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Author(s): J. Richard Udry

Source: *American Sociological Review*, Vol. 65, No. 3 (Jun., 2000), pp. 443-457

Published by: American Sociological Association

Stable URL: <https://www.jstor.org/stable/2657466>

Accessed: 04-03-2019 13:14 UTC

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# BIOLOGICAL LIMITS OF GENDER CONSTRUCTION

J. RICHARD UDRY

*University of North Carolina at Chapel Hill*

*A biosocial theory of gender is constructed on both the macro and micro levels. A micro-model of within-sex differences among females integrates the biological model current in primatology with the prevailing social science model. It shows how sex differences in hormone experience from gestation to adulthood shape gendered behavior (that is, behavior that differs by sex). On the macro level, this model also illustrates how socialization and environment shape gendered behavior. It then demonstrates how hormone experiences can facilitate or dampen the effects of socialization and environment on gendered behavior. Data are from a sample of women who were studied from before they were born to the end of their third decade. I speculate about the constraints placed by biology on the social reconstruction of gender.*

**B**IOSOCIAL interaction models explaining the motivation and control of social behavior are built into our culture and religion. Laymen have always imagined that to some degree humans are “born” with propensities for behavior that are socially undesirable. In response to this they have seldom hesitated to structure social environments to control the expression of these propensities. Yet they presume that controls will often fail on the most predisposed.

Likewise (at least until the last few decades) parents have traditionally believed

Direct all correspondence to J. Richard Udry, Carolina Population Center, CB# 8120 University Square, Chapel Hill NC 27516-3997 (udry@unc.edu). Data were collected under grant R01 HD23454 from the National Institute of Child Health and Human Development (NICHD), with assistance from the Laboratories for Reproductive Biology at the University of North Carolina Medical School, and center grant P30 HD05798 from NICHD. Thanks to Naomi M. Morris for her collaboration on the original research, to Judith Kovenock for data analysis, and to Nancy Dole Runkle for project management. Thanks to Bea van den Berg for arranging access to respondents. Preliminary versions of this article were presented at the Johns Hopkins University Population Research Seminar series, the meeting of the Society for Menstrual Cycle Research, and the annual meeting of the International Academy of Sex Research, all in 1997.

that sex differences in behavior are biologically based. They have nonetheless applied socialization mechanisms to refine the behavior of males and females along dimensions of difference that are culturally approved and parentally preferred. Yet they assume that the “natural” proclivities of some children are hard to overcome.

These cultural models are the lay counterpart of the late nineteenth-century scholarly concerns, recently revived (Maryanski and Turner 1992), about the fit between human nature and social structure. Durkheim ([1893] 1964) laid out his own biosocial interaction problem. He took for granted that humans have individual differences in hereditary behavior dispositions. He argued, “If these are ceaselessly disturbed by our daily occupations, we shall suffer and seek a way of putting an end to our suffering” (p. 375). Durkheim saw a smoothly functioning society as one that allows the individual to fit activities to natural dispositions. He saw the absence of “fit mechanisms” as a source of social malaise.

This article is one of a series of articles that struggles toward a theory of gendered behavior. The first article applied a primate model to the explanation of within-sex differences in humans (Udry, Morris, and Kovenock 1995). The second article explored the fit between the distribution of

sex-dimorphic behavior predispositions of biological origin and hypothetical normative structures of societies (Udry 1994). (Sex-dimorphic means different distributions by sex. Sex-dimorphic distributions usually have a large overlap.) The present article applies this theory to the constraints imposed on socialization effects by the biological processes that determine the gendered predispositions of individuals. This problem is a special case within the category of biosocial interactions. Identification of biosocial interactions is the scientific antidote to both biological determinism and environmental determinism.

My primary hypothesis is that the effect on women of their childhood gender socialization is constrained by the biological process that produces natural behavior predispositions. This extension is stimulated by and modeled after parallel experimental work on primates and humans.

#### A HORMONE PRIMER

The theoretical biological model for sex-dimorphic and reproductive behavior is often discussed as though it were applicable only to primates because the comparisons to humans are more direct. The generic model is actually broadly applicable to all vertebrates and has been empirically explored for many species, from humans (Reinisch and Sanders 1987) to the red-sided garter snake (Crews 1991). The model states that exposure to androgens (male hormones) during a critical developmental period masculinizes the individual's reproductive organs and nervous system and potentiates subsequent appropriate reproductive behavior. The critical period differs across species, but in primates this critical period is mid-gestation.

Androgens masculinize species-specific sex-dimorphic behaviors for both males and females, but because the androgen levels produced by males are many times greater than those produced by females, the effects on females are more subtle, and species-typical female behavior occurs in the absence of androgens. Testosterone is the androgen that masculinizes behavior. It is produced in males primarily by the testes starting in mid-gestation; in females it is produced by the adrenal glands and the ovaries.

As a general rule, its effect on behavior in humans and other animals is limited in each species to behaviors that are sex-dimorphic for that species (Goy, Bercovitch, and McBair 1988). Sex hormone binding globulin (SHBG) is a large protein molecule (produced in the liver) that binds testosterone. It "transports" testosterone in the blood, and also prevents bound testosterone molecules from binding to testosterone receptors in the brain, where it exerts its behavioral effects. SHBG, on average, binds more than 90 percent of testosterone.

