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Testosterone Intake and Aggressiveness: Real Effect or Anticipation?

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In a double-blind experiment, human males (n = 27) were given either testosterone (40 mg/day), placebo, or no treatment, over a one week period. Subjective and observer assessed mood estimations were conducted before and after treatment. Testosterone levels in saliva were measured with radioimmunoassay. The results revealed a significant placebo effect [c.f. Medicine and Science in Sports 4:124–126]: After treatment, the placebo group scored higher than both the testosterone and the control group on self-estimated anger, irritation, impulsivity, and frustration. Observer-estimated mood yielded similar results. The lack of a placebo effect in the testosterone group is intriguing, and may be due to secondary effects caused by suppression of the body's own testosterone production, since recorded non-protein bound testosterone did not significantly rise due to treatment. The results suggest that androgen usage causes expectations, rather than an actual increase of aggressiveness.

Key words: testosterone, aggression, radioimmunoassay, mood, steroids

INTRODUCTION

Reports of homicide committed by sportsmen abusing anabolic steroids are, every now and then, presented in the news media. Such cases are typically described as having no apparent motive and as accompanied by uncontrolled fits of rage. In 1984, a Finnish athlete who killed his girlfriend blamed the usage of steroids for his irrational behavior. That same year, a steroid-using Swedish athlete killed his best friend, and on December 6, 1992, a TV news report on TV announced that a Swedish steroid abuser had shot and injured six people. These are but examples, and similar instances can easily be found in countries outside Scandinavia.

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The media present the connection between the use of steroids and aggressiveness as if it were a well established fact. The press and TV, naturally, do not report abusers not killing or not becoming aggressive. Likewise, when non-abusers commit homicide or some other violent crime, the prefix "non-abuser" is not contained in the report. Negative cases and false positives never pass the news threshold and thus, every reported incident earns disproportionate coverage. It has never been shown that athletes abusing steroids commit homicide significantly more often than nonabusing athletes, and even if this were true, it would not be proof of a cause-effect relationship.

Controversy over the use of anabolic steroids has unquestionably created a great dilemma in the world of sports. Already two decades ago, Cooper [1972] reported that 80% of weight lifters, javelin throwers and shot-putters regularly used illegal steroids. Recent cases, such as Ben Johnson and Kristina Krabbe, indicate that steroid usage is now common among short distance runners, too. Whether steroid intake really improves performance has not been scientifically established, although sportsmen seeking fast recovery after intensive training and rapid muscle development seem convinced that it does [e.g., Lucking, 1982; Perry et al., 1990]. In a double-blind experiment, Freed et al. [1975] found a slight improvement in the performance of weight lifters. The only change in physical performance Hannan et al. [1991] could establish was an improvement in the first trial of a pegboard task. Ariel and Saville [1972] found placebo to be more effective than steroids.

The relationship between testosterone levels and aggression in subhuman species has been studied extensively, and findings suggest a connection between the two [for reviews see, for instance, Bouissou, 1983, and Brain, 1977]. Castration is known to reduce physical aggression among animals, including subhuman primates [Bernstein et al., 1983]. There are, however, good reasons to believe that levels of testosterone are primarily related to social status and dominance. Rejeski et al. [1988] gave injections of testosterone to an experiment group of male cynomolgus monkeys, while a control group received sham injections. Although the administration of testosterone resulted in a significant increase in aggression, more important was the finding that changes in behavior are mediated by social status; the incidence of both contact and noncontact aggression in dominant monkeys was far greater than the frequency of these behaviors in subordinate monkeys [Rejeski et al., 1988].

As far as humans are concerned, the question of whether the level of testosterone in males is related to aggression is more open to debate. While some studies indicate a relationship, others do not. For example, Olweus et al. [1980] found a correlation between unbound plasma testosterone and certain aspects of aggressiveness and impulsivity. Ehrenkranz et al. [1974] using prisoners as subjects, found plasma testosterone to be correlated not only with aggression, but with social dominance, too. Rada et al. [1974] investigated plasma testosterone levels in rapists but were cautious about interpreting their results as indicating a causal relationship with rape. Lindman et al. [1992] did not find a correlation between serum testosterone level and aggression in males arrested for spouse abuse, neither at the time of arrest nor in a sober control state.

