shape and much larger (roughly two to three times wider and almost an order of magnitude larger in volume than those of X cells). The mean width and range of terminal zones for X cell axons are 140 μm and 100 to 170 μm, and for Y cell axons are 293 μm and 220 to 410 μm. On average, more of the terminal zone of Y cell axons is distributed in the bottom half of lamina A or A1, whereas no such division is seen for the X cell axons. Within the medial interlaminar nucleus, Y cell terminations spread predominantly dorsoventrally.

5) The terminal boutons of X cell and Y cell axons generally differ (Figs. 1, D and E, and 2, D and E). Both have small spherical (< 2 μm in diameter), medium-sized spherical (2 to 5 μm), and large (> 5 μm) crenulated boutons (*9*). The X cell terminals more homogeneously consist of the medium-sized boutons, whereas the Y cell terminals are more heterogeneous. Furthermore, X cell boutons tend to cluster together, whereas those of Y cells tend to occur singly or on short, fine stalks (Figs. 1E and 2E). Consequently, X cell axons tend to have fewer boutons that occur in higher density than do those of Y cells.

Several of these morphological features can be related to those described for geniculate neurons (*2, 10*). (i) The narrower and more vertically arranged terminal zones of X cell axons closely match the dendritic geometry of geniculate X cells, whereas the wider terminal zones of Y cell axons are more closely related to the radial arrangement of geniculate Y cell dendrites. (ii) The observation that Y cell terminal zones are larger and more widely distributed than X cell terminal zones is consistent with the suggestion that more divergence occurs in the retinogeniculate Y cell pathway than in the X cell pathway, so that more geniculate Y cells than X cells are innervated by each optic tract axon. (iii) The clustering of X cell terminal boutons might be related to the clustered dendritic appendages that are a much more common feature of geniculate X cells than of Y cells.

The clear morphological differences between X and Y cell axons indicate that the X and Y cell pathways differ markedly in their retinogeniculate projections. Previously, morphological differences have been described between geniculate X and Y cells (*2*) and their cortical terminations (*11*). These data, then, reinforce the significance of the organization of the central visual pathways into cell types that can be physiologically distinguished. The anatomical differences in the X and Y cell pathways show that these indeed represent distinctly different neural circuits.

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Hemispheric Asymmetries in the Behavioral and Hormonal Effects of Sexually Differentiating Mammalian Brain

Abstract. *Estrogen pellets were placed in either the right or left hypothalamus of newborn female rats so that only one side of this brain area was exposed to the postnatal masculinizing and defeminizing effects of the hormone. The effects of estrogen on gonadotropin secretion and reproductive behavior depended on both the region and the side of implantation. Exposure of the left hypothalamus to estrogen resulted in defeminized development. Exposure of the right hypothalamus to estrogen resulted in masculinized development. Thus the response of the developing hypothalamus to gonadal steroids may be asymmetric.*

During a restricted period of perinatal development, gonadal steroids act on the mammalian hypothalamus to masculinize or defeminize reproductive functions, including behavior (*1*). Masculinization increases male sexual behavior in adult males or females exposed to testosterone or its metabolite, estradiol (E_2); defeminization decreases female sexual behavior and eliminates positive-feedback effects on the secretion of luteinizing hormone (LH) in adults exposed to E_2. These two aspects of sexual differentiation are independent processes involving separate regions of the hypothalamus (*2*). We suggest here that the hypothalamus develops asymmetrically with respect to sexual differentiation. When we exposed only the left side of the hypothalamus of neonatal rats to gonadal steroid, development was defeminized. When we exposed only the right side of the hypothalamus to gonadal steroid, development was masculinized.

Between 24 and 48 hours after birth, 91 female rat pups received bilateral intrahypothalamic implants of steroid (*3*). Thirty control pups received implants of cholesterol. Experimental females received E_2 on one side (31 right, 30 left) and cholesterol on the other. We used E_2 as the hormonal stimulus for sexual differentiation because it is the active metabolite of testosterone for this process

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(*1*). Some implants were aimed at the developing preoptic area (POA). The rest were aimed at the mediobasal hypothalamus, particularly the ventromedial nucleus (VMN). The POA and VMN participate in the hormonal control of sexual behavior and LH secretion, both developmentally (*2*) and in adulthood (*4*). Our goal was to examine regional specificities in the control of these reproductive functions and to relate the behavioral consequences of sexual differentiation to specific changes in neuroanatomy.

