Effects of Girls' Hormonal Status on Depressive and Aggressive Symptoms Over the Course of One Year

Roberta L. Paikoff,¹ Jeanne Brooks-Gunn,² and Michelle P. Warren³

Associations between hormonal and physical status and girls' depressive affect, aggressive affect, and delinquent behavior were studied over the course of one year. Seventy-two White girls, aged 10-14 at initial data collection, were seen twice. Endocrinological status (estradiol, luteinizing hormone [LH], folicle stimulating hormone [FSH], testosterone, and dehydroepiandosterone sulfate [DHEAS] at Time 1, physical development (menarche, secondary sexual characteristics) and maturational timing at Times 1 and 2 were used to predict self- and maternal reports of depressive affect, and self-reports of aggressive affect and delinquent behavior at Time 2. It was posited that initial endocrinological status, as represented by hormonal categories derived by Warren and Brooks-Gunn [(1989) "Mood and Behavior at Adolescence: Evidence for Hormonal Factors," Journal of Clinical Endocrinology and Metabolism, Vol. 69, pp. 77-83] and reflecting estradiol, LH, and FSH levels, would be associated with affective expression and delinquent behavior one vear later, DHEAS, however, was not expected to be associated with affective expression a year later as it is thought to be more indicative of adrenal changes that are in part environmentally mediated. Initial affective expression was hypothesized to account for more of the variation than prior hormonal status. Hormonal categories predicted depressive and aggressive affect

¹Associate Research Scientist, Educational Testing Service, Princeton, NJ 08541. Received Ph.D. from Institute of Child Development, University of Minnesota. Research interests include the interpaly among developmental processes during the transitions into and out of adolescence. To whom reprint requests should be addressed.

²Senior Research Scientists, Educational Testing Service, Princeton, NJ 08541. Received Ph.D. from University of Pennsylvania. Research interests include Girl's psychological adaptation to pubertal change, biosocial aspects of female reproductive events, development of biological and socially at risk children and adolescents.

³Associate Professor Clinical Obstetrics and Gynecology and Clinical Medicine, Columbia University College of Physicians and Surgeons; Co-Director of Gynecologic Endocrinology. St. Luke's-Roosevelt Hospital Center. M.D. from Cornell University. Research interests include: Reproductive endocrinology, anorexia nervosa, neuroendocrinology, adolescence.

a year later, while DHEAS, physical status, and maturational timing did not. Initial hormonal categories were associated with subsequent reports of delinquent behavior, although not after physical status or prior reports of delinquency were entered into the regression. Initial reports of affective expression accounted for a far greater proportion of the variance in aggressive affect and delinquent behavior than did hormonal or physical status.

INTRODUCTION

Increases in emotionality and moodiness are often thought to typify the adolescent experience. While extreme moodiness and emotional lability are commonly thought of as normative during adolescence, most research focuses on negative mood states, not on emotional lability (see, as a notable exception, Larson and Lampman-Petraitis, 1989). In fact, considerable interindividual variation in negative affect is seen, although the average level of affective expression does increase from childhood to adolescence (Csikszentmihalyi and Larson, 1984; Larson and Lampman-Petraitis, 1989, Rutter et al., 1976; Simmons et al., 1973). Gender differences in negative mood states are often seen in early to middle adolescence, with depressive affect and disorder more common in young women than in young men (Petersen et al., this volume: Radloff, this volume: Cantwell and Baker, this volume; Rutter et al., 1976, 1986). Conversely, aggressive behavior problems are more common in males than in females; however, gender differences in aggressive affect have not been studied extensively (Cairns et al., 1989; Parke and Slaby, 1983; Susman et al., 1987). Emotional excitability or level of affective expression may influence depressive and aggressive affect or delinquent behavioral manifestations in both genders, although little research has been conducted to address this issue.

Researchers interested in the development of young adolescents have long postulated relations between physiological changes and emotionality or moodiness (A. Freud, 1948; S. Freud, 1905/1953; Hall, 1904); however, it is only recently that systematic investigations into physiological change-affective expression associations have begun (Brooks-Gunn and Warren, 1989; Buchanan, Eccles, and Becker 1990; Paikoff and Brooks-Gunn, 1990; Susman *et al.*, 1987).

Physiological processes may contribute to affective expression in young adolescents in several different ways. First, physiological change may act directly to influence emotional state. For example, changes in either pubertal hormone concentration or fluctuation at early adolescence could render an adolescent more excitable or easily aroused, and thus more prone to sudden mood shifts or to negative mood states (Buchanan and Eccles, 1990; Paikoff and Brooks-Gunn, 1990). Hormonal changes also may interact in as yet unspecified ways with the endogeneous opiate or neurotransmitter systems.

Hormones and Girls' Affective Expression

On the other hand, physiological change could impact upon affective expression indirectly (via the secondary sex changes of puberty) due to changing reactions of others or to heightened self-consciousness or self-awareness during the early pubertal years (Koff *et al.*, 1981; Paikoff and Brooks-Gunn, in press). It also may be the case that physiological and physical changes each exert independent effects upon emotional states in young adolescents, that their effects are mediated by prior experience or by life events, or that some combination of these occurs. Few conceptualizations, however, have considered possible longer term effects of the biological changes occurring in young adolescents.

The current study examines possible effects of hormonal and physical status on affective expression and delinquent behavior in adolescent females in a short-term longitudinal study. Initial hormonal status, pubertal status, timing of maturation, and initial reports of affective expression were considered in association with depressive and aggressive affect as well as delinquent behavior, one year later. The following questions were addressed:

1. Are initial hormonal levels associated with affective expression or delinquent behavior one year later?

2. Are pubertal status and/or timing variables associated with affective expression or delinquent behavior one year later?

3. What is the relative contribution of hormonal levels, pubertal status, and initial affective expression or delinquent behavior to reported affective expression or delinquent behavior one year later?

In terms of the first question, hormonal changes during adolescence may have activating effects on both behavioral and affective changes in young adolescents (Beach, 1975; Tieger, 1980; but see also Coe *et al.*, 1988). Activational effects of hormones on adolescent affective expression have just begun to be studied. Associations found thus far have been small, often overridden by environmental circumstances, and specific to only a few behavioral domains having to do with emotional expression, sexual behavior, and aggressive behavior (Brooks-Gunn and Warren, 1989; Buchanan *et al.*, 1990; Olweus *et al.*, 1988; Paikoff and Brooks-Gunn, 1990; Susman *et al.*, 1987; Udry, 1988). We have postulated that increases in excitability, arousability, and/or negative mood states may occur when the hormonal system is undergoing its initial rise during the transition from prepubertal to pubertal functioning (Brooks-Gunn and Warren, 1989). Longitudinal analyses on the possible persistence of these effects, however, have not yet been conducted.

Several issues must be addressed when studying possible direct hormone effects upon affect or behavior. First, one must consider which hormones would be likely to exert influence on the affective or behavioral domain(s) of interest. Hormonal associations have been reported for depressive affect, aggressive affect, aggressive behavior, and sexual behavior in human adolescents. Changes in estradiol level may be associated with depressive affect in adolescent girls (Brooks-Gunn and Warren, 1989; as nonconfirmatory evidence, however, see Susman *et al.*, 1987). Testosterone has been associated with sexual arousal in boys and girls, and with sexual behavior and aggression in boys (Olweus *et al.*, 1980, 1988; Udry, 1988; Udry *et al.*, 1985). Both androstenedione (in the forms of dehydroepiandosterone [DHEA] and its sulfate DHEAS) and estradiol have been associated with aggressive behavior in adolescent girls (Inoff-Germain *et al.*, 1988; Brooks-Gunn and Warren, 1989; Susman *et al.*, 1987; Warren and Brooks-Gunn, 1989).