These examples show that some studies do indicate a relationship between testosterone and aggressiveness, others have failed to find a correlation. It is well known that positive results are more readily published than null findings, and many researchers do not even bother to submit manuscripts reporting nonconfirmed hypotheses. Accordingly, many studies investigating the testosterone-aggression relationship yielding a negative result are likely never to have been submitted and/or published.

In view of the inconsistent findings of research on this topic, the conclusions drawn by reviewers also tend to be in disagreement. While some claim that a connection can be established [e.g., Donovan, 1985], others [such as Benton, 1992] are of the opinion that, on the basis of existing data, there is no reason to suggest that human aggression is related to level of testosterone. Benton [1983a, 1983b] emphasized that the claim of a relationship between testosterone and aggression is based primarily on animal data. Extrapolations from animal to human behavior are questionable, since, among humans, social and cognitive mechanisms play a much greater role than physiological factors. The closer animal is to man on the phylogenetic scale, the smaller the influence of testosterone on aggression. Benton's [1992] conclusion is that in man, aggressive behavior is a reflection of psychosocial history, and differences in aggressiveness can be attributed to the level of testosterone only to a very limited extent, if at all.

If Benton is correct—and the present authors think he is—then the main source of variation in aggressiveness among humans is really to be found in the psychohistory of the individual. What, then, is the explanation for the immense interest in the testosterone-aggression connection? Is it primarily a reflection of hardy attitudes about the aggressive male and the submissive female, still prevalent within our society?

If males within subhuman vertebrate species are more aggressive than females (which is not true for all species) [Adams, 1992], among humans, this seems to be reflected only in greater *physical* aggression. In regard to direct verbal aggression, human females are as aggressive as males, and with respect to indirect aggression, even more so [Björkqvist et al., 1992a; Björkqvist et al., 1992b; Lagerspetz et al., 1988]. The greater amount of physical aggression among males is more likely to be a consequence of learning mechanisms and greater physical strength, rather than of testosterone [Björkqvist and Niemelä, 1992].

Most research on aggression is conducted by men, with male subjects, perhaps resulting in a biased, "male" perspective on the subject. For instance, Frodi et al. [1977] found that of 314 experimental studies on aggression, 54% were concerned exclusively with men, and only 8% could be found specifically investigating aggression in women.

Another revealing example is the different interest aroused by two experiments conducted by Edwards [Edwards, 1969, and Edwards and Herndon, 1970]. In Edwards [1969], it was reported that female mice treated with androgens at birth were more aggressive as adults. This result is well-known and well-publicized. The second study [Edwards and Herndon, 1970] showed that newborn female mice treated with *estrogen* at birth also fought more as adults! This finding has gone almost unnoticed, since it is not in accord with the generally accepted.

The aim of the present study was to investigate whether there is any truth in the reports of increased irritability and aggressiveness in males after steroid intake [Perry et al., 1990]. The study was a double-blind experiment. The subjects, university sportsmen who volunteered for the experiment, were divided into three groups: testosterone, placebo, and no treatment. Testosterone was administered perorally for one week. The dose (40 mg/day) was well within the levels administered for therapeutic reasons and, taken for such a limited period, the treatment could not possibly cause the subjects physical harm. The subjects were, however, under strict medical supervision for the duration of the experiment.

There are findings suggesting that it is the level of non-protein-bound testosterone which is decisive in behavioral manifestations [cf. Vermeulen and Verdonck, 1972]. Unbound testosterone constitutes 1-4% of the total level [Sannikka et al., 1983]. In the

present research, unbound testosterone levels were measured with radioimmunoassay of the saliva, before and after the treatment period [Furugyama et al., 1970; deGomez et al., 1981; Ismail et al., 1972]. This method offers a more benign way of measuring testosterone than blood sampling. Measurements in saliva correlate highly with the non-protein-bound serum testosterone level: .75 according to Sannikka et al. [1983], and .94 according to Wang et al. [1981].

