
Pergamon

Aggression and Violent Behavior
6 (2001) 579–599

AGGRESSION AND VIOLENT BEHAVIOR

The relationship between testosterone and aggression: a meta-analysis

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Received 15 November 1999; accepted 30 March 2000

Abstract

In non-human animals, the relationship between testosterone and aggression is well established. In humans, the relationship is more controversial. To clarify the relationship, Archer conducted three meta-analyses and found a weak, positive relationship between testosterone and aggression. Unfortunately, each of the analyses included only five to six studies. The aim of the present study was to re-examine the relationship between testosterone and aggression with a larger sample of studies. The present analyses are based on 45 independent studies ($N = 9760$) with 54 independent effect sizes. Only studies that reported a p-value or effect size were included in the analyses and the sample may underestimate the proportion of non-significant findings in the population. Correlations ranged from -0.28 to 0.71. The mean weighted correlation ($r = 0.14$) corroborates Archer's finding of a weak positive relationship.

Keywords: Violence; Aggression; Testosterone

The relationship between testosterone and aggression has been of interest to researchers for decades. A positive relationship is well established in non-human animals, but there is no consensus in the case of humans (Archer, 1991). Correlations run the gamut and replication has been difficult. The purpose of this paper is to establish the size of the relationship between testosterone and aggression among humans using meta-analytic techniques. A secondary goal was to determine whether there are variables that moderate the relationship.

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1359-1789/01/$ – see front matter
PII: S1359-1789(00)00032-X

1. Rationale for the relationship

Testosterone is an androgen that has been implicated in the development and maintenance of masculine characteristics in a variety of species (Mazur & Booth, 1998). It has been documented that the females of most species are less aggressive and have far lower testosterone levels than do males; this is taken as evidence of a link between testosterone and aggression (Archer, 1991). How exactly testosterone affects aggression and dominance behavior is unknown (Mazur, 1983), but multiple pathways have been proposed (Simon, McKenna, Lu, & Collager-Clifford, 1996).

In an effort to explain the influence of testosterone on aggressive behavior, Vom Saal (1983) proposed the Organization/Activation Model of testosterone. According to this model, androgen influences in the prenatal period organize the neural networks that mediate aggressive behavior. When these androgen-organized networks are exposed to androgens again in early adulthood, the neural networks are activated and aggression is elicited by relevant environmental stimuli. Support for this model comes from numerous experiments with mice demonstrating that prenatal exposure is necessary for testosterone to mediate a higher level of aggressive behavior in adulthood (Vom Saal, 1983). Vom Saal furthers this idea in the Sensitivity Model of inter-male aggression, where each presentation of testosterone results in a higher sensitivity to subsequent exposure to the hormone. Individuals who are exposed to lower levels of testosterone apparently require a higher dose of testosterone later in life for aggression to be elicited, than do individuals with a greater amount of previous exposure.

2. Evidence for the relationship in non-human animals

In non-human animals, the relationship between testosterone and aggression (where aggression is operationalized through observable aggressive behavior) has been demonstrated through correlational and experimental studies, involving manipulation of testosterone levels through castration and injection of testosterone. Results overwhelmingly indicate that testosterone and aggression are related (Turner, 1994).

Individual correlational studies demonstrate this relationship in a wide range of birds (Harding, 1983) and many species of mammals, including rats, monkeys, hamsters, dogs, and deer (Rada, Kellner, & Winslow, 1976). For example, in male mice, aggression is positively correlated with an increase in circulating testosterone (Vom Saal, 1983). Similarly, male rhesus monkeys with relatively high levels of testosterone tend to engage in more dominant and aggressive displays than their lower testosterone counterparts (Rose, Holladay, & Bernstein, 1971). Higher testosterone levels also correlate with territorial behavior in a variety of birds (Schwabl & Kriner, 1991; Wingfield & Hahn, 1994).

Experimental results further suggest that testosterone is a causal factor in aggressive non-human animal behavior. Castration of male mice (Vom Saal, 1983) and lizards (Greenberg & Crews, 1983) results in a decrease in aggressive behavior, regardless of how aggression is measured (Vom Saal, 1983). Similarly, it has been shown that the injection of testosterone

consistently increases aggressiveness in a variety of species (Brain, 1983; Monoghan & Glickman, 1992).

While the relationship between testosterone and aggression seems clear, the connection becomes more complex as the range of species being studied widens (Rubinow & Schmidt, 1996). Extremely social mammals, for example, rely on a variety of social and environmental cues that aid in determining appropriate action for particular situations (Turner, 1994). Because aggressive behavior is the product of a complex sequence of physiological, emotional, and cognitive components (Floody, 1983), it is likely that several variables moderate the relationship between testosterone and aggression. Consequently, it is plausible that the relationship may be attenuated by other factors among more social animals.

3. Evidence for the relationship in humans

As mentioned previously, results from human studies are widely discrepant, although the relationship between testosterone and aggression in humans has been investigated a great deal (Archer, 1991). Some studies find a strong positive relationship, others a negative one, and still others find no effect whatsoever (Archer, 1991). This lack of consensus is sometimes evident even within studies. A good example is Turner (1994), where testosterone was positively correlated with norm-violating (aggressive) behavior in 12- and 13-year-old boys, but not in 15- and 16-year-old boys (Turner, 1994).

There are likely several factors that have contributed to the inconsistency of findings on this topic. One factor may be poor reliability in measuring testosterone levels. In part, this is because testosterone is released in spurts from moment to moment (Mazur & Booth, 1998). There are both circadian cycles in testosterone levels and substantial variation over 8 to 20 daily cycles (Doering, Kraemer, Brodie, & Hamburg, 1974). These variations contribute to substantial measurement error because most researchers sample participants' testosterone levels only once.