Second, one must consider the possibility that general rises in all of the gonadal and hypothalamic pituitary hormones may account for some activational effects, rather than rises in any one hormone by itself. All pubertal hormones are rising at approximately the same time although the changes are more pronounced in some than in others and the timing of the rises differs somewhat. Intercorrelations among the pubertal hormones are quite high in young adolescents, ranging from the .30s to the .60s, making it hard to disentangle effects of separate hormones. At the same time, effects may be more general in that rapid endocrine rises may affect internal states or other psychological or physiological substrates (Nottelmann et al., 1987; Warren and Brooks-Gunn, 1989). To capture these general endocrine changes, we classified endocrine levels into four categories (Warren and Brooks-Gunn, 1989). Although these categories were based on estradiol levels, they also "capture" rises in luteinizing hormone (LH) and follicle stimulating hormone (FSH) levels (see Warren and Brooks-Gunn, 1989, Figure 1, p. 79). In the present study, these categories, as well as two androgens – DHEAS and testosterone-were examined to see how they were associated with depressive affect, aggressive affect, and delinquent behavior.

While both testoterone and DHEAS have been shown to have effects on aggression, DHEAS is an andrenal androgen while testosterone is for the most part a gonadal androgen; thus the two may operate differently and have different effects on behavior. For example, DHEAS is likely to influence aggression and responses to provocation in the short term, as it may be more responsive to environmental stressors than is testosterone (Parker et al., 1985; Sassenrath, 1970; Susman et al., this volume; Suomi, this volume; Zumoff et al., 1983). Thus, the nature of the association between hormone level and affective expression or behavior must be considered as well. When specific hormones are entered into straightforward linear regression models few, if any, associations with affective expression in girls are found (Susman et al., 1987). These analyses, however, do not test for the possibility that associations might not be captured by a linear model. In our earlier study, estradiol was associated with girls' reports of depressive and aggressive affect in a curvilinear fashion: dividing hormonal levels into four categories, depressive affect was higher at Level 2 (time of initial pubertal hormonal rise) than Level

1, but plateaued at Level 3, and may have even decreased at Level 4. The current study examines both linear and nonlinear effects of hormones on affective expression and delinquent behavior.

Given our reading of the current, albeit preliminary literature, several hypotheses related to the first question (effects of initial hormone levels on affect and behavior one year later) were advanced. First, hormonal effects on depressive and aggressive affect seen in the cross-sectional study were expected to persist over one year, using the categories developed earlier (based on estradiol but also capturing LH and FSH). Girls in the lowest estradiol category at Time 1 were expected to continue to have the lowest scores on depressive and aggressive affect at Time 2. Depressive affect scores show relatively high across time stability in adolescence. For example, in a nine-year follow-up of 14- to 18-year-olds, correlation coefficients were .44 and .35 for girls and boys respectively on a measure of depressive affect (Kandel and Davies, 1986). Continuity emerges even earlier: in a four-year annual followup of over 100 girls aged 10-12 at first-year testing (some of whom were also in the hormonal study), correlations between years ranged from the .30s to the .60s for measures of depressive affect (Baydar et al., 1989). Consequently, a trajectory of negative affect, once begun, is likely to continue, requiring a large change or highly significant event to alter it. In addition, girls in the lowest estradiol category are likely, in a rank order sense, to remain the least advanced vis-à-vis pubertal growth. Consequently, their depressive and aggressive affect scores may still be lower than other girls as their physical development has not progressed as far.⁴

Second, effects of DHEAS on aggressive affect seen in the crosssectional study were not expected to persist. As mentioned earlier, adrenal androgens may be more influenced by environmental events than are gonadal or hypothalamic pituitary hormones (and may be influenced by hypothalamic pituitary hormones as well). The adrenal androgens are associated with the cortisone adrenal system, which is known to be highly reactive to environmental events and ill health (see Parker *et al.*, 1985; Susman *et al.*, this volume; Suomi, this volume; Zumoff *et al.*, 1983). Consequently, our initial finding is posited to have been time linked.

Our second question concerned possible effects of secondary sexual development. For this question, we had two hypotheses. The development of secondary sex characteristics, as assessed by the Tanner (1962) rating scale, was posited not to be associated with reports of delinquent behavior, nor with depressive or aggressive affect. No concurrent effects of pubertal sta-

⁴Whether or not our curvilinear effect would persist over a year, however, was not clear. On the one hand, if hormonal effects were strong and consistent, the curvilinear relationship should replicate after one year. On the other hand, dependent upon the rate of hormonal changes (as well as the duration of hormonal effect upon negative affect), any number of other associations could be found.

tus upon depressive or aggressive affect were found in our other studies (Baydar *et al.*, 1989; Brooks-Gunn and Warren, 1989) or in the work of others (Olweus *et al.*, 1988; Susman *et al.*, 1987).

Pubertal timing, however, was expected to be associated with delinquent behavior, and possibly with affective expression as well. Delinquent and aggressive behavior have been associated concurrently with early pubertal maturation (Magnusson *et al.*, 1985). Additionally, later breast development has been associated with lower depressive affect in adolescent girls (Baydar *et al.*, 1989). It is possible that early maturers demand increased freedom and autonomy and that parents, in fact, do modify their expectations for early maturing adolescents (Simmons and Blyth, 1987). More physically advanced adolescents may be more likely to engage in deviant activities, be involved with an older peer group, and thus be exposed to deviant activities earlier. Thus, timing of maturation was expected to be associated with delinquent behavior and possibly aggressive and depressive affect.

Our final question relates to a concern of long-term work in any area, and to the relative importance of a variety of factors in prediction. In this study, we were particularly interested in the amount of variance accounted for by prior reports of affect and behavior, as well as potential interactive effects between negative affect, delinquent behavior, and hormonal or pubertal status. That is, in our earlier study, while hormonal factors were associated with affective expression, social factors (e.g., life events) accounted for a relatively larger proportion of the variance (8-18%) than did hormonal ones (1-4%). Here, we expected prior reports of affective expression to account for more variance than the hormonal factors. Given stability of self-reports of affective expression cited earlier (Kandel and Davies, 1986; Baydar et al., 1989), initial reports ought to account for more variance than any of the pubertal events (hormonal or physical). As mentioned earlier, hormonal effects upon depressive and aggressive affect were expected to persist on some level due to consistency of reports of depressive and aggressive affect through the adolescent years. Thus, it was not clear whether reports of prior depressive or aggressive affect would wipe out prior hormonal effects, interact with these effects, or act independently of these effects. It was expected, however, that prior depressive and aggressive affect would contribute more to current affect than prior hormonal level given the short-term stability reported by others.

METHOD

Subjects

Seventy-two White girls who were 10-14 years of age at initial data collection (M = 12.16, SD = .88) were participants in the current follow-up.

Hormones and Girls' Affective Expression

These girls are a subsample of 103 girls upon whom endocrinological data were collected, and who, when contacted one year later, participated in additional psychological assessments. All girls attended one of several private day schools in New York City; parental education and family social class are similar in this sample to those in affluent suburban school systems.

The girls are from well-educated, middle to upper middle-class families. Virtually all of the families were in the two highest of the five Hollingshead social classes (96%; Hollingshead and Redlich, 1958), 85% of the mothers had graduated from college, and 60% of the mothers were working part or full time. No demographic differences were found for age or grade in school.

