Testosterone, conflict style, and depression in late adolescent couples: a dyadic predictive model.

Heidemarie Kaiser

University of Massachusetts Amherst

Follow this and additional works at: https://scholarworks.umass.edu/theses

Retrieved from https://scholarworks.umass.edu/theses/2409
TESTOSTERONE, CONFLICT STYLE, AND DEPRESSION IN LATE ADOLESCENT COUPLES: A DYADIC PREDICTIVE MODEL

A Thesis Presented

by

HEIDEMARIE KAISER

Approved as to style and content by:

Sally Powers, Chair

Jerrold Meyer, Member

Maureen Perry-Jenkins, Member

Melinda Novak, Department Head
Psychology
ABSTRACT

TESTOSTERONE, CONFLICT STYLE, AND DEPRESSION IN LATE ADOLESCENT COUPLES: A DYADIC PREDICTIVE MODEL

September 2003

HEIDEMARIE KAISER, B.A., SCRIPPS COLLEGE
M.S., UNIVERSITY OF MASSACHUSETTS AMHERST

Directed by: Professor Sally Powers

The current study examined predictive relationships among testosterone levels, conflict tactics, and depression in 18-20-year-old heterosexual couples. It was hypothesized that aggression within the couple would mediate a relationship between testosterone (T) and depression. The first link of the model (T to aggression) was expected to be stronger for males, whereas the second link (aggression to depression) was thought to be stronger for females. Data were analyzed using hierarchical linear modeling to take into account the interdependency of his and her scores within the couple. Results failed to support the mediational model since testosterone did not relate significantly to depression, but the other pieces of the model yielded findings that largely supported hypotheses. The interaction of his and her T levels predicted the male’s frequency of physical assault within the relationship; when both he and she were concordant for higher or lower levels of T for their gender group, he was more physically aggressive than if they had complementary levels. For females, the psychological aggression of both parties (higher levels on her part and lower levels on his part), as well as greater physical assault by her partner, predicted her depression.
LIST OF TABLES

Table                                                                 Page
1. Means and standard deviations for couple variables..........................40
2. Regression coefficient estimation for predicting CESD depression ..........40
3. Variance component estimation for predicting CESD depression .............40
4. Regression coefficient estimation for predicting frequency of physical assault, poisson model with over-dispersion (population-average model).................................................................41
5. Variance component estimation for predicting frequency of physical assault, poisson model with over-dispersion (population-average model).................................................................41
### LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Physical assault by female testosterone, high and low values of male testosterone (unit-specific model)</td>
<td>42</td>
</tr>
<tr>
<td>2. Physical assault by male testosterone, high and low values of female testosterone (unit-specific model)</td>
<td>43</td>
</tr>
<tr>
<td>3. Physical assault female testosterone, high and low values of male testosterone (population-average model)</td>
<td>44</td>
</tr>
<tr>
<td>4. Physical assault by male testosterone, high and low values of female testosterone (population-average model)</td>
<td>45</td>
</tr>
</tbody>
</table>
CHAPTER 1  
INTRODUCTION

Late adolescence, as the period of transition from childhood dependence to an adult identity and responsibility, is an important time of life. It is also the period at which both the prevalence and the gender difference in depression greatly increases (Hankin, Abramson, Moffitt, Silva, McGree, & Angell, 1998) and so could be a critical time to examine the mechanisms leading to depression in both males and females. Adolescent depression is also an important topic of study in its own right; not only does it cause problems in functioning at the time, it also predicts negative outcomes in later life, including adult depression (Kandel & Davies, 1986), and even subclinical levels predict psychosocial problems and psychopathology in the future (Gotlib, Lewinsohn, & Seeley, 1995).

The literature on depression supports both a biological and a psychosocial basis for the disorder, but much work remains to link these lines of research. Hormone levels appear to be important in affective states, as well as in personality and behavior, through both direct and indirect effects. The mechanism for hormonal action may be organizational, meaning that prenatal exposure influences the formation of the central nervous system and subsequent sensitivity to hormones, and/or activational, which refers to more immediate effects (i.e., through activity at brain receptors). Both the stress hormone cortisol and the female sex steroid estradiol have been linked to depression, but there is less definitive research about the possible role of the male sex steroid testosterone. The current investigation follows up on indications of direct and indirect relationships between testosterone and depression in adolescents to help clarify
the biopsychosocial nature of the disorder and, ultimately, to suggest possible gender-specific points of intervention.

**Hormonal Changes at Adolescence**

Before and during adolescence, components of the hormonal system in both males and females are activated, reaching maturity by late adolescence (Panter-Brick & Worthman, 1999). First, a pre-pubertal rise in adrenal steroids (such as androstenedione) signals increased activity of the hypothalamic-pituitary-adrenal (HPA) axis. At puberty, the hypothalamic-pituitary-gonadal (HPG) axis becomes more active with increased gonadotropin (luteinizing hormone and follicle-stimulating hormone) release, which in turn increases secretion of the sex steroids (estradiol and testosterone). In girls, estradiol (E) increases from age 9 or 10 to 13 or 14 and begins cycling with progesterone, which leads to menarche. Because estradiol is derived from testosterone metabolism, testosterone also increases, but to a lesser extent. In boys, testosterone (T) is the primary gonadal hormone, and it increases between the ages of 10 and 17, with the sharpest rise from 12-14 years. Again, because of the metabolic link between testosterone and estradiol, E also increases, but the T/E ratio is greater in males and increases with age. Another interesting difference is that females have higher levels of sex hormone binding globulin from early childhood until late adulthood, along with lesser absolute amounts of gonadal output, so males experience greater amounts and variation in circulating free gonadal steroid.

During early adolescence, then, the hormonal system is in flux, and it takes time for hormone levels to stabilize and for the body to adapt to these new levels. This may explain why many of the hormone-related studies on younger adolescents yield
contradictory findings, both when compared to adult studies and to other adolescent studies. Another value of the present study on older adolescents is thus to explore how their pattern of hormone-behavior interaction fits on the developmental spectrum from puberty to adulthood.

**Testosterone and Mood**

Although testosterone is not known as a “mood hormone,” it has been related to human affective states, particularly in the work of Susman and her colleagues in a series of studies on young adolescents between the ages of 9 and 14 (Nottelmann et al., 1987; Susman, Dorn, & Chrousos, 1991; Susman et al., 1985). Among boys, controlling for pubertal stage and age, lower levels of T and higher levels of andrenostenedione predicted negative affect. Furthermore, these hormone levels predicted negative affect one year later. The authors suggested that this pattern makes sense because it reflects a state of stress; in the stress response, the HPA axis is activated, which involves adrenal androgen (such as androstenedione) secretion, while the HPG axis is suppressed, which means inhibition of sex steroid release (such as testosterone). In this way, low testosterone level in boys could be associated with depressive symptoms as a stress marker. Another point is that this hormone profile was associated with lower pubertal stage and higher chronological age, and these later-maturing boys were found to have poorer psychosocial functioning, which could generate stress.

For the girls studied, on the other hand, negative affect was predicted by higher T (as well as by high cortisol). This may mean that T works differently for females and/or that its association with stress differs. At the same time, the relationships were in general weaker than for males, and the authors speculated that because boys are
exposed to higher androgen levels prenatally, their brains might be organized to respond more sensitively to those hormones, whereas girls are more sensitive to environmental influences. Exposure to higher levels of testosterone at puberty might also influence subsequent sensitivity to the hormone’s effects; a comparison of hippocampal slices of male and female rat brains showed that gonadally intact males had the highest androgen receptor immunoreactivity, but castrated males had the lowest, and treatment with testosterone propionate increased immunoreactivity in both sexes (Xiao & Jordan, 2002). In support of the importance of T for male mood, a randomized, placebo-controlled study of testosterone gel therapy for men with refractory depression and low T levels found that subjects receiving the extra T showed significantly greater improvement on depression scales (Pope, Cohane, Kanayama, Siegel, & Hudson, 2003).