As a result of the inconsistent findings in human participants, some researchers only use the results of animal studies to substantiate the argument for the relationship in humans. Others, such as Archer (1991), question whether comparative data are generalizable to humans at all because of the numerous methodological differences between human and animal studies. Specifically, researchers studying humans often have a more inclusive definition of the construct of aggression, measure aggression as a personality trait, and obtain ratings of aggressiveness both through behavioral observations and self-report methods in experimental and natural settings (Archer, 1991).

Archer published three meta-analyses in 1991. He chose to do three separate analyses, because studies were highly variable in terms of methodology. More specifically, type of participant and measures of aggression and testosterone varied substantially. Archer found a weak, positive relationship between testosterone and aggression, despite the large range of results in the literature. It is important to note, however, that each analysis was based on only five to six investigations. The purpose of the present meta-analysis is to more definitively establish the relationship between testosterone and aggression among humans with a greatly increased number of studies.

4. Possible moderating variables

The research literature suggests many possible moderators of the relationship between testosterone and aggression, including metabolism, age, sex, circadian rhythm, stress, past experience, and social rank (Rubinow & Schmidt, 1996). Hormonal moderators, such as cortisol, have also been proposed (Brain, 1983; Leshner, 1983). Although it would be useful to investigate the effects of these moderators on the relationship between testosterone and aggression, typically, insufficient data are available to allow an evaluation via meta-analytic methods. However, enough information exists to test whether cyclic fluctuations in testosterone, age, sex, offender status, type of aggression measure, and testosterone sampling method influences the strength of the relationship between testosterone and aggression. The male testosterone cycle, age, sex, and the reciprocal nature of the relationship are described in detail here. Other potential moderators, such as offender status, are dealt with in the Predictions section.

4.1. Cyclic fluctuations of testosterone

Previous research suggests that testosterone levels fluctuate daily and seasonally (Bernstein, Gordon, & Rose, 1983), and that individual testosterone levels are higher and most variable in the morning and lower and more stable in the afternoon. Although testosterone levels decline throughout the day, the largest decline occurs between 7:00 and 10:00 a.m. (Dabbs, 1990).

This variation in testosterone levels has prompted researchers to measure testosterone as early in the morning as possible. Presumably, researchers do this to get testosterone estimates when levels are highest. This practice is problematic, however, precisely because morning testosterone levels are more variable than at any other time of the day. The time of measurement during the daily cycle likely affects the magnitude of the observed relationship between testosterone and aggression. The consequence of this practice for studies measuring testosterone levels in the morning is that the estimate of the relationship is likely to be confounded with a higher degree of measurement error.

4.2. Age

For males, another possible moderator is the age of the participant. This is because both the effects of testosterone and testosterone levels vary over the lifespan (Mazur & Booth, 1998).

At the prenatal stage, testosterone has an organizational influence, differentiating male from female neural architectures. Prenatal testosterone levels also affect behavior later in life. In humans, for example, fetal hormones increase assertive and aggressive behavior (Turner, 1994) as well as rough and tumble play and dominant behavior (Archer, 1991). This behavioral effect is also true for rhesus monkeys, where males are more likely to engage in threats and rough and tumble play than are females. Interestingly, this difference between male and female rhesus monkeys is not apparent if the females are also exposed to testosterone in utero (Mazur & Booth, 1998). At puberty, when testosterone levels are highest and the relationship between testosterone and aggression is strongest (Susman,

Granger, Murowchick, Ponirakis, & Worrall, 1996), testosterone has more of an activational influence (Mazur & Booth, 1998). It works by activating pre-existing structures and neural networks and facilitates the long-term reorganization of the body. After the early twenties, testosterone levels begin to decline slowly (Mazur & Booth, 1998).

4.3. Sex

Sex is another possible moderator in the relationship between testosterone and aggression because women have relatively lower levels of testosterone and are much less physically aggressive than are men (Archer, 1991). In support of this differentiation, Mazur and Booth (1998) found that while men showed a rise in testosterone levels prior to a contest, there was no corresponding rise for women. They concluded that the effect of competition (often aggressive) for increasing testosterone levels appears to be specific to men. There is evidence to the contrary, however. Kemper (1990), for example, provides evidence to suggest that the relationship between testosterone and aggression is similar in strength and direction across the sexes. Further support is provided by the fact that injection of androgens into non-human female primates results in an increase in aggressive behavior (Floody, 1983). That these results are generalizable to human females is suggested by the results of Van Goozen, Frijda, and Van de Poll (1994), who found that similar androgen treatments result in increased anger among women.

Similarly, Persky, Smith, and Basu (1971) found reliable positive correlations between testosterone and aggression during early follicular and late luteal phases of menses. Nonetheless, it is currently unclear whether sex moderates the relationship, or whether it is an issue of differential variation in amounts of testosterone that may attenuate the observed correlations.

4.4. Past experience

The reciprocal nature of the relationship between testosterone and aggression adds potential complications. Mazur and Booth (1998) outlined two models of the relationship between testosterone and aggression. The first is known as the Basal Model. This theory asserts that testosterone levels directionally affect aggression with no feedback loops to testosterone. An alternative is the Reciprocal Model wherein testosterone and dominance/aggression reinforce one another (Mazur, 1983). The Reciprocal Model is supported by the fact that the relationship interacts with previous experience in mice (Archer, 1991) and primates (Mazur, 1985). Indeed, in some cases, past experience explains more of the variance in aggression than does testosterone level. Further, changes in dominance or social status can result in changes in testosterone levels; there is strong evidence for this effect in humans (Mazur & Booth, 1998; Schaal, Tremblay, Soussignan, & Susman, 1996).