Procedure

Girls were recruited from the initial sample of 103 who participated in hormonal and psychological assessments: 70% of the initial sample was seen a year later. The girls who participated in the longitudinal study did not differ from those who did not participate on initial physical development and selected aspects of psychological development⁵ (as measured by variants of subscales of the Self-Image Questionnaire for Young Adolescents; Brooks-Gunn *et al.*, 1989; Offer *et al.*, 1982; Petersen *et al.*, 1984). Girls were seen in the late spring and summer of 1983, and again in late spring and summer of 1984. The data were gathered in a physical examination, by questionnaire, and by interview. Girls were paid for their laboratory visit. Additionally, mothers of girls who participated in the studies were asked to complete questionnaires mailed to their homes. The majority of mothers (90%; N = 65) did so.

Measures

Self-report and maternal report scales were used to tap depressive affect. Only self-report was used for aggressive affect and delinquency. Pubertal status was measured via physical examination and hormonal status via blood sample. Table I presents the means and standard deviations of all measures used in the study; Table II presents their intercorrelations.

Depressive Affect

The Depressed-Withdrawal Scale of the Youth Behavior Profile, developed from the Child Behavior Checklist (Achenbach and Edelbrock, 1983,

⁵Slight differences (e.g., .3 of one point) between girls who participated in longitudinal assessments and those who did not were found, however, on measures of emotional tone and social relationships at Time 1.

Variable	Time	Mean	SD
Depressed-Withdrawal	1	6.72	3.73
Depressed-Withdrawal	2	6.62	3.13
CES-D	2	32.02	9.13
PI-DMI	1	9.08	2.38
PI-DMI	2	9.43	2.88
Aggression	1	11.10	5.84
Aggression	2	10.22	4.62
Delinquency	1	4.69	3.64
Delinquency	2	7.05	4.14
Estradiol	1	41.70	37.59
Testosterone	1	.43	.19
DHEAS	1	108.34	56.05

Table I. Means and Standard Deviations of All Measures

1987)⁶ was used to assess adolescents' reports of negative affect at Times 1 and 2. In addition, the Center for Epidemiological Studies Depression Scale (CES-D) was added at Time 2 (Radloff, 1977, this volume), and mothers filled out the Psychiatric Institute Depressive Mood Inventory (PI-DMI; Kandel, 1983) at both Time 1 and Time 2.

On the depressed-withdrawal items of the Youth Behavior Profile, girls indicated on a 3-point Likert scale whether each of 11 items were *not at all true, somewhat true,* or *very true* of themselves. Possible scores range from 0 to 22. Items on the depressed-withdrawal scale include "I refuse to talk" and "I am unhappy, sad, or depressed." Alpha coefficients were .68 at Time 1 and .63 at Time 2, and scores at Time 1 were correlated with Time 2 scores (r = .44; p < .001).

On the CES-D, the adolescent rates a series of 20 items (such as "I was bothered by things that don't usually bother me," and "I wasn't able to feel happy") on a 4-point scale (from *rare* to *very often*) in terms of their occurrence within the past week. Possible scores range from 20 to 80. Interitem reliability within the sample was high (Cronbach's alpha = .88) and similar to other studies using the instrument (Radloff, 1977, this volume).

The PI-DMI is a 6-item scale where the mother is asked to rate how much her daughter has been bothered or troubled by items such as "feeling too tired to do things," or "feeling hopeless about the future" on a 3-point scale, from *much* to *somewhat* to *not at all*. Possible scale scores in this study range from 6 to 18. Interitem reliabilities were good for the current sample

⁶Due to a large number of cases missing one or two items on the Achenbach, missing values were inputed using the modal response of an individual on a particular scale. In addition, although Achenbach scales discussed here use adolescent self-reports, items were put into scales using the parent report scales on the Youth Behavior Profile. At the time these data were collected, norms and subscales for the Youth Self-Report were not yet available.

	101101101101								7 1111 T 10	
	Estradiol	ΓН	FSH	DHEAS	Testosterone	D-W	CES-D	PI-DMI	Aggression	Delinquency
Estradiol	1	.63	.37°	.18°	.53°	.07	.26 ^d	.07	06	.08
LH		I	.69 ^e	.38°	.68°	$.16^{b}$.12	,19 ^b	.07	.03
FSH			I	.17°	.45°	.21°	.05	.24 ^c	.18 ^b	.26 ^d
DHEAS				I	.61°	$.16^{b}$.14	.14	03	15^{b}
Testosterone					I	.18 ^b	.20°	80.	03	06
Depressed-Withdrawal (D-W)						I	.47°	.26 ^d	.45°	<u>*</u> 44.
CES-D							I	.18°	.41°	.42°
PI-DMI								l	.15 ^b	.15 ^b
Aggression									۱	.59°
Delinquency										I
^a Since particular N's vary slig	shtly, borderli	ine sign	ificance	levels vary	as well.					
p < .10										
$a_p^d > .01.$										
${}^{e}p < .001.$										

Table II Intercorrelations Between Hormonal Measures at Time 1 and Psychological Variables at Time 2ª

(Time 1 alpha = .65, Time 2 alpha = .76), and cross-time correlation was high (r = .71; p < .001).

Aggressive Affect and Delinquent Behavior

Aggression and Delinquency scales were derived from the Youth Behavior Profile at Times 1 and 2. Aggression and delinquency are two subscales used to describe externalizing behavior problems in adolescent girls that discriminate between children referred for psychosocial problems and nonreferred children (Achenbach and Edelbrock, 1981; Boyle and Jones, 1985). The Aggression Scale used here consists of 21 items, with possible scores ranging from 0 to 42. Examples from this scale include "I physically attack people" and "I feel that others are out to get me."⁷ The Delinquency Scale consists of 16 items, with possible scale scores ranging from 0 to 32. Examples from this scale include "I lie or cheat" and "I steal things from places other than home." For the current study, redundant items (N = 2)on the two scales were omitted from the Aggressive Affect Scale and left on the delinquent scale, making aggressive affect into a 19-item scale. Interitem reliabilities for both scales are high (for Aggression, at Time 1 alpha = .85; at Time 2 alpha = .83; for Delinquency, at Time 1 alpha = .70; at Time 2 alpha = .81). The two dimensions are highly correlated in our sample (r = .59 at Time 1 and .68 at Time 2, p < .001), and cross-time correlations within scales are high as well (r = .58 and p < .001 for Aggression, and r = .73, p < .001 for Delinquency).

Pubertal Development

Each girl was given a physical examination at both Time 1 and Time 2. Tanner ratings were obtained at this time; pubic hair and breast development were rated on a 5-point scale, from prepubertal to postpubertal (Marshall and Tanner, 1969; Reynolds and Wines, 1948). The examinations were conducted by the third author or by a nurse practitioner. Breast and pubic hair scores were averaged for this study, with half-stages rounded down. At Time 1, the majority of girls were in Tanner Stages 1 (26%) or 2 (30%), with a minority in Stages 3 (28%), 4 (15%), and 5 (1%). By Time 2, the majority of girls were in Stages 3 (24%), 4 (29%), and 5 (21%), with a minority remaining in Stages 1 (6%) and 2 (19%). Interobserver reliabilities be-

⁷It is important to note here that the Aggression Scale of the Achenbach contains items assessing both affective and behavioral dimensions of aggression. Since the majority of items tap more affective dimensions of aggression while the delinquency scale is more behaviorally oriented, we have chosen to label the Aggression Scale an affective one.

tween the two were obtained by an independent assessment of 10 girls; agreement was 100% on pubic hair and 90% on breast development. Questions were asked about menarcheal age; such reports are quite accurate, even for this age group (Brooks-Gunn *et al.*, 1987; Damon *et al.*, 1969).