At the same time, other researchers (Baucom, Besch, & Callahan, 1985) found that women low in testosterone experience more anxious and dejected mood, which would argue for a single inverse relationship between T level and depression across genders, as found by Daitzmann and Zuckerman (1980). The specific actions of neuroactive steroids, including testosterone, are of growing interest in neuropsychopharmacology, and it appears that they can affect anxiety- and depression-related symptoms through modulation of GABA_A receptors, as well as through antagonism of certain serotonin receptors (5-HT3) and glutamate receptors (Rupprecht, 2003). Furthermore, a disequilibrium of certain 3α-reduced neuroactive steroids is found during major depression, which is corrected by successful treatment with antidepressant drugs, suggesting a non-gender-specific interplay between this class of
hormones and mood symptoms. Again, the current study will touch on this issue by exploring testosterone-depression relationships separately in males and females.

**Testosterone and Personality**

Testosterone (like estrogen) has an activating effect, and its absence has been found to induce behavioral despair in rats (Bernardi, Genedani, Tagliavini, & Bertolini, 1989). In humans, similarly, T is associated with a more active personality style; high behavioral activation was associated with higher levels of testosterone in male military veterans (Windle, 1994). Udry and Talbert (1988) distilled a personality factor highly correlated with T in both males and females, and it included such traits as outgoing, extraverted, and dominant. This followed on the finding that more “masculine” women who perceived themselves as enterprising, resourceful, and action-oriented, were higher in testosterone, whereas women who viewed themselves as conventional and “feminine” were lower in testosterone (Baueom et al., 1985). In the same line, testosterone has been positively correlated with self-image in both boys (Nottelmann et al., 1987) and girls (Cashdan, 1995) and negatively correlated with neuroticism and pessimism (Dabbs, Hopper, & Jurkovic, 1990). Together, these studies suggest that higher testosterone relates to a positive, depression-resistant personality structure that opposes the passive, unassertive style of behavioral coping found to predict depression (Allgood-Merten, Lewinsohn, & Hops, 1990). However, these data are all correlational, so no causal statements can be made, and testosterone’s activational nature can also lead to negative consequences, as in aggression.
Testosterone and Aggression

Animal studies have tended to show an association between testosterone and aggressive behavior, both by decreased aggression in castrated animals and increased aggression in hormone-treated animals (Beatty, 1979). More specifically, T implants in the medial hypothalamus have been found to increase attack behavior in castrated rats (Albert, Dyson, & Walsh, 1987), and aggression appears to have to do with a more complex interaction of low serotonin in the hypothalamus and amygdala and testosterone action at these sites (Bernhardt, 1997). According to this model, T promotes dominance behavior, which puts the animal in situations where frustration can occur, and low serotonin then makes the animal hyperresponsive to the aversive stimuli and potentially aggressive. In line with this theory, CSF free T in nonhuman primates was found to correlate positively with overall aggressiveness, while CSF 5-hydroxyindoleacetic acid (a serotonin metabolite) was negatively correlated with impulsive behavior and severe, unrestrained aggression, but not with overall rates of aggression (Higley, Mehlman, Poland, & Taub, 1996). Another clue to the mechanisms of T action is provided by a series of experiments on the modulatory effect of T on vasopressin receptors in the hypothalamus. The researchers found that T treatment of castrated hamsters increased V-sub-1 vasopressin (AVP) receptor binding in the ventrolateral hypothalamus, that AVP injections to that area accelerated the onset of aggression in T-treated animals, and that these injections failed to activate offensive aggression in non-T-treated animals (Delville, Mansour, & Ferris, 1996).

Even in animals, though, aggressive behavior has been shown to depend on social context and not on any one hormone level alone. The aggression-facilitating
effects of T have generally been found in a situation of social mixing and the establishment of dominance relationships (Ruiz-de-la-Torre & Manteca, 1999) or physical provocation (McGinnis, Lumia, Breuer, & Possidente, 2002). In fact, repeated exposure to unfamiliar males as well as cohabitation with a female activated testosterone-dependent social aggression in castrated rats (Albert, Dyson, Walsh, & Petrovic, 1988). Aggressive behavior activation may also depend on early hormonal exposure; initial exposure to elevated T levels was found to increase sensitivity to brief elevations in plasma T levels induced by social challenge in black-headed gulls (Ros, Dieleman, & Groothuis, 2002). Finally, it may be that different types of aggression depend not only on testosterone, but also on relative levels of other hormones; naturalistic studies of free-ranging rhesus monkeys found higher basal cortisol levels in defensive aggression, while offensive aggression related to both higher T levels and lower cortisol levels (Kalin, 1999).

In adolescent males, there does seem to be a relationship between testosterone and aggression, but a path analytic study only found a direct relationship for provoked aggression (Olweus, Mattsson, Schalling, & Löw, 1986). For unprovoked aggression, there seemed to be an indirect relationship mediated by lower frustration tolerance. Thus, it may take a frustrating event, such as an interpersonal conflict, to elicit testosterone-related aggression. In adolescence, body mass may also play a role; in a sample of boys followed from 6 to 13 years, T level and body mass were found to additively predict social dominance, whereas only body mass predicted physical aggression (Tremblay et al., 1998). The researchers interpreted their findings to mean that the association between T level and physical aggression is probably observed in
contexts where physical aggression lead to social dominance. Also, testosterone may not play a decisive role in all forms of aggression; although Christiansen and Knussmann (1987) found that T correlated with spontaneous aggression in males, there was no relationship with sexual aggression.

Again, the relationship may differ for women. One study found a negative correlation between T and aggression in a non-patient female sample (Gladue, 1991), and the authors suggested that in women behavior is more socially moderated. Of course, the measures of aggression used in most of these studies may be biased toward masculine manifestations of the construct. At the same time, in a sample of female outpatients, the violent aggressive group had higher T than did the nonaggressive group (Ehlers, Richler, & Hovey, 1980), so it may be the behavioral inhibition of “normal” women in society that makes the T-aggression relationship seem to differ from that found in men. Also, relationships for women may vary by menstrual cycle phase; one study found a relationship between T and aggressive responses only during the midfollicular phase (Dougherty, Brok, Moeller, & Swann, 1997). This may be why another study of healthy premenopausal women found a significant correlation between free T measured at the mid-cycle phase of the menstrual cycle and physical and verbal aggression on a self-report questionnaire (von der Pahlen, Lindman, Sarkola, Mackisalo, & Eriksson, 2002). Other factors, such as type of androgen being measured and total vs. free hormone, might complicate the hormone-aggression relationship in women; Cashdan (2003) found that women’s expression of competitive aggression correlated positively with androstenedione and total testosterone (but not free T), and failing to express overt aggression correlated negatively with both hormones. However,
when the hormone values were converted to ranks to reduce the influence of
participants with extreme values, the relationship with testosterone became
nonsignificant, while androstenedione remained significant, leading her to suggest that
the latter hormone is more important for female aggression.

Aggression relates to depression in several ways. Within a single individual,
aggressive or antisocial behavior has been associated with depression as both a
concomitant factor (Rohde, Lewinsohn, & Seeley, 1991) and as a predictor of future
difficulties (Capaldi, 1992). In fact, a mixed temperament subtype (high in both
behavioral activation and inhibition) characterized by higher levels of T, aggression,
and hostility, was found to be associated with higher levels of both internalizing and
externalizing lifetime psychiatric disorders (Windle, 1994). In an interpersonal context,
aggressive behavior and conflict predict negative consequences for all parties involved,
and this paper will explore both direct and interactional pathways of an aggression-
depression relationship in a romantic relationship.

Testosterone and Aggression in Relationships

Even in youth, testosterone relates to conflictual interaction; in adolescent boys,
high T relates to more parent-child arguments and lower frequency of calm discussion
(Steinberg, 1987). Such early aggressive patterns – specifically, aversive
communication in the family and antisocial behavior – in turn predict couple
dysfunction in adulthood (Andrews, Foster, Capaldi, & Hops, 2000). In adult
relationships, high testosterone in men relates to poorer quality marital relationships
(Julian & McKenry, 1989), with less interaction with spouse, more separations, and a
higher likelihood of divorce (Booth & Dabbs, 1993). As the preceding arguments
suggest, this relationship weakness may be due to the higher levels of verbal and physical aggression toward a female partner associated with high T levels (Soler, Vinayak, & Quadagno, 2000).

