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SEXUAL DIMORPHISM AND HOMOSEXUAL GENDER IDENTITY [1]

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To classify homosexuality as hereditary or constitutional versus acquired is outmoded. The differentiation should be between chronic, obligative, or essential versus transient, facultative, or optional. Cytogenetics and statistical genetics do not throw light on etiology, but new research on fetal hormonal differentiation of sexual morphology, and especially of sexually dimorphic hypothalamic differentiation offers promising leads. The sum total of the sex-differential effects of assignment and rearing on gender identity differentiation is known, through observations of hermaphrodites, to be profound. Postpubertally, homosexuality does not correlate with hormonal measures presently available or with assessments of neuroperceptual sex differences in erotic arousal. Sexual dimorphism of brain functioning in gender identity is the end product of sequential events at critical periods, with prenatal and postnatal effects interacting, and the end product being extremely durable.

Whatever the degree of an individual's homosexual commitment, the behavior concerned may be in some degree hereditary, constitutional, and biological in its determination, and in some degree environmental, learned, and sociological. It is not a question of either/or with respect to each of these categories, or a question of how much; the basic question is, Which type? The chronic and obligative, essential or idiopathic homosexual may be a product of the confluence of heredity and environment, constitution and learning, biology and sociology. Likewise with the transitory, facultative, optional, and induced homosexual.[3]

In the etiology of homosexuality, one may look for prenatal preordained factors either in the genetic code or in the metabolism of the intrauterine environment. It is remotely possible that one may find also a factor associated with birth—or birth injury. Postnatally, one must look for critical period experiences or exposures that may leave a permanent imprint. One needs an open mind regarding the nature of such experiences. For all that is known at present, these factors may range from a specific nutritional insufficiency to deprivation of sensory stimulation, as in congenital hearing loss, to pathology of behavioral interaction within the family, or to incapacity to relate freely with children of similar age.

Despite the fact that science and medicine do not yet have the answer to what determines psychosexual differentiation as homosexual, heterosexual, or bisexual, the body of knowledge is constantly expanding. The remainder of this paper reviews the present state of knowledge regarding genetic, fetal, hormonal, and central nervous system factors that may be related to behavioral homosexuality.

Cytogenetics: The Sex Chromosomes

The discovery that a difference between the sexes is visibly evident in individual cells of the body dates from as recently as 1949. It was then that Barr and Bertram at London, Ontario, discovered the sex-chromatin spot (Moore, 1966), since known as the Barr body, present in the nucleus of cells from female mammals but not in those from males. Subsequently, it was discovered that the cells of certain morphologic-appearing males, namely those with Klinefelter's syndrome, have a Barr body in them. The significance of this finding

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[3] Exactly the same statements may be made of the heterosexual.

remained obscure until after 1956, at which time it first became possible actually to visualize and count the chromosomes in a single cell (see reviews by Bartalos & Baramki, 1967; Ferguson-Smith, 1961; Hirschhorn & Cooper, 1961; Sohval, 1961). The chromosome count of a phenotypic male with Klinefelter's syndrome shows him to have a supernumerary X chromosome. Instead of the normal male count of 44 + XY chromosomes, or the normal female count of 44 + XX, the Klinefelter male has 44 + XXY (47, XXY). In some cases, there is more than one supernumerary X chromosome. To the extra X chromosome is attributed responsibility for the stigmata of the syndrome: namely, a tendency to eunuchoidism of body build; late onset of puberty; weak pubertal virilization with possible breast development, as in a female; small penis, small sterile testes, and subnormal libido or sexual drive. The central nervous system seems also to be involved: Witness an elevated frequency of the occurrence of mental deficiency and of various forms of psychopathology. The latter includes psychosexual pathologies, despite the low power of sexual drive, including homosexuality and its related conditions of transvestism and transexualism (Money & Pollitt, 1964).

It cannot be said that the extra X chromosome of Klinefelter's syndrome induces a degree of psychic femininity in all affected individuals, but only that the incidence of effeminacy is increased. Some individuals escape entirely. The best way to make sense of this state of affairs is to postulate a genetically determined condition of vulnerability to error in psychosexual differentiation. The developmental period of greatest risk would appear to be postnatal, during the preschool years, which is when psychosexual differentiation chiefly is accomplished (Money, Hampson, & Hampson, 1955, 1957). At this time all development is susceptible to influence from the social environment—for example, the acquisition of a native language. That the differentiation of a gender identity is also powerfully influenced by social experience can be clearly demonstrated in certain cases of hermaphroditism (see subsequent discussion). As in the case of a native language, however, the process of psychosexual differentiation is clearly one of interactionism between brain and social stimulus. The child with injury to language centers of the dominant hemisphere may be defective in language acquisition. One cannot be very specific about the human brain in matters of psychosexual identity, except to note that psychosexual changes may, in rare instances, be associated with temporal lobe malfunction or injury; and that these changes sometimes are reversed as a consequence of successful brain surgery (Blumer, 1969; Epstein, 1961, 1969). It may also be of significance that not only psychosexual pathology, but also electroencephalographic abnormality (Hambert & Frey, 1964) has an elevated frequency incidence in Klinefelter's syndrome.

Other syndromes in which the sex chromosomes are implicated are the female triple-X syndrome, Turner's syndrome, and the XYY syndrome in males (Bartalos & Baramki, 1967; Gardner, 1969; Wilkins, 1965). Triple-X females are morphologically and psychosexually unremarkable.

Patients with Turner's syndrome are morphologic females who are dwarfed, without gonads, and subject to a variety of other birth defects. The chromosomal error is most typically an absence of one of the pair of X chromosomes (45, X). Girls with this condition need hormonal replacement therapy at the age of puberty in order to mature sexually. Psychosexually they represent virtually the obverse of homosexuality which is conspicuous by its absence in this syndrome. The girls not only conform to the style of femininity idealized in our cultural definition of femininity, but they also are (long before they know the prognosis of their condition) maternal in their childhood play and adult aspirations. This very complete feminine gender identity and absence of homosexual traits may have its origins in a total absence of gonadal hormones during fetal development, so that there is no malelike hormonal effect on the sex-regulating centers of the developing brain (Ehrhardt, 1969; Money & Mittenthal, 1970).

