A BIOSOCIAL PARADIGM FOR WOMEN'S GENDER ROLES

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This research was supported by a research grant (HD23454) from the National Institute of Child Health and Human Development. Prepared for presentation at the annual meetings of the Population Association of America to be held in Denver, CO, April 30 - May 2, 1992.

ABSTRACT

This study applies a paradigm from behavioral endocrinology derived from primate studies to the explanation of the variance in adult women's gender roles. The object is to predict women's choices between traditional and non-traditional gender roles on the basis of prenatal and adult hormone experience combined with personal and environmental characteristics derived from social science theories of gender role development. A sample of women was followed from their own prenatal period through the third decade of life. Adult gender roles were identified through interviews at ages 27-30. The predictive model uses measures of fetal hormone environment and adult measures of steroid hormones. The model is successful in predicting a substantial amount of variance in adult gender roles. This is strong evidence for a biological foundation underlying the variance in women's gender roles. Many childhood characteristics were also predictive of women's later gender role behavior. The most important was the effect of high intelligence toward non-traditional roles.

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J. Richard Udry

This research program integrates a sociological/psychological theory of gender roles (primarily a socialization theory) with a theory from behavioral endocrinology that is generic to mammals. The social science theory we use is almost universal in sociology and psychology, and appears in the work of Bem (1987) in the form of Gender Schema Theory:

Gender Schema Theory states that "as a result of historical accident, the culture has clustered a rather heterogeneous collection of personality attributes" into the categories masculine and feminine. The content of these categories is entirely arbitrary. The culture places so much emphasis on these categories as opposed to any other scheme of classification of people and attributes that everyone learns the classification scheme and uses it to organize a perception of the world and the self. Individuals who have learned the schema select from the world of their observations behaviors that are appropriate to the schema, and avoid those that are inappropriate. Individuals differ from one another, based primarily on their family socialization experiences, in the extent to which they use this schema to organize their own personalities and behavior. Sex typed individuals (those most motivated to use the schema) select behaviors that are consistent with their gender and avoid those that are inconsistent with it. Gender schema theory can be seen as a contemporary version of Kohlberg's (1966) cognitive construction theory of the acquisition of gender roles.

The theory from behavioral endocrinology is based on three decades of studies showing that in all mammalian species studied, all dimensions of

sex-dimorphic behavior are controlled by steroid hormone experience. A two-phase process is involved. In primates, differential exposure to prenatal androgens causes a permanent differential neural organization of the central nervous system. This differential neural organization provides the sub-structure for differential behavior predispositions, and for differential behavioral response to adult steroid steroid hormone levels. Adult gender role behavior is therefore influenced by prenatal androgens and adult steroid hormones, both through main effects and two types of interaction: prenatal/adult hormone interactions, and hormone/environment interactions.

The empirical support in human behavior for each of these theories remains sketchy for two reasons. First, the theories are still rudimentary. Second, sound empirical studies are difficult to design because of data requirements, and therefore few existing studies provide more than general direction for the future. Bem says, "it is not possible at this point to state whether individual differences in gender-schematic processing do in fact derive from differences in emphasis placed on the gender dichotomy in individuals' socialization histories, or to describe concretely the particular kinds of socialization histories that enhance or diminish gender-schematic processing" (Bem, 1987, p. 213).

The primary weakness in empirical support for the behavior endocrinology theory is the fact that most existing support comes from clinical studies, and hence the contribution of normal variation in steroid hormones to normal adult gendered behavior in women remains in doubt. Furthermore, no previous studies have had available both prenatal and adult hormone measures on the same sample of women.

The integration of socialization theory with the theory of behavioral endocrinology involves the modification of socialization theory by adding

the following propositions: (1) hormones, both prenatal and adult, may have main effects on gender roles in addition to effects of childhood experience; (2) hormones may lead to behavior predispositions that lead to selection of certain environments, and therefore environments may be intermediate variables for the effects of hormones; (3) hormones may interact with both environmental effects and other hormones to influence adult gender roles.