In the present experiment, pre- and-post treatment behavior, mood, and physical fitness were assessed by multiple measures, including self-estimated and observer-evaluated mood, observer-estimated behavior, and interviews. Each subject was observed in a group situation with two other subjects. All sessions were videotaped. In this paper, only the effect of treatment on mood will be reported in detail.

MATERIALS AND METHODS

Subjects

Twenty-seven university students, all males, and all but one active in sports, participated in the experiment. Most of them were members of the sports association of Åbo Akademi University, Turku, Finland. All had volunteered for the experiment, and received a remuneration of 100,- FIM for their participation. Their mean age was 23.9 years (age range 21–31 years). The mean weight of the subjects was 73.6 kgs (range 62–88 kgs), and the mean height was 180.3 cm (range 170–197 cm). None of them admitted to having had pesonal experience of androgens or other steroids. Subjects were divided into three treatment groups: *testosterone, placebo,* and *control,* on the basis of matched levels of testosterone in the saliva. The main criterion for selection was that the testosterone level of the three groups should correspond as closely as possible. The physician conducting a medical examination to ascertain that there were no medical objections for participation in the experiment was responsible for the selection of subjects. Psychological counterindications were also checked by interview and personality assessment. Of 30 volunteers, three were rejected. Every precaution possible was taken, both prior to and during the experiment, to ensure that subjects would come to no harm. The actual experimenters were ignorant as to which group each subject belonged, in accordance with the double-blind design of this experiment.

Sessions

The subjects attended three sessions in the laboratory: These will be referred to as sessions I, II, and III, respectively. Sessions I and II were separated by 2 to 3 weeks, and the intermediate period between sessions II and III was 10 days. Testosterone/placebo intake began exactly 7 days before session III. Session II delivered pre-treatment data, and post-treatment data were obtained from session III.

Testosterone and Placebo

The administered testosterone consisted of 40 mg pills of Panteston®, produced by Oregon. Panteston is administered perorally, i.e., it has to be kept under the tongue until dissolved, it is not to be swallowed. Administered perorally, it works through the lymphatic circulation and is not broken down by the liver. The placebo pills were calcium carbonate pills which were administered in the same way.

Radioimmunoassay (RIA) of the Saliva

Subjects delivered 5 ml saliva into a test tube after first rinsing their mouth with pure water and then chewing on a parafilm in order to stimulate the secretion of saliva. The delivery always took place at the same time (19.00) in order to avoid differences due to circadian rhythm. The samples were immediately frozen and stored until the time of actual analysis. Analysis was conducted according to the method described by Sannikka et al. [1983].

The Self-Estimated Mood Check List

The mood check list utilized successive magnitude scales [Lindman, 1985]. Subjects were requested to indicate the degree to which they confirmed experiencing certain moods (e.g. relaxed) on a straight 100 mm line, with zero confirmation at one end, and 100% confirmation at the other.

Twelve items (expressing mood) were included: Relaxed, irritated, excited, angry, indifferent, interested, frustrated, elated, nervous, impulsive, self-confident, and active.

Observer-Evaluated Mood

All sessions, except for the medical examination, were videotaped for the subsequent assessment of subjects' mood during the session by two independent observers. The observers were unaware of the group to which subjects belonged. A similar successive magnitude scale and the same 12 items as in the self-evaluated mood check list were used. The inter-rater reliability was .86 ($P < .01$).

Session I

The sole purpose of the first session was to investigate the psychological and medical suitability of those volunteering for the experiment. Session I consisted of four parts: 1) delivery of saliva for the RIA test, 2) a medical examination, 3) an interview, 4) personality assessment.

The purpose of the saliva sample was to establish baseline testosterone data for allocation into groups. The purpose of the medical examination was to investigate medical suitability, while the interview and the personality assessment were conducted in order to test for psychological counterindications to participation in the study. Three subjects were excluded on basis of medical and psychological grounds. The whole session, except for the medical examination, was videotaped.