We used an additional group of eight pups to assess the interhemispheric diffusion of E_2 (*5*). These pups received unilateral implants of [^{3}H]E_2. Within 24 hours, nearly 90 percent of the E_2 in the implant was released. The side of the hypothalamus opposite the implant contained about 5 percent as much E_2 as the implanted side (*5*). Thus the hormonal stimulation was confined almost completely to one side of the brain.

When the females were 65 to 80 days old, their ovaries were removed and examined for the presence of corpora lutea, a sign of ovulation. In rats exposed to steroids during the neonatal period, anovulatory sterility, or a lack of corpora lutea, implies that the pattern of LH secretion is defeminized (*2, 6*). The rats were then tested for female sexual behavior in response to systemic injections of estradiol benzoate (EB) and progesterone. How receptive they were to sexual advances of males was determined by recording how often they assumed a lordosis or mating posture when mounted repeatedly by a stimulus-male partner. These scores were converted to lordosis quotients or LQ's (*7*). Low LQ's indicate behavioral defeminization. The females were then tested for male sexual behavior while receiving EB and dihydrotestosterone propionate (DHTP) and again while receiving EB alone. We used EB to stimulate male sexual behavior because E_2 is a behaviorally active metabolite of testosterone (*4*). Dihydrotestosterone, another metabolite of testosterone, potentiates E_2's effects (*8*). Masculinization was assessed by noting how quickly and how often the female mounted a highly receptive female partner (*9*).

At the end of behavioral testing, the females were perfused under heavy anesthesia with saline and Formalin. Their brains were sectioned to determine precisely the locations of the neonatal implants. Implant sites fell into two distinct, nonoverlapping clusters: one near the POA, the other near the VMN (Fig. 1A). We examined thionin-stained sections of 32 females in detail to determine the volume of the sexually dimorphic nucleus (SDN) of the POA (*10*). The volume of the SDN is one of the most