Research on testosterone in marital interaction (Cohan, Booth, & Granger, 2003) further reveals that it is not the absolute level of testosterone itself, but rather the level relative first to the average for one’s gender and then to one’s partner’s (relative) level, that makes a difference in adaptiveness of interactions. In this study, men showed less positive marital problem-solving behavior when they had higher T and their wives had lower T (for their gender) and more positive behavior when both were lower T. Similarly, men showed less negative behavior when both partners had higher T, suggesting that men interact best in a relationship when they and their wives have concordant T levels. Wives, on the other hand, provided more positive support when they had higher T and their husbands had lower T, suggesting that women do better when they have complementary levels, with theirs being relatively higher. How such findings might apply to adolescent couples is not certain, but this dyadic approach to hormone-behavior relationships informs the present study.

Regardless of the biological correlates, aggression in a relationship may be expressed differently by women than by men. In a study by Harncd (2001) of college-age dating couples, men and women reported comparable amounts of overall aggression from their partners, but women reported more sexual victimization, while men reported being the victims of more psychological aggression. Another study of dating aggression (Jenkins & Aube, 2002) found that men reported higher victimization levels for both physical and psychological aggression, and women reported greater
perpetration of symbolic aggression (defined in this study as a subset of psychological aggression, including verbal and nonverbal acts that symbolically hurt or threaten the other). The authors offered several explanations for this surprising finding of greater female aggression in relationships. On the one hand, male aggression might tend to be underreported because it is expected based on gender role norms, whereas female aggression is salient because it is not as expected based on these same norms. On the other hand, societal stereotypes that women are relatively harmless could work to make minor aggression more tolerated in women than in men, leading the latter to underreport their use of such tactics.

Whether one partner or the other actually exhibits more aggression, women appear to be more impacted by violence in relationships; in the Harned study, gender interacted with both psychological and physical abuse such that at lower levels of victimization, men and women reported comparable levels of anxiety, depression, and posttraumatic stress, but as the levels of aggression increased, women reported more severe outcomes. Based on this and other studies showing similar gender differences in effects of abuse (e.g., Follingstad, Wright, Lloyd, & Sebastian, 1991; Stets & Straus, 1990), aggressive interactions should relate more strongly to her adjustment than to his.

Relationship Interaction and Depression

Research on couples shows that conflictual interaction tends to predict depression for one or both partners. In a comparison of couples with an outpatient depressed wife and community controls, the former group reported greater marital distress and more destructive and less constructive conflict resolution tactics (Coyne, Thompson, & Palmer, 2002). In addition, the husbands of the depressed women were
significantly more psychologically distressed than those of control women, and psychological distress and destructive conflict tactics were correlated between husband and wife, highlighting the reciprocal nature of behavior and adjustment in the relationship. The direction of the relationship could go both ways (i.e., destructive conflict leads to depression and/or depression leads to destructive conflict), but at least one prospective study of never-depressed women found that while negative marital events (including physical aggression) and marital discord predicted subsequent depression, depressive symptoms did not predict later marital discord (Christian-Herman, O’Leary, & Avery-Leaf, 2001).

As mentioned earlier, the impact of such negative relationship variables may be greater for females than for males, following the belief that women are more relationship-oriented and men more focused on autonomy. Also, women may be more affected by negative relationships because they are less likely to be in positions of power either within or outside of relationships. Although marital discord was found to be strongly associated with depressive symptomatology in both men and women, when marital discord was controlled for, physical aggression by her partner and less assertiveness with her husband added significantly to predicting depression in women, while no relationship variables added to prediction in men (Christian, O’Leary, & Vivian, 1994). This study also suggests that the combination of an unassertive female and a dominating male in a conflictual relationship constitutes a risk factor for depression in the former.

Among early adolescents, interpersonal conflict has also been found to predict depression symptoms more strongly for girls than for boys (Jenkins, Goodness, &
Buhrmester, 2002), while conflict management self-efficacy was more important for boys; for the latter, it was not so much the amount of conflict that mattered as their sense of being able to deal with it. Girls also showed a much stronger negative relationship between conflict and intimate support, indicating a greater sense of loss in relationship resources when conflict occurs. If girls are socialized to base more of their self-esteem on maintaining relationships and they believe that these relationships must remain conflict-free to be successful, it would make sense that conflict and the subsequent feelings of failure would relate more strongly to depression for them.

Finally, while conflict may have a depressenogenic effect on females, depression may breed conflictual behavior in males, further complicating the picture. An analysis of the relationship between externalizing and internalizing behavior and depression in adolescents found that the former predicted depression in males, but not in females (Price & Lavercombe, 2000). This association between externalizing behavior and depression in males appears to emerge in adolescence; Weiss and Weisz (1988) found neither the externalizing-depression relationship nor the gender difference in a child sample, but they did in an adolescent sample. Thus, conflict and aggression could be expected relate to depression in both males and females, but the direction of causality seems to differ.

From the above literature, it appears that testosterone could relate to depression both on an individual and a dyadic basis through its association with certain personality and conflict styles. For males, higher levels of T may indicate an absence of stress and better social functioning, including concordance with their partners, which would lead to better adjustment. For females, the association may be more through the implications
of her and her partner’s levels for styles of coping with relationship conflict; the fit between her and her partner’s level might be translated into more or less adaptive conflict styles, which would in turn affect her level of depression. The current paper studies heterosexual late adolescent couples to try to connect these hormonal and behavioral factors in the development of depression in this age group. A mediational path model is tested, by which his and her testosterone levels predict conflict style, which in turn predict the mental health outcomes of each. In the current study, conflict style is characterized by aggression as a negative component and assertiveness as a positive component, and the predictive value of testosterone is thought to be moderated by one’s partner’s T. The model proposed looks as follows:
In an unrestricted population, it was expected that testosterone would predict depression in a curvilinear way, such that there is a certain optimal level for each gender, with extremely high or extremely low levels being maladaptive. However, extreme high or low values were not expected in this sample, so it would probably be generally more adaptive to have relatively higher levels of T. Both a linear and a quadratic model of T-adjustment relationships were tested to ascertain whether this would be the case.

Also, one must remember that testosterone, and the assertive personality style with which it is associated, exists within a social context. Gender roles dictate that women should be more yielding to others, whereas men are encouraged to be more aggressive, so higher testosterone was expected to predict aggressive conflict tactics more strongly for men than for women.

Finally, the testosterone level of one’s partner was expected to moderate the effects of one’s own T level. On the basis of the previous literature, the hypothesized outcomes of such dyadic interactions are:

As she increases and he decreases in T, she will be more assertive but not dominating, and the relationship will be characterized by constructive conflict tactics (less verbal and physical aggression), leading to less depression for both.

As he increases and she decreases in T, he will dominate the relationship with an aggressive style, and the relationship will be characterized by harmful conflict tactics (more aggression), leading to greater depression, especially for her.
CHAPTER 2

METHOD

Participants

Participants for this study are 90 heterosexual older adolescent couples from Sally Powers’ NIMH-funded study on adolescent depression. Ages range from 18 to 20, with a mean age of 19.3 years (SD = .87). Of the 180 participants, 9 (5%) identified as Asian American or Pacific Islander, 1 (.6%) as Native American, 2 (1.1%) as African American, 12 (6.7%) as Latino/a, 137 (76.1%) as European American, and 23 (12.8%) as “other” or gave no response. This ethnic distribution is similar to that of youth in the community, based on the 2000 census data statistics collected by the Massachusetts Institute for Social and Economic Research (6.1% Asian American or Native American, 3.3% African American, 3.9% Latino/a, and 86.7% European American).