Session II

The subjects were taken to the laboratory in groups of three, each from a different treatment (testosterone/placebo/no treatment) group. The session consisted of two parts: 1) completion of the self-estimated mood check list, to obtain pre-treatment data on self-estimated mood, 2) a physical performance test. The whole sesson was videotaped for subsequent observer assessment of subjects' mood. Physical performance was evaluated during session II, for comparison with post-treatment performance during session III, by exercises demanding physical strength as well as fine motor skills. Subjects started taking testosterone/placebo 3 days after session II, and 7 days before session III.

Session III

Subjects attended session III in a different group of three to that of session II. The session included 1) delivery of saliva for RIA (at exactly 19:00), 2) completion of the

self-estimated mood check list (post-treatment data), 3) a physical performance test (post-treatment data), 4) completion of the mood check list a second time (post-treatment, after-exercise data), 5) an interview. The whole session was again videotaped, and the mood of subjects subsequently evaluated by observers.

RESULTS

Effects on Self-Evaluated Mood

The results were somewhat surprising: A significant placebo effect was found on four (one third) of the twelve items of the self-evaluated mood check list, but no similar effect was obtained for the testosterone group! Subjects belonging to the placebo group experienced themselves as more angry [$F(2,23) = 6.01$, $P < .01$], frustrated [$F(2,23) = 5.85$, $P < .01$], impulsive [$F(2,23) = 4.30$, $P < .02$], and irritated [$F(2,23) = 6.99$, $P < .01$]. Pre and post data of two of these items (angry and irritated) are presented as examples in Figures 1 and 2.

Effects on Observer-Evaluated Mood

Observer-evaluations were consistent with the self-estimated data, thus supporting the validity of the findings. For example, observer-evaluated frustration revealed a sig-

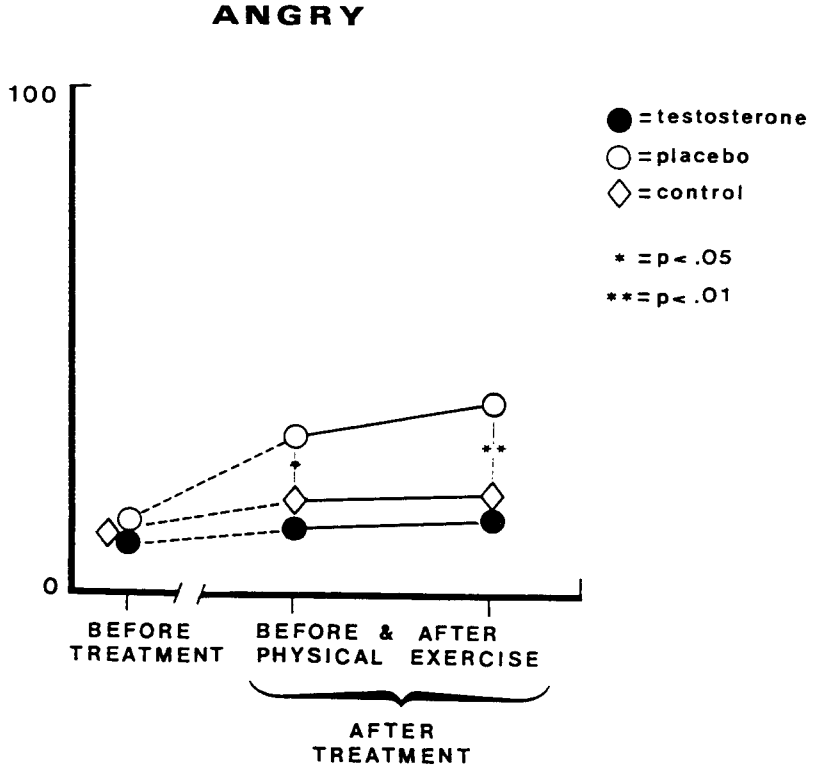

Fig. 1. Self-evaluated anger before and after treatment in the three experimental groups.