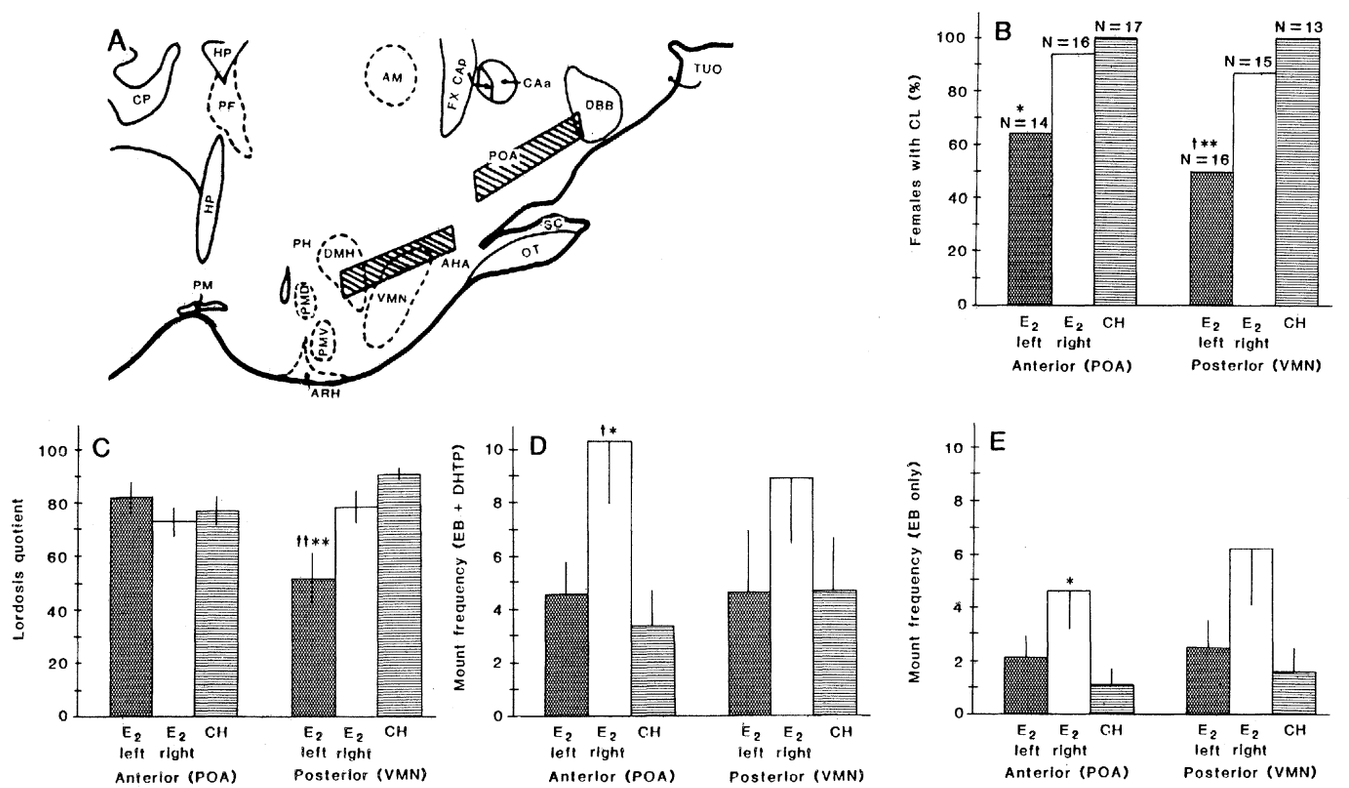

Fig. 1. Locations and effects of E_2 pellets implanted unilaterally into the brains of newborn female rats. (A) Schematic drawing, after (*21*), of a parasagittal section through the hypothalamus of an adult rat. Cross-hatched bars show locations of E_2 implants designated anterior (*POA*) and posterior (*VMN*) in (B) through (F). All implants were within 0.5 mm of the midline; none ruptured the third ventricle. For abbreviations, see (*22*). (B) Percentage of females with corpora lutea (*CL*), an index of ovulation. Ovulating females had well vascularized ovaries with many corpora lutea. Anovulatory females had at most one corpus luteum per ovary; most had none. *CH* denotes controls given bilateral implants of cholesterol. (C) LQ's during treatment with EB plus progesterone. (D) Mount frequency during treatment with EB plus DHTP. (E) Mount frequency and (F) mount latency during treatment with EB alone. Asterisks indicate that the group differed from controls implanted with cholesterol in the same region; daggers indicate that the group differed from the group that received E_2 on the other side of the brain in the same hypothalamic region; * or ‡, $P < .05$; ** or ‡‡, $P < .01$.

striking neuroanatomical sex differences known in mammals and has been proposed as a neural correlate of masculinization (*11*).

As we expected, the neuroendocrine effects of the neonatal implants depended on which hypothalamic region was exposed to hormone. What was unexpected was that the side of E_2 implantation had an equal or greater impact on sexual differentiation. For defeminizing LH secretion, left-side implants were most effective. Five of 14 females given E_2 in the left POA were sterile compared to 1 of 16 females given E_2 on the right (Fisher exact, $P < .06$). Two of 15 females that received E_2 in the right VMN were anovulatory as opposed to 8 of 16 that received E_2 on the left ($P < .05$). For each region, females exposed to E_2 on the left were less likely to ovulate than controls ($P < .02$ for POA; $P < .01$ for VMN); this was not true for females exposed to E_2 on the right (Fig. 1B).

Female sexual behavior was defeminized only by E_2 implants in the left VMN (Fig. 1C). There were no differences in LQ scores among females given E_2 or cholesterol in the POA (*12*), but LQ did vary among females with implants in the VMN [$F(2,41) = 8.47$, $P < .001$]. Females that received E_2 in the left VMN had the lowest LQ's (51.3 ± 9.5) (*13*). They were less receptive than either VMN controls (LQ = 90.7 ± 1.9, $P < .01$) or females exposed to E_2 on the right (LQ = 78.7 ± 5.8, $P < .01$). The last two groups did not differ from each other.