The XYY syndrome (see review by Money, Gaskin, & Hull, 1970) has so recently been discovered that information regarding gender identity in men with this chromosomal aberration (47, XYY) is still tentative. The condition became newsworthy after it was found

to be frequent among tall and slender men detained in institutions for delinquents and criminals. Therefore, it has been conjectured that the extra Y chromosome may have some bearing on poorly regulated or impulsive behavior, sexual behavior included. The incidence of homosexual experience is high among institutionalized XYY males and occurs also when they are not institutionalized.

The examination of syndromes gives one-half of the story (the half that is traditionally neglected) of the relationship between genetics, specifically chromosomal genetics, and homosexuality. The other half of the story comes from testing a sample group of homosexuals themselves.

Nuclear sex-chromatin surveys of homosexuals and eonists have disclosed no discrepancies between them and control groups of men with normal masculine gender identity (Bleuler & Wiedemann, 1956; Pare, 1956; Raboch & Nedoma, 1958). In chromosome counting, there are no reports of discrepancies consistently related to homosexuality or to either the transvestite or transexual form of eonism (Pritchard, 1962), though there are known sporadic combinations of either homosexuality or eonism with the XXY chromosome complex of Klinefelter's syndrome (as discussed earlier in this review). Though the total number of cases studied has been modest, deviations from normal expectancy have always been in patients whose other clinical signs indicated the probability of a cytogenetic error in advance of the actual test. Homosexual men whose physical examination reveals no bodily abnormality have not been found to have a Barr body, indicative of an extra X chromosome; nor when the more time-consuming chromosome count has been performed have chromosomal errors been directly visualized. Since men with the XYY syndrome were overlooked until recent years, one must allow the possibility that a chromosome-counting (karyotyping) survey of a large sample of homosexuals might disclose some hitherto unsuspected abnormality in some individuals. Meantime, on the basis of techniques so far employed, there is no way of implicating an error of the chromosomes themselves in the etiology of ordinary homosexuality. Whether genes rather than entire chromosomes may be implicated is an altogether different matter. There is no technique yet available for visualizing, counting, or otherwise directly implicating certain genes in the etiology of anything. Such implication must always be by inference, is only rarely possible, and has not yet been achieved in any part of behavior genetics, to say nothing of sexual behavior genetics.

Statistical Genetics

The attempt to implicate hereditary mechanisms in homosexuality at the genic, if not the chromosomal, level long antedates the new era of cytogenetics. The older, statistical methods are those of the sex ratio, ordinal position, and twin comparisons.

In sex-ratio studies, the male:female ratio in the sibships of male homosexuals was compared with the expected ratio of 106:100 (Darke, 1948; Jensch, 1941a, 1941b; Kallman, 1952; Lang, 1940; Slater, 1958). Each study turned up a different ratio, some with and some without statistical significance, ranging from 106:100 in Darke's small sample to 125:100 in Kallman's twin study. The most often quoted study is that of Lang, based on 1,015 cases. His ratio was 121:100, which could be subdivided to 128:100 for those cases over the age of 25, and to 113:100 for the younger age group. It is possible that the results of all these studies have no more significance than if the figures had been drawn from a random number table, a procedure which sometimes yields statistically significant differences. On the other hand, the results may reflect a tendency for homosexual men to have more brothers in the family than expected. In this case, the findings may signify not a genetic predisposition to homosexuality, but a tendency for an effeminate gender identity to develop more easily in boys whose families have a shortage of sisters and daughters. From this latter point of view, it is provocative that Slater found a brother:sister ratio in the families of male exhibitionists of 109:144—an excess of female sibs to form an audience for their show-off brother!

Extending his observations from family constellation to ordinal position of siblings, Martensen-Larsen (1957) somewhat casually re-

ported findings on a sample of 63 homosexuals, which not only confirmed a preponderance of brothers over sisters, but also showed the homosexuals to have a predominance of ordinal position in the lowest third of the sibship. There was also a preponderance of brothers in the families of the 42 fathers and 21 grandfathers studied, whereas the 25 grandmothers and 45 mothers had a preponderance of sisters. In the 44 homosexual women and the 39 of their mothers and 16 maternal grandmothers studied, sisters predominated in the sibships with no data reported for the fathers. The homosexual women came from the upper and lower thirds of the sibship more often than the middle.

In the matter of ordinal position, Slater's data (1958, 1962) showed that compared to exhibitionists, homosexuals tended to be born late in the sibship and of older mothers.

Another type of study purporting to implicate a genetic mechanism for homosexuality is that of Kallman (1952) on homosexual twins. Kallman reported a high degree of concordance for homosexuality in each of 37 pairs of identical monozygotic twins. The figure for fraternal dizygotic twins was quite different. There were 26 such pairs of twin brothers studied, with the index case known to be overtly homosexual; 58% of the dizygotic co-twins revealed no evidence of homosexual experience after the age of adolescence, whereas the remaining 42% ranged over the full scale of 1 to 6 on the Kinsey homosexuality ratings. Only 11.5% of the co-twins (3 of the 26) were homosexual enough to get a rating as high as 5 or 6 (which requires a high or exclusive degree of homosexuality for at least 3 years between the ages of 16 and 35). Kinsey's corresponding percentage for ratings of 5 or 6 in the general male population was 10%.

Kallman's identical twins were not reared apart. Their concordance for homosexuality may, therefore, represent a tendency in monozygotic twins to be replicas of one another while growing up and responding to their jointly shared life experiences. In this case, their homosexuality would not be a primary genetic unfolding, but secondary to the unison in which the pair encountered life's transactions.