Based on animal and human studies, the effects of testosterone in the prenatal period are thought of as "organizational," involving more or less permanent effects on the structure of the brain. In fact, these structural effects have been experimentally induced in animal brains. They increase the later probabilities of masculine species-typical sex-dimorphic behaviors, both in childhood and later in adulthood. In adulthood, the effects of testosterone are partly dependent on the degree of prenatal masculinization of the brain. Levels of the circulating female hormones, estrogen and progesterone, are probably not central to the development of sex-dimorphic behaviors.

Testosterone at and after puberty is supposed to act on genes in the central nervous system that control the production of neurotransmitters (Panksepp 1998, chap. 12). In this way it increases or decreases the probability of masculine sex-dimorphic behaviors.

Some behaviors alter some hormone levels, in both animals and humans. The literature on this effect is extensive and generically confirmatory. While most research on the effects of testosterone on behavior show similar or analogous effects on males and females, the effects of behavior on testosterone levels sometimes differ by sex. Testosterone levels are affected by stress, sometimes in one direction and sometimes in the other (Dulac et al. 1986; Krantz and Manuck 1984; Rejeski et al. 1990).

Kemper (1990), in a provocative theoretical treatise, incorporates behavior→hormone effects with hormone→behavior effects into a socio→bio→social behavior model. He argues, for example, that in recent decades, as women moved into roles

eliciting more aggressive and control behaviors, the average testosterone levels of a whole generation of women may have risen, leading to further increases in women's assertive behavior. He speculates that rises in the female testosterone levels in response to the rebirth of the women's movement may have caused the rise in divorce rates in the United States around 1960. Testing the effects of behaviors on hormones is beyond the design possibilities of this project, but such effects always lurk as competitive hypotheses. (I make no presumption of the dominance of either hormone effects or socialization effects.)

Both maternal testosterone and SHBG increase as a pregnancy proceeds. Researchers think that SHBG prevents physiological masculinizing of the mother from her own increasing testosterone in late pregnancy. At any particular physiological juncture, statistical effects of testosterone on behavior may be seen as associated with SHBG or with testosterone or both, depending on the relative variance of each in the sample population.

At puberty in humans, testosterone increases greatly in males and increases somewhat in females. Its peak level is reached early in the third decade of life and subsequently declines in both sexes. Behavioral effects of testosterone at puberty and after are thought of as "activating" the prenatal structures.

Work on primates was recently reviewed by Wallen (1996). It shows how rearing environments modify the effects of prenatal hormone experience on the sex-dimorphic behaviors in rhesus monkeys. Research on humans is summarized by Reinisch, Ziemba-Davis, and Sanders (1991) and Collaer and Hines (1995). I have chosen a sociological audience for this article because gender has become central to social science discourse. It is important that sociologists reconcile their social constructionist models of gender with prevailing theories emerging in the biological sciences.

Departure from the social constructionist view in sociology is now under way in main-line texts on gender. For example, Lindsey (1997) says,

[E]ven if hormones predispose the sexes to different behavior, societal factors will ultimately activate this behavior. . . . Overall,

the relationship between hormones and distinctive social behavior exhibited by the two sexes is one of mutual interaction. (P. 27)

This statement represents a step toward biosocial integration. Here I take the next step and indicate what might be meant by such a statement. The first section recapitulates a model that demonstrates a biological foundation for within-sex gendered behavior in adult women parallel to that found in rhesus monkeys. The second section shows how biology constrains the effects of childhood gender socialization.

### MODELS OF WOMEN'S GENDERED BEHAVIOR

The concept of women's gendered behavior refers to the degree to which a woman's behavior is more "masculine" or more "feminine" for those behaviors on which women and men typically differ. The research begins by applying a primate model of sex-dimorphic behavior to women, and then integrates this model with a traditional social science model. Traditional social science models of gender begin with the postulate that in humans, males and females are born neutral with respect to sex-dimorphic behavior predispositions. These models assume that behavioral differences between the sexes emerge as a consequence of socialization and social structure (Maccoby 1998).

The primate model of sex-dimorphic behavior has emerged over the past 40 years as an empirically well-documented explanation of how male and female primates come to display different behavior patterns (Ehrhardt and Meyer-Bahlburg 1981). Previous applications of this theory to humans have often used clinical syndromes as the human material. The best known work assesses the prenatal androgenization component of the model by comparing the gendered behavior of girls with congenital adrenal hyperplasia (CAH) to unaffected control girls (Dittmann et al. 1990). Girls with CAH create an abnormally large amount of androgens (male hormones) prenatally because of a metabolic disorder and later show masculinized behavior (Berenbaum and Hines 1992). Prenatal androgen exposures due to medication (Reinisch 1977, 1981) and other genetic anomalies have also shown results consistent

with this theoretical model (Reinisch and Sanders 1987).

The present study takes as its design template the experiments by Goy (1970) in which pregnant female rhesus monkeys were administered exogenous androgens. The sex-dimorphic behavior of their female offspring was compared with that of the offspring of control pregnant females who received either other treatments or no treatment. These experiments showed that the female offspring of androgen-treated mothers exhibited more masculine behavior as juveniles than did the control offspring. Such experiments, in which pregnant rhesus monkeys are injected with androgens, indicate that the effects of exogenous prenatal androgenization of female fetuses on their external genitalia and their subsequent juvenile behavior are exquisitely sensitive to the dose, duration, and timing of hormone treatment during gestation (Wallen 1996). Early and large doses produce masculinized genitalia as well as behavioral masculinization. Later and smaller doses masculinize juvenile behavior but produce no masculinization of genitalia.

I assume that the greatest sensitivity to prenatal androgen differences in humans would occur in the second trimester, when fetal sex differences in androgen production are the largest. The present study is the first to model the effects of both prenatal and adult androgen on the gendered behavior of normal adult women. The basic biological model I used is published in Udry et al. (1995).