Participants were recruited from five colleges and the surrounding community through posters and sign-up sheets in psychology classes with the following conditions: both partners had to be between the ages of 18 and 20, and they had to be in a romantic relationship for at least 2 months. Each participant was paid $20 for participating in a 3-hour session, and participants who were in introductory psychology classes had the additional option of receiving six research credits toward their final grade.

Procedure

All data used in the current study were obtained during the first of three data-collection sessions conducted for the larger NIMH study. For this first session, participants were asked to come to the lab with their romantic partners, and in the interests of obtaining accurate hormone measurements, they were instructed not to drink
alcohol, use illegal drugs, or visit the dentist within the 24 hours prior to the study, or to exercise, eat, drink (except water), smoke cigarettes, or brush their teeth up to 2 hours prior.

Participants were seated at computers on which they filled out computerized questionnaires, and a divider between them ensured that they could not see each other’s responses or discuss their answers. First, they filled out an Admissions Questionnaire containing questions about variables that could affect hormonal levels, such as the number of hours of sleep the previous night, daily medications or vitamins, the use of oral contraceptives, phase of menstrual cycle, and the possibility of pregnancy. If participants had an elevated temperature or felt ill, reported that they have used alcohol or illegal drugs or had any mouth or gum abrasion in the past 24 hours, or reported that they had brushed their teeth, eaten, drunk caffeinated beverages or exercised in the past two hours, they were scheduled to return at a later date. Participants also rinsed their mouths thoroughly with water 10 minutes before giving the first saliva sample to minimize the potential for contamination.

After completing the admissions questions, participants gave the saliva sample upon which testosterone measurements for the present study are based. Participants were instructed to passively drool down a straw into a small plastic vial with their heads tilted forward until the required amount of saliva is collected. The vial was then sealed and immediately placed in frozen storage (-20 degrees C) until shipment to Salimetrics, LLC (on dry ice) for analysis. This baseline measure preceded a conflict negotiation task, after which additional samples were taken, but they are not used in these analyses since T level is being treated as a trait, rather than a state, for each individual.
Following the conflict task, participants were seated at the computers again to fill out a series of questionnaires, including the measures used in this paper.

**Measures**

Testosterone Levels as Assessed by Saliva Samples

Because testosterone samples were obtained at the same time of day for each participant (sessions start uniformly at 4pm), fluctuations due to daily rhythms -- higher in the morning, lower in the afternoon -- should not introduce error variance. Seasonal fluctuations in T were tested and controlled for if found to be substantial. Also, blood contamination can falsely elevate salivary analyte levels because the levels of most analytes are higher in the circulation than in saliva. Therefore, samples were first tested for blood contamination by Salimetrics before assay for testosterone level (contaminated samples are not used for analyses). Saliva samples were assayed for Testosterone using an enzyme immunoassay (EIA) specifically designed for use with saliva according to the manufacturer's recommended protocol (Salimetrics, State College, PA). The assay has a range of sensitivity from 1.5 to 360 pg/mL, and average intra- and inter-assay coefficients of variation less than 10% and 15%, respectively. All saliva samples were assayed in duplicate, and the average of the duplicates were used in all analyses. Because the literature indicates that personality effects are associated with different testosterone increments in men and women (Udry & Talbert, 1988), making a comparison of absolute levels problematic, T was be expressed as a z score for the participant’s gender group to show where his/her level fit into the distribution of males or females.
Assertiveness Scale

This 8-item scale measures the Assertiveness facet of the NEO Extraversion factor (Costa & McCrae, 1992). Each item is rated from 0 to 4, with higher scores indicating more assertiveness. Assertiveness as measured by this scale has been found to predict life satisfaction, especially for men (for women, the Positive Emotion facet better predicted life satisfaction) (Herringer, 1998). Similarly, in an investigation of personality correlates of evaluative dimensions, Assertiveness was related to positive valence, as opposed to negative valence, which related to Depression (McCrae & Costa, 1995). Another finding relevant to this study is that a gender difference in Assertiveness favoring males was replicated across 26 different cultures, with the most pronounced gender differences in European and American cultures (Costa, Terracciano, & McCrae, 2001). In this sample, the reliability (alpha) of the scale was .69.

Revised Conflict Tactics Scale (CTS2)

This is a 39-item self-report measure that assesses the frequency on a scale of 0-6 of physical and verbal aggressive experiences of the respondent. The original Conflict Tactics Scale (CTS) was developed by Straus (1979) to clarify the nature of intrafamily conflict and violence by measuring the use of reasoning, verbal aggression, and violence within the family. In 1996, Straus, Hamby, Boney-McCoy, and Sugarman developed a revised scale (CTS2) to measure psychological and physical attacks on a partner, as well as the use of negotiation, in a marital, cohabiting, or dating relationship. Items were added to increase content validity and reliability, and wording was revised for better clarity and specificity. Also, new scales were added to tap sexual coercion and physical injury. Based on a sample of 317 college students, the authors found
sufficient evidence for construct validity, and internal consistency reliabilities for the five subscales ranged from .79 to .95.

Newton, Connelly, and Landsverk (2001) investigated the factor validity of the CTS2 based and found that a 5-factor model -- with the factors Negotiation, Minor Psychological Aggression, Severe Psychological Aggression, Minor Physical Assault, and Severe Physical Assault -- yielded a better fit than did the original 3-factor model (which did not differentiate levels of severity). In the current sample, "severe" items were endorsed so infrequently that prediction based on these subscales was impossible. Therefore, only the minor physical and psychological aggression subscales are used to assess participants' conflict tactics for these analyses. Based on this sample, the internal consistency (alpha) of the subscales used was .68 for Minor Psychological Aggression and .47 for Minor Physical Assault ($\alpha = .28$ for Severe Psychological Aggression and .38 for Severe Physical Assault). Also, because the focus in this study was on conflict factors thought to be related to testosterone, such as aggression and assertiveness, Negotiation was not used in analyses.

Center for Epidemiologic Studies Depression Scale (CES-D)

This 20-item questionnaire was developed by researchers at the Center for Epidemiologic Studies at NIMH (Radloff, 1977) to measure depressive symptoms among adults in community surveys. A total score for depressive symptoms is computed based on the sum of the frequency of occurrence rating of the 20 symptoms. The current sample yielded an alpha reliability of .82. The CESD has been found to discriminate significantly between subjects with and without major depression (Geisser, Roth, & Robinson, 1997), and it was found to have a specificity of 90% and sensitivity
of 86% in identifying depression in stroke patients (Parikh, Eden, Price, & Robinson, 1988). Higher CESD scores have been associated with certain psychosocial variables -- less perceived life control, less perceived accomplishment, higher derived identity, lower social support -- as well as certain demographic variables -- lower education, lower or unemployment, younger age, and lower family income (Warren & McEachren, 1983). Because demographics will be fairly constant in this sample, variance in CESD scores will probably depend more on the psychosocial variables, which in turn are assumed to relate to testosterone levels and/or conflict style.

Data Analytic Strategy

Although ordinary least-squares regression can be used to look at gender differences by estimating males’ and females’ outcomes separately (and this is used for initial exploration of the data in this study), this approach does not take into account the fact that the male’s and the female’s scores within a dyad are in some way dependent on each other. Hierarchical linear modeling, as described by Raudenbush and Bryk (1986), is better suited to exploring relationships with such dependent data. In an HLM model such as the one used here, the couple is approached as the unit of analysis, with a female outcome score and a male outcome score nested within the couple, and information about the association between the scores in the couple is used to reduce the standard error in testing regression coefficients. A further advantage to this technique is that it takes into account measurement error, splitting each outcome into a true score and an error term, allowing for clearer estimation of effects. HLM also allows simultaneous estimation of male and female outcomes based on different sets of
predictors, which is useful if the hypothesized explanatory variables are different for men and women.