5. Adaptationist reasoning behind the proposed relationship

It has been suggested that testosterone levels interact with environmental influences to produce behavior (Vom Saal, 1983) and that aggression is primarily an adaptive behavior in

response to stimuli that could be construed as threatening to one's fitness, such as territory violations, and/or survival (Daly & Wilson, 1994). In support of these ideas, for example, the aggressive behavior of birds increases in frequency when resources are scarce (Harding, 1983). In the context of this research, however, it is important to highlight that some researchers (e.g., Mazur & Booth, 1998) suggest that testosterone may be related to dominance, and not aggression per se, except where dominance "happens to be asserted aggressively" (p. 354). The previously mentioned Olweus (1986) study supports this notion. Inventory items that most highly correlated with testosterone levels were those involving non-physical aggressive responses to provocation (e.g., "When a teacher criticizes me, I tend to answer back and protest", p. 53). In this framework, dominance-promoting mechanisms, such as aggression and intimidation through aggression, are adaptive because dominant animals, including humans, are more likely to acquire valued resources, such as mates, and increase in status than their less dominant competitors (Mazur & Booth, 1998). Consistent with this reasoning, the literature supports the idea that the primary purpose of aggressive behavior is to secure access to mates, other valued resources, and status (Daly & Wilson, 1994).

Also consistent with this framework are reports that higher levels of testosterone are related to increased inter-male aggression in mice (Maxson, Shrenker, & Vigue, 1983) and in humans (Archer, 1991; Hinde, 1986). That inter-male competition mediates testosterone levels, and thus also aggression levels, is further reinforced by evidence that the presence of females has an influence on both testosterone and aggression levels. For example, Bernstein et al. (1983) found that, in non-human primates, cyclical changes in testosterone levels only occur when females are present. That women tend to admire men with relatively dominant features (Mazur & Booth, 1998) suggests that female choice has had an impact on the selection for these traits. In humans, additional support comes from evidence that testosterone and aggression decline around middle age (Julian & McKenry, 1989). This is consistent with the inter-male competition theory because this decrease in testosterone corresponds not only to an increase in expressiveness and parental and marital satisfaction, but also to a decrease in inter-male competition. This decrease in competition for females and an increase in caring for offspring would promote the survival of the father's existing offspring.

5.1. Competition and testosterone

Inter-male competition takes many forms, and the literature, consequently, examines a wide variety of competitive situations. Mazur and Booth (1998) found that male testosterone rises prior to competitive matches, as if in anticipation of the conflict. Mazur and Booth suggested that this rise might increase risk-taking and improve coordination, cognitive performance, and concentration. Indeed, following a match, winners maintained higher testosterone levels relative to losers for approximately 1 to 2 h. This pattern has been observed in a number of sports, including wrestling and tennis (Elias, 1981). Why would winners have an increase relative to losers? One possible explanation is that winners are likely to be involved in subsequent competitions. Relatively high testosterone may prepare them for this eventuality (Mazur & Booth, 1998). The lower testosterone levels in individuals who have lost a competition may encourage withdrawal from future competition, thereby avoiding injury or further loss in status (Mazur & Booth, 1998).

Human males also compete in non-physical ways. Presumably, this competition occurs for many reasons. However, one likely reason is that winning often increases one's status. In this context, it is not surprising that testosterone is also implicated in these non-physical aggression scenarios. It is also likely that testosterone levels rise with increased status. Mazur and Lamb's (1980) results that the testosterone levels of medical students were low during internship and rose following graduation (symbolic of success) suggest this. This pattern has also been demonstrated in participants of chess matches and reaction time contests (Mazur, Booth, & Dabbs, 1992; Mazur, Susman, & Edelbrock, 1997). One particularly interesting study conducted by Fielden, Lutter, and Dabbs (1994, as cited in Mazur & Booth, 1998) showed that fans of a winning team exhibited an increase in testosterone levels, while fans of a losing team showed a decrease.

5.2. Testosterone and mating

If mating success were the primary adaptive value of the positive relationship between testosterone and aggression, mated status would also be expected to moderate the relationship. Support for this comes from Mazur and Michalek (1995, as cited in Mazur & Booth, 1998) who found that testosterone levels fall and remain lower when a man gets married and subsequently rises if the man divorces. Under the competition theory that is developed here, this change in testosterone levels would occur because a man is again competing for mates and other resources.

5.3. Universality of the relationship

For a behavior to be considered adaptive, it is useful to show that it is exhibited in many different species; this has been demonstrated (Benton, 1992). It is also important to look at the relationship in a cross-cultural context. One study of 114 !Kung San men of Namibia (Christiansen & Winkler, 1992) found that individuals with relatively high testosterone had significantly more scars from interpersonal conflicts (mostly inter-male) than individuals with lower levels of testosterone. In other words, testosterone levels were higher in individuals who engaged in a disproportionately high number of fights with other males. Another cross-cultural study involved Caucasian and Metis young offenders who were labeled either violent or non-violent (Brooks & Reddon, 1996). In both ethnic groups, violent offenders had significantly higher testosterone levels than their non-violent counterparts (there were no differences between the two ethnic groups).