The power of the research being conducted under this grant is due primarily to the unique strengths of the Child Health and Development Study (CHDS). The CHDS, organized by the University of California at Berkeley in collaboration with Kaiser Plan hospitals, enrolled all patients presenting for prenatal care and delivery in Kaiser Plan hospitals in the California Bay Area beginning in 1960 and extending several years. Those entering the study from the spring of 1960 to the spring of 1963 were followed up by the CHDS at offspring age 5, 9-11, and 15-17. For these cohorts, serum samples were obtained from the pregnant woman during the first, second, and third trimesters. The serum was frozen and stored for 30 years. We located and re-interviewed the white female offspring of these cohorts in 1990-91. Out of 470 eligible cases, we interviewed 350. By relocating and interviewing these women, and obtaining blood samples at ages 27-30, we have put together a powerful data set capable of addressing the crucial points of both theories. The maternal sera and daughter's sera were assayed in our laboratory for the hormones most relevant to the theory.

Many aspects of the first phase of our work remain incomplete. We have limited our analysis to preliminary testing of the generic hypotheses of the study. These preliminary tests in general confirm all the basic hypothesis. In the following summary, we will only illustrate the results we have

obtained.

RESULTS

1. Confirmation of the biological theory

a. Current androgenic hormones are related to women's gender roles. We have confirmed that either or both adult testosterone and androstenedione are positively related to non-traditional gender roles in our sample. We looked at 7 dimensions of behavior (ever-married, importance of career, Socio-Economic Index (Featherman) of latest occupation, proportion female in latest occupation, gender role attitudes, importance of caring for children, and number of children) and found that adult androgens predict non-traditional behavior on each dimension, except gender role attitudes. Using these dimensions, a composite factor was constructed and called GENDER ROLE. (High on GENDER ROLE is traditional feminine behavior.) Both adult testosterone and androstenedione predict non-traditional GENDER ROLE at a level of statistical significance of .01 or better.

As an illustration of the unexpected power of interviewer ratings, we asked our interviewers to rate each respondent on her general demeanor and presentation during the interview on a multi-point scale from very masculine to very feminine. The interviewers were given no instructions or definition for the rating. Interviewers' ratings could not be contaminated by the woman's questionnaire responses, since all questionnaires were self-administered, and interviewers were instructed not to examine them. But a model of adult androgens predicted about ten per cent of the variance of the masculinity/femininity ratings of interviewers, and was highly significant ($p = .0009$). Adult androgens also inversely predict interviewer ratings of attractiveness and and 8-item use-of-cosmetics scale.

b. Prenatal androgen exposure alters the effect of adult androgens on women's gender roles. This hypothesis, while proposed by the theory, has never been tested before because no study has ever been able to put the proper data together. The first observation based on our data is that the prenatal androgens from the mother correlate positively and significantly with the daughter's adult androgens. No doubt this is a genetic relationship.

The second observation is that when the models above are constructed using both adult and prenatal testosterone, there are inverse main effects of both adult and prenatal androgens on GENDER ROLE, and an interaction effect. The effect of adult androgens depends on the level of prenatal testosterone. Both adult androgens and prenatal testosterone jointly cause shifts to non-traditional roles, but the higher the prenatal testosterone, the less the effect of adult androgens. This means that high prenatal androgens desensitize the woman to adult androgen effects. This is consistent with previous observations that women are much more sensitive to androgens than men, presumably because of their much lower exposure to prenatal androgens. Models using specific elements of GENDER ROLE produce essentially the same conclusions, but less powerful models. Various hormone models of GENDER ROLE and its components yield R squares in the range of .08 to .26.

The OLS model in Table 1 is typical of those obtained predicting GENDER ROLE.

TABLE 1 ABOUT HERE

Considering the facts that we have only one assay of adult hormones on one cycle day of hormones that vary by cycle day, from cycle to cycle, and across the life span of adulthood, that we are working within the range of normal variation of prenatal hormones, and have no clinical cases in the sample, these are surprisingly strong first findings.