The level 1 model presents each outcome (i.e., depression or conflict tactics) as a function of the male’s and the female’s true scores plus measurement error:

$$Y_{ij} = \beta_{1j} \text{ (female)} + \beta_{2j} \text{ (male)} + e_{ij}$$

where $Y_{ij}$ is the subscale score $i$ for couple $j$ on the outcome, with $i = 1, \ldots, 4$ (two subscales of the outcome for the woman, two for the man) and $j = 1, \ldots, 90$ couples. The variables “female” and “male” are dummy variables coded 1 or 0 to indicate which partner a particular score belongs to. Thus, $\beta_{1j}$ represents the true score for the woman in couple $j$, and $\beta_{2j}$ represents the true score for the man. The errors are represented by the $e$’s – $e_{1j}$ and $e_{2j}$ for the female’s scores, $e_{3j}$ and $e_{4j}$ for the male’s scores – and are assumed to have a constant variance, $\sigma^2$.

The level 2 model attempts to predict male and female true scores based on a set of explanatory variables that characterize the couple (or individual members of the couple) as follows:

$$\beta_{ij} = \gamma_{10} + \sum \gamma_{1q}W_{1qj} + u_{ij} \text{ for females and}$$

$$\beta_{2j} = \gamma_{20} + \sum \gamma_{2q}W_{2qj} + u_{2j} \text{ for males}$$

where $\gamma_{10}$ and $\gamma_{20}$ are the intercepts (averages) for females and males, $W_{1qj}$ and $W_{2qj}$ are the $q$ th predictors for each, and residuals $u_{ij}$ and $u_{2j}$ are assumed to be normally distributed across couples with variances $\tau_{11}$ and $\tau_{22}$, respectively, and covariance $\tau_{12}$. 
CHAPTER 3
RESULTS

Descriptive Characteristics of the Sample

As shown in Table 1, males and females in the sample showed quite similar means on depression and conflict style factors addressed in this study. As seen in the table, the occurrence of severe psychological and physical aggression in this sample was quite rare, with standard errors higher than the means, so using the minor scales only for analyses continued to make sense. Women did not differ significantly from men on levels of psychological aggression, $t(168) = -1.11, p = .27$, or physical assault, $t(168) = .78, p = .44$. Similarly, the assertiveness and depression scores did not show a significant gender difference, $t(176) = -.66, p = .51$ and $t(178) = -.32, p = .75$, respectively, with most scores falling in the moderate to high functioning range for these scales. As would be expected, males showed a significantly higher average testosterone level than females, $t(167) = 10.48, p < .001$, although there was some overlap between the distributions. For each gender group, there was one extreme outlier — a male with avg. T of 970.37 and a female with avg. T of 211.11 — so analyses were done both with and without those cases to make sure that they did not substantially alter the results (they did not). Without these outliers, the range of testosterone was 94.47-324.44 for males and 19.69-141.21 for females.

Control Variables

Possible factors that might need to be controlled for in analyses involving testosterone were investigated through regression, $t$-test, or ANOVA, depending on the type of variable. In this sample, at least, most of the variables that could potentially
make a difference were unrelated to testosterone. Testosterone did not vary significantly by season, either for the overall sample, $F (3, 165) = 1.57, p = .20$, or for men or women separately, $F (3, 79) = .95, p = .42$ and $F (3, 82) = 1.36, p = .26$, respectively. After controlling for gender, neither participant age nor age of puberty predicted testosterone level ($\beta = -.089, p = .14$ and $\beta = .056, p = .36$, respectively). Although there was no direct measure of frequency of sexual activity, participants’ report of average frequency of nights spent together with partner also failed to predict testosterone ($\beta = -.007, p = .91$). Other variables that made no difference in testosterone included hours of sleep in the past 24 hours ($\beta = .084, p = .17$) and whether the participant had taken any drugs or alcohol in the past 24 hours, $t (157) = -1.23, p = .22$. Among females, testosterone levels did not differ by menstrual phase, $F (2, 61) = .22, p = .81$, or by whether she was taking oral contraceptives, $t (82) = 1.03, p = .31$. Of the other medications checked, including antibiotics, cold medicine, and allergy medicine, as well as many psychotropic medications, the only one that seemed to show a significant relationship to testosterone was asthma medication, $t (163) = -4.22, p < .001$; however, this relationship was found to be an artifact of a single case that both took asthma medication and had very high testosterone. When this case was removed from analyses, the relationship disappeared, $t (162) = -1.20, p = .23$, so asthma medication was not considered a necessary control variable for the sample.

The one factor that did emerge as significantly related to testosterone was smoking cigarettes; participants who said they had smoked that day had higher testosterone than those who had not, $t (159) = -3.06, p = .003$. Although there is little evidence that smoking caused higher testosterone or vice versa, because this variable
was extraneous to the model being tested, it was retained as a control variable in analyses involving testosterone.

**Hierarchical Linear Modeling (HLM) Analyses**

Based on preliminary regression analyses, it was determined that Assertiveness did not relate meaningfully to either depression or to conflict tactics in this sample, so this scale was not used in the final HLM analyses. Similarly, although Physical Assault was found to relate to testosterone levels, Psychological Aggression did not, so only the former was used for the testosterone→conflict tactics piece of the model (p. 14). The significant positive skew of both of these Conflict Tactics subscales poses a problem when the variable is used as an outcome, which will be addressed below. In a predictor, on the other hand, such skew is less worrying as it tends to attenuate rather than bias results, and so these variables were used as untransformed predictors in analyses.

In order to fit a regression line in HLM, at least two outcome scores are required, but for the outcomes being predicted here (depression and conflict tactics), only one score was obtained for each member of the couple. To create two scores, the scales – CESD total depression and Minor Physical Assault from the Conflict Tactics Scale – were divided into two functionally equivalent scales. The standard deviation for each item was computed, and items were assigned to each scale – the highest and lowest standard deviation items to one scale, the next highest and lowest to the other, etc. – so that the total variances for the two created scales were as similar as possible.

Finally, because there can be no missing data at level 2 when running HLM, missing data values were imputed using regression equations. Both minor
psychological aggression and minor physical assault were regressed on related predictors, including other conflict tactics and partner values, and these regression equations were used to calculate reasonable values to fill in the gaps (7 missing data points in one scale, 4 in the other). One couple was missing too much data to be able to impute a reasonable estimate, and they were dropped in running the analyses.

Predicting CESD Depression

**Baseline Model** (Table 2, Model 1)

The baseline model predicts male and female true depression scores based solely on intercepts with no explanatory variables. Because the CESD scale was divided into two scales, the figures in the HLM models represent half of the original scale, which is why the intercept values (5.37 for females, 5.36 for males) are smaller than the means given in the descriptives (10.72 for females, 10.39 for males). With this adjustment, though, the intercepts present the same picture as that achieved by a descriptive analysis of the sample -- men and women have similar depression levels overall. At the same time, this baseline model shows that there is significant variation around these averages (see Table 3) among both men, $\chi^2(79) = 443.45, p < .001$, and women, $\chi^2(79) = 485.44, p < .001$. The intraclass correlations further showed that 70.6% of the variance in males’ and 69% of the variance in females’ depression scores could be attributed to level 2 (couple) variables, making the case for adding explanatory variables.

Interestingly, the correlation between male and female depression true scores within the couple was only .04, indicating essentially no association between his and her depression levels.
**Testosterone Model** (not shown)

To begin setting up the mediational model proposed, male and female testosterone levels (expressed as z-scores by gender) were entered as level 2 predictors of their depression scores. None of the testosterone predictors, including the interaction of male and female testosterone within the couple, proved significant, and the deviance test showed this model to be no better than the baseline for fitting the data, \( \chi^2 (6) = 2.34 \), \( p > .50 \). A quadratic model, by which each partner’s depression was predicted by his/her T and \( T^2 \) was also fit to see if there might be a curvilinear relationship, but again none of the predictors were significant, and the model as a whole was not an improvement over baseline, \( \chi^2 (2) = .59 \), \( p > .50 \). The idea of a mediational model thus had to be abandoned, and analyses were continued in two parts: one part predicting depression from conflict tactics, the other predicting conflict tactics (physical assault) from testosterone.