5.4. "Young male syndrome"

Daly and Wilson (1994) discussed what is known as the "Young Male Syndrome," referring to the fact that males between the ages of 12 and 25 are the principal perpetrators and victims of violence. Studies such as one by Mazur (1983) suggest that this may be due to the rapidly rising testosterone levels at precisely that age. Puberty also signals an increase in dominance promoting behavior, aggression, and inter-male competition. It has been suggested that since sexual "maturity" also occurs at puberty, the adaptive value of an increase

in testosterone at this period revolves around mating strategies (Hinde, 1986). According to Daly and Wilson (p. 277), males at this age "constitute the demographic class upon which there was the most intense selection for confrontational competitive abilities."

5.5. Summary

In the inter-male competition theory of the relationship between testosterone and aggression, testosterone is the mechanism that facilitates aggressive behavior. According to this perspective, individuals with testosterone levels that fluctuated in response to appropriate environmental cues would have been selected for in ancestral environments, if, and only if, the aggressive behavior incurred a fitness benefit. The above information suggests that the rise in testosterone and thus aggressive behavior at puberty coincides with a time of intense competition for mates and/or status. The proposed moderator variables, thus, provide support for an adaptationist viewpoint.

6. Predictions

First, it is predicted that the relationship between testosterone and aggression would be weak and positive based on the results from past meta-analyses and the expectation that the relationship is moderated by other variables (Archer, 1991). The rest of the predictions relate to the effects of those moderator variables. For example, it is predicted that the relationship between testosterone and aggression should decrease with increasing age. This is expected because of the range restriction in testosterone levels prior to puberty (Halpern, Udry, Campbell, & Suchindran, 1995) and in old age (Ehrhardt & Meyer-Bahlburg, 1981) relative to adolescents and young adults. In other words, because testosterone levels are very low prior to puberty and at old age, little variance in testosterone levels is expected to attenuate any existing relationship.

Another prediction was related to sex. Specifically, we expected females to show a much smaller relationship between testosterone and aggression than males, again, due to range restriction.

Time of measurement during the day was included as another predictor because testosterone levels fluctuate during the daily cycle. A priori, however, it was unclear what period of the daily cycle would have a significant influence on the relationship. Previous research by Dabbs (1990) is suggestive because testosterone levels in Dabbs' (1990) study were highest and most variable in the morning. Of course, in the situation where variances are unequal across different periods of the day, reliability and restriction of range may be relevant. Because of these issues, it would be valuable to determine whether it is best to measure testosterone in the morning, afternoon, or evening.

Offender status may also affect the observed relationship. It is sometimes assumed that offenders are more violent as a group than non-offenders and should exhibit a stronger relationship between testosterone and aggression. Offenders are, however, heterogeneous with respect to aggressiveness. For example, it is likely that offenders who have committed a very serious violent offense against persons in the context of a non-criminal lifestyle will

have lower testosterone levels than chronically aggressive offenders (e.g., Quinsey, Maguire, & Varney, 1983). Studies often do not discriminate between chronically aggressive offenders and those with only a violent admission offense.

Method of hormone measurement (using blood, urine, or saliva samples) was included because of Archer's (1991) suggestion that this is a relevant factor, although other research by Wang, Plymate, Nieschlag, and Paulsen (1981) is not supportive of this assertion. Specifically, Wang et al. found salivary and serum methods to be equally valid and that the two methods exhibit strong positive correlations with one another.

In contrast to method of hormone measurement, the method of measuring aggression was expected to be an influential moderator. Specifically, it was predicted that the relationship would be more robust (and more valid) for studies where aggression was measured behaviorally rather than through self-report, as is often done in studies using human subjects (Archer, 1991).

7. Method

7.1. Studies

One hundred and six articles were examined to determine the nature and strength of the relationship between testosterone and aggression. Articles were located utilizing a number of databases (PsycInfo, Biological Abstracts, General Science Index, and Current Contents), as well as through referrals from colleagues. Reference sections were also perused for additional studies to include. Aggression was broadly defined, involving competition, self-report measures of state and trait aggression (verbal and physical), as well as a number of behavioral measures (including past crime records, ratings of aggressive behavior from individuals who knew the participants fairly well, and experimental manipulations). Of the reviewed studies, 45 were included in the analysis. Most of the remaining articles were excluded due to a lack of information. Information necessary for inclusion in the meta-analysis included effect size information (or enough information with which to calculate effect size) and details on the method of testosterone and aggression measurement. Many studies stated that no significant relationship was found and then neglected to give specific statistics or probability levels. The exclusion of these studies, in particular, could affect the average effect size because most of them found non-significant effects. The other issue in sampling was the lack of unpublished studies in the analysis. Researchers do not tend to publish non-significant results. This is known as the "file-drawer phenomenon" (Wolf, 1986) and may affect the results as well.

Non-independence was another issue in this sample. In the case where two studies used data from the same participants, one was excluded from the analysis. In the between-group studies, the groups were usually defined as high versus low in aggression, or high versus low in testosterone. When groupings were otherwise, such as males versus females, it was determined that the study did not ask the same question, namely, "What is the relationship between testosterone and aggression?" In the case of males versus females, the question was whether testosterone levels were different in males and females and

Table 1
Sample size, effect size, and study characteristics of studies included in meta-analysis

