown as bold boxes. Top portion depicts the Male Circuit involved in processing sexually relevant chemosensory information has been well studied in the male rodent, and involves the pathway from the main and accessory olfactory bulbs to the amygdala, particularly the medial division, and then via the bed nucleus of the stria terminalis and an alternative route via the ventral amygdalofugal pathway to the medial preoptic area. Exactly what happens to this information once it reaches the medial preoptic area is anyone's guess, but is presumed to result in the decision to attempt to mount. Once the mount is established, events in both the female and male involve reflex arcs mediated by well-characterized neural circuits, shown by bold arrows. Bottom portion depicts the Female Circuit underlying lordosis in the female rat and involves the transfer of sensory information from the male's mounting and thrusting movements to the lumbar spinal cord, when it ascends to the brainstem motor nuclei responsible for integrating the muscular motor pattern of lordosis. Descending control over this reflex arc is exerted by the ventromedial hypothalamus via the periaqueductal gray and the midbrain reticular formation.

from the preoptic region is to reduce feminine-typical behavior and to increase male-typical behavior.' Neuronal activity increases in the VMH during sexual receptivity in the female rat, and is reduced when there is increased activity in the mPOA. Effects of excitatory and inhibitory amino acid neurotransmitters are opposite in the VMN and mPOA. Specifically, in the VMN, GABA is facilitatory, while NMDA is inhibitory, to lordosis, and in the POA, GABA inhibits and NMDA facilitates, lordosis in hormone-primed female. So, ought the two centers to be considered as two independent neuroanatomical anatomical units, one of which will be chosen by developmental events, to determine the sexual phenotype of the animal, while the other languishes? This model should be rejected in favor of a mutual inhibition model in which the centers work actively together antagonistically in both sexes, in a way more analogous to the interactions of two political parties, the balance of whose power determines their joint decisions.

Looking to Nature

The central issue in science is the support (or lack thereof) of the hypothesis. In this particular case, however, there are inherent and seemingly insurmountable obstacles to proving whether the there are dual neural circuits in each individual, with one predominant in one sex and the another in the opposite sex, or whether there is a single sex behavior circuit that is modulated to produce one of two outputs. This hypothesis cannot be addressed in mammals and any other species having heritable sex

chromosomes (e.g., XX females and XY males). Not only do the sexes differ in an elemental gene, but males and females develop and age in entirely different endocrine milieus and, as a consequence, have different life history experiences.

Fortunately, we can look to nature for the necessary evidence. Hermaphroditic species come in several varieties. Simultaneous hermaphrodites are species in which each individual produces both sperm and eggs, but curiously, never at the same time. When breeding, one individual will assume the 'male' role and shed sperm and its partner the 'female' role and shed eggs. In the next spawning, even the roles are completely reversed. In sequentially hermaphroditic species, the individual begins as one sex, but transforms into the opposite sex if the appropriate social events present themselves. Clearly, in both instances, the brain of each individual is bisexual in its organization and performance. In the only experiment that has been done to date, Leo Demski demonstrated that stimulating one brain area of the sea bass, a simultaneous hermaphrodite, would cause sperm release while stimulation in another brain area resulted in egg release.

But what about the 'higher' vertebrates, that is the reptiles, birds, and mammals that constitute the amniote vertebrates? Particularly revealing insights into the relationship between the sexual dimorphism of the brain (or lack of) and the display of sex-typical behaviors are afforded by my work on parthenogenetic whiptails of the genus *Cnemidophorus*. Some species of the genus are gonochoristic with male and female individuals that behave in a sexually dimorphic manner (i.e., males mount receptive females), while some species are parthenogenetic, all individuals being morphologically female and reproducing clonally. In the gonochoristic species, the brain is sexually differentiated in a typical vertebrate pattern. For example, in *C. inornatus*, males mount while females do not, and male mounting is dependent on androgens acting on the mPOA. Female *C. inornatus* do not mount, but exhibit receptivity dependent upon estrogen acting at the level of the VMN. Individuals of the parthenogenetic species engage, at different times, in behaviors that physically are identical to both the male- and female-typical behaviors of their sexual congeners (albeit with the exception of intromission and insemination, hence called 'pseudosexual behavior'). When pairs of animals are observed displaying these complementary behaviors, there is a tight relationship between the behavior displayed and the ovarian state of the animal (i.e., the individual mounting and displaying other male-like copulatory behavior (pseudocopulation)) is generally postovulatory and has elevated progesterone levels, while the receptive individual is preovulatory, having high estrogen levels. Any given individual will thus display both behaviors at different points in the ovarian cycle.