**Psychological Aggression Model** (Table 2, Model 2)

Males’ and females’ reported psychological aggression in the relationship were next entered as explanatory variables for each of their depression scores. For women, both her and her partner’s psychological aggression contributed significantly to her depression, but in opposite ways; the more depressed she was, the more psychologically aggressive she was, but the less psychologically aggressive he was. For men, neither his nor her psychological aggression related significantly to his depression, and a contrast test of whether the psychological aggression predictors were equivalent for her and for him confirmed this, \( \chi^2 (4) = 13.14 \), \( p = .01 \). The overall model proved a
significant improvement in fit over baseline, as seen in the deviance test, $\chi^2 (4) = 12.34$, $p = .015$, and it explained 12.3% of the variance in females’ depression.

**Physical Assault Model** (Table 2, Model 3)

The next step involved entering men’s and women’s reported physical assault within the couple as predictors of each of their depression scores. In this model, the only parameter that approached significance was males’ physical assault predicting his own depression, and the deviance test indicated that it was a nonsignificant improvement over the baseline model, $\chi^2 (4) = 7.87, p = .095$. However, the fact that male physical assault was marginally significant ($p = .07$) and explained 9.1% of the variance in male depression made it worthwhile to retain the physical assault predictors for a full predictive model before weeding out unnecessary variables.

**Full Model** (Table 2, Model 4)

For the full model, all of the psychological and physical aggression predictors from the above two models were entered at level 2 to explain male and female depression within the couple. It was expected that entering these variables together might affect the coefficients since controlling for other factors would influence individual relationships, and although there were no radical changes, some of the predictors actually showed up stronger once the influence of the others was taken into account. Male physical assault came closer to significantly predicting his depression ($p = .052$), and it also emerged as a significant predictor of her depression. This full model explained 17.6% of the variance in females’ depression and 9.9% of males’ depression, which is fairly substantial for this type of variable. At the same time, some of the
variables seemed to be carrying much more of the predictive weight than others, and a more parsimonious model was sought.

**Reduced Model (Table 2, Model 5)**

On the one hand, the full model with physical assault predictors did not prove itself a significant improvement in fit over the psychological aggression model, $\chi^2(4) = 8.27, p = .081$, which would suggest that the set of physical assault variables as a whole are not necessary for the model. On the other hand, male physical assault seemed to be important, particularly in explaining his depression, so a contrast test was performed to ascertain whether male physical assault (both in predicting her and his depression) was different from zero. The result, $\chi^2(2) = 8.69, p = .01$, affirms that physical assault, at least on his part, is not negligible in explaining depression. Finally, because psychological aggression variables clearly predicted her depression but never seemed to contribute to his, they were removed from his part of the model. The deviance test, $\chi^2(4) = .65, p > .5$, confirmed that these reductions did not cause a decrement in fit, so this more parsimonious model was kept as the final one.

**Predicting CTS Physical Assault**

The Conflict Tactics Scales, and the Physical Assault subscales in particular, were extremely positively skewed, with a modal response of zero and a scattering of higher reported frequencies of aggressive behavior. None of the transformations attempted (i.e., reciprocal, log) seemed to normalize the distribution, so a non-linear Poisson model was used to predict expected frequencies of physical assault within the past year. The distribution was further characterized by over-dispersion, with a standard deviation greater than the mean, so an over-dispersion option was included in
fitting the model. Finally, the Poisson model gives both unit-specific and population-average versions; since this study was conducted, ultimately, to draw conclusions about relationships within the population of late adolescent couples as a whole, the latter is reported here.

**Baseline** (Table 4, Model 1)

With no explanatory predictors, the intercepts show that females are somewhat less likely to physically assault their partner than are males. The expected frequencies (found by an inverse natural log transformation of the coefficients) of .65 for women and .98 for men are only half of the true frequencies because of the split scale noted above, so the final estimate of physical assault in this unconditional model is 1.3 times per year for women and 1.96 times per year for men. Again, there is significant variance around these means, $\chi^2(79) = 553.82, p < .001$ for females, $\chi^2(79) = 2170.07, p < .001$ for males, and the intraclass correlations of .654 for females and .760 for males further confirm that there is a great deal of variability that could be explained at level 2. The correlation between the male and female physical assault true scores within the couple was .48, indicating a substantial positive relationship between his and her physical aggression.

**Own Testosterone** (Table 4, Model 2)

The first step in prediction was to enter each participant’s own testosterone to explain his/her physical assault, controlling for cigarette smoking. One’s own testosterone did not prove a significant predictor for either men or women, but this initial model did explain 7.4% of the variance in men’s physical assault, and the
deviance test showed it to be a significant improvement in fit over baseline, $\chi^2(4) = 22.68, p < .001.

**Own and Partner Testosterone** (Table 4, Model 3)

The next step was to include partner’s testosterone (controlling for cigarette smoking), as well, to take into account the contribution of each member of the dyad. Although his testosterone, controlling for her own, emerged as a significant negative predictor of the female’s physical assault, the individual parameters were not of the greatest interest here because this model was viewed more as a step toward a truly interactive model, which could change the direct relationships markedly. This model did not prove a significant improvement in fit over the previous one, $\chi^2(4) = 4.54, p = .34$, but the addition of partner’s testosterone was not to be rejected before testing the interactive model, and changes in pseudo-$R^2$’s from basically zero to 5.2% of the variability in females’ physical assault and from 7.4% to 9.9% in males’ suggested that there might be some value in these predictors.

**Full Model with Interaction** (Table 4, Model 4)

Adding an interaction term of his $x$ her testosterone proved the most important piece of explaining physical assault in relationships, at least for males, and it shifted the predictive power of the other variables, as well. Although his and her testosterone levels by themselves did not predict men’s physical assault, their interaction was a strong positive predictor; for every one standard deviation above the mean they both were in testosterone, he was likely to commit almost 10 acts of minor physical assault per year. Further examination of the interaction showed that for males high in testosterone, as his partner increased in testosterone, his physical assault increased,
whereas for males low in testosterone, the reverse was true (see Figures 1 and 3 for unit-specific and population-average graphs, respectively). Alternatively, when his partner is high in testosterone, the higher the male’s testosterone, the more often he uses physical assault, whereas when his partner is low in testosterone, the opposite is true (see Figures 2 and 4). This effect is particularly noticeable at the higher end of the distribution, where high levels of testosterone on either her or his part, combined with higher levels on the other’s part, are associated with much more physical assault. Conversely, if both he and she are low in testosterone, he also is likely to show more physical assault than if one of them is relatively higher and the other relatively lower for his/her gender.

While cigarette smoking was not a focus of this paper and was treated as a control variable, it is interesting to note that if he smokes, both he and his partner are likely to commit significantly more acts of physical assault on each other, and if she smokes, he is also predicted to be more violent than if she does not. The model as a whole proved a significantly better fit than the previous one without the interaction term, $\chi^2 (2) = 29.44, p < .0001$, due primarily to the added explanatory value for male physical assault; the model explained only 6.2% of the variability in her physical assault, but 31.5% of the variability in his.
Although the findings of the present study do not bear out the mediational model hypothesized, they do support certain links within the model that highlight the importance of a dyadic predictive framework. The mediational model failed because testosterone levels did not predict depression for either member of the couple. There are several possible reasons that this relationship, found in previous studies, did not emerge here. One is that there could be moderators of the relationship that were not addressed; for example, Booth et al. (2000) found that relationships between testosterone and adjustment in adolescents depended not only on gender of the participant, but also on the quality of different relationships in the adolescent’s life (good or poor relations with mother and father predicted different outcomes). Another potential problem involves the testosterone measurements used; saliva samples may not provide as good an index in this case as serum, and correlations between serum T and salivary T have been found to be particularly low for females (Shirtcliff, Granger, & Likos, 2002). Also, the restricted range of T levels in this college sample made prediction more difficult, so relationships in the broader population may not have been apparent here.