## SUBJECTS AND PROCEDURES

Ideally, this test requires second-trimester fetal measures of testosterone for a sample of females who are now adults so both their adult gendered behavior and their adult levels of testosterone can be assessed. I use a sample from the Child Health and Development Study (CHDS) (van den Berg, Christianson, and Oechsli 1988). Pregnant women presenting for prenatal care at Kaiser Plan facilities in the San Francisco Bay area were entered into the CHDS from 1960 through 1969. They were interviewed, serum samples from the women were collected in each trimester, and these samples were fro-

zen and banked for 30 years. Somewhat fewer second-trimester samples were available than for other trimesters because of no prenatal visits or because specimens were later assigned to a different trimester. Dates of blood draws were available from the prenatal records. By using the physician-estimated date of last menstrual period and physician-estimated gestational age, each blood draw was assigned to a trimester. Data from these prenatal samples provide our proxy measure for prenatal testosterone exposure.

CHDS conducted various follow-up interviews with mothers and their offspring of the index pregnancies, the last at child's age 15 to 17. In 1990 to 1991 we followed up on the white daughters born in 1960 to 1963—when they were 27 to 30 years old. These daughters were interviewed and blood samples were taken in the late afternoon and early evening during the early follicular (pre-ovulatory) phase of their menstrual cycles, in the same hospital where most of them were born three decades earlier. The mother's prenatal and daughter's adult blood samples were assayed for testosterone and sex hormone binding globulin (SHBG).

## SAMPLE

We selected mothers who had at least two prenatal blood samples (collected in 1960 to 1963) and who had a CHDS daughter interviewed at age 15 to 17. We used the CHDS public-use data set to identify eligible daughters by anonymous ID number. These ID numbers were sent to the CHDS principal investigator. Her staff sought current addresses from multiple sources—the respondents had last been interviewed as adolescents 12 years earlier. Letters were sent to inform located respondents about the study and seeking their agreement to participate. Telephone follow-ups were used when necessary. If respondents agreed to participate, interviewers located at the Kaiser Plan research center phoned, answered questions, and made appointments. Interviews were timed according to the research plan. ID numbers from the public-use data set matched the completed questionnaires with the blood samples—our research team never had access to the identities of the respondents, thus anonymity was maintained. Preg-

nant women were omitted because hormone levels are altered during pregnancy and for many weeks afterward. Also, nonwhites were omitted because pilot work with another sample indicated that female adult hormone levels and their correlations with behavior differ by race. There were not enough eligible Blacks and Asians to justify their recruitment to the sample, as their numbers would not sustain an analysis by race.

Of 470 daughters who met the eligibility criteria, 351 (75 percent) completed questionnaires. At the time of recruitment they were ages 27 to 30. The nonparticipants ( $N = 119$ ) were accounted for as follows: No address ever located by CHDS = 30; address located, but no response received = 45; response received, but Kaiser could not contact = 17; refused to participate = 20; agreed to participate, but interview was not completed by closing = 7.

Some who agreed to participate lived too far away to come to the research office for an interview. Those living near other Kaiser facilities were invited to visit to complete the required blood draw and to fill out the questionnaire unsupervised. Other participants visited the research office, but declined to give blood samples. Those not providing a blood sample were omitted from the analysis. Of the 351 who provided questionnaires, 282 had prenatal serum samples classified as second trimester, and 246 provided adult blood samples; but only 195 had both. Of these 195, 32 lacked interviewer ratings (used to compute a primary gender factor) because they were not seen in person. This left 163 women who met all the criteria for inclusion in the present analysis. Substitute values for missing data were not assigned.

Some subjects were lost from the CHDS between the prenatal period and the adolescent interview. The remaining sample of adolescent respondents had parents of somewhat higher socioeconomic status at study entry (1960 to 1963) than did those who were lost to follow up, but the two groups did not differ on other prenatal data. Sample bias was assessed simply because for all originally eligible cases we have some data, even if no adult data were obtained on the respondent. Participating and nonparticipating eligibles did not differ on

prenatal testosterone or SHBG. Participating respondents with and without adult blood samples did not differ on prenatal hormones. Women with and without values on interview-rated components of the gender factors did not differ on gender factor components on which both had values. In short, no discernible biases were found for the many comparisons made.

#### OFFICE AND LABORATORY PROCEDURES

Testosterone and SHBG have known patterns of day-to-day variation, time-of-day variation, time-of-menstrual-cycle variation, week-of-gestation variation, and so on. Time-of-day variation was controlled by scheduling interviews between the hours of 4:00 and 7:00 P.M., when testosterone levels are known to be flat and at their daily nadir. We controlled for menstrual-cycle-day variation in testosterone by scheduling interviews only on cycle days 2 through 7, when testosterone values are at their cycle nadir for women. At this time of the cycle, between-women variation is not affected by differences in cycle length or differences in oral contraceptive use. Otherwise menstrual cycle hormone variation would have made adult hormone values questionable. Respondents were asked to call the office to let interviewers know that menses had begun and to schedule interviews over the dinner hour during the required cycle window.