A further word of caution, applicable not only to homosexual but to all twin studies, is very much in order; namely, identical twins are not necessarily cytogenetically identical. That is to say, twins who qualify as identical by all the usual criteria (including blood type, dermatoglyphics, and skin transplant) may not have the same number of chromosomes. This surprising finding has emerged from the study of mongolism (trisomy-21) and Turner's syndrome, when identical and apparently monozygous twins proved to be cytogenetically discordant (Bruins, van Bolhuis, Bijlsma, & Nijenhuis, 1963; Lejeune, Lafourcade, Schärer, de Wolff, Salmon, Haines, & Turpin, 1962; Ross, Tjio, & Lipsett, 1969; Russell, Moschos, Butler, & Abraham, 1966; Turpin, Lejeune, Lafourcade, Chigot, & Salmon, 1961). These findings may require a rather extensive revision of current concepts of perfect genetic identity in monozygous twins, since they may be identical for all but one chromosome or one pool of genes. The missing (or added) part of the genetic code may be the very part that makes all the difference with respect to the trait, like homosexuality, which is the subject of behavioral interest in a twin study.

Fetal Differentiation of Sexual Morphology

In clinical medicine and experimental biology, the principles governing sexual differentiation of the embryo and fetus (see review by Federman, 1967) have been elucidated during the past quarter century. Errors of differentiation, notably in both clinical and experimentally induced hermaphroditism, are important to the theory of homosexuality. In some instances these errors result in what is, in effect, homosexuality by experiment or by experiment of nature.

In the development of the embryo, nature's first choice or primal impulse is to differentiate a female. The anlagen of the sex organs when they first appear are identical for both sexes. The principle of differentiation is always that to obtain a male, something must be added. Subtract that something, and the result will be a female. Castrate the fetus in utero prior to the critical period when sexual morphology will be differentiated, and all the offspring, whether genetic males or females,

will be born with female external genitalia. The first demonstration of this principle was by means of surgical castration of fetal rabbits (Jost, 1961). Jost's original work was in the early 1950s. The same effect can now be achieved nonsurgically by means of functional hormonal castration (Neumann & Elger, 1966). The hormone used is cyproterone acetate, an antiandrogen or androgen antagonist. Injected into the pregnant mother, it reaches the fetus and renders all of its cells androgen insensitive. Thenceforth, those cells of a genetic male dependent on male hormone, in order to differentiate the masculine sexual morphology, behave as if they were in a genetic female. The result is that a genetic male animal is born with the perfect facsimile of external female morphology, including a vagina large enough in adulthood for copulation. The gonads are testes. The uterus and fallopian tubes are absent, as in a normal male. The embryonic differentiation of these organs is not under the control of androgen. They therefore do not develop under the influence of antiandrogen. At puberty, the testes will function in the normal male fashion. To obtain a feminizing puberty, they will need to be removed or their influence counteracted by further administration of antiandrogen. Then female hormones will need to be administered. Properly regulated in the female fashion, the animals (rats) used in Neumann's experiment exhibited estrus, behaved in mating in a manner indistinguishable from normal females, and were accepted as such by normal males in the colony.

These antiandrogenized rats are an experimental counterpart of a human clinical condition, the syndrome of androgen insensitivity or testicular feminization (Federman, 1967, Chapter 8). Children born with this syndrome are a perfect female facsimile, externally. Therefore, they are invariably assigned as females. Their gonads are two testes, always lacking spermatogenesis, which may be completely undescended or may push down toward the labia. The uterus is vestigial, making menstruation impossible. Either menstrual failure or the lumps of the testes in the groins are the usual reason for referral and diagnosis. The vagina in some cases is too short for comfortable intercourse and so will need surgical lengthening in middle teenage or sometime later. The onset of puberty is at the usual time and is invariably feminizing, since the cells of the body are unable to respond to the normal masculine output of androgen from the testes. They respond instead to the normal amount of estrogen produced in a male by the testes, which is enough to produce a degree of feminization so complete as to be quite compatible with a career as a fashion model. It is a characteristic of the syndrome that the girls are tall. Some of them are unable to grow sexual or axillary hair. The nature of the resistance to androgen at the cellular level is unknown, but is presumed to be enzymatic—either a missing enzyme or a superfluous and toxic one. The condition is hereditary, probably a sex-linked dominant, being found in the aunts, nieces, cousins, and siblings of affected individuals. Psychosexual differentiation is invariably feminine, usually uncompromisingly feminine, with a strong degree of maternalism which makes for very good adoptive motherhood (Money, Ehrhardt, & Masica, 1968).

Human beings with the androgen insensitivity syndrome and antiandrogenized rats make it very clear, for those who ever doubted it after the opening sections of this paper, that genetic sex alone does not exercise a direct and peremptory power over psychosexual differentiation. The genetic sex difference can express itself in psychosexual differentiation only if the intervening steps of hormonal and morphological differentiation follow a phylogenetically prescribed course. Quite possibly, neural differentiation within the brain is yet another necessary intervening step (see subsequent discussion).

There is a semantic and conceptual lesson to be learned from women with the androgen insensitivity syndrome. Genetically and gonadally they are male. Therefore, when they marry a man, both partners are in a relationship of genetic and gonadal homosexuality. Morphologically, hormonally, and psychosexually, they are heterosexual in their relationship. Legally and in the popular conscience also they are heterosexual, for the evidence of common sense is that two people are homosexual not when they have the same chromosomal sex or the same hidden sexual struc-

tures internally, but when they have the same copulatory organs externally, and a psychosexual disposition to use them in an erotic relationship together.