The testosterone-aggression link did hold true for males in the sample, but not in a direct way, and only for physical aggression. Contrary to Cohan et al.’s (2003) findings in marital interactions, concordance for either low or high T (but especially the latter) predicted more destructive conflict tactics (physical assault) on his part, and it actually seemed to be better for the partners to have complementary T levels. If women
with relatively higher testosterone were more aggressive and/or assertive, it might make sense for high testosterone men to behave more aggressively toward them, but there was no relationship between T and either of these variables for females, so there may be other personality variables correlated with testosterone in women that impact couple interaction. The lack of a clear connection between T and conflict tactics in women fits with the hypothesis that compliance with social norms would dampen any hormone-related aggressive tendencies more in females than in males. As noted earlier, a T-aggression relationship could also be obscured because of less accurate salivary T assay in women, and testosterone may also be the wrong hormone to look at in female aggression (estrogen or androstenedione could be more important for her).

Another perspective on why testosterone complementarity may be associated with less male aggression comes from the animal literature. While high-ranking male primates are not more aggressive than others during stable periods in the social hierarchy (when their dominance is unquestioned), they show more aggression during unstable periods of social challenge and higher absolute levels of testosterone, though less relative T elevation during stress (Sapolsky, 1983; 1991). At the other end of the scale, low-ranking males were also found likely to initiate fights or displace aggression onto a weaker 3rd party (Vigin & Sapolsky, 1997). It could be that having similar gender-relative levels of T as one’s mate creates a dominance struggle that males respond to by using aggression, whereas a more clearly established rank relationship based on having T levels at different ends of the spectrum allows for a more harmonious interaction. Another implication from this primate research is that, although the testosterone-aggression relationship is discussed primarily in terms of T’s
impact on his aggression, the direction of causality could also go the other way, such that greater physical assault on his part raises both his and her T levels.

Surprisingly, most of the potential control variables expected to relate to testosterone were unimportant in this sample, with the exception of cigarette smoking. In a relationship in which the male smokes, both he and she tend to be more physically aggressive, and her smoking further contributes to his aggression. It seems likely that more aggressive people in the sample tended to smoke, both for its calming effect and because of its association with counterculture tendencies in youth, rather than that smoking causes greater aggression. The fact that, for both men and women, partner's smoking predicted greater aggression on one's own part allows for the possibility that smoking irritates the partner more and predisposes him/her to greater aggression, but there is no way of confirming this.

The lack of T differences by time of year, menstrual cycle phase, and nights spent together ran counter to some evidence from the literature, but this could be due to imprecision of some of the measures. Although T has been found to vary seasonally within the individual, the current study is based on measurements from one sample from each individual, and the variations among individuals may have been greater than that associated with seasonal changes. For women, it was somewhat surprising that T level did not seem to vary by menstrual phase since androgen levels tend to be higher at mid-cycle, but the cycle phase was determined from the participant's report of how many days it had been since her last period, which is not a very exact measure, considering the variability in cycle length both within and between women. Even more indirect was the approximation of frequency of sexual activity by number of nights participants
reported spending with partners -- not only do college students engage in sexual activity without necessarily spending the night together, some of them may be engaging in sex with someone other than their partner -- so this attempt at control probably did not address what it was intended to.

Another unexpected finding was that assertiveness, at least as measured by the subscale from the big-5 inventory, did not relate to either depression or conflict tactics in this sample. It could be that the measure was simply too general to predict specific areas of functioning. Another possible reason for this disconnect, contrary to both theoretical considerations and previous research, is that since the Assertiveness questionnaire came after a series of other scales, participants were fatigued and less careful about their responses on this scale. In any case, whether because of measurement problems or a true null relationship, this construct could not be usefully integrated into the model presented in this study.

Much as expected based on previous research, conflict variables within the couple related more strongly to women’s depression than to men’s. His physical aggression directly contributed to her depression, as indicated in other studies, which could be due to a power imbalance in the relationship and her sense of helplessness, or his aggression could be a sign of other troubles within the relationship that impact her mood. Psychological aggression also proved to be important in her depression, but the relationships were more complex. While higher levels of psychological aggression on her part related positively to her depression, his psychological aggression (when controlling for hers) related negatively to her depression. There are several ways to view this somewhat puzzling discrepancy. On the one hand, the socialization of women
to base more of their self-esteem on maintaining harmonious relationships might cause her to feel that she has failed if she perceives herself as contributing more to the aggression within the relationship. The psychologically aggressive behavior on her part might also be a reflection of her depression; previous research has found that depressed people are less socially skillful and more hostile, demanding, and verbally aggressive (Kahn, Coyne, & Margolin, 1985). He might, in turn, react to her depression by backing off a bit psychologically, similarly to Nelson and Beach’s (1990) findings that depressed partners were “rewarded” by their spouse’s inhibition of hostile and irritable behaviors. Another explanation relies on the indications from previous studies and, to some extent, in the present study, that women tend to use psychological aggression more than men do. Thus, lower levels on her part and higher levels on his might represent more of an equalizing relationship in which they exhibit more of a balance of psychological aggression between them.

For men, as predicted, the evidence for a relationship between conflict and depression was much weaker than that for women. The only conflict tactics variable that related to his depression, and that with marginal significance, was his level of physical assault. As discussed earlier, this probably reflects the tendency of men who are depressed to exhibit more externalizing behavior, but it could also work in the other direction if he feels guilty for aggressing against his partner, which could further his depression. In contrast to the females in the sample, the males were unaffected by their partners’ conflict tactics, underlining the preferential impact of relationship conflict on women, at least in the area of depression.
The broader implications of this study involve both a theoretical explanation of why gender differences in depression emerge in adolescence and some practical guidelines for dealing with adolescent couples. For the former, it seems that a part of why females grow more depressed starting in adolescence has to do with greater vulnerability to relational conflict as they take on the female gender role. If women are socialized to maintain a sense of self through relationships and learn that conflict will cut them off from interpersonal support, aggressive interactions with a partner are naturally more threatening to them, beyond the possible physical consequences of his aggression. The expectation that the woman is the caretaker of the relationship also sets her up to take responsibility for problems in the relationship, which further contributes to depression on her part. Even though the full mediational model of hormones to conflict to depression was not confirmed, the fact that the interaction of his and her T levels related to his physical aggression further suggests that hormone relationships that do not necessarily affect her directly could impact her through her partner’s aggressive tendencies.

In practice, this means that those who are dealing with adolescents, especially in a couples context, should be aware of the harmful consequences of aggression in relationships (especially for her mental health) and help couples to find alternative ways of working out conflict. Both partners should be encouraged to take responsibility for maintaining the relationship so that it does not fall so much to the female, and girls should be supported in building self-esteem outside of relationships so that conflict and interpersonal ruptures are not as devastating. Although there is rarely a reason to look
at adolescents’ hormone levels, the results of this study also imply that particular care should be taken when both partners are relatively high in testosterone.