Pubertal Timing

Study participants were classified as early, on time, or late using Tanner breast and pubic hair stage at Time 1 and Time 2. See Gargiulo *et al.*, 1987, for a full discussion of the procedures used to assess pubertal timing. At Time 1, 9% were classified as early, 55% were classified as on time, and 37% were classified as late maturers. At Time 2, 30% of participants were classified as early, 53% as on time, and 18% as late maturers. Since rate of maturation may change over the pubertal process, these timing distinctions represent a dynamic phenomenon, and thus change from year to year (Eichorn, 1980).

Hormonal Assessment

Measurements of serum FSH, LH, estradiol, DHEAS, and testosterone by specific radioimmunoassay (RIA) were made for blood samples collected at Time 1 (Warren, 1980; Warren *et al.*, 1977).⁸ All samples were determined in duplicate, and interassay and intraassay coefficients are reported in Warren (1980) and Warren and Brooks-Gunn (1989). Blood was taken in the late afternoon from all girls to reduce the chance of possible diurnal variations in hormonal levels. Zero-order correlations among hormonal levels are presented in Table II. As can be seen, all were positively associated, although the magnitude of the relationship was often modest. Those of moderate magnitude (i.e., r's over .50) were estradiol and LH, LH and FSH, LH and testosterone, and testosterone and DHEAS. Since concurrent relations had been found only between depressive and aggressive affect and estradiol and DHEAS in our sample at Time 1, as well as with testosterone in other samples, only these hormones were used in the following analyses. Mean hormone levels were 41.70 pg/ml (SD = 37.59) for estradiol, .43 pg/ml

⁸Blood samples were collected at Time 2 as well; however, possible effects of cyclicity on our data become more worrisome at this time since the majority of girls in the sample are postmenstrual. We have thus used menarcheal status at Time 2 rather than hormonal status as a proxy for biological change. At Time 1, 80% of the sample were premenarche, and 20% postmenarche. By Time 2, only 41% were premanarche with 59% postmenarche. We present the data using our estradiol categories separately for pre- and postmenarcheal girls at Time 2 to make sure any differences found are not due to eventual physical status (see Question 2 in our introduction).

(SD = .19) for testosterone, and 108.34 pg/ml (SD = 56.05) for DHEAS, similar to other studies of girls in this age range. As mentioned earlier, girls' estradiol data were coded into categories created by Warren and Brooks-Gunn (1989). Estradiol was the focus of study due to previous findings as well, as the fact that it is the principal gonadal hormone in girls and the one exhibiting the most dramatic increase during puberty (Apter, 1980; Apter and Vihko, 1977; Faiman and Winter, 1974; Grumbach, 1975; Root, 1973).

The following four estradiol levels were used:

Level	Amount (pg/ml)
1	0-25.00 pg/ml
2	25.01-50.00 pg/ml
3	50.01-75.00 pg/ml
4	75.01-high value

In general, levels of less than 25 pg/ml estradiol are known to have minimal effect on the human female. Levels of 25–50 pg/ml have early visible physiological effects such as secondary sexual development and effects on the vagina; however, estrogens at this level are not usually significant enough to cause proliferation of the endometrium and withdrawal bleeding with a progesterone challenge. Levels of 50–75 pg/ml are commensurate with middle or late puberty and early follicular levels in menstruating girls. These levels have significant effects on endometrial growth and other organs such as the breast. Girls with levels in this range would generally experience withdrawal bleeding to progesterone and/or have spontaneous periods. Levels greater than 75 pg/ml are considered mature levels and associated with cyclicity in females (Warren and Brooks-Gunn, 1989). The percentages of girls in each category at Time 1 are 41% (Level 1), 25% (Level 2), 15% (Level 3), and 19% (Level 4).

RESULTS

Question One

Are hormonal levels associated with affect and/or behavior one year later?

Stepwise hierarchial regressions were used to answer this question. Separate equations were run for each dependent variable (Depressed-Withdrawal, CES-D, PI-DMI, Aggression, and Delinquency). The following variables were entered into the initial equation: Step 1: Estradiol (4 categories); Step 2: Estradiol² (also by 4 categories); Step 3: DHEAS, testosterone; Step 4: age at Time 1 (as a control for possible age effects). DHEAS and testosterone were entered jointly into regression equations, since both were expected to tap possible androgenic effects upon affective expression.

Depressive Affect

A significant linear effect for estradiol Time 1 was found for adolescents' reports on the CES-D (Multiple r = .263; r^2 change = .069; p < .05; beta = .26). Mean depression scores on the CES-D rose consistently from Estradiol Level 1 (M = 30.53; SD = 7.18) to Levels 2 and 3 (M's are 33.31 and 33.08 for Levels 2 and 3, respectively, with SD's of 13.08 and 9.70) and then to Level 4 (M = 37.55; SD = 11.54; see Table III and Fig. 1). DHEAS and testosterone did not enter this equation. Contrary to the earlier concurrent analyses, estradiol categories were not associated with adolescent reports of depressed-withdrawal on the Youth Behavior Profile one year later. Maternal reports of daughter depressive affect on the PI-DMI measure were not associated with hormones, just as they were not in the earlier concurrent analyses (Warren and Brooks-Gunn, 1989).

Step	Variable	Multiple r	R ² change	Beta
Α	CES-D			
1	Estradiol	.263	.069ª	.263
2	Estradiol ²	.266	.002	064
3	DHEAS/	.282	.009	.086
	testosterone			.015
4	Age Time 1	.284	.001	036
В	Aggression			
1	Estradiol	.057	.003	070
2	Estradiol ²	.250	.059ª	365
3	DHEAS/	.261	.005	.007
	testosterone			095
	Age Time 1	.261	.001	.009
С	Delinquency			
1	Estradiol	.139	.019	.139
2	Estradiol ²	.295	.067 ^a	389
3	DHEAS/	.393	.067 ^b	076
	testosterone			257
4	Age Time 1	.423	.024	.183

 Table III. Significant Regression Equations, Question 1: Associations Between Hormonal Levels, Affective Expression, and Delinquent Behavior

 ${}^{a}p < .05.$ ${}^{b}p < .10.$



Fig. 1. CES-D time 2 by hormonal category Time 1 and menarcheal status Time 2. (Note: Only 2 girls were premenarcheal at Time 2 and in hormonal category 3; thus, they were not included in the figure. No girls were premenarcheal at Time 2 and in hormonal category 4.)

Aggressive Affect

As was found in our concurrent analyses, a nonlinear effect of estradiol reached significance in adolescent reports of aggression on the Youth Behavior Profile (Multiple r = .25, r^2 change = .059; p < .05, beta = -.37). Scores on aggressive affect increased from estradiol Stage 1 (M = 10.41;



Fig. 2. Aggressive affect at Time 2 by hormonal category Time 1 and menarcheal status Time 2. (Note: Only 2 girls were premenarcheal at Time 2 and in hormonal category 3; thus, they were not included in the figure. No girls were premenarcheal at Time 2 and in hormonal category 4.)

SD = 4.56) to Stage 2 (M = 12.24; SD = 6.83), decreased from Stage 2 to Stage 3 (M = 10.75; SD = 4.16), and decreased further from 3 to 4 (M = 9.00; SD = 7.82; see Table III and Fig. 2). Neither DHEAS nor testosterone entered the regression equation.