Hormonal and neuroanatomical correlates of the two kinds of behavior in these animals parallel those observed in males and females of more commonly studied vertebrates. Examination of the mPOA and the VMN of the parthenogenetic lizards indicates that these nuclei do not change in cell size or number during these different behavioral phases, nor do these parameters respond to exogenous hormone treatment. However, they do differ in metabolic activity in predictable ways (**Figure 4**): Rand and Crews showed that during the male-like pseudocopulatory behavior metabolic activity as measured by 2-deoxyglucose uptake (2DG) is high in the mPOA but below baseline in the VMH (indicating suppression of activity); during female-like pseudoreceptive behavior, the opposite occurs, with 2DG suppressed in the POA and enhanced in the VMN. Intracranial implantation of

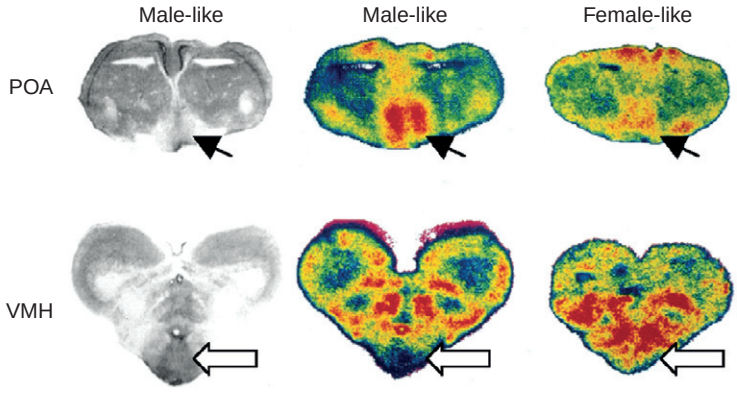

Figure 4 Metabolic activity during pseudosexual behavior in the unisexual lizard. Brains in two individual lizards engaged in a pseudocopulation. In the left column are light micrographs of brain sections at the level of the medial preoptic area (mPOA) (top row) and the ventromedial nucleus of the hypothalamus (VMH) (bottom row). Other columns are pseudocolor images where red denotes maximum accumulation of 2DG and green the lowest accumulation. Middle column is the brain of the individual exhibiting male-like pseudosexual behavior (same brain sections as on left), while the right column is the brain of the lizard exhibiting female-like pseudosexual behavior. Rand MS and Crews D (1994) The bisexual brain: Sex behavior differences and sex differences in parthenogenetic and sexual lizards. *Brain Research* 663: 163–167.

androgen (and progesterone) into the mPOA of both male *C. inornatus* and *C. uniparens* elicits mounting behavior, but fails to elicit either mounting or receptive behavior when placed in the VMN. On the other hand, while implantation of estrogen into the VMH elicits receptive behavior in female *C. inornatus* and in *C. uniparens*, it fails to do so in male *C. inornatus*, suggesting that the brains of these animals are not bisexual, but rather that either sex is capable of expressing male-typical behavior.

Both the mPOA and the VMN are dimorphic in size, with the mPOA being larger, and the VMN smaller, in sexually active male *C. inornatus* than in females or in the descendant parthenogenetic species. Castration of male *C. inornatus* causes the mPOA to decrease, and the VMN to increase to female size; androgen replacement restores the sex difference. The overall change in nuclear volume is paralleled in soma size of individual neurons in both areas, suggesting that the size of these neurons changes to reflect their functional activity. However, once again, this sex difference appears to be a correlate, rather than a necessary substrate of the expression of male-typical behavior, since *C. uniparens* exhibiting male-like pseudocopulatory behavior (either as intact postovulatory or ovariectomized, testosterone-treated animals) do not show an increase in regional or somal area of the mPOA. Dias and Crews found that differences in the mPOA thus observed between the parthenogens displaying male- and female-typical behaviors have also been subtle at the levels of gene expression and neurotransmitter levels. The parthenogenetic whiptails thus oblige us, while continuing to accept the existence of sex differences in brain morphology, to consider the possibility that such developmentally long-term differences in morphology are less important in determining the behavior exhibited than is the short-term activity of the brain, which is determined by external stimuli as well as by immediate physiological state. However, in these animals, as in others studied, this sexual phenotype-determining 'activity' can be profitably studied by focusing on the interaction between the mPOA and the VMN.

Conclusions

Beach only reluctantly accepted the idea of sexually dimorphic central structures, not because he was stubborn, but rather because he was hesitant to concede that such organizational actions might be the mechanism underlying activational gating of the bisexual brain.

> . . . the specificity of the mating patterns for the two sexes, although probably inherited, is not rigidly dictated by the innately organized substratum. Although there may be a strong preference for the normal copulatory response it is obvious that in a few individuals at least, there exists the innate organization essential to the mediation of the mating pattern of either sex. The presence or absence of such duplicative arrangement within all individuals is a matter for speculation. It is obvious, however, that the mating behavior to be displayed by a member of either sex may in part or (in the cases reported), entirely predetermined by the behavior of the partner. (Beach, 1938, p. 324)

Beach thus delineated four essential points: first, that both male and female individuals are capable of displaying the sexual behaviors of the opposite sex; second, that the brain must have the neural circuitry sufficient to support these opposite behaviors although third, each sex is predisposed to exhibit the behavior consistent with its sex; and fourth, that the stimulus animal is essential in eliciting the complementary behavior. If one accepts Beach's conclusions, one expects that male- and female-typical copulatory behaviors are mediated by brain structures that are present and (at least latently) fully functional in both sexes, that is, not sexually dimorphic. Experimentally one is then forced to examine how males and females can behave differently, and what, if not to mediate sex-typical copulatory behavior, are the functions of the observed sexual dimorphisms in brain structure. I propose that the neural mechanisms mediating both male and female copulatory behavior are under tonic inhibition from a range of sources, and that activation constitutes relief from some of these inhibitory inputs. Major sexual dimorphisms in brain structure are seen as mostly sex-specific sources of additional inhibition so that, for example, the large mPOA typical of males is responsible not for mediating male-typical copulatory behavior, but for allowing a more sophisticated pattern of inhibition. Sex differences, in other words, should not be seen as sex differences, but as male- and female-typical features that enable males and females to do better the things they do, rather than enabling them to do something that the other sex cannot. Either sex, the evidence shows, is intrinsically capable of doing either thing.