Genetic males who qualify as females have their counterpart in genetic females who qualify as males. This latter condition is found clinically in human beings, as is its counterpart, and it can also be induced experimentally in animals by injection of the pregnant mother with male sex hormone. In human beings the condition of complete masculinization occurs sometimes in the adrenogenital syndrome of female hermaphroditism (Federman, 1967, Chapter 9; Money, 1968b). The source of excess fetal androgens is from the baby's own adrenal cortex which, on the basis of a genetically recessive trait, functions abnormally. From its undifferentiated state, the genital tubercle enlarges to become a penis instead of a clitoris. The skin that would have constituted the hood of the clitoris and the labia minora, follows the masculine alternative of wrapping around the penis and fusing to form the urethal tube. The labioscrotal folds do not stay divided to form labia majora, but fuse in the midline to form an empty scrotum. The gonads are ovaries and remain internal. The uterus opens into a shortened vagina which opens into the urethra near the neck of the bladder, instead of having an external opening in the normal position adjacent to the urinary orifice.

This same masculinized sexual anatomy can be found also in genetic females whose mothers were given one of the new synthetic progestins to prevent miscarriage. Rather rarely when they are administered, these hormones which have an androgenic biochemical structure will have an androgenic physiologic effect on the female fetus.

As may be expected, some of these masculinized genetic females with a penis pass as cryptorchid males and are declared and reared as boys (Money, 1955). Some of them may escape further medical attention until they are too old for a sex reannouncement. They remain living as boys and are given the appropriate hormonal therapy to prevent early virilization and the various other signs and symptoms associated with the adrenogenital syndrome, if they have that diagnosis. Additional surgical treatment is also given to prevent the pubertal appearance of menstrual bleeding, spontaneously, in the progestin-induced syndrome and, as a sequel to cortisone therapy in the adrenogenital syndrome, if such has been instituted. Such boys are also given androgenic hormonal therapy to induce masculine pubertal development. So regulated surgically and hormonally, these patients differentiate a masculine psychosexual identity.

The sexual behavior in adulthood of experimentally masculinized genetic female animals will depend partly on the regime of hormonal regulation on which they are maintained. The most data have come from guinea pigs and rats (see review by Money, 1965). The general trend was for their behavior to resemble more closely that of normal male than normal female controls, but their scores were not identical with those of normal males. The rhesus monkey has also been used in these masculinizing experiments, but the animals are still too scarce or too young for all the critical questions to have been answered regarding adult sexual behavior. In childhood, however, the masculinized girl monkeys behaved like tomboys, engaging in more rough and tumble play than the normal controls, plus more initiation of play, and more threats, chasing play and sexual play with attempted mounting.

One should not look too closely for an etiologic comparison between a genetic female with a penis and a typical female homosexual, since the latter differentiates a psychosexual identity as the possessor not of a penis, but a normal female vulva. A more fair comparison might be between a female homosexual and a genetic female fetally exposed to androgen without getting a penis, or one whose masculinized, or partially masculinized external genitals were surgically corrected at birth. There are cases, clinical and experimental, that more or less fulfill this requirement. The same holds true in the analogous comparison of the androgen insensitivity syndrome with typical male homosexuality. But more about this in the next section.

Sex Differentiation and Brain

The most up-to-the-minute new knowledge in sex research concerns a sex difference in

the influence of fetal hormones on that part of the brain, the hypothalamus, which will subsequently regulate the cycles of sexual functioning in the female or their absence in the male. (See reviews by Doerner, 1967; Harris, 1964; Money, 1965; Neumann, Steinbeck, & Hahn, in press.) The work of several different investigators in this country and Europe has converged to produce this new knowledge, most of it obtained from species that manifest the phenomenon of estrus. The basic principle to emerge is the same as that which applies to the differentiation of genital morphology (see earlier discussion in this review), namely, that nature's basic premise is to make a female. To make a male, something must be added. Once again, that something is male sex hormone. When radioactive-labeled male hormone is administered to the fetus either through the mother or directly, in the case of the rat which is born immature, its uptake can be traced to various organs, including cells in the hypothalamus. When the hormone is administered within the time limit of the effective critical period in development, then if the fetus is a genetic female, the hypothalamic nuclei will be masculinized. In consequence, these cells will never, in the future, be able to release their neurohumoral messages to the pituitary in the cyclic fashion characteristic of the female. Like the male, the animal will therefore always be uncyclical, though unlike the male will most likely be in a state of constant estrus but without ovulation. In addition, because other nearby hypothalamic nuclei that regulate sexual behavior in phase with the sexual cycle will have been affected by the male sex hormone, the animal's sexual behavior will not conform to that of the normal female. Typically, the animal will be disoriented and disorganized in its sexual response, or will repulse the male.

The converse of this story is found in the male that is castrated before the critical period when androgen should leave its imprint on the hypothalamus. Animals so treated show, when they are tested by means of an ovarian graft implanted behind the lens of the eye, that the pituitary functions cyclically, in the female fashion. The ovarian graft can be seen to pass through ovarian cycles as in a normal female. In sexual behavior in adulthood, these early castrated animals are significantly more feminine in their responses to mounting by intact males than are their experimental controls, though they are likely to be sexually apathetic unless first primed with estrogen and progesterone or, paradoxically, with testosterone (see reviews by Whalen, 1968; Gorski, in press).

In the foregoing experiments, interference with the normal progress of growth and development occurred after the fetuses had passed through the phase of external sex-organ differentiation. In gross external anatomy, they appeared normal. Only their pituitary-gonadal cycling was abnormal and also their behavior. If the experimental interference with normal fetal development is timed earlier, then the morphologic differentiation of the external organs is rendered hermaphroditic and abnormal, as in the extreme cases of antiandrogenization and masculinization already described. Rodent females exposed to androgen from this earlier stage until after birth, and not subsequently castrated, reject the male in adulthood, and display more masculine mounting behavior toward receptive females than do their normal controls. Antiandrogenized males, their testes intact, show bisexual behavior in adulthood, dependent on the sex of the partner (Neumann & Elger, 1965).