Delinquent Behavior

A significant nonlinear effect of estradiol was found in initial analyses of associations between hormonal factors and delinquency (Multiple r =.295; r^2 change = .067; p < .05, beta = -.39). Additionally, DHEAS and testosterone jointly approached significance in self-reports of delinquent behavior (Multiple r = .393; r^2 change = .067; p < .07, beta DHEAS = -.08; beta testosterone = -.25; see Table III and Fig. 3).

Question Two

Are pubertal status and/or timing variables associated with affect and/or behavior one year later?

Stepwise hierarchial regressions also were performed to address questions concerning effects of pubertal status and timing on adolescent's behavior and affective expression. Separate regression equations were conducted for each independent variable, as well as for each of the following dependent variables: Tanner Stage, Time 1; Tanner Stage, Time 2; Pubertal Timing,



Fig. 3. Delinquency at Time 2 by hormonal category at Time 2 and menarcheal status at Time 2. (Note: Only 2 girls were premenarcheal at Time 2 and in hormonal category 3; thus, they were not included in the figure. No girls were premenarcheal at Time 2 and in hormonal category 4.)

Time 1; Pubertal Timing, Time 2; Menarcheal Status, Time 1; Menarcheal Status, Time 2. Problems of colinearity necessitated the use of so many independent equations; because so many independent regression equations were run, the Bonferroni statistic was calculated to assess significance of our regression equations. This created a significance level of .01 (rounded up from .008) for tests of pubertal status and timing effects. Specific sequences used were in part determined by initial hormonal analyses (see below). Analyses were conducted with Time 2 pubertal measures to make sure that any hormonal effects were not just due to girls being physically more advanced a year later. Nonlinear effects as well as linear effects of pubertal timing were explored, with no changes in results.

Depressive Affect

The following sequence was used in stepwise regression equations concerning associations between hormonal and pubertal status and negative affect: Step 1: Estradiol; Step 2: Pubertal Status, and Menarche, OR timing, Time 1 OR Time 2; Step 3: Age.

None of the pubertal status variables examined were associated with any measures of depressive affect. In all equations, the effect of estradiol on CES-D was maintained at the .05 level reported previously.

Aggressive Affect

The following stepwise regression sequence was used to test for effects of physical status on self-reports of aggressive affect: Step 2: Estradiol²; Step 2: Pubertal status and Menarche OR timing at Time 1 OR Time 2; Step 3: Age. None of the physical status variables were significantly associated with reports of aggressive affect. In each case, however, when a physical status variable was entered into the regression equation, the estradiol effect was somewhat dampened (p < .10).

Delinquent Behavior

The following sequence was used to test associations between hormonal and pubertal status and delinquent behavior: Step 1: Estradiol²; DHEAS and testosterone; Step 2: Pubertal status and Menarche OR timing at Time 1 OR Time 2; Step 4: Age. Breast and pubic hair stage at Time 2 was significantly associated with reports of delinquency ((Multiple $r = .388; r^2$ change = .122; p < .01, betas are .198 and .336 for breast and pubic hair, respectively). Results for Time 1 pubertal status were similar, but did not meet the stringent Bonferroni criteria for statistical significance (see Table IV and Fig. 4).

Table IV. Significant Regression Equations, Question 2: Associa-tions Between Puberal Status and Timing, Affective Expression, andDelinquent Behavior

Step	Variable	Multiple r	R^2 change	Beta
1	Estradiol	.069	.005	069
2	DHEAS/ testosterone	.168	.023	183 .063
3	Tanner Breast/ Pubic Time 2	.388	.122ª	.198 .336
4	Age Time 1	.389	.001	.052

 ${}^{a}p < .01.$



Fig. 4. Delinquency scores at Time 2 by Tanner Stage, Times 1 and 2. (Note: Only 2 girls were premenarcheal at Time 2 and in hormonal category 3; thus, they were not included in the figure. No girls were premenarcheal at Time 2 and in hormonal category 4.)

Question Three

What is the relative value of hormonal and pubertal status variables as opposed to prior behavior and affect?

As would be predicted, responses to most measures at Time 1 were highly associated with responses at Time 2. In the case of depressive affect, however, CES-D was not highly associated with prior or concurrent reports on the Depressed-Withdrawal Scale (used as a proxy for CES-D in these analyses, as CES-D was not available at Time 1). Effects of estradiol on depressive and aggressive affect were not changed by the inclusion of prior reports of affective expression, and no interactions between hormones and prior reports were found. For depressed-withdrawal, aggressive affect, and delin-

gression Estradiol ² Aggression Time 1 (T1) Aggression × Estradiol ² Age Time 1 pressed-Withdrawal (D-W) Estradiol D-W T1 × Estradiol Age Time 1 DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.205 .588 .588 .589 .71 .546 .546 .546 .546 .548 .069 .070 .160	.042 ^a .304 ^b .001 .001 .005 .293 ^b .001 .002 .005 .001	205 .562 025 .024 .071 .546 .045 .044 .069 006
Estradiol ² Aggression Time 1 (T1) Aggression × Estradiol ² Age Time 1 pressed-Withdrawal (D-W) Estradiol D-W T1 D-W T1 × Estradiol Age Time 1 DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.205 .588 .588 .589 .71 .546 .546 .546 .548 .069 .070 .160	.042 ^a .304 ^b .001 .001 .005 .293 ^b .001 .002 .005 .001	205 .562 025 .024 .071 .546 .045 .044 .069 006
Aggression Time 1 (T1) Aggression × Estradiol ² Age Time 1 pressed-Withdrawal (D-W) Estradiol D-W T1 × Estradiol Age Time 1 DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.588 .589 .71 .546 .546 .546 .548 .069 .070	.304 ^b .001 .001 .005 .293 ^b .001 .002 .005 .001	.562 025 .024 .071 .546 .045 .044 .069 006
Aggression × Estradiol ² Age Time 1 (D-W) Estradiol D-W T1 D-W T1 × Estradiol Age Time 1 DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.588 .589 .71 .546 .546 .546 .548 .069 .070 .160	.001 .001 .005 .293 ^b .001 .002 .005 .001	025 .024 .071 .546 .045 .044 .069 006
Age Time 1 pressed-Withdrawal (D-W) Estradiol D-W T1 D-W T1 × Estradiol Age Time 1 DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.589 .71 .546 .546 .548 .069 .070 .160	.001 .005 .293 ^b .001 .002 .005 .001	.024 .071 .546 .045 .044 .069 006
pressed-Withdrawal (D-W) Estradiol D-W T1 D-W T1 × Estradiol Age Time 1 DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.71 .546 .546 .548 .069 .070 .160	.005 .293 ^b .001 .002 .005 .001	.071 .546 .045 .044 .069 006
Estradiol D-W T1 D-W T1 × Estradiol Age Time 1 DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.71 .546 .546 .548 .069 .070	.005 .293 ^b .001 .002 .005 .001	.071 .546 .045 .044 .069 006
D-W T1 D-W T1 × Estradiol Age Time 1 DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.546 .546 .548 .069 .070	.293 ^b .001 .002 .005 .001	.546 .045 .044 .069 006
D-W T1 × Estradiol Age Time 1 DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.546 .548 .069 .070 .160	.001 .002 .005 .001	.045 .044 .069 006
Age Time 1 DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.548 .069 .070 .160	.002 .005 .001	.044 .069 006
DMI Estradiol Menses Time 1 Menses Time 1 × Estradiol	.069 .070 .160	.005 .001	.069 006
Estradiol Menses Time 1 Menses Time 1 × Estradiol	.069 .070 .160	.005	.069. 006
Menses Time 1 Menses Time 1 × Estradiol	.070 .160	.001	006
Menses Time 1 × Estradiol	.160	010	
Estradiol	.160	010	
		.019	273
PI-DMI Time 1	.728	.506 ^b	.724
PI-DMI Time 1			
\times Estradiol	.802	.114	-1.227
Age Time 1	.802	.001	.018
linquency			
fanner Time 1	.134	.018	.134
Fanner Time 2 (T2)	.170	.011	.173
Delinquency T1	.738	.520 ^b	.720
Delinquency T1			
× Tanner T1	.739	.001	064
Delinguency T1			
× Tanner T2	.760	.032 ^b	1.62
e Time 1	.764	.007	12
	Age 11me 1 linquency Fanner Time 1 Fanner Time 2 (T2) Delinquency T1 X Tanner T1 Delinquency T1 X Tanner T2 e Time 1	Age Time T.802linquency Fanner Time 1.134Fanner Time 2 (T2).170Delinquency T1.738Delinquency T1.739Delinquency T1.739Delinquency T1.760e Time 1.764	Age Time T $.802$ $.001$ linquency Fanner Time 1 $.134$ $.018$ Fanner Time 2 (T2) $.170$ $.011$ Delinquency T1 $.738$ $.520^b$ Delinquency T1 $.739$ $.001$ Delinquency T1 $.760$ $.032^b$ e Time 1 $.764$ $.007$