An interviewer explained the procedures. Respondents completed self-administered questionnaires in a private room; the interviewer was available for assistance. In addition to the study questionnaire, respondents also completed the Personality Research Form, the Adjective Check List, the Bem Sex Role Inventory, and the Strong Vocational Interest Inventory. Upon completion, a phlebotomist drew a 10 ml venous blood sample, which was clotted and spun down; the serum was poured into a glass tube and frozen at  $-20^{\circ}$  C. These specimens were shipped in dry ice to the investigators. Respondents were paid \$85 for completing all aspects of the study, less for partial completion. Assays for hormones were completed at the Laboratories for Reproductive Biology, University of North Carolina at Chapel Hill. Standard commer-

**Table 1. Definitions and Factor Assignments for Measures of Adult Gendered Behavior**

Factor	Description
Importance of home	<p>Ever married to a man: Yes response is feminine. (1 item)</p> <p>Number of live births: High number is feminine. (1 item)</p> <p>Index of sex role orientation (Dreyer, Woods, and Sherman 1991): Traditional is feminine. (16-item scale of gender role attitudes)</p> <p>Importance of career: Not important is feminine. (1 item)</p> <p>Importance of children: Important is feminine. (1 item)</p> <p>Domestic division of labor scale for current or last relationship: E.g., cooking, childcare, car repair, home repair. (14 items)</p> <p>Sex-typed activities scale listing activities in current or last relationship: E.g., who drove, who paid, who decided. (6-item questionnaire)</p>
Feminine interests	<p>Importance of marriage: Important is feminine. (1 item)</p> <p>Feminine appearance factor: Interviewer ratings of feminine demeanor, facial attractiveness, use-of-jewelry scale, use-of-cosmetics scale.</p> <p>Strong Vocational Interest Inventory (20 items that most discriminate males and females): High score is feminine (respondent has occupational interest responses like those of most females). (Hansen and Campbell 1985)</p> <p>Likes baby care. (16 items selected from maternal attitude questionnaire) (Miller 1980)</p>
Job status	<p>Proportion female in current occupation or, if not employed at present, in last occupation. (1980 census)</p> <p>Featherman socioeconomic index of current or last occupation: Low score is feminine. (Stevens and Featherman 1981)</p> <p>Proportion female in work unit on last job.</p>
Masculinity–femininity	<p>Bem Sex Role Inventory: feminine scale. (10 female items) (Bem 1981)</p> <p>Bem Sex Role Inventory: masculine scale. (10 male items) (Bem 1981)</p> <p>Adjective Check List, scored as percent masculine items selected: High score is masculine; indicates that respondent checked adjectives to describe self that a higher proportion of Americans rate as masculine (mean of 300 possible items). (Williams and Best 1990)</p> <p>Personality Research Form: masculinity score. (Berzins, Welling, and Wetter 1978)</p> <p>Personality Research Form: femininity score. (Berzins, Welling, and Wetter 1978)</p>

cial kits available at the time (1990 to 1991) were used for radioimmunoassay of both the prenatal and adult serum samples.

The self-administered questionnaire collected measures of 20 gendered behaviors. A gendered behavior is one on which males and females differ. Principal-components factor analysis produced four unrotated primary factors with eigenvalues above 1. The behaviors and their factors are defined in Table 1. A single second-order factor based on the four primary factors is the dependent variable used here—"gendered adult behavior." The loadings on the second-order factor are "importance of home" .78, "feminine

interest" .65, "job status" .66, and "masculinity–femininity" .56. The higher the score on the dependent variable, the more feminine the respondent. This measure is based on an explicitly bipolar concept of gender. I use a bipolar concept of sex-dimorphism (gender) because it is consistent with the generic biological theory.<sup>1</sup>

<sup>1</sup> The orthogonal concept conceptualizes masculinity and femininity as uncorrelated characteristics rather than as opposite poles on a continuum (Bem 1981). For a discussion of the fit between gender measures and gender theories, see Appendix A.

**Table 2. Basic Hormone Model: Unstandardized Coefficients from the OLS Regression Predicting the Effects of Prenatal and Adult Hormone Levels on Gendered Behavior in Adulthood**

Independent Variable	<i>b</i>	Standard Error
Adult testosterone (ng/dl)	-.014**	(.005)
Adult SHBG (ng/dl)	.001	(.002)
Prenatal testosterone (std)	-.299	(.206)
Prenatal SHBG (std)	.313***	(.074)
Prenatal testosterone × adult testosterone	.013*	(.006)
Adjusted R <sup>2</sup> = .16		

\**p* < .05 \*\**p* < .01 \*\*\**p* < .001 (one-tailed tests)

## RESULTS

The logic of this simple test of the primate model of sex-dimorphic behavior on human females is as follows. The daughter's second-order factor score ("gendered adult behavior") is the dependent variable. To predict it, I enter the mother's testosterone and SHBG values during the second trimester of pregnancy as indicators of the level of fetal androgen exposure—how much testosterone was transmitted from the mother to the fetus (the "organizing" effect on behavior). I have the daughter's testosterone and SHBG values from adulthood—how much testosterone was acting on the brain of the adult daughter (the "activating" effect on behavior) when the dependent variable was measured. I hypothesize that the higher the level of prenatal exposure to testosterone, the less sensitive the daughter's behavior will be to her own testosterone level in adulthood. Thus, a multiplicative interaction term is entered, "prenatal testosterone" × "adult testosterone." Table 2 shows the results of this simple test.

The basic hormone model shows that in this sample, mothers' prenatal hormones have an effect on the gendered behavior of the daughters three decades later. The effect of prenatal testosterone is picked up by variance in prenatal SHBG. So SHBG is treated as an inverse measure of testosterone exposure because the more SHBG in the mother,

the less testosterone gets through to the daughter. SHBG has no effects on behavior except through binding testosterone. The more SHBG, the less testosterone effect and thus the more feminine the woman's behavior in adulthood. There is also a significant main effect of adult testosterone. The interaction of prenatal testosterone and adult testosterone shows that the more prenatal testosterone, the smaller the masculinizing effect of adult testosterone on gendered behavior. The model explains about 16 percent of the variance in gendered behavior.