Phylogenetically, it is a big leap from estrous rodents to menstrual primates, especially with respect to a system so species-variable as the reproductive system. One may not draw parallels and inferences indiscriminately. For example, in the rhesus monkey and in man, fetal exposure of the female to androgen does not predictably interfere with the pituitary's regulation of normal menstrual cycling. Provided the evidence is interpreted with caution, however, there are some inferences to be made regarding the masculinization of behavior in human females fetally exposed to androgen, as in congenital virilizing adrenal hyperplasia (the adrenogenital syndrome of female hermaphroditism) and girls with progestin-induced masculinized genitalia.

For present purposes, the most instructive human cases are those in which the condition was recognized at birth and the necessary hormonal and surgical corrections immediately instituted (Ehrhardt, Epstein, & Money,

1968; Ehrhardt & Money, 1967). Under these circumstances, the possible effect of a prenatal masculinizing effect on the central nervous system is not contaminated by either the postnatal effect of masculine-looking abnormal genitalia or by the persistence of incongruous hormonal functioning.

In both groups of girls, there was a high incidence of tomboyism as defined by themselves, their mothers, and their playmates—far higher than found in the control group. This tomboyism manifested itself primarily in athletic energy expenditure that included, but was not restricted to, boys' sports and activities. Boys' toys were preferred over dolls. The rehearsal of motherhood in childhood play was missing or low in the priority of interests. In anticipating the future, romance and marriage, though not ruled out, were subordinated to career ambitions. Girlhood clothing and hair style showed a marked preference for utilitarianism and functionalism rather than being chic, pretty, or fashionably feminine. There was no evidence of a directly or incipiently lesbian type of erotic interest—simply a low priority rating for interest in boys as compared with the control group. An unexpected side-finding was an excessively high incidence of high IQs, which probably is not attributable to an artifact of sampling.

In the clinical study of human beings, there are no male counterparts to the above groups, namely, genetic males who were antiandrogenized or unandrogenized prenatally and then hormonally and surgically remasculinized for assignment at birth. The nearest approximation is found in cases of agenesis of the penis which may be total, or the organ may be a microphallus with or without hypospadias. At birth, some children of this type are assigned and surgically corrected as females. Others are assigned as males. Some few of them may ultimately request to be reassigned as females, but the majority will accept themselves in the sex of original assignment (Money, unpublished data).

In each foregoing type of case, it does seem likely that there may be some residual influence of the hormonal effect on the developing fetal brain—probably the hypothalamus but possibly also the temporal lobe. The residual effect does not, however, automatically dictate the postnatal differentiation of a gender identity as a homosexual. Postnatal events exercise their own power on gender identity differentiation. Nonetheless, one may be justified in making the inference that had the fetally masculinized girls been assigned and corrected as boys, they would not have had much difficulty in differentiating psychosexually as boys. There are indeed cases, as is evident from the next section of this review, where such has been observed. The same thing holds in converse for the incompletely masculinized boys.

As applied to homosexuality, these clinical findings suggest that an apparently normal baby destined to be a homosexual may have some hidden predisposition, perhaps lurking in the neurohumoral system of the brain, that makes him or her more vulnerable to differentiate a psychosexual identity as a homosexual —not in any preordained, automatic, or mechanistic sense, but only if the social environment happens to provide the right confluence of circumstances.

Sex Assignment and Gender Identity

None of the foregoing evidence regarding genetic or fetal-hormonal influences on sex differences in morphology or neural organization is incompatible with the evidence of an extraordinary contribution from the postnatal environment of social exposure to the differentiation of a person's gender identity. There is no more convincing evidence of the power of social interaction on gender-identity differentiation than in the case of congenital hermaphrodites who are of the same diagnosis and similar degree of hermaphroditism, but are differently assigned and with a different postnatal medical and life history (Money, 1963b, 1970a; Money & Ehrhardt, 1968). It is possible, for example, to have four genetic females, each ultimately diagnosed as having the adrenogenital syndrome of female hermaphroditism: one is assigned as and reared as a girl, one as a boy, one provisionally as a boy, and one provisionally as a girl. The subsequent medical and social histories differ, relative to the sex of assignment or provisional assignment and rearing. Psychosexual differentiation takes place, respectively, in the four cases as feminine, masculine, ambivalently

wanting to be changed to a girl, and ambivalently wanting to be changed to a boy (Money, 1968c, 1970b, and unpublished case histories).

The lesson of these four cases does not need to be belabored. They indicate that the outcome in psychosexual differentiation was entirely independent of genetic sex which was the same in all four, and also independent of hormonal sex—the boy who changed to live as a girl was already prematurely and strongly virilized (as is usual in the syndrome) before cortisone therapy was instituted to induce breast development, menstruation, and ovulation. The first two cases illustrate also the congruence between sex of assignment and the differentiation of gender identity when the parents and other significant people in the social environment are not ambivalent about the sex to which their child belongs; whereas the second two cases illustrate what may happen when the opposite is true.

Requests for sex reassignment among hermaphrodites are the exception rather than the rule. The request may be from male to female or vice versa, irrespective of genetic or gonadal sex, but requests from female to male are more common. There is a good explanation for this preponderance of female to male requests. Such requests are typically made by individuals with a large and visible phallus who were not surgically feminized early in life, and so grew up with visible evidence of ambiguity. At puberty, further masculinization is evidenced, regardless of genetic sex, because female hermaphrodites with a large, phallic-looking clitoris typically have the virilizing adrenogenital syndrome (untreated); and in males, the larger the unfinished hermaphroditic penis, the greater the chance that they will virilize at puberty instead of feminize or remain eunuchoid.

There is, at the present stage of medical history, a rather remote possibility that some few male hermaphrodites without a large phallus and not pubertally masculinized, may, if reared as a girl, feel like a lesbian who should have been a boy. Many if not all of such cases, however, have a history of undercurrents of ambiguity in the minds of the parents as to their true sex. The preponderance of evidence from hermaphroditism relative to the theory of homosexuality points to the importance of subtleties in the sex of rearing in shaping the differentiation of gender identity as male, female, or ambivalent.