Table V. Significant Regression Equations, Question 3: Contribution of Hormones, Pubertal Status, Initial Affective Expression, and Initial Delinquent Behavior

quent behavior, prior reports accounted for a larger proportion of variance than did hormonal factors (see Table V).

DISCUSSION

Associations between hormonal levels, affective expression, and delinquent behavior one year later were found. Findings will be discussed separately for the domains of depressive affect, aggressive affect, and delinquent behavior.

Depressive Affect

The CES-D was significantly associated with prior hormonal levels, while the Depressed Withdrawal Scale and the PI-DMI measure (mother report) were not. The association of the CES-D with prior hormonal level was linear (see Fig. 1), not curvilinear, as in the concurrent Time 1 hormonal analyses. However, the CES-D was not administered at Time 1. Time 1 reports of depressed-withdrawal were not associated with Time 2 reports on the CES-D, and did not change the association between CES-D and estradiol.

Although the CES-D and the Depressed-Withdrawal Scale are both measures of depressive affect, they appear not to tap identical dimensions. The correlation between the two measures is .47, relatively low for two measures of the same construct. The Depressed-Withdrawal Scale contains items reflective of both depressive affect and isolation or loneliness, such as "I like to be alone" and "I am secretive or keep things to myself." The CES-D, however, has a clearer behavioral and temporal emphasis, since respondents are asked to indicate behavior over the past week in response to items such as "I felt down and unhappy this week" and "I felt lonely, like I didn't have any friends." Finally, while the CES-D is used as a measure of clinical depression (Radloff, 1977, this volume; Weissman *et al.*, 1987), the Depressed-Withdrawal Scale is a subscale of a larger measure designed to assess general psychopathology (Achenbach and Edelbrock, 1983).

While prior hormonal level was associated with reports of depressive affect on the CES-D, pubertal status was not. This finding is consistent with other work from our research program (Baydar *et al.*, 1989; Brooks-Gunn and Warren, 1989) as well as the work of others (Susman *et al.*, 1987). However, contrary to the work of others (Baydar *et al.*, 1989; Petersen, 1988), pubertal timing was not associated with depressive affect. Since these other studies did not examine hormonal factors, however, it may be that our hormonal effect supercedes any possible effects of pubertal timing.

As was expected, prior reports of depressed-withdrawal affect were highly associated with current depressed-withdrawal affect. No interactions were reported between prior depression and estradiol category. Thus, prior depressed withdrawal was associated with later depressed-withdrawal while prior estradiol level was associated with the CES-D.

Finally, while daughters' reports of depressive affect on the CES-D were associated with hormonal status, mothers' reports of their daughters' depressive affect (using the PI-DMI) were not associated with the hormonal or physical status of their daughters. Indeed, mother reports of depressive affect were only moderately associated with daughter reports (r = .26 and .18 for the Depressed-Withdrawal Scale and the CES-D Scale, respectively). While it is possible that the PI-DMI, a third measure of depressive affect, taps a

different dimension of the construct of negative affect than either of the other two measures, similar discrepancies between mother and daughter responses on the CES-D have been reported in other samples (Paikoff *et al.*, 1989; Weissman *et al.*, 1987). It seems likely that differences in reports of mothers and daughters, rather than in the measures used, account for the variation in these associations. It makes sense, in fact, that daughter rather than mother reports of depressive affect would be associated with daughter's hormonal status, since mothers would not have access to as much knowledge about daughter's depressive affect nor to any internal physiological states possibly associated with hormonal levels.

Aggressive Affect

As in concurrent findings (Brooks-Gunn and Warren, 1989), a curvilinear association was found between hormonal levels and aggressive affect one year later. Unlike our prior study, however, no association was found between DHEAS level at Time 1 and reports of aggressive affect at Time 2. These findings support our initial hypothesis concerning persistent hormonal effects where estradiol is concerned, but not when DHEAS is the focus. As in other work, pubertal and menarcheal status were not related to reports of aggressive behavior. Prior reports of aggressive affect accounted for a much greater portion of the variance (35%) than prior hormonal status (4%). While hormonal status was still associated with aggressive affect when prior reports were entered into the equation, the association was somewhat dampened. The interaction between hormonal status and prior reports, however, was not significant.

Delinquent Behavior

Delinquent behavior was associated with hormonal status in our initial regression equations; however, when pubertal status was entered into the equation, or when prior reports were entered into the same regression equations, no significant associations with hormones were found. This finding was not anticipated, given others' report that pubertal timing, rather than status, is associated with delinquent behavior in girls (Magnusson *et al.*, 1985). It suggests, however, as does the timing literature, that effects of physiological processes (such as hormonal changes) are mediated through secondary sexual characteristic development in associations with delinquent behavior for young girls.

CONCLUSION

The current study demonstrates that associations between hormonal levels and affective expression are likely to persist, at least over the course of one year. Several limitations, however, affect the generalizability of these findings. First, although longitudinal data on the psychological measures were analyzed, longitudinal endocrine data on the current sample are not yet available. Second, only one endocrine assessment was conducted at Time 1. Most research groups using blood assays report only slight variability in different hormone assays over several hours or weeks (Olweus, 1980, 1988; Udry, 1987, 1988), providing partial justification for the methods used in this study. However, it is possible that single assays obscure day-to-day variations in hormonal levels that may be occurring and may influence affective expression. In one study using salivary rather than blood assays, day-to-day variability was reported (Eccles et al., 1988); however, Udry and Halpern (1990) recently reported that salivary measurements may be less reliable, and hence more variable, than blood measurements. If anything, using a single point in time assay may make it less rather than more likely that associations would be found.

A final issue concerns the study sample. The current study participants were a nonrandom sample of upper middle-class girls from a major metropolitan area. Other hormone studies are similar in their inclusion of middle- to upper middle-class subjects (Eccles *et al.*, 1988; Susman *et al.*, 1987). While the means of our psychological and endocrine measures look similar to those of other upper middle-class samples (Brooks-Gunn *et al.*, 1985; Petersen *et al.*, 1984; Radloff, 1977, this volume) as well as some more heterogenous samples (Achenbach and Edelbrock, 1978), no other studies have examined the particular associations between measures that have been examined in this study. Thus, the associations reported may be particular to females within a particular social class or geographical area.