Study/authors	Sample size	Effect size	Study characteristics
Bain, Langevin, Dickey, and Ben-Aron (1987)	28	−0.11	3 0 1 2 1 1
Banks and Dabbs (1996)	65	0.53	3 2 2 0 1 2
Berman, Gladue, and Taylor (1993)	202	0.42	2 0 0 3 ? 1
Booth and Dabbs (1993)	3928	0.13	4 0 0 2 1 1
Booth, Shelley, Mazur, Tharp, and Kittok (1989)	72	0.28	3 0 0 3 4 1
Brooks and Reddon (1996)	112	0.14	2 0 1 2 1 1
	65	0.15	2 0 1 2 1 1
Christiansen and Knussman (1987)	117	0.02	3 0 0 2 1 1
Christiansen and Winkler (1992)	11	0.25	3 0 0 2 1 1
Dabbs, Carr, Frady, and Riad (1995)	200	0.16	2 0 1 3 1 1
	490	0.21	2 0 1 3 1 1
Dabbs, Frady, Carr, and Besch (1987)	89	0.19	2 0 1 3 1 1
Dabbs and Hargrove (1997)	87	0.27	3 1 0 0 1 1
Dabbs, Hargrove, and Heusel (1996)	98	0.26	2 0 0 3 ? 1
Dabbs, Hopper, and Jurkovic (1990)	102	−0.03	? 2 0 0 ? 2
	57	0.06	? 2 0 0 ? 2
	63	−0.09	? 2 0 0 ? 2
Dabbs, Jurkovic, and Frady (1991)	113	0.20	2 0 1 3 1 1
Dabbs, Ruback, Frady, Hopper, and Sgoutas (1988)	20	0.58	? 1 1 0 1 2
Daitzman and Zuckerman (1980)	40	0.31	2 0 0 2 1 2
Ehrenkranz, Bliss, and Sheard (1974)	24	0.51	3 0 1 2 1 1
Elias (1981)	13	0.51	2 0 0 2 ? 1
Gladue (1991)	155	0.22	2 0 0 2 2 2
	150	−0.28	2 1 0 2 2 2
Gladue, Boechler, and McCaul (1989)	39	0.71	3 0 0 3 2 1
Gray, Jackson, and McKinlay (1991)	1679	0.04	4 0 0 2 1 1
Halpern et al. (1995)	127	0.40	2 0 0 2 3 2
Harris, Cook, Walker, Read, and Riad-Fahmy (1989)	60	−0.27	4 0 0 3 ? 2
Harris, Rushton, Hampson, and Jackson (1996)	155	0.36	3 0 0 3 1 2
	151	0.41	3 1 0 3 1 2
Houser (1979)	5	0.11	3 0 0 2 ? 2
Kreuz and Rose (1972)	21	−0.16	3 0 1 2 1 1
Lindman, von der Pahlen, Öst, and Eriksson (1992)	55	0.16	4 0 2 2 1 1
Mattsson, Schalling, Olweus, Low, and Svensson (1980)	16	0.07	2 0 0 2 1 2
Mazur et al. (1997)	28	0.37	2 0 0 3 3 1
	32	0.40	2 1 0 3 3 1
Meyer-Bahlburg, Nat, Boon, Sharma, and Edwards (1974)	11	0.04	3 0 0 2 1 2
Monti, Brown, and Corriveau (1977)	101	−0.01	3 0 0 2 1 2
Olweus (1986)	58	0.33	2 0 0 2 ? 2
Olweus, Mattsson, Schalling, and Low (1988)	58	0.35	2 0 0 2 ? 2
Olweus, Mattsson, Schalling and Low (1980)	58	0.22	2 0 0 2 1 2
Orengo, Kunik, Ghusn, and Yudofsky (1997)	13	0.33	4 0 0 2 1 1
Paikoff, Brooks-Gunn, and Warren (1991)	72	0.40	1 1 0 2 1 2
Persky et al. (1971)	18	0.52	3 0 0 2 1 2
Quinsey (unpublished)	219	0.01	2 0 0 3 ? 2
	107	−0.13	3 0 0 3 ? 2

(continued on next page)

Table 1 (*continued*)

Study/authors	Sample size	Effect size	Study characteristics
Rejeski, Gagne, Parker, and Koritnik (1989)	20	0.88	2 0 0 2 2 2
Salvador, Simón, Suay, and Liorens (1987)	39	0.20	2 0 0 2 4 1
Scaramella and Brown (1978)	14	0.28	3 0 0 2 2 2
Scerbo and Kolko (1994)	40	0.40	1 2 0 3 1 2
Schaal et al. (1996)	178	0.22	2 0 0 3 4 1
Susman et al. (1987)	55	0.06	2 0 0 2 1 2
	52	− 0.21	2 1 0 2 1 2
Udry (1990)	101	0.29	2 0 0 2 3 1

Sample size indicates the sample used for the effect size estimate. This may differ from the overall sample size for a study. Effect size is Rosenthal's *r*-value. Coding of study characteristics (age, sex, offender status, hormone measure, time, and aggression measure). For all variables where information was not supplied a "?" has been inserted.

Age: 1 = under 13, 2 = 13–21, 3 = 22–35, 4 = over 35; *Sex*: 0 = male 1 = female, 2 = male and female; *Offender status*: 0 = nonoffenders, 1 = offenders, 2 = mixed group; *Hormone measurement*: 1 = saliva, 2 = urine, 3 = blood; *Time of measurement*: 1 = 6 a.m. to 12 p.m., 2 = 12 p.m. to 5 p.m., 3 = 5 p.m. to 12 a.m., 4 = throughout the day; *Aggression measure*: 1 = behavioral, 2 = self-report.

whether aggression was different. These types of studies did not look at the relationship between the two variables.

The combined sample size for the 45 included studies was 9760. Table 1 presents a list of study characteristics and effect sizes. It should be noted that all effect sizes (*r*, *t*, or *F*) were transformed into standardized correlations in order to complete all analyses. The values in the table are the transformed values.