See also: Animal Behavior: Antiquity to the Sixteenth Century; Animal Behavior: The Seventeenth to the Twentieth Centuries; Comparative Animal Behavior – 1920–1973; Development, Evolution and Behavior; Endocrinology and Behavior: Methods; Ethology in Europe; Female Sexual Behavior and Hormones in Non-Mammalian Vertebrates; Future of Animal Behavior: Predicting Trends; Integration of Proximate and Ultimate Causes; Male Sexual Behavior and Hormones in Non-Mammalian Vertebrates; Mammalian Female Sexual Behavior and Hormones; Mate Choice in Males and Females; Mating Signals; Nervous System: Evolution in Relation to Behavior; Neurobiology, Endocrinology and Behavior; Neuroethology: Methods; Pair-Bonding, Mating Systems and Hormones; Psychology of Animals; Reproductive

Skew, Cooperative Breeding, and Eusociality in Vertebrates: Hormones; Sexual Behavior and Hormones in Male Mammals; Sexual Selection and Speciation.

Further Reading

Beach FA (1938) Sex reversals in the mating pattern of the rat. *Journal of Genetic Psychology* 53: 329–334.

Beach FA (1948) *Hormones and Behavior*. New York: Paul B. Hoeber.

Beach FA (1971) Hormonal factors controlling the differentiation, development, and display of copulatory behavior in the ramstergig and related species. In: Tobach E, Aronson LR, and Shaw E (eds.) *The Biopsychology of Development*, pp. 249–296. New York: Academic Press.

Crews D (2005) Evolution of neuroendocrine mechanisms that regulate sexual behavior. *Trends in Endocrinology and Metabolism* 16: 351–361.

Crews D and Moore MC (1986) Evolution of mechanisms controlling mating behavior. *Science* 231: 121–125.

DeVries GJ and Simerly RB (2002) Anatomy, development, and function of sexually dimorphic neural circuits in the mammalian brain. In: Pfaff DW, Arnold AP, Etgen AM, Fahrbach SE, and Rubin RT (eds.) *Hormones, Brain and Behavior* vol. 1, pp. 137–191. San Diego, CA: Academic Press.

Dias BG and Crews D (2008) Regulation of pseudosexual behavior in the parthenogenetic whiptail lizard, *Cnemidophorus uniparens*. *Endocrinology* 149: 4622–4631.

McEwen BS (1981) Neural gonadal steroid actions. *Science* 211: 1303–1311.

Newman SW (1999) The medial extended amygdala in male reproductive behavior: A node in the mammalian social behavior network. *Annals of the New York Academy of Sciences* 877: 242–257.

Pfaff DW, Schwartz-Giblin S, McCarthy MM, and Kow LM (1994) Cellular and molecular mechanisms of female reproductive behaviors. In: Knobil E and Neil J (eds.) *The Physiology of Reproduction,* 2nd edn., pp. 107–220. New York: Raven Press.

Phoenix CH, Goy RW, Gerall AA, and Young WC (1959) Organizing action of prenatally administered testosterone propionate on the tissues mediating mating behavior in the female guinea pig. *Endocrinology* 65: 369–381.

Rand MS and Crews D (1994) The bisexual brain: Sex behavior differences and sex differences in parthenogenetic and sexual lizards. *Brain Research* 665: 163–167.

Sengoopta C (2006) *The Most Secret Quintessence of Life: Sex, Glands, and Hormones, 1850–1950*. Chicago: University of Chicago Press.

Södersten P (1984) Sexual differentiation: Do males differ from females in behavioral sensitivity to gonadal hormones? *Progress in Brain Research* 61: 257–270.

Södersten P (1987) How different are male and female brains? *Trends in Neuroscience* 10: 197–198.

Tinbergen N (1951) *The Study of Instinct*. Oxford: Clarendon Press.

Wallen K and Baum MJ (2002) Masculinization and defeminization in altricial and precocial mammals: Comparative aspects of steroid hormone action. In: Pfaff DW, Arnold AP, Etgen AM, Fahrbach SE, and Rubin RT (eds.) *Hormones, Brain and Behavior,* vol. 4, pp. 385–423. San Diego, CA: Academic Press.

Whalen RE (1974) Sexual differentiation: Models, methods, and mechanisms. In: Friedman RC, Richart RM, and Van de Wiele RL (eds.) *Sex Differences in Behavior*, pp. 467–481. New York: Wiley.