Development was masculinized only by right-side implants; statistically significant effects were limited to the POA. Females given E_2 or cholesterol in the VMN did not differ significantly in mounting behavior when exposed to EB plus DHTP or to EB alone (*14*). Females with POA implants differed in mount frequencies under both hormone regimens [$F(2,44) = 4.57$, $P < .02$ for EB plus DHTP; $F(2,43) = 3.59$, $P < .05$ for EB] (*15*). They also differed in mount latency while receiving EB [$F(2,43) = 4.20$, $P < .05$]. In each case, females given E_2 on the right differed from POA controls, but females given E_2 on the left did not. For example, females with E_2 implants in the right POA mounted 10.3 ± 2.3 times per test during treatment with EB plus DHTP. This was three times the mount frequency of controls (3.4 ± 1.3, $P < .05$) and twice that of females given E_2 in the left POA (4.6 ± 1.2, $P < .05$; see Fig. 1D). Mount latencies during treatment with EB plus DHTP followed the same pattern (17.88 ± 2.18 minutes for E_2 in the right POA; 21.53 ± 2.13 minutes for E_2 in the left POA; 23.67 ± 2.21 for POA controls), but the differences were not statistically significant. During EB treatment, females with E_2 implants in the right POA began mounting 18.42 ± 2.61 minutes after tests began and averaged 4.6 ± 1.4 mounts per test. They therefore mounted more quickly and more often than controls (27.04 ± 1.47 minutes, $P < .05$; 1.1 ± 0.6 mounts per test, $P < .05$; see Fig. 1, E and F). Females with E_2 implants in the left POA had intermediate scores (23.13 ± 2.37 minutes; 2.1 ± 0.8 mounts per test) and did not differ from either of the other two groups.

Table 1. Effects of E_2 on SDN volume relative to its effects on masculinization. All of these females ovulated. For comparison, four males had SDN volumes of 12.14 ± 1.43 on the left and 12.35 ± 0.71 on the right.

Neonatal implants	*N*	LQ	Mounts	Volume of SDN ($\times 10^{-3}$ mm^3)	
				E_2 side	Cholesterol side
Cholesterol (both sides)	10	73 ± 8	0	1.55 ± 0.15*	1.34 ± 0.18†
E_2 and cholesterol	7	83 ± 6	0	1.75 ± 0.22	1.76 ± 0.22
E_2 and cholesterol	15	80 ± 6	16 ± 2‡	1.59 ± 0.12	1.41 ± 0.12

*Result for left side. †Result for right side. ‡Mount frequency during treatment with EB plus DHTP.

Neither behavioral masculinization nor neonatal E_2 implantation altered the volume of the SDN (Table 1). The volume of the SDN on the side of the E_2 implant was identical to that on the other side of the brain and was comparable to SDN volume in controls, whether or not the females mounted.

Asymmetries in the effects of sexual differentiation suggest that the two sides of the developing hypothalamus differ in their sensitivity to steroids. There is ample reason to believe that the critical period for masculinization begins and ends earlier than the critical period for defeminization (*1*). If maturation is also asymmetric and favors accelerated neural development on the left (*16*), then the right and left sides of the hypothalamus would pass through these critical periods at different times. Shortly after birth, right hypothalamic neurons that mediate male sexual behavior would still be in the critical period for masculinization, but the corresponding neurons on the left would have already passed through it. Right-side neurons that control LH secretion or female sexual behavior would not yet be susceptible to defeminizing effects of steroids, but the corresponding neurons on the left would respond. If the hemispheres differed in maturation rate, sexually differentiated areas of the brain would probably develop asymmetrically because the critical period phenomenon might crystallize or even magnify the original asymmetry. It is not known whether the control of reproductive functions in normal adult rats is lateralized. Presumably, both sides of the hypothalamus of normal males are exposed to testicular androgens during the critical period for masculinization (*17*). Whether accelerated neural development on the left would still lead to left-side dominance of male sexual behavior is not clear. We suggest, however, that lateralization can occur when steroids are present for only part of a critical period. Prenatal masculinization (*18*) should involve the left hypothalamus; postnatal masculinization should involve predominantly the right. Similarly, prenatal defeminization (*19*) should be limited to the left hypothalamus since only neurons on the more mature left side would reach the critical period for defeminization before birth. Data indicating that the left VMN of normal adult females is less sensitive to E_2 than the right support this view. Davis *et al.* (*20*) found that bilateral implants of E_2 in the VMN induced sexual receptivity in 6 of 11 female rats, with the LQ of responders being 45.8 ± 14.3. Unilateral implants in the right VMN were equally effective (7 of 12 females responded with an LQ of 42.9 ± 7.5), but left side implants were not (only 3 of 12 females responded, with an LQ of 25.0 ± 7.6). These data are only suggestive because they rely on comparisons across experiments. Nonetheless, they reinforce our hypothesis that the consequences of sexual differentiation may not be the same on the two sides of the brain.