All models showed that prenatal androgen exposures from the second trimester affect gendered behavior, but not exposures from the first or third trimesters. This is as the theory predicts (Pilgrim and Reisert 1992): The second trimester is the period of greatest sensitivity to the effects of androgens, and is also the period during which male and female fetuses have the biggest difference in prenatal exposure to androgens.

### CONSTRUCTING A SIMPLE BIOSOCIAL MODEL

In a general way, variance in gendered behavior in human females can be explained with the primate model. Next I introduce an integrated biosocial model. The idea that socialization by parents begins the social process of shaping gendered behavior is pretty much accepted; whether the reader prefers a learning theory model or a cognitive construction model makes no difference for this test. The factor construction of the variable for gendered behavior is consistent with the cognitive construction model, and the cognitive theory is built explicitly on the logical process of children creating a generalized concept of gender (Maccoby and Jacklin 1974:360–66).

Respondents were given a list of 26 parental behaviors related to gender socialization and were asked to indicate which behaviors their mothers encouraged when they were children (ages 5 to 15). Sample masculine items were: "encouraged you to defend yourself physically," "to repair things around the house," "to be athletic," "to have an interest in math." Sample feminine items were: "encouraged you to wear jewelry," "to wear dresses," "to have an interest in sew-



**Table 3. Biosocial Model: Unstandardized Coefficients from the OLS Regression of the Effect of Childhood Gender Socialization on Adult Gendered Behavior by Level of Prenatal Androgen Exposure**

Independent Variable	<i>b</i>	Standard Error
Adult testosterone (ng/dl)	-.015**	(.005)
Adult SHBG (ng/dl)	.001	(.001)
Prenatal testosterone (std)	-.271	(.201)
Prenatal SHBG (std)	.296***	(.074)
Prenatal testosterone × adult testosterone	.011*	(.006)
Mother encouraged femininity	-.461*	(.280)
Prenatal SHBG × mother encouraged femininity	-.591*	(.276)
Adjusted R <sup>2</sup> = .20		

\**p* < .05 \*\**p* < .01 \*\*\**p* < .001 (one-tailed tests)

ing,” “to take dancing lessons,” and “to plan to have children.”

The feminine items were subtracted from the masculine items to create a variable called “mother encouraged femininity.” We added this variable to the biological model (not shown) and found that mothers’ encouragement of femininity significantly increased adult feminine behaviors of their daughters. The increase in R<sup>2</sup> is .02. The model with “mother encouraged femininity” is stronger than the model including only biological variables, and its inclusion has no effect on the coefficients in the biological model.

I then hypothesized that prenatal androgen exposure, because it is presumed to permanently organize the brain and therefore permanently alter the natural gender predisposition, should affect the sensitivity of the individual to feminine socialization. I predicted that women with high prenatal androgen exposure would be less sensitive to mothers’ socialization efforts. Table 3 adds an interaction of “prenatal SHBG” × “mother encouraged femininity.”

Table 3 shows that for the biosocial model, the SHBG × biosocial interaction is significant. Figure 1 displays the interaction from Table 3 in graphic form. Prenatal SHBG is

read as an inverse androgen effect: High prenatal SHBG indicates low prenatal androgen exposure and low prenatal SHBG indicates high prenatal androgen exposure. The main effect of prenatal androgen exposure is indicated by the differences in the general level of the lines. The slope of each line indicates the effect of mothers’ socialization efforts at each level of prenatal androgen exposure.

The top line of Figure 1 shows the effect of increasing mother’s encouragement of femininity for women with low exposure to androgen. For these women with low exposure to androgen, mother’s encouragement of femininity has a strong effect on gendered behavior in adulthood.

The bottom line in Figure 1 shows the effect of increasing mother encouragement on femininity for women with high exposure to androgen. The line is generally flat, indicating that no matter how much encouragement the mother provides it has little effect, and the daughter remains more masculine than average. Thus, Figure 1 shows that high prenatal androgen exposure “immunizes” daughters to the effects of feminine socialization.

The limits of female gender socialization can be illustrated another way. After respondents were asked to indicate which behaviors their parents encouraged, they were asked to indicate for each behavior whether their parents encouraged the behavior in order to reinforce their daughter’s natural tendencies or because their daughter was below average on the behavior. The number of times the respondent checked encouragement of female-typical and male-typical behaviors because she was below average on the behavior were counted separately, and the female score was subtracted from the male score to create a variable called “remedial socialization.” The higher the remedial socialization score, the more the parents appeared to be working to encourage female behaviors because the daughter was “insufficiently feminine.”