Postpubertal Hormonal Differences

It has long been taught that childhood is a period of hormonal dormancy, but that belief is currently open to doubt and reexamination, particularly in the wake of a new technique (Raiti & Davis, 1969; Raiti, Johanson, Light, Migeon, & Blizzard, 1969; Raiti, Light, & Blizzard, 1969) for measuring the pituitary gonadotrophic hormones that stimulate the ovaries and testes. Changes in the level of these hormones, follicle stimulating hormone (FSH) and luteinizing hormone (LH), are detectable in advance of clinically evident puberty. Nonetheless, the chief fact of puberty is the release of sex hormones by the gonads. Contrary to popular belief, males and females make some of all three sex hormones, androgen, estrogen, and progestin. The difference between the sexes is not absolute and all-or-none, but a matter of relativity or degree. Further, in biochemical structure, the sex hormones are all "first cousins." It is possible for the body to convert one into the other. Alternatively, it may retain a surplus of a hormone or its precursor which might be destroyed. For example, in males estrogen produced by the testes should be destroyed in the liver. Failure of this mechanism may be responsible for some cases of breast development (gynecomastia) in adolescent boys.

In the 1940s, the era when the sex hormones had first been synthesized, there was a flurry of excitement concerning the relationship of the level of hormones in the blood or urine to homosexuality (male) and heterosexuality; and conversely, concerning the use of hormones in the treatment of homosexuality (see review by Money, 1961). The excitement was all doomed to disappointment. Methods of measurement were crude then. In the present day of more refined measurement techniques, no one is very interested in large-scale studies of homosexual blood and urine, since detailed individual studies, for example, of spermatic vein blood taken from a male transexual at operation, indicate that there is no reason to suspect a statistical difference

from hormone levels in control blood and urine (Migeon, Rivarola, & Forest, 1969).

Clinically, it is true that some homosexuals have a history of chromosomal error, sexual birth deformity, undescended testes, small penis, delayed puberty, gonadal insufficiency, poorly developed or contradictory sexual dimorphism of body build, gynecomastia (in boys), and hirsutism (in girls). But the frequency of any one of these disorders among homosexuals is so sporadic as to not create special hormonal research vigilance at the present time. Conversely, the incidence of homosexuality in the clinical population of each one of the listed disorders is so sporadic that one cannot seriously entertain the hypothesis of a primary hormonal cause–effect link between the physical symptoms, on the one hand, and homosexuality on the other. If there is any link, it is more likely to be secondary. There is far and away more homosexuality among organists, hairdressers, actors, interior decorators, or antique dealers than among patients with any given endocrine diagnosis! One may fairly safely interpret today's clinical evidence to mean that the sex-hormone levels of adulthood have very little to do with the etiology of homosexuality.[4]

The story with regard to therapy is a little different, insofar as male hormone administered to either a male or female homosexual, enhances the level of libido and leads to a probable increase in homosexual activity. Androgen is a libido-enhancing hormone for both sexes (Everitt & Herbert, 1969; Herbert, 1970; Money, 1961). By contrast, estrogen is a functional castrating agent in the male. It thus may lower the intensity of libido and lead to a decrease of sexual initiative and activity. In effect, it is an erotic tranquilizer in the male. Its therapeutic use is limited by its promotion of breast growth,[5] which is a positive advantage in the case of the male transexual who is not disturbed by having his sexual initiative reduced by estrogen therapy. In the female, estrogen appears to be more important to the cycle of nidation and gestation than to libido and eroticism per se, except that the unestrogenized vagina is unlubricated and painful in intercourse.

Neuroperceptual Sex Differences

Knowledge of differences between the sexes at the neuropsychologic—neurosensory, neuroperceptual, or neurocognitional—level is fragmentary, being mostly derived from anecdotal observations with only a few systematic observations or experiments. The earliest systematic data are those from the Kinsey interviews (Kinsey, Pomeroy, Martin, & Gebhard, 1953). They point to a higher frequency of erotic (genitopelvic) arousal in the male than the female, in response to visual and narrative erotic stimuli. The relative frequencies varied in accordance with the type of stimulus and, no doubt, in accordance with individual preference and personal history.

A more recent study and an experimental one is that of Schmidt and Sigusch (1970). Using a specially prepared movie demonstrating sexual relations, they found that college women responded sexually to this form of erotic stimulation in a fashion similar to men.

When men and women are sexually aroused by visual or narrative material, the nature of the carry-through may be different. Men are, in the popular view, more readily aroused than women to a casual or promiscuous liaison, if such is available. Women are more dependent on longer-term romantic and sentimental ties with a partner, especially in connection with stimulation through the tactile senses.[6]

[4] A special case might be made for women with the congenital adrenogenital syndrome who, in the era before cortisone therapy, reached adulthood heavily masculinized by their own adrenal androgens. Among a group of 23 such women, 10 could report having dreamed bisexually, and 4 of the 10 had had bisexual experience (Ehrhardt, Evers, & Money, 1968). These women were all markedly masculinized before birth, however, as well as subsequently, so the possible relationship between male hormone and masculine (homosexual) behavior cannot be attributed to a pubertal and adult hormonal effect per se.

[5] Two other steroids have the same antiandrogenic and erotic-tranquilizing effect without inducing breast growth. They are cyproterone acetate and medroxyprogesterone acetate. Both have been used, in recent therapeutic investigations, for the successful regulation of criminal sexual behavior in sex-offender disorders (Laschet, 1969; Money, 1968a, 1970b).

[6] There is some conversely corroborative evidence from the hormonally virilized women referred to in Footnote 4. These women reported a ready arousal from visual and narrative, as well as tactile stimulation (Ehrhardt, Evers, & Money, 1968).