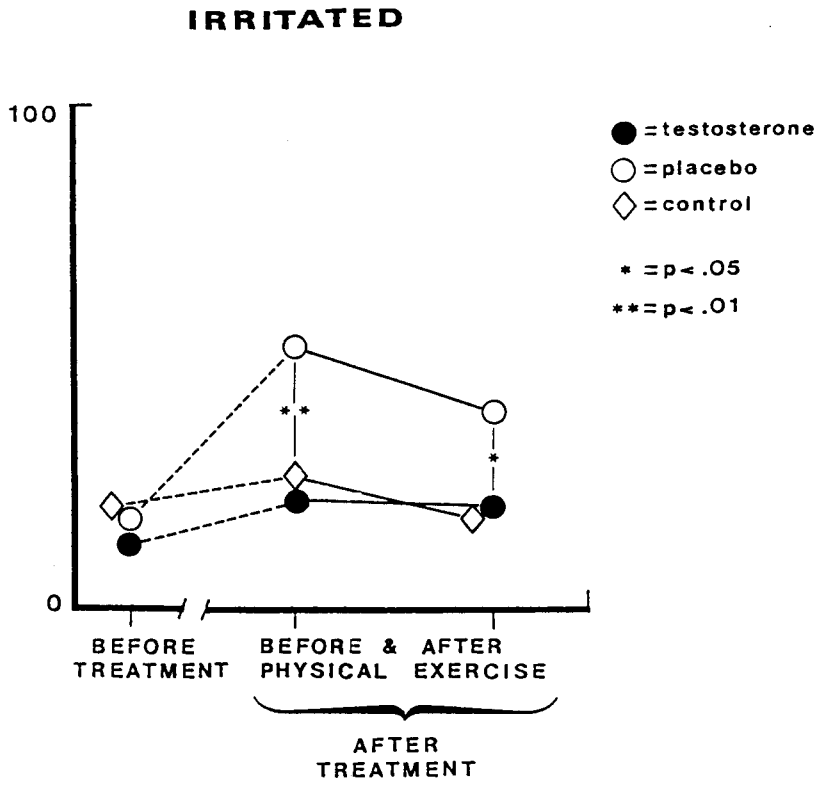

Fig. 2. Self-evaluated irritation before and after treatment in the three experimental groups.

nificant placebo effect [$F(2,23) = 7.05$, $P < .01$]. The remaining items also revealed group differences consistent with self-estimated mood, but these were, however, not significant.

When observer-evaluated anger, irritation, and frustration were added together to create a summed variable (the internal consistency of which was $\alpha = .90$), the summed variable revealed a significant placebo effect [$F(2,23) = 4.05$, $P < .05$], too. Why were group differences based on observer-evaluation scores smaller than group differences based on self-estimations? Subjects were aware of the fact that they were being videotaped: they may have attempted to hide their emotions from the camera. In other words, they may have been camera-shy.

Interviews

Data obtained in the interviews revealed the same trends. For instance, one individual belonging to the placebo group expressed himself with the following typical words: "I have, during this week, felt touchy and irascible, I got irritated almost for no reason. I was very tired at the beginning of the week. I got mad at others, and it showed. I'm not usually like that. My physical training has been more successful than ever. I felt self-confident in competition, which is unusual for me."

RIA Data

No significant between groups differences in testosterone level was found after 1 week's treatment. The testosterone group had slightly increased levels, but not more than might be explained by chance fluctuation.

Physical Performance

There were no between groups differences in physical performance.

DISCUSSION

The results demonstrate a clear placebo effect: Subjects, although they denied this in the interview, obviously expected to become more irritable, frustrated and angry. What is surprising and calls for an explanation, is the absence of a placebo effect in the group receiving testosterone. This finding is so consistent that it is difficult to conceive of it as a coincidence. Although the present authors do not claim to have an explanation for this finding, the following suggestion is offered: Contrary to expectation, testosterone levels did not differ significantly between the groups after 1 week's treatment. What may have happened is that the artificially induced testosterone suppressed the body's own production of the steroid: If the treatment had been carried out for a longer period, or larger doses had been administered, differences in testosterone would probably have appeared. The suppression of the body's own production may have had secondary psychological effects, depressing