Table 4 shows the results when the remedial socialization variable is added to the hormone model (shown in Table 2). Its effect is significant, generally additive to the model, and it has a positive coefficient, indicating that the more the parents worked to improve below average femininity, the less

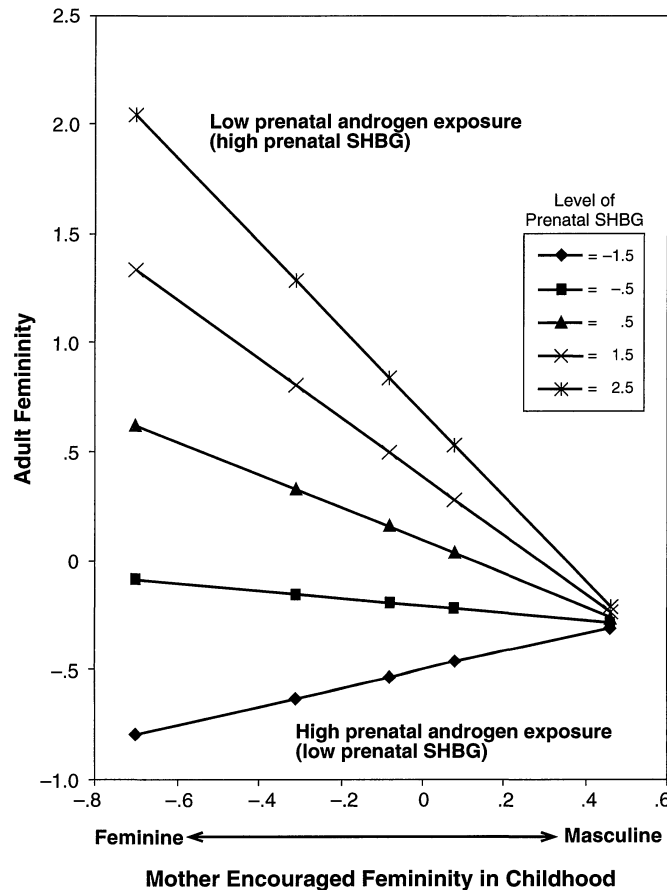


Figure 1. Effect of Childhood Gender Socialization on Adult Gendered Behavior by Level of Prenatal Androgen Exposure

successful they were; the more they tried, the less feminine the daughters were in adulthood. Respondent answers indicate that if a daughter has natural tendencies to be feminine, encouragement will enhance femininity; but if she has below average femininity in childhood, encouraging her to be more feminine will have no effect.

The daughters were also interviewed at ages 15 to 17 by CHDS. Then they were asked how important it would be 10 years in the future for them to spend a lot of time with their family. (This variable was scored from 1 to 4, with 1 as not important at all and 4 as very important.) The more highly they valued spending time with their families in the future, the higher their femininity measure in their adult interviews about 12 years later. This result is consistent with

other findings from the study, which show that daughters who end up high in femininity in adulthood also show patterns in adolescence of more traditional, conforming behaviors, with lower levels of deviant behavior, more traditional attitudes, and better relations with their families.

Table 5 shows that the importance of time spent with family interacts with second-trimester SHBG (our inverse testosterone effect) in predicting adult femininity. This interaction is graphed in Figure 2, in which the equation for Table 5 is evaluated at five levels of SHBG in standardized units.

Figure 2 shows that for those who, as adolescents, answered that time with their families would be very important to them a decade later (a distinctly feminine response), their values on gendered behavior at adult-

**Table 4. Biosocial Model: Unstandardized Coefficients from the OLS Regression of the Effect of Remedial Socialization on Adult Gendered Behavior**

Independent Variable	<i>b</i>	Standard Error
Adult testosterone (ng/dl)	-.015**	(.005)
Adult SHBG (ng/dl)	.001	(.001)
Prenatal testosterone (std)	-.251	(.206)
Prenatal SHBG (std)	.293***	(.074)
Prenatal testosterone × adult testosterone	.011*	(.006)
Remedial socialization	.530*	(.278)
Adjusted R <sup>2</sup> = .17		

\**p* < .05 \*\**p* < .01 \*\*\**p* < .001 (one-tailed tests)

hood were clustered and slightly above average in femininity, showing only a moderate effect of their differential prenatal androgen exposure. But for those who as adolescents said that time with their families would not be important at all to them a decade later, their values on gendered behavior as adults are widely dispersed and depend heavily on their prenatal androgen exposure. Those most highly androgenized in the second trimester are three standard deviations more masculine than those least androgenized.

Figure 2 demonstrates how prenatal hormone experience continues to influence the trajectories of women's gendered behavior during adulthood. A decade of young adult life separates the adolescent attitudes and the adult measure of gendered behavior. This is a decade during which many opportunities are encountered and many choices are made. During this period, those who held equally nonfamily-oriented attitudes in adolescence arrived at quite different gendered behavior by the end of their third decade of life. Those most androgenized prenatally drifted most toward more masculine behavior.

## SPECULATION

### IMPLICATIONS FOR MALES

I now explore the implications of the fact that the models predicting gendered behavior show that high prenatal androgenization of

**Table 5. Biosocial Model: Effects of Importance of Time with Family on Adult Gendered Behavior by Level of Prenatal Androgen Exposure (OLS Regression)**

Independent Variable	<i>b</i>	Standard Error
Adult testosterone (ng/dl)	-.015**	(.005)
Adult SHBG (ng/dl)	.000	(.001)
Prenatal testosterone (std)	-.290	(.194)
Prenatal SHBG (std)	-.104	(.169)
Prenatal testosterone × adult testosterone	.012*	(.006)
Importance of time with family	.332***	(.081)
Prenatal SHBG × importance of time with family	-.240**	(.088)
Adjusted R <sup>2</sup> = .26		

\**p* < .05 \*\**p* < .01 \*\*\**p* < .001 (one-tailed tests)

females not only masculinizes their gendered behavior predispositions at later ages, but immunizes them against socialization toward typical feminine behavior. Generalizing this effect to males, we should predict that males' much higher prenatal androgenization (perhaps tenfold that of females), caused by testosterone from their own testes, not only masculinizes their later gendered behavior predispositions, but also immunizes them against later feminizing socialization. The only males that would not be highly immunized against feminizing socialization would be those who as fetuses had androgen exposures as low as females. These would be rare clinical cases. So in a general way simply by *being* male, males can be thought of as highly immunized against feminine socialization by prenatal androgenization.