There is another potential sex difference in response to the visual image. Men respond to a visual image, for example a pinup girl, as to a substitute object of sexual desire. Women may respond to the same stimulus, but not, if they do, as to an object of desire: the woman projects herself into the image and becomes the seductive one. Men, in general, do not project themselves into a picture of a man in this way. Only the homosexual man responds to pictures of other males, and then as sexual objects.

Apart from the preferred sex of the image, homosexuals do not, so far as is known, differ from other members of their morphologic sex in response to visual or narrative erotic stimulation. There is some evidence (Money & Brennan, 1969; Money & Primrose, 1969) to indicate that transexuals, despite the intensity of their identification with the sex of their desired reassignment, also do not differ from members of their morphologic sex of birth in visual and narrative erotic responsivity.

Another line of evidence supporting a sex difference in erotic arousal pertains to dreams. The Kinsey studies showed that only 37% of women reported orgasm dreams, whereas 83% of men did. Further, in women, dreams culminating in orgasm occurred with greatest frequency between the ages of 30 and 50, but in men they occurred during the immediate postpubertal teens and the 20s. On the basis of anecdotal impression, a similar difference holds up in a comparison of homosexual women and men. Obviously a systematic study is needed.

Perceptual distractibility is another variable that shows a sex difference. More than the female, the male in his erotic pursuits is fairly promiscuously distractible from one love object to another, especially over a period of time, except perhaps when he is in the vortex of having just fallen desperately in love. The female is more steadfastly tied to a single romantic object or concept. In the act of copulation, by contrast, it is the male who has a singleness of purpose, perhaps oblivious even to noxious stimuli, and who is likely to be unable to continue if successfully distracted by a competing stimulus. This sex difference appears to hold widely in the animal kingdom (Beach, 1947, p. 264). Horsley Gantt (1949), director emeritus of the well known Pavlovian Laboratories at Johns Hopkins, wrote:

> A marked difference between the male and female cat is that the female's interest in food is not inhibited by the sexual excitation of copulation, for she, as well as a bitch, will accept food not only after coitus but even during the act! . . . The female is, however, much more strongly oriented about the offspring than about the sexual act; she undergoes a great inhibition of conditional reflexes and of some unconditional reflexes postpartum, a fact which has been demonstrated several times in my laboratory with dogs [p. 37].

The male's ready arousal by perceiving a new erotic stimulus perhaps relates to his greater expenditure of energy in the service of sexual searching, pursuit, and consummation. Expenditure extends also to adventurous exploratory roaming, to assertiveness and aggression, and to the defense of territorial rights.

Once again, there is no systematic evidence concerning homosexuals and their ratings on these variables of distractibility and energy expenditure. Students of homosexuality would probably agree, however, that male and female homosexuals are not different from ordinary males and females regarding distractibility. In energy expenditure, there is quite possibly a difference with regard to aggression and territorial rights, insofar as a certain rather languid, strongly female-identifying homosexual males are persecuted by their age-mates in childhood because they do not fight for a position in the boyhood pecking order. The relationship of this nonbelligerency to the nervous system is still conjectural.

The sense of smell (see reviews by Money, 1960, 1963a) is characterized by a rather remarkable sex difference which is hormone controlled. Smell acuity, at least for some compounds, appears to be regulated by estrogen level. Women generally have greater acuity than men. There may be some exceptions, but acuity is at its peak for most women when they are not in their progestin (menstruating) phase of the menstrual cycle. Their acuity is lost after ovariectomy, but regained if they are given estrogen. In male rats, estradiol (an estrogen) will suppress acuity completely. Castration will lessen, but not abolish, an adult male rat's capacity to distinguish the estrous female from the dies-

trous; male juveniles have a little of this discriminatory ability. Surgical removal of the pituitary gland suppresses ovarian function and brings about loss of the sense of smell. There is a rare clinical syndrome (Kallman's syndrome), more frequent in males than females, in which congenital absence of the sense of smell is a pathognomonic sign, along with hypogonadism and sterility.

Whether all these data on the sense of smell have any bearing on homosexuality is not known. One possibility that might warrant further investigation pertains to neonatal life and early childhood. Perhaps human sex differences in the sense of smell are already in evidence at the age when, in some lower mammals, smell is the mechanism for bonding the mother-child relationship. One might conjecture that a neural impairment in the baby's sense of smell, or in the mother's stimulus (too much or too little, or the wrong odor), or even in the father's stimulus, will perhaps lead to an error in psychosexual differentiation. The extraordinary potency of odors in regulating body function in mammals was not known until Parkes and Bruce (1961) published their findings. They showed that an odor may be a pheromone (a long-distance exciter) for mammals as well as insects. They demonstrated that if a newly mated female mouse was exposed to the smell of an alien stud male, for an appropriate number of hours, or even to the box in which he had been caged, then she would fail to implant the eggs, would fail even to have the expected pseudopregnancy, but would return to estrus as though coitus had not occurred.

The effect of odor was further demonstrated; crowding female mice together in small groups induced pseudopregnancies and, in large groups, anestrus. These effects could be prevented by excision of the olfactory bulbs or by individual housing. Overcrowding of both sexes together is known to produce a range of social pathologies in rats (Calhoun, 1962), including destructive attack on subordinates, starvation of subordinates, male cannibalism of the young, and male homosexuality. Moreover, overcrowding of female mice during pregnancy was shown (Keeley, 1962) to have an adverse effect on the offspring as manifested in behavioral development. The mechanism of transmission is presumably hormonal.

Clearly, pheromonal and density research are pertinent for further research on the genesis of psychosexual differentiation. The sex-difference effect of vocal sound in the neonatal and early childhood period might also be looked into, and the sense of touch as experienced in rocking, patting, stroking, cuddling, clinging, and nestling.