Hormonal levels (most frequently estradiol hormonal categories taking into account estradiol, LH, and FSH) were associated with adolescent reports of depressive and aggressive affect. Delinquent behavior was not associated with hormonal levels when pubertal status was entered into the equation. Pubertal timing was not associated with depressive and aggressive affect, nor with deliquent behavior. Initial and current pubertal status was not an important factor in association with depressive or aggressive affect. Prior reports of depressive and aggressive affect and delinquent behavior accounted for more variance than prior hormonal status. In sum, our findings suggest that individual trajectories for development of negative affect and delinquent behavior may be influenced by pubertal hormonal processes, as well as by prior experience within these domains, and that these trajectories may operate somewhat independently from physical development changes. Future research should help delineate the consequences of these associations for the development of negative affect, delinquent behavior, and associated problems through adolescence.

ACKNOWLEDGMENTS

The research reported in this paper was funded by the National Institutes of Health and the W. T. Grant Foundation; we thank them for their support. We also wish to acknowledge those who contributed their time and energy to the Adolescent Study Program: J. Gargiulo, D. Friedman, L. Ferrington, and M. Samelson for collecting the data; L. Yen and E. Ewen for conducting the assays; J. Rosso for help with data analyses; A. Lechowicz, R. Deibler, and F. Kelly for manuscript preparation. The thoughtful comments of Christy M. Buchanan and Anne C. Petersen on an earlier draft of the manuscript are greatly appreciated. The schools and the families who participated in the research are, of course, to be thanked most of all. The first author was a Postdoctoral Fellow at Educational Testing Service while write-up of this article was completed; the second author was a Visiting Scholar at the Russell Sage Foundation. We appreciate these institutions' support.

REFERENCES

- Achenbach, T. M., and Edelbrock, C. S. (1978). The classification of child psychopathology: A review and analysis of empirical effects. *Psychol. Bull.* 85: 1275-1301.
- Achenbach, T. M., and Edelbrock, C. S. (1981). Behavioral problems and competencies reported by the parents of normal and disturbed children aged 4 through 16. *Monogr. Soc. Res. Child Develop.* 46(1) (Serial No. 188).
- Achenbach, T. M., and Edelbrock, C. (1983). Manual for the Child Behavior Checklist and Revised Child Behavior Profile. Queen City Printers, Burlington, VT.
- Achenbach, T. M., and Edelbrock, C. S. (1987). Manual for the Youth Self-Report Checklist and Profile. Queen City Printers, Burlington, VT.
- Apter, D. (1980). Serum steroids and pituitary hormone in female puberty: A partly longitudinal study. Clin. Endocrinol. 12: 107-120.
- Apter, D., and Vihko, R. (1977). Serum pregnenolone, progesterone, 17-hydroxy-progesterone, testosterone and 5a-dihydrotestosterone during female puberty. J. Clin. Endocrininol. Metab. 45: 1039-1048.
- Baydar, N., Brooks-Gunn, J., and Warren, M. P. (1989). Determinants of depressive symptoms in adolescent girls: A four-year longitudinal study. Unpublished manuscript, The Educational Testing Service, Princeton, NJ.
- Beach, F. A. (1975). Behavioral endocrinology: An emerging discipline. Am. Scient. 63: 178-187.
- Boyle, M. H., and Jones, C. S. (1985). Selecting measures of emotional and behavioral disorders of childhood for use in general populations. J. Child Psychol. Psychiatr. 20: 137-159.
- Brooks-Gunn, J., Petersen, A. C., and Eichorn, D. (1985). The study of maturational timing effects in adolescence. J. Youth Adolesc. 14: 149-161.
- Brooks-Gunn, J., Rock, D., and Warren, M. P. (1989). Comparability of constructs across the adolescent years. *Develop. Psychol.* 25: 51-60.
- Brooks-Gunn, J., and Warren, M. P. (1989). Biological contributions to affective expression in young adolescent girls. *Child Develop.* 60: 372-385.

- Brooks-Gunn, J., Warren, M. P., Rosso, J., and Gargiulo, J. (1987). Validity of self-report measures of girls' pubertal status. *Child Develop.* 58: 829-841.
- Buchanan, C. M., Eccles, J. S., and Becker, J. B. (1990). Are adolescents the victims of raging hormones?: Evidence for activational effects of hormones on moods and behavior at adolescence. Unpublished manuscript, Stanford University, Stanford, CA.
- Cairns, R. B., Cairns, B. D., Neckerman, H. J., Ferguson, L. L., and Gariepy, J. L. (1989). Growth and aggression: 1. Childhood to early adolescence. *Develop. Psychol.* 25: 320-330.
- Coe, C. L., Kayashi, K. T., and Levine, S. (1988). Hormones and behavior at puberty. Activation or concatenation? In Gunnar, M., and Collins, W. A. (eds.), *Minnesota Symposium on Child Development, Vol. 21*. Erlbaum, Hillsdale, NJ.
- Csikszentmihalyi, M., and Larson, R. (1984). Being Adolescent: Conflict and Growth in the Teenage Years. Basic Books, New York.
- Damon, A., Damon, S. T., Reed, R. B., and Valadian, I. (1969). Age at menarche of mothers and daughters with a note on accuracy of recall. *Human Biol.* 41: 161-175.
- Eccles, J. S., Miller, C., Tucker, M. L., Becker, J., Schramm, W., Midgley, R., Holmes, W., Pasch, L., and Miller, M. (1988, March). Hormones and affect at early adolescence. Paper presented at the biannual meeting of the Society for Research on Adolescence, Alexandria, VA.
- Eichorn, D. H. (1980). The school. In Johnson, M. (ed.), Toward Adolescence: The Middle School Years. Seventy-ninth Yearbook of the National Society for the Study of Education, Part I. The University of Chicago Press, Chicago.
- Faiman, C., and Winter, J. S. (1974). Gonadotropins and sex hormone patterns in puberty: Clinical data. In Grumbach, M. M., Grave, G. D., and Mayer, F. E. (eds.), *Control* of the Onset of Puberty, John Wiley & Sons, New York.
- Freud, A. (1948). The Ego and the Mechanisms of Defense. International Universities Press, New York.
- Freud, S. (1905/1953). A General Introduction to Psychoanalysis (Riviere, J., trans.). Permabooks, New York.
- Gargiulo, J., Attie, I., Brooks-Gunn, J., and Warren, M. P. (1987). Girls' dating behavior as a function of social context and maturation. *Develop. Psychol.* 23: 730-737.
- Grumbach, M. M. (1975). Onset of puberty. In Berenberg, S. R. (ed.), Puberty, Biologic and Social Components. H. E. Stenfert Kroese, BV, Leiden.
- Hall, G. R. (1904). Adolescence: Its Psychology and Its Relations to Psychology, Anthropology, Sociology, Sex, Crime, Religion and Education. Prentice-Hall, Englewood Cliffs, NJ.
- Hollingshead, A. B., and Redlich, F. C. (1958). Social Class and Mental Illness: A Community Study. John Wiley & Sons, New York.
- Inoff-Germain, G., Arnold, G. S., Nottlemann, E. D., Susman, E. J., Cutler, G. B., and Chrousos, G. P. (1988). Relations between hormone levels and observational measures of aggressive behavior of young adolescents in family interactions. *Develop. Psychol.* 24: 129-139.
- Kandel, D. B. (1983). Psychiatric Institute Depressive Mood Inventory (PI-DMI). Columbia University, New York.
- Kandel, D. B., and Davies, M. (1986). Adult sequelae of adolescent depressive symptoms. Arch. Gen. Psychiatr. 43: 225-262.
- Koff, E., Rierdan, J., and Jacobson, S. (1981). The personal and interpersonal significance of menarche. J. Am. Acad. Child Psychiatr. 20: 148-158.
- Larson, R., and Lampman-Petraitis, C. (1989). Daily emotional states as reported by children and adolescents. *Child Develop.* 60: 1250-1260.
- Magnusson, D., Stattin, H., and Allen, V. L. (1985). Biological maturation and social development: A longitudinal study of some adjustment processes from midadolescence to adulthood. J. Youth Adolesc. 14: 267-283.
- Marshall, W. A., and Tanner, J. M. (1969). Variations in pattern of pubertal changes in girls. Arch. Dis. Child. 44: 291-303.
- Nottlemann, E. D., Susman, E. J., Blue, J. H., Inoff-Germain, G., Dorn, L. D., Loriaux, D. L., Cutler, G. B., Jr., and Chrousos, G. P. (1987). Gonadal and adrenal hormone correlates of adjustment in early adolescence. In Lerner, R. M., and Foch, T. T. (eds.), *Biological-Psychosocial Interactions in Early Adolescence*. Erlbaum, Hillsdale, NJ.
- Olweus, D., Mattsson, A., Schalling, D., and Low, H. (1980). Testosterone, aggression, physical, and personality dimensions in normal adolescent males. *Psychosom. Med.* 42: 253-269.