2. Confirmation of The Social Science Theory

The data set allowed us to examine the childhood and adolescent environmental and temperamental characteristics of respondents to predict adult gender roles. While the theory predicts the categories of variables that should be of interest as precursors of women's gender roles, it is not very specific about exactly what those variables should be. We have been guided by the idea that the most choice of gendered behavior should be available to those that come from privileged backgrounds, are more intelligent, show early non-traditional gendered personality, and are less family oriented as children. Table 2 shows that we were highly successful in predicting gender roles from childhood characteristics. This in itself is a first. While many aspects of the data we have are not yet ready to enter analysis, we constructed a GENDER ROLE factor from aspects of the main questionnaire only. HIGH on GENDER ROLE signifies traditional female gender role. This factor includes the following variables and has a satisfactory eigenvalue: Importance of career, importance of rearing children, importance of marriage, ever-married, number of living children, Featherman-Stevens SEI of current or last occupation, proportion female in current or last occupation, and a gender role attitude scale. The strongest single predictor was child's Peabody score, a verbal IQ equivalent. (Related findings appear in Dittmann et al. (1990) and previous studies based on clinical samples. According to Dittman et al., and our preliminary

analysis, this is evidently not a hormone effect.) Parental characteristics (father's age at birth of child, father's and mother's education, and mother's Peabody score) were good predictors of gender role and most of its components. Father's age is a strong predictor net of parents' education and other SES variables. The theoretical significance of this finding needs further exploration. Child's temperament/personality components at ages 5 and 9 (especially the mother's descriptors of the child's behavior indicating the girl has temperamental traits not typically ascribed to girls by mothers) are highly predictive of adult gender roles. By combining parental characteristics, and child's characteristics at ages 5 and 9, we were able to predict a third of the variance in GENDER ROLE (R square = .36).

Adding the girl's relationships to her parents at adolescence, and various adolescent attitudes based on an interview with the girls at adolescence, no additional predictive power was obtained. Similar but less powerful results were obtained for most components entering into the GENDER ROLE factor.

No predictive power for gender roles was obtained from any aspect of family structure, nor any of several measures of the mother's work behavior (for example, worked outside the home for pay, or number of hours worked). Girls with nontraditional gender roles as adults spent less time with their fathers as adolescents, thought it was unimportant to obey parents or to spend time with their families.

TABLE 2 ABOUT HERE

The power of the variables in predicting GENDER ROLES (mean = 0, SD = 1) can be demonstrated using a cross-classification of the sample on two variables: Child's Peabody and father's education (each dichotomized at its mean), as in Table 3.

TABLE 3 ABOUT HERE

Using the same cross-classification, the mean number of children born to women with Low Father's Education and Low Peabody is 1.01, while for those with High father's education and high Peabody it is .43 (others .70).

3. Confirmation of Joint Hormone effects and biosocial interactions

Effects of both prenatal and adult hormones have been found on Premenstrual Syndrome (PMS). PMS is measured by a summative index based on a 12-item set of questions about symptoms. The effect of estradiol on PMS is conditioned by the level of prenatal androgen. High prenatal androgens protect the woman from the PMS-inducing effects of adult estradiol observed in women with low prenatal androgens. In a model predicting minor lawbreaking (got speeding ticket, shoplifted, stolen something besides shoplifting, and arrested for other than shoplifting) we found that mother's education and the woman's adult testosterone predicted the minor lawbreaking behavior. But a strong interaction showed that among women with low-education mothers, increasing testosterone was associated with more lawbreaking, while among those with high-education mothers, high testosterone women did not differ from low testosterone women on minor lawbreaking. (High mother's education protects women against the lawbreaking effect of high testosterone.) This result is similar to Dabbs' (1990) finding that high testosterone is associated with anti-social

behavior in low SES but not in high SES adult males.

To see the effect of two variables, one the most powerful single hormone effect, and one the most powerful single childhood precursor on GENDER ROLE, we cross-classified the sample into four categories: high Peabody/high (adult) androgen, high Peabody/low androgen, low Peabody/high androgen, and low Peabody/low androgen. Each variable was dichotomized at its mean. GENDER ROLE is measured in SD units. The results in Table 4 were obtained.

TABLE 4 ABOUT HERE

The effect of each variable on GENDER ROLE is stronger in the group high on the other variable. A similar model is obtained substituting adult androstenedione for testosterone. The joint effect of the two cross-classified variables is to shift the GENDER ROLE mean nearly a full standard deviation. This result is based on a median split of each variable, using the whole sample, and is not based on extreme cases only.

The present research is not subject to most of the objections of earlier critics (Quadagno et al, 1977; Hines, 1982) concerning the application of the behavior endocrinology model to human gender roles. First, it is based on a general, non-clinical population, and does not involve inference to the general population by comparison of clinical cases to unaffected controls, as in published studies of girls with congenital adrenal hyperplasia. Second, there is no confusion related to cortisol treatment of the clinical cases, since there is none. Third, there is no possibility of differential parental socialization based on known clinical

cases, early genital masculinization, etc.