Brain and Dimorphism of Gender Identity

The differentiation of one gender identity instead of the other may follow a paradigm not unlike that of the development of the internal sexual morphology (Money, 1968b). Two separate anatomic systems exist, the Wolffian and the Muellerian, both of which are intact early in embryonic development. Under hormonal influence, one system proliferates at a critical point in development into the functional organs of one sex, and the other regresses to a vestigial state. The correct balance is not always maintained, as in the sexual ambiguity of hermaphroditism where both systems may appear in an incomplete form. An analogous ambiguity in psychosexual differentiation might result in sexual deviations such as homosexuality, transvestism, or more rarely, transexualism.

In the world of a growing child, both male and female systems of behavior exist as models, carrying a positive or negative valence according to the sex of the child's rearing. Whatever may be the possible unlearned assistance from constitutional sources, the child's psychosexual identity is not written, unlearned, in the genetic code, the hormonal system, or the nervous system at birth. The child becomes conditioned to adhere to the positive model, that is, the one congruous with his rearing which, in the normal course of events, is congruous with his anatomy. There are some children who very early encounter difficulty with the positive model and a correct gender identity. The signs may be unmistakably manifested as young as the age of 3. For most children, however, positive and negative are clearly distinguishable. The model belonging to the other sex takes on a

negative valence and is a constant reminder of how *not* to act. With its negative valence this opposite-sex model does exist in the brain, however, as a neurocognitional entity. For those individuals destined to be heterosexual and incapable of homosexual acts, under any circumstances, the negative valence will be incapable of lifting. For other individuals, the negative valence may never become properly established. These are the individuals who will trace their homosexuality as far back as they can remember. For yet other individuals, the negative valence may be unstable so that the opposite-sex model of gender identity may, under special circumstances, become operational. These special circumstances may be developmental or in response to a traumatic emotional event. They may be the product of brain pathology: homosexuality or other forms of sexual pathology, quite incongruous with the earlier life history, may appear in cases of deteriorative or senile brain disease. There could conceivably, if one interpolates from brain stimulation experiments on animals, be special experimental circumstances which would induce homosexuality in human beings. The special circumstances which render operational the opposite-sex model are, for certain individuals, social—though the model's operation is only in fragmented form. These are the circumstances in which some people are able to temporize with homosexuality while heterosexually deprived, or chiefly for purposes of financial gain.

Investigation of the origin and regulation of the activating and inhibiting mechanisms in the hypothalamus and elsewhere in the human brain still remains to be undertaken. In the future, it may be found that a theory of neural inhibition can be extended to include a faulty postnatal inhibitory mechanism in homosexuals which allows opposite-sex behavior patterns, including those built up through social learning, to be activated. The activation of dormant, sexually dimorphic behavior was demonstrated experimentally in the rat by Fisher (1966). Male sexual behavior was elicited from either male or female animals. Fisher injected sodium testosterone sulfate directly through a microcanula implanted in the midlateral preoptic region of the hypothalamus. Similar implants of testosterone in the medial preoptic region produced maternal-like behavior. Again, the effect was obtained in either male or female animals.

The operational existence of the two sex models, or rather the patient's version of them, is nicely evident in some cases of transexual homosexualism. In what are probably only rare cases, the two models may be activated in alternating episodes of short duration. Each is identified by a separate name, as in one case, Marylou and Robert. In this case, Marylou went into a kind of staring trance when she was questioned about Robert's sex-reassignment operation, which was taboo knowledge for her. From the trance Robert emerged, with his different, deep-pitched speaking voice and his more down-to-earth ways. Marylou was coy and demure. She was gradually taking over. Robert believed that she would soon displace him completely.

The trancelike phenomenon ushering in an alternation of personality is not common among transexuals, who, by the very nature of their condition, usually stabilize as full-time members of their reassigned sex. Non-transexual transvestite homosexuals more frequently alternate their egos, episodically, along with their clothes. One such patient would take on his alternate personality, Pattie, in a kind of metamorphosis from Jack to Pattie as he sat before the mirror while dressing and applying makeup.

A great many transexual homosexuals claim that they have always had only one personality, namely, the feminine one for as long as they can remember. Usually there is no one to dispute them—no one who observed them sufficiently closely over the years to be able to detect and report a personality change. Occasionally, one has the chance to follow a transexual over the years. There was one patient in whom the feminine personality appeared only episodically at first, and not when the patient was dressed in boys' clothes which was, by family edict, most of the time. Over a period of 3 years, Tommy, also known as Kelvin, phased out as Kathryn, was then old enough to be independent of family, and established herself on a full-time basis, completely sure that she would eventually get

surgically transformed. Kathryn had a different speaking voice than Tommy, more high-pitched and with a minimal lisp. She was also different in demeanor, being more languid and tender in nature.

There are reports in the literature to indicate that, occasionally, a change of personality toward transvestism may have its onset in conjunction with the development of temporal lobe seizures (Epstein, 1961). In one case, the change was reversed by a temporal lobectomy which got rid of both the seizures and the transvestism.

This case is a reminder that in the absence of seizures, one does not yet know anything of the neurophysiology of the phenomenon of multiple personality or dissociation, or of the emergence of an alternate personality, as in transexualism, transvestism, or other manifestations of homosexuality. It is there, however, in the developmental neurophysiology and neuropsychology of behavior that the answer will one day be found to lie.

The male transexual, being the extreme form of homosexual that he is, is able to live, work, think, and make love as a woman. His female personality is, in part, his developmental conception of those traits and behavior patterns which typically constitute femininity. It is a conception that excludes some traits, such as an urge to fondle the newborn, and a propensity to erotic arousal less by visual and narrative stimuli than by touch, because those traits are intrinsic to feminine experience unfamiliar to him. The male transexual conforms to the conception of femininity he has assimilated, however, until by most standards, his personality—*her* personality—is female and is completely dissociated from male identity. There is no more extraordinary example of what homosexuality means and of how the human organism can react.

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