### IMPLICATIONS FOR SOCIAL CHANGE

Now some speculation about secular changes in gendered behavior in a society based on extending the theory put forth here. If a society should decide that it wanted to reduce sex differences in gendered behaviors, it could alter the socialization patterns to give females less feminine or more masculine socialization. Our results indicate that most females are to some degree responsive

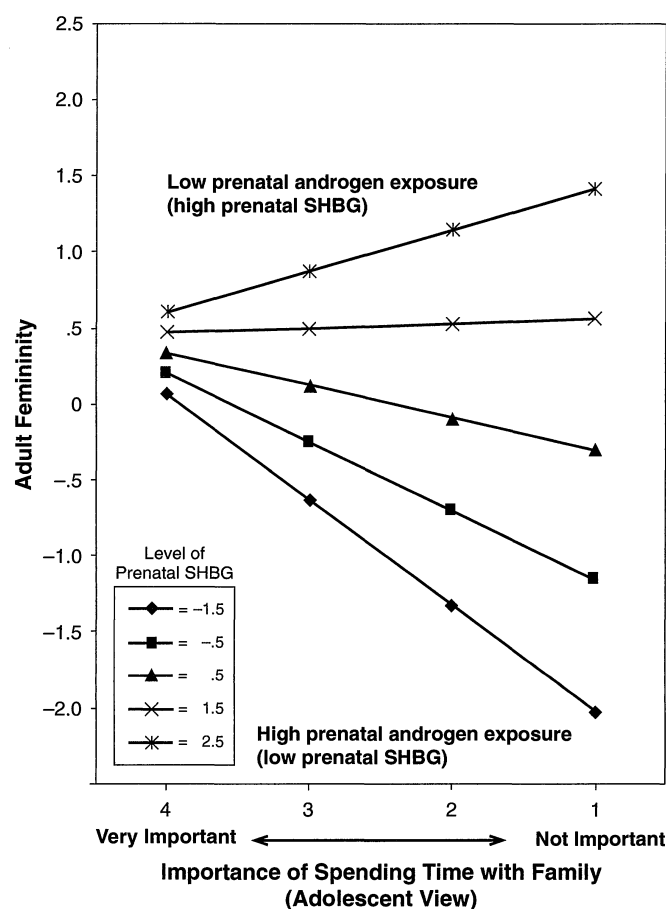


Figure 2. Effect of Adolescent Family Attitudes on Adult Gendered Behavior by Level of Prenatal Androgen Exposure

to variations in gender socialization (see Figure 1) and so would respond by displaying more masculine or less feminine behavior. Those highly androgenized prenatally would already have more masculine behavior. But if males by being males, are highly immunized against feminizing socialization experiences, then attempts at feminizing their socialization would be less effective. With these hypothetical changes in the social regimen of gender, males would change little, while females would change to exhibit more masculine or less feminine behaviors. Thus, females would be thought of as more responsive to shifts toward masculine socialization, and males would be thought of as less responsive to shifts toward feminine socialization.

I make no judgment here as to whether it is morally good to reduce sex differences, or to leave them alone. I do not hold to the view that if a behavior pattern is natural (that is, biologically underwritten), it is morally desirable. I am certainly willing to mess with Mother Nature. Assuming there is a moral consensus in society that sex differences should be reduced, I take no moral stand on whether this should be achieved by masculinizing females or feminizing males, or a little of each, or some other way. But some approaches may be easier than others.

## CONCLUSIONS

Neither the biological theory of gender nor the socialization theory is an original contri-

bution of this paper. Each theory is accepted and researched in its own field. The idea of integrating the two theories has been proposed previously by sociologists (Lindsey 1997). My sole contribution is to put together a data set that illustrates one way to test the integrated theory.

I show that the seemingly contradictory theories of sex-dimorphic behavior from the two diverse scientific traditions are not, in fact, incompatible—they are well on the way to being integrated. This integration is possible on both the macro and micro levels. On each level, sociologists need only incorporate the element from the primate theory that indicates that sex-dimorphic behavior has biological foundations. Admittedly, this is the toughest obstacle to overcome for most sociologists.

Nothing in this integration requires sociologists to reject or transform any existing sociological or psychological gender theory, however social constructionist, once the postulate is added that biology sets limits to the macro-construction of gender and also sets individual limits to the effects of gender socialization. Even those theories that seem least amenable to integration are not difficult. For example, if the statement, "Gender is a socially constructed power device invented by males to exploit females," is treated as a premise, an integrated macro model can explain why it is a male rather than a female invention. Differential female exposure to androgens can explain the differential response of females to their disadvantage.

Broadening the theory I have sketched here leads toward explanation of the cross-cultural similarities of gender structure while leaving intact the sociological explanation of cross-cultural variation in terms of technology and ecological variation. We can

theorize about the escape of exceptional women from even the most stultifying and restrictive boundaries of women's roles while not give up an inch of constructionist territory in explaining the structure from which they escaped.

A biosocial macro theory is simple: Humans form their social structures around gender because males and females have different and biologically influenced behavioral predispositions. Gendered social structure is a universal accommodation to this biological fact. Societies demonstrate wide latitude in this accommodation—they can accentuate gender, minimize it, or leave it alone. If they ignore it, it doesn't go away. If they depart too far from the underlying sex-dimorphism of biological predispositions, they will generate social malaise and social pressures to drift back toward closer alignment with biology. A social engineering program to degender society would require a Maoist approach: continuous renewal of revolutionary resolve and a tolerance for conflict. But if a degendered (or post-gendered) society is the goal, our micro-models offer some guidelines. It may be easier to degender society by changing female behavior to more closely coincide with the present behavior of males rather than the reverse.