7.2. Coding

Studies were coded on several characteristics by independent raters. Inter-rater reliability was high for most of the variables and adequate for a few (Table 2). The characteristics included sample size, mean age of participants, sex of participants, offender status of

Table 2
Inter-rater reliability for coded variables

Variable	Test	Value
Time of hormone measurement	r_s	1.00
Aggression measure	κ	0.70
Sex of participants	r_s	0.76
Age	r	0.98
Offender status	r_s	0.88
Method of hormone measurement	r_s	0.80
Effect size	r	0.96
Sample size	r	0.99

participants, measure of testosterone, time of hormone measurement, mean and standard deviation of testosterone, aggression measure, type of aggression measure, aggression mean and standard deviation, observed effect size, and the significance level. Table 1 lists the codes given for each particular study.

7.3. Analysis

The independence of individual effect sizes was considered before the analyses. Studies listed up to 14 different effect sizes. Where these effect sizes were based on information from the same subjects, an average was calculated. While this is not always the preferred approach, there was no objective way to choose between the different effect sizes. Often, the measures within studies tended to assess the same construct. Where multiple effect sizes were found to be independent (i.e., based on separate samples of individuals), they remained in the analysis on their own. A total of 54 independent effect sizes were included in the analysis.

The meta-analysis was conducted using methods outlined by Rosenthal (1991) and Wolf (1986). A Fisher's r to z transformation was performed before the analyses were undertaken.

8. Results

8.1. Effect sizes

The aggregate unweighted mean correlation across all the included studies was 0.230. When effect size was weighted by number of participants, the mean correlation was significant ($Z_c = 13.730$, $p < 0.01$), but of smaller magnitude ($r(53) = 0.140$). Table 3 gives additional descriptive information for the weighted and unweighted solutions. Out of 54 effect sizes, 45 were in a positive direction (Fig. 1). In other words, a positive relationship was observed in 83% of the cases.

Table 3
Descriptive statistics for sample of effect sizes

Effect sizes (Z_r) unweighted	
Mean	0.226
Confidence interval	0.157 to 0.296
Median	0.215
Standard deviation	0.258
Effect sizes (Z_r) weighted	
Mean	0.141
Confidence interval	0.138 to 0.144
Median	0.130
Standard deviation	0.149

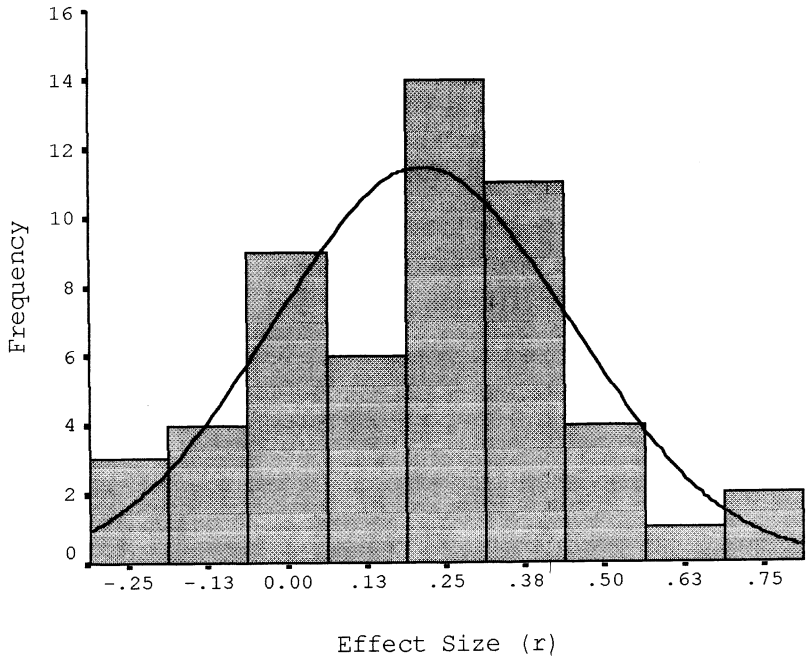

Fig. 1. Histogram for unweighted effect sizes (Z_r).

8.2. Effects of moderator variables

Before an analysis of possible moderators was carried out, the heterogeneity of the effect sizes was determined. The set of effect sizes was found to be heterogeneous ($\chi^2(54) = 202.778$, $p < 0.01$). Because heterogeneity was evident, it was possible to search for moderators and to describe their effect on the correlation between testosterone and aggression.

Moderating variables were identified using chi-square analyses because of the large sample size. Had parametric techniques been utilized, all statistics would have been significant. A number of variables were tested in regard to the number of expected and observed significant correlations.

The effect of the time at which hormones were sampled was significant ($\chi_{(2)}{}^2 = 10.426$, $p = 0.015$) (Fig. 2). Thirty studies measured testosterone in the morning (between 6 a.m. and 12 p.m.). Twenty-six of these yielded positive effect sizes. The unweighted mean effect size was 0.200 ($s = 0.210$). Effect sizes ranged from -0.210 to 0.680. The distribution appeared relatively normal ($z_{skew} = 0.690$, $z_{kurt} = -0.090$). Five of the six studies measuring testosterone in the afternoon (12 to 5 p.m.) found positive relationships. Effect sizes ranged between -0.290 and 0.910. The unweighted mean was 0.450 ($s = 0.460$). This distribution displayed the characteristics of normality ($z_{skew} = 0.78$, $z_{kurt} = -0.25$). All four of the studies measuring testosterone in the evening (5 p.m. to 12 a.m.) yielded positive values. These ranged from 0.300 to 0.420. The unweighted mean was 0.380 ($s = 0.060$). The distribution was relatively normal ($z_{skew} = -0.640$, $z_{kurt} = 0.998$).