The initial phase of our data analysis has been able to confirm the basic features of the behavior endocrinological theory of women's gender roles. We have also shown strong confirmation for the sociological model of gender roles. Finally, integrated biosocial models demonstrate that the elements of the two theories combine to produce both additive and interactive relationships between the theories. We have therefore laid the foundations for a new paradigm for studying women's gender roles.

CONCLUSIONS

There is a biological foundation for differences among women in gender role behavior. This foundation is laid in the prenatal period through fetal androgen exposure to maternal androgens. This does not mean that endogenous androgens in the fetus do not also contribute to the foundation of gender roles. We simply have no measures of fetal androgens. Our measures of prenatal maternal androgens may only provide a faint shadow of the total effect of fetal androgen exposure on gendered behavior. Studies of girls with congenital adrenal hyperplasia show that endogenous fetal androgens play a pivotal role in their later gendered behavior. Adult androgens also strongly shape adult gender role behavior. The effect of adult androgens is mediated by the level of fetal androgens. This probably means that fetal androgen exposure permanently alters the brain's sensitivity to androgens.

A frequent response of social scientists to such a theory is to question how this can be so, and at the same time gender role behavior of women in a society can shift dramatically over a few decades. Surely everyone agrees that the secular trends in women's gender roles are caused by changing sociological forces, not changing biology. The biological

theory is only designed to explain variance in behavior among a cohort of women. The causes of secular change and the causes of variance in a cohort of women are not necessarily related.

Our findings with respect to the biological foundations of women's gendered behavior in no way undermines the importance of socialization as a contributor to variance in gender roles. Our theory states and our data reveal that the sociological factors are both additive to and interact with the biological substrate of individual behavior predispositions. It is the integration of the biological and sociological theories that provides a new paradigm for studying gender roles. While this paper has attended only to women's behavior, the biosocial theory is equally applicable to males, and ultimately of course to the behavior differences between males and females.

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TABLE 1. EFFECTS OF PRENATAL AND ADULT HORMONES ON GENDER ROLE (OLS)

Variable	B	SE	p
Intercept	.36	.40	.36
Adult Androstenedione	-.003	.001	.00
Adult SHBG	-.002	.002	.40
Prenatal Testost. (trimester 2)	-.42	.24	.08
Prenatal SHBG (trimester 2)	.002	.0005	.00
Andro x Prenatal Testos.	.002	.001	.05

R square = .12

(Note on metrics: Androstenedione - ng/dl; SHBG - nm/l; Prenatal Testosterone - standard deviate (unit is SD of Testosterone distribution.)

TABLE 2. CHILDHOOD PREDICTORS OF ADULT GENDER ROLE (OLS MULTIPLE REGRESSION)

CATEGORY OF VARIABLES	F STATISTIC
1. Parental characteristics (R Square = .14)	
Father's age at child's birth (-)	11.74
Father's education (-)	11.83
Mother's Peabody score (-)	4.21
2. Child's test scores (R square = .11)	
Peabody score (-)	31.83
3. Child's behavior/temperament (R square = .17)	
Has nightmares (-)	3.90
Likes school (+)	9.15
Typical girl index (+)	14.58
Good Student (-)	5.34
Hates to share possessions (-)	12.37
Prefers play w/younger children (+)	4.31
4. Adolescent relationship with parents (R square = .05)	
Hours spent with father (+)	6.20
Father wants to know where I am (-)	5.64
5. Adolescent attitudes (R square = .11)	
Important to be ambitious (+)	4.12
Important to obey parents (+)	9.31
Important spend time w/family (+)	6.89
Can't change important things (+)	6.71

TABLE 3. JOINT EFFECT OF FATHER'S EDUCATION AND CHILD'S PEABODY ON GENDER ROLE

Father's Educ	Child Peabody	Mean GENDER ROLE
Low	Low	+.51
Low	High	+.07
High	Low	+.01
High	High	-.30

TABLE 4. JOINT EFFECT OF ADULT TESTOSTERONE AND CHILD PEABODY ON GENDER ROLE

Testosterone	Child Peabody	Mean GENDER ROLE
Low	Low	+.30
Low	High	.00
High	Low	+.07
High	High	-.59