*J. Richard Udry is Kenan Professor in the Department of Maternal and Child Health and the Department of Sociology at the University of North Carolina at Chapel Hill. He is a Fellow at the Carolina Population Center. His long-term scientific interest is integrating biological factors into sociological models. He is Principal Investigator for the National Longitudinal Study of Adolescent Health (Add Health), a program project in its third wave of data collection. His Add Health analysis focuses on sexual behavior and gender.*

#### Appendix A. Fitting Gender Measures to Gender Theories

Definitions of the concepts in theories are an integral part of the theories—concepts and their measures should not be thought of as exogenous to a theory, merely imported for testing the theory. The theory guiding the present research is derived from a biological model explaining primate sex-dimorphic behavior. That model attributes differences in gendered behavior to the same single hormone process for both sexes. According to this theory, then,

it is logical to incorporate a bipolar concept of gendered behavior.

The history of measures of masculinity-femininity in psychology began with a bipolar scale consisting of questionnaire items that showed sex differences (Terman and Miles 1936). This scale enjoyed wide use for more than three decades. Many standardized personality tests included implicitly bipolar masculinity-femininity scales or were retrofitted

Table A-1. Hormone Model Predicting Personality Measures of Gender

Independent Variable	Bem Sex Role Inventory		Personality Research Form		Adjective Check List <sup>a</sup>
	Male	Female	Male	Female	
Adult testosterone	n.s.	n.s.	n.s.	n.s.	n.s.
Adult SHBG	n.s.	n.s.	n.s.	+	-*
Prenatal testosterone	n.s.	-*	n.s.	n.s.	n.s.
Prenatal SHBG	n.s.	n.s.	-*	+	+**
Prenatal testosterone × adult testosterone	n.s.	+	n.s.	n.s.	n.s.
Model probability (F)	.66	.11	.19	.05	.00
Adjusted R <sup>2</sup>	.00	.02	.01	.03	.06

Sources: See Table 1.

Note: Signs of significant coefficients are indicated by "+" and "-"; "n.s." indicates nonsignificant coefficients.

<sup>a</sup> Percentage of responses indicating masculinity.

\* $p < .05$     \*\* $p < .01$  (one-tailed tests)

with such scales on the basis of sex differences in response. In an early example of an orthogonal measure, Brim (1958) retrofitted a two-scale approach to an existing data set. He asked judges to assign adjectives to either "instrumental" or "expressive" categories, and called the resulting scales "masculinity" and "femininity" respectively. He found that different sibling configurations affected masculinity and femininity in different ways—a finding that would have been concealed by a bipolar approach.

After a widely cited article by Constantinople (1973) argued that gender was multidimensional, many different multidimensional approaches to the measurement of masculinity and femininity appeared. Bem (1974) developed the Bem Sex Role Inventory, which contained separate measures of masculinity and femininity that were deliberately constructed to be orthogonal. This inventory has been widely used. Many researchers developed factorial approaches that produced from 2 to 16 gender factors (Cohen and Burdsal 1978; Huston 1985; Robinson and Follingstad 1985). Little attention was devoted, however, to the gender theory underlying these models. At the same time, researchers working with the rapidly developing primate hormone model using human clinical samples were using bipolar scales (Dittman et al. 1990; Meyer-Bahlberg et al. 1984).

Nothing about the primate theory states that sex-dimorphic behaviors cannot arise from other, non-hormonal, processes as well. In both humans and other primates, environmental variations produce sex differences that are not testosterone dependent and can modify testosterone-dependent behavior patterns. Whether these differences would be hypothesized to be organized along a single bipolar dimension or multiple orthogonal dimensions would depend on the processes hypothesized to produce them.

The dependent variable used here, gendered adult behavior, contains one primary factor that consists

of two orthogonal pairs and one bipolar measure of personality-type masculinity-femininity scales (see Table 1). Table A-1 shows how the hormone model in Table 2 predicts each of these five commonly used gender scales when the scales are the dependent variables. While the orthogonal pairs have a shadow of the findings shown by using the bipolar second-order factor derived from Table 1 and used as the dependent variable in Table 2, the only significant model is for the bipolar scale derived from the Adjective Check List. This analysis shows that for the present theory much is lost when using either of the commonly used orthogonal scales as dependent variables. At the same time, however, the basic findings of the hormone model can still be discerned.

It is possible to construct bipolar, orthogonal, or multidimensional scales measuring sex-dimorphism as free creations of the human intellect, derived from theories of diverse origins. But the theories in which they become imbedded and the structure of real behavior restrict their range of usefulness. At the same time, the scales can diagnose the fit between scales and theories. If it is not possible to take a broad range of sex differences and construct a coherent bipolar scale, then any theory leading to such a scale begins to fall apart. If scales based on theories implying orthogonal dimensions are in fact not orthogonal, it is hard to fit them into an orthogonal theory.

In conclusion, gender concepts and their measurement are integral to the theories that give birth to them. Within the empirical constraints imposed by the structure of our observations and the theories in which we use them, we may need, and therefore may construct, concepts of gender that call for a scale that is unidimensional (and bipolar), or multidimensional (either correlated or orthogonal). Is it better to use a single bipolar gender scale, two orthogonal scales, or multidimensional scales? The best answer is what does your theory imply?

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