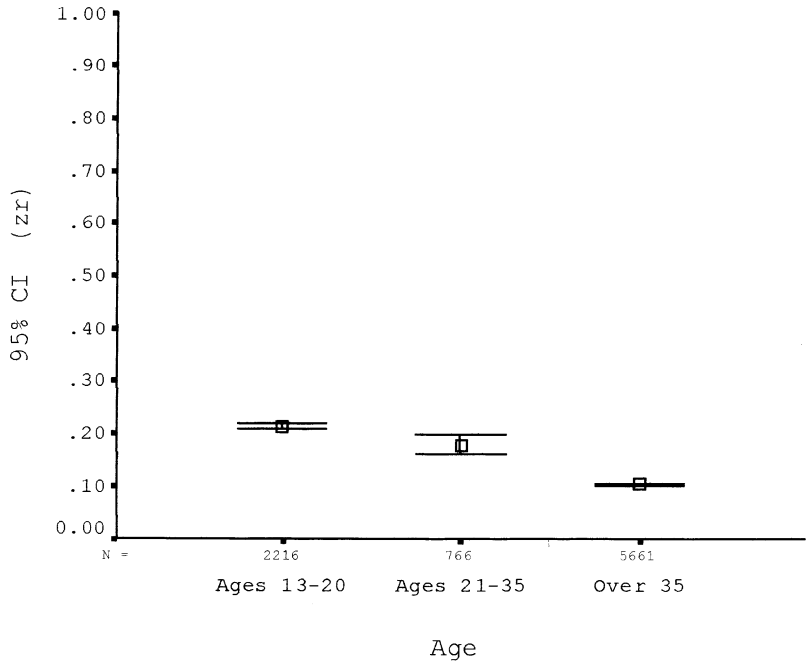

Fig. 2. Relationship between testosterone and aggression in males moderated by age.

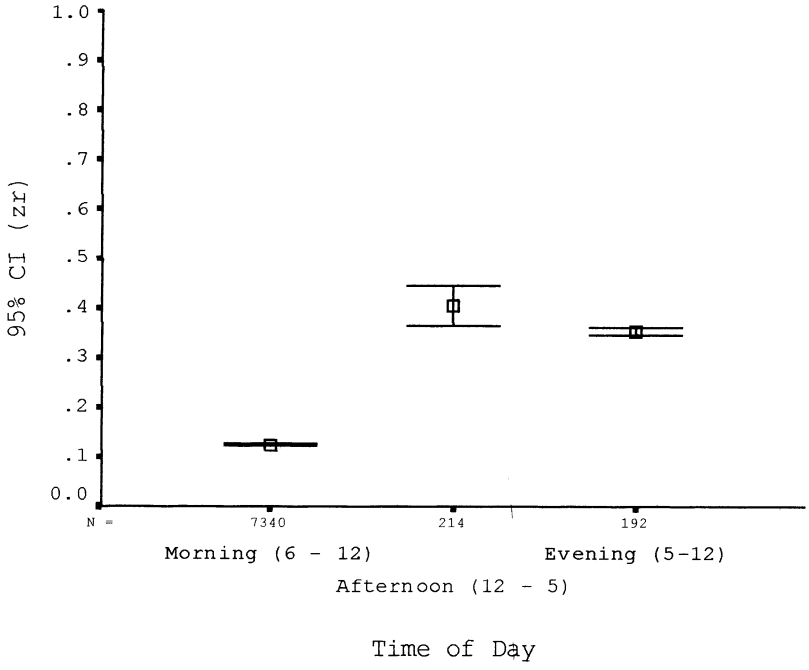

Fig. 3. Relationship between testosterone and aggression in males moderated by time of day.

Table 4
Chi square statistics for testing of moderator variables

Variable	*N*	χ^2	*df*	Significance
Age	52	8.591	3	0.035
Time of day	43	10.426	3	0.015
Sex	54	1.461	2	0.482
Offender status	54	3.267	2	0.195
Hormone measure	54	0.867	1	0.352
Aggression measure	54	0.564	1	0.453

N refers to the number of effect sizes included in the analysis. Where effect sizes were excluded, the appropriate data were missing or vague.

As mentioned previously, it was predicted that the relationship between testosterone and aggression would be moderated by age, but only for males. Thus, separate analyses were carried out for males and females. Age was shown to influence the effect size in males ($\chi^2(2) = 6.995$, $p = 0.030$) but not in females ($\chi^2(2) = 3.733$, $p = 0.155$). The change in the relationship for the different age groups is displayed graphically in Fig. 3. In the 23 studies examining males aged 13 to 20, the average unweighted effect size was 0.277 ($s = 0.191$). Effect sizes ranged from 0.010 to 0.910, meaning that all effect sizes were in the positive direction. The distribution was positively skewed ($z_{skew} = 3.570$) and leptokurtic ($z_{kurt} = 4.880$). When effect sizes were weighted by sample size ($N = 2216$), the average effect size was a bit lower ($Z_r = 0.210$, $s = 0.120$). In the 13 studies involving participants ages 21 to 35, the average unweighted effect size was 0.210 ($s = 0.320$). The effect sizes ranged from -0.160 to 0.890. Out of the 13 effect sizes, 9 were in a positive direction. The distribution of effect sizes was found to be normal ($z_{skew} = 1.330$, $z_{kurt} = 0.060$). When the values were weighted by sample size ($N = 766$), the mean effect size was, again, lowered ($Z_r = 0.180$, $s = 0.270$). Finally, seven studies used participants over 35 years of age. Six of these yielded positive effect sizes. The unweighted mean effect size was 0.080 ($s = 0.190$). Effect sizes ranged from -0.280 to 0.340. The distribution was normal ($z_{skew} = 1.270$, $z_{kurt} = 1.820$). The effect size increased when studies were weighted by sample size ($N = 5661$, $Z_r = 0.100$, $s = 0.050$).