The current study has both strengths and weaknesses that should be used to inform future research. Probably the major strengths of this study are the inclusion of hormone measures to make the model truly biopsychosocial and the use of HLM to examine explanatory variables in a couples context. At the same time, there are certainly improvements that could be made. Sampling a broader range of testosterone values and using serum T could make testosterone relationships clearer, and measures of other hormones (such as androstenedione, estrogen, luteinizing hormone) and ratios between various hormones would give a better idea of the biological piece of the picture. For the social piece, more measures of relationship quality and the context of conflict might be useful, and even using participants’ reports of victimization, as well as perpetration, on the CTS would add another facet that could be important. As far as psychological measures go, the CESD is a widely used measure, but there are other aspects of depression, and of psychological adjustment more generally, that might help clarify the correlates of relational aggression for both men and women. Finally, the design of this study did not allow for a definite distinction between antecedents and consequences of depression in predictive relationships; although most of these relationships are probably to some extent bidirectional, ascertaining the dominant causal tendencies would have practical importance for intervention. Future studies with longitudinal designs and/or SEM analysis could help further tease apart the reasons for and effects of depression in young men and women, as well as how they affect each other.
Table 1: Means and standard deviations for couple variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Women</th>
<th></th>
<th></th>
<th>Men</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>Conflict Tactics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minor Psychological Aggression</td>
<td>16.34</td>
<td>15.07</td>
<td>13.71</td>
<td>16.50</td>
<td></td>
</tr>
<tr>
<td>Severe Psychological Aggression</td>
<td>.75</td>
<td>2.37</td>
<td>.59</td>
<td>1.41</td>
<td></td>
</tr>
<tr>
<td>Minor Physical Assault</td>
<td>1.27</td>
<td>2.62</td>
<td>1.38</td>
<td>3.66</td>
<td></td>
</tr>
<tr>
<td>Severe Physical Assault</td>
<td>.13</td>
<td>.87</td>
<td>.52</td>
<td>3.74</td>
<td></td>
</tr>
<tr>
<td>Assertiveness</td>
<td>17.48</td>
<td>4.99</td>
<td>17.05</td>
<td>3.64</td>
<td></td>
</tr>
<tr>
<td>CESD Depression</td>
<td>10.72</td>
<td>7.00</td>
<td>10.39</td>
<td>6.74</td>
<td></td>
</tr>
<tr>
<td>Testosterone**</td>
<td>62.94</td>
<td>32.28</td>
<td>185.42</td>
<td>103.31</td>
<td>** significant gender difference at .05 level.</td>
</tr>
</tbody>
</table>

Table 2: Regression coefficient estimation for predicting CESD depression.

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Model 1 Baseline</th>
<th>Model 2 Psych</th>
<th>Model 3 Phys</th>
<th>Model 4 Full</th>
<th>Model 5 Reduced</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient</td>
<td>SE</td>
<td>Coefficient</td>
<td>SE</td>
<td>Coefficient</td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>5.368**</td>
<td>.375</td>
<td>4.684**</td>
<td>.488</td>
<td>5.083**</td>
</tr>
<tr>
<td>Fpsych</td>
<td>.094**</td>
<td>.031</td>
<td></td>
<td></td>
<td>.097**</td>
</tr>
<tr>
<td>Mpsych</td>
<td>-.064*</td>
<td>.025</td>
<td></td>
<td></td>
<td>-.098**</td>
</tr>
<tr>
<td>Fphys</td>
<td>.129</td>
<td>.125</td>
<td>.036</td>
<td>.163</td>
<td></td>
</tr>
<tr>
<td>Mphys</td>
<td>.086</td>
<td>.122</td>
<td>.234*</td>
<td>.099</td>
<td>.231*</td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>5.362**</td>
<td>.375</td>
<td>5.246**</td>
<td>.588</td>
<td>5.024**</td>
</tr>
<tr>
<td>Fpsych</td>
<td>-.025</td>
<td>.037</td>
<td>-.021</td>
<td>.038</td>
<td></td>
</tr>
<tr>
<td>Mpsych</td>
<td>.039</td>
<td>.043</td>
<td>-.001</td>
<td>.031</td>
<td></td>
</tr>
<tr>
<td>Fphys</td>
<td>-.011</td>
<td>.132</td>
<td>.046</td>
<td>.145</td>
<td></td>
</tr>
<tr>
<td>Mphys</td>
<td>.255</td>
<td>.141</td>
<td>.276</td>
<td>.142</td>
<td>.254</td>
</tr>
</tbody>
</table>

* p < .05; ** p < .01.

Table 3: Variance component estimation for predicting CESD depression.

<table>
<thead>
<tr>
<th>Variance Component</th>
<th>Model 1 Baseline</th>
<th>Model 2 Psych</th>
<th>Model 3 Phys</th>
<th>Model 4 Full</th>
<th>Model 5 Reduced</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female variance</td>
<td>10.15</td>
<td>8.81</td>
<td>9.92</td>
<td>8.36</td>
<td>8.37</td>
</tr>
<tr>
<td>Male variance</td>
<td>9.40</td>
<td>9.13</td>
<td>8.54</td>
<td>8.47</td>
<td>8.54</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Proportion of variance explained</th>
<th>Females</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>.123</td>
<td>.022</td>
<td>.176</td>
<td>.176</td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>.028</td>
<td>.091</td>
<td>.099</td>
<td>.091</td>
<td></td>
</tr>
</tbody>
</table>
Table 4: Regression coefficient estimation for predicting frequency of physical assault, poisson model with over-dispersion (population-average model).

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Model 1 Baseline</th>
<th>Model 2 Own T</th>
<th>Model 3 Own + Partner T</th>
<th>Model 4 Full with Interaction</th>
<th>Expected Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient</td>
<td>SE</td>
<td>Coefficient</td>
<td>SE</td>
<td>Coefficient</td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>-.430*</td>
<td>.181</td>
<td>-.379</td>
<td>.203</td>
<td>-.684**</td>
</tr>
<tr>
<td>Feig</td>
<td>-.399</td>
<td>.410</td>
<td>-.522</td>
<td>.369</td>
<td>-.619</td>
</tr>
<tr>
<td>M cig</td>
<td>1.524**</td>
<td>.554</td>
<td>1.427**</td>
<td>.523</td>
<td></td>
</tr>
<tr>
<td>Ftest z</td>
<td>-.115</td>
<td>.186</td>
<td>.157</td>
<td>.163</td>
<td>.102</td>
</tr>
<tr>
<td>Mtest z</td>
<td>.271*</td>
<td>.136</td>
<td>-.481</td>
<td>.329</td>
<td></td>
</tr>
<tr>
<td>F x M</td>
<td></td>
<td></td>
<td>.076</td>
<td>.076</td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>-.021</td>
<td>.344</td>
<td>-.396</td>
<td>.283</td>
<td>-.536</td>
</tr>
<tr>
<td>Feig</td>
<td>1.431</td>
<td>.941</td>
<td>1.994</td>
<td>1.132</td>
<td>1.176*</td>
</tr>
<tr>
<td>F test z</td>
<td>.244</td>
<td>.478</td>
<td>.080</td>
<td>.290</td>
<td></td>
</tr>
<tr>
<td>M test z</td>
<td>.183</td>
<td>.700</td>
<td>.031</td>
<td>.809</td>
<td>-.418</td>
</tr>
<tr>
<td>F x M</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p < .05; ** p < .01.

Table 5: Variance component estimation for predicting frequency of physical assault, poisson model with over-dispersion (population-average model).

<table>
<thead>
<tr>
<th>Variance Component</th>
<th>Model 1 Baseline</th>
<th>Model 2 Own T</th>
<th>Model 3 Own + Partner T</th>
<th>Model 4 Full with Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female variance</td>
<td>1.654</td>
<td>1.691</td>
<td>1.568</td>
<td>1.551</td>
</tr>
<tr>
<td>Male variance</td>
<td>2.771</td>
<td>2.565</td>
<td>2.496</td>
<td>1.898</td>
</tr>
</tbody>
</table>

Proportion of variance explained

<table>
<thead>
<tr>
<th>Females</th>
<th>Males</th>
</tr>
</thead>
<tbody>
<tr>
<td>.074</td>
<td>.099</td>
</tr>
<tr>
<td>.099</td>
<td>.315</td>
</tr>
</tbody>
</table>
Figure 1: Physical assault by female testosterone, high and low values of male testosterone (unit-specific model).
Figure 2: Physical assault by male testosterone, high and low values of female testosterone (unit-specific model).
Figure 3: Physical assault by female testosterone, high and low values of male testosterone (population-average model).
Figure 4: Physical assault by male testosterone, high and low values of female testosterone (population-average model).
REFERENCES


Costa, P.T. Jr., & McCrae, R.R. *Revised NEO Personality Inventory (NEO-PI-R) and NEO Five-Factor Inventory (NEO-FFI) professional manual.* (Odessa, FL: Psychological Assessment Resources)


Steinberg, L. (1987, April). Pubertal status, hormonal levels, and family relations. (In E. J. Susman [Chair], *Hormone status at puberty: Consequences for adolescents and their families*. Symposium conducted at the biennial meeting of the Society for Research in Child Development, Baltimore.)