- Olweus, D., Mattsson, A., Schalling, D., and Low, H. (1988). Circulating testosterone levels and aggression in adolescent males: A casual analysis. *Psychosom. Med.* 3: 261-272.
- Offer, D., Ostrov, E., and Howard, K. I. (1982). The Offer Self-Image Questionnaire for Adolescents: A manual (3rd ed.). Michael Reese Hospital, Chicago.
- Paikoff, R. L., and Brooks-Gunn, J. (1990). Physiological processes: What role do they play during the transition to adolescence? In Montemayor, R., Adams, G., and Gullotta, T. (eds.), Advances in Adolescent Development, Vol. 2: The Transition from Childhood to Adolescence. Sage Publications, Newbury Park, CA.
- Paikoff, R. L., and Brooks-Gunn, J. (in press). Do parent-child relationships change during puberty? Psychol. Bull.
- Paikoff, R. L., Carlton-Ford, S., and Brooks-Gunn, J. (1989, August). Variation in motherdaughter agreement and psychological adjustment in adolescent females. Paper presented in a symposium entitled, "Sociology of Childhood and Youth: A Research Agenda on Adolescent Issues," at the American Sociological Association meetings, San Francisco, CA.
- Parke, R. D., and Slaby, R. G. (1983). The development of aggression. In P. H. Mussen, P. H., and Hetherington, E. M. (eds.), *The Handbook of Child Psychology: Socialization, Personality, and Social Development*. John Wiley & Sons, New York.
- Parker, L. N., Levin, E. R., and Lifrak, E. T. (1985). Evidence for adrenocortical adaptation to severe illness. J. Clin. Endocrinol. Metab. 60: 947-952.
- Petersen, A. C. (1988, November). Coping with adolescence: It's depressing! Keynote address at an interdisciplinary research conference "Adolescent Stress, Social Relationships, and Mental Health," sponsored by the University of Massachusetts at Boston and the William T. Grant Foundation, Boston, MA.
- Petersen, A. C., Schulenberg, J. E., Abramowitz, R. H., Offer, D., and Jarcho, H. D. (1984). A self-image questionnaire for young adolescents (SIQYA): Reliability and validity studies. J. Youth Adolesc. 13: 93-111.
- Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. Appl. Psychol. Measurem. 1: 385-401.
- Reynolds, E. L., and Wines, J. V. (1948). Individual differences in physical changes associated with adolescence in girls. Am. J. Dis. Child. 75: 329-350.
- Root, A. W. (1973). Endocrinology of puberty. Am. J. Pediatr. 17: 35-56.
- Rutter, M., Graham, P., Chadwick, O. F., and Yule, W. (1976). Adolescent turmoil: Fact or fiction. J. Child Psychol. Psychiatr. 17: 35-56.
- Rutter, M., Izard, C. E., and Read, P. B. (eds.) (1986). Depression in Young People: Developmental and Clinical Perspectives. The Guilford Press, New York.
- Sassenrath, E. N. (1970). Increased adrenal responsiveness related to social stress in Rhesus monkeys. *Hormones Behav.* 1: 283-298.
- Simmons, R. G., and Blyth, D. A. (1987). Moving into Adolescence: The Impact of Pubertal Change and School Context. Adline De Gruyter, New York.
- Simmons, R. G., Rosenberg, F., and Rosenberg, M. (1973). Disturbance in the self-image at adolescence. Am. Sociol. Rev. 38: 553-568.
- Susman, E. J., Inoff-Germain, G., Nottlemann, E. D., Loriaux, D. L., Cutler, G. B., and Chrousos, G. P. (1987). Hormones, emotional dispositions, and aggressive attributes in young adolescents. *Child Develop.* 58: 1114-1134.
- Tanner, J. M. (1962). Growth at Adolescence (2nd ed.). Lippincott, New York.
- Tieger, I. (1980). On the biological basis of sex differences in aggression. *Child Develop.* 5: 943-963.
- Udry, J. R. (1987). Biosocial models of adolescent problem behaviors. Unpublished manuscript.
- Udry, J. R. (1988). Biological predispositions and social control in adolescent sexual behaviors. Am. Sociol. Rev. 53: 709-722.
- Udry, J. R., Billy, J. O. G., Morris, N. M., Groff, T. R., and Raj, M. H. (1985). Serum androgenic hormones motivate sexual behavior in adolescent boys. *Fertil. and Steril.* 43: 90-94.
- Udry, J. R., and Halpern, C. (1990, March). Validity of adolescent hormone measures for behavior research. paper presented at the 3rd biennial meetings of The Society for Research on Adolescence, Atlanta, GA.
- Warren, M. P. (1980). The effects of exercise on pubertal progression and reproductive function in girls. J. Clin. Endocrinol. Metab. 51: 1150-1157.

Hormones and Girls' Affective Expression

- Warren, M. P., and Brooks-Gunn, J. (1989). Mood and behavior at adolescence: Evidence for hormonal factors. J. Clin. Endocrinol. Metabol. 69: 77-83.
- Warren, M. P., Siris, E., alnd Petrovich, C. (1977). The influence of severe illness on gonadotropin secretion in the post menopausal female. J. Endocrinol. Metabol. 45: 99-104.
- Weissman, M. M., Wickramaratne, P., Warner, V., John, K., Prusoff, B. A., Merikangas, K. R., and Gammon, G. D. (1987). Assessing psychiatric disorders in children: Discrepancies between mothers' and children's reports. Arch. Gen. Psychiatr. 44: 747-753.
- Zumoff, B., Walsh, B. T., Katz, J. L., Levin, J., Rosenfeld, B. S., Kream, J., and Weiner, H. (1983). Subnormal plasma dehydroisoandrosterone to cortisal ratio in anorexia nervosa: A secondary hormonal parameter of ontogenic regression. J. Clin. Endocrinol. Metabol. 56: 668-670.