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10. Brains were sectioned and stained as in (*11*). Camera-lucida drawings of the part of the POA that stained as darkly as the suprachiasmatic nuclei were retraced on a digitizing tablet that computed their areas. The SDN volume is the sum of the areas (corrected for magnification) × 60 μm (section thickness).
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Mapping the Primate Visual System with [2-^{14}C]Deoxyglucose

Abstract. *The [2-^{14}C]deoxyglucose method was used to identify the cerebral areas related to vision in the rhesus monkey (*Macaca mulatta*). This was achieved by comparing glucose utilization in a visually stimulated with that in a visually deafferented hemisphere. The cortical areas related to vision included the entire expanse of striate, prestriate, and inferior temporal cortex as far forward as the temporal pole, the posterior part of the inferior parietal lobule, and the prearcuate and inferior prefrontal cortex. Subcortically, in addition to the dorsal lateral geniculate nucleus and superficial layers of the superior colliculus, the structures related to vision included large parts of the pulvinar, caudate, putamen, claustrum, and amygdala. These results, which are consonant with a model of visual function that postulates an occipito-temporo-prefrontal pathway for object vision and an occipito-parieto-prefrontal pathway for spatial vision, reveal the full extent of those pathways and identify their points of contact with limbic, striatal, and diencephalic structures.*

Many structures involved in processing visual information in the monkey have been identified through the combined use of neurobehavioral, electrophysiological, and anatomical techniques. Converging evidence has revealed a sequential pathway for processing information about visual objects that begins with retino-geniculate input to the striate cortex, proceeds through the prestriate cortex, and continues along a corticocortical route to the inferior temporal and then the inferior prefrontal cortex (*1, 2*). Classical mapping techniques have also identified another cortical visual pathway, which again begins with retino-geniculate input to the striate cortex but appears to be specialized for processing information about visual space; the corticocortical route in this case is through the prestriate, posterior parietal, and prearcuate cortex (*1, 3, 4*). Both pathways receive projections from the superior colliculus via the pulvinar and send projections to various limbic, striatal, and diencephalic structures. Despite a range of research efforts, however, the full extent of these two cortical pathways and their exact subcortical targets remain undefined. In the studies described here we used the [2-^{14}C]deoxyglucose (*5*) method to measure local cerebral glucose utilization (LCGU). This method enabled us to prepare a comprehensive map of the functioning visual system in a single preparation (*6*).

Four rhesus monkeys (*Macaca mulatta*) weighing 2.5 to 4.5 kg were subjected to unilateral (right) section of the optic tract about 6 weeks before we measured the LCGU. Metabolic activity produced by direct retinal stimulation was thereby limited to one hemisphere, and it was thus possible to compare LCGU in a "seeing" and a "blind" hemisphere in the same animal (*7*).

On the day of the experiment each monkey was lightly anesthetized with halothane and nitrous oxide, catheters for deoxyglucose administration and blood sampling were inserted into the femoral vessels, and the animal was seated in a primate chair. After at least 4 hours of recovery from anesthesia, the monkey was placed within an encircling screen, which displayed a brightly illuminated, high-contrast geometric pattern that rotated counterclockwise at 5 to 7 rev/min. The monkey focused its gaze on this visual stimulus throughout most of the experimental session, tracking it with head and eye movements. To begin the experiment we gave an intravenous injection of [2-^{14}C]deoxyglucose (100 μCi/kg). Forty-five minutes later the animal was killed, and the brain was removed, frozen, and prepared for quantitative autoradiography. Other details of the method have been described (*5, 8*).

The hemispheric differences that we found in optical density (see Fig. 1) reflected only the relative rates of glucose utilization. To quantify the rates we used a computerized image-processing system which, supplied with the plasma variables monitored during the experiment and the kinetic constants for the species (*5*), transform the autoradiographs into color-coded maps of the actual LCGU values (*9*). Determination of the LCGU values for every structure identified in Fig. 1 was based on readings of areas 2 to 3 mm square at all of the points indicated. In Table 1, the structures are categorized as "visual" or "nonvisual" on the basis of the presence or absence of a statistically significant degree of hemi-

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