Sex, offender status, method of testosterone measurement and the way in which aggression was measured did not affect the observed relationship significantly (Table 4).

9. Discussion

9.1. Overall relationship

The finding of a weak, positive correlation between testosterone and aggression is consistent with past meta-analyses (Archer, 1991). However, it should be noted that only studies that reported a *p*-value or effect size were included in the analyses. A possible consequence is that this sample underestimates the proportion of non-significant findings in the population.

9.2. Moderator variables

Two variables were found to influence the size of the relationship between testosterone and aggression: (1) age of participant and (2) time of day that the testosterone was measured.

Age affected the relationship significantly. Specifically, the effect size was largest in the 13- to 20-year-old males, declining with age. This is not surprising, as that is when the testosterone and aggression curves both peak (Olweus, 1986). Prior to puberty and later in life, testosterone levels are lower. This possible range restriction in testosterone levels and/or aggression could result in attenuation of effect size. Evolutionary considerations lead to the expectation that the relationship would remain constant over the lifespan. Because testosterone and aggression appear to follow the same timeline, the relationship between the two should not vary over time except for the influence of confounding variables, such as range restriction in testosterone levels.

Why would it be advantageous to have a decrease in testosterone later in life, if higher levels lead to more dominance-promoting behavior? According to evolutionary psychology, an individual has a finite amount of energy that can be directed toward mating effort, survival, or parenting (Low, 1998). It is possible that as a male ages, he is more likely to have mated and accumulated offspring and will be expending more energy in parenting, (thus, less will be available for mating tactics). In support of this, Mazur and Michalek (1995, as cited in Mazur & Booth, 1998) have shown that mated status corresponds to a decrease in testosterone levels. Age appears to confound the relationship between testosterone and aggression in males. Researchers should take advantage of the increased variability following puberty, as this may give a clearer picture of the magnitude of the relationship. It was expected that females would not show the same age confound because female testosterone levels do not fluctuate as dramatically over the lifespan. As expected, age did not affect the relationship between testosterone and aggression in females.

The time of day that the testosterone was measured was found to be a significant moderator in the relationship between testosterone and aggression. This is not surprising given the diurnal variation in the reliability of testosterone levels (Dabbs, 1990). What is surprising is that so many researchers measure testosterone in the morning, when the testosterone levels of individuals are most variable. On any given trial, then, the level of testosterone that is observed could change markedly. This would tend to attenuate the magnitude of the observed relationship. For males, the observed relationship between testosterone and aggression is highest in the afternoon, even though the mean level of testosterone is lower. This could be due to increased reliability of the testosterone measurement.

Offender status did not affect the relationship significantly. However, it is important that many offenders are not violent and, as mentioned, researchers have often used the violence of the admission offence to create the aggressive–nonaggressive typology, without taking into account the offenders' histories (Quinsey et al., 1983).

That sex did not affect the relationship between testosterone and aggression attests to universality. Just because females have less testosterone and exhibit less aggressive behavior does not mean that the relationship does not exist. In fact, the correspondence of low testosterone and low aggression fits with the proposed relationship. It is higher levels of testosterone that lead to more aggressive behavior. What is strange is that the lower

testosterone levels did not create the same range restriction problem that occurred with the different age groups in males; correlations between testosterone and aggression in females should have been attenuated by a lack of variation between subjects. Perhaps it is easier to get accurate testosterone measurements in females (less diurnal variation?). This type of phenomenon may counteract the range restriction.

As expected, method of hormone measurement did not alter the effect size. Thus, researchers are free to choose the more economical and easier to use method of measurement (saliva). All methods of hormone measurement appear to be equally reliable (Dabbs, 1990).

The final moderator that was examined was type of aggression measure. Contrary to the prediction, the nature of the measure of aggression (behavioral or self-report) did not have any effect on the relationship between testosterone and aggression. This is good news for researchers because it is much easier to administer a paper and pencil test than to observe behavioral indicators of aggression. This finding may also support Mazur and Booth's (1998) contention that the relationship is actually between testosterone and dominance. This is an interesting idea and should be developed further. Future research could involve posturing and threats as alternatives to aggressive behavior.

In summary, there were two possible moderators in the relationship between testosterone and aggression: participant age and time of day. It is possible, however, that the effects of age and time of testosterone measurement are artifactual. More research needs to be conducted examining the reliability of testosterone measurements at different ages and times of day to permit estimates of statistical power. As well, the effect of time of day and age could be clarified via repeated measurement and longitudinal designs.

Future research might profitably address the evolutionary hypotheses concerning inter-male competition by concentrating on inter-male aggression in competitive situations, especially in the presence of females. This research might be informed by the finding of a time lag in which testosterone levels most accurately predict aggressive behavior 6 h prior to its measurement (Van Honk et al., 1999). One final issue deserving of investigation is the reciprocal nature of the relationship between testosterone and aggression, already established in non-human animals (Archer, 1991; Mazur, 1985; Mazur & Booth, 1998).

Acknowledgments

We wish to thank Grant Harris, Martin Lalumière, and Marnie Rice for their comments on an earlier version of this paper, Martin Daly, Lee Fabrigar, Kevin Parker, and John R. Reddon for their valuable advice, and Kusum Sharma for assisting with collection of the data. This research was supported by a contract from the Kingston Psychiatric Hospital and a Senior Research Fellowship from the Ontario Mental Health Foundation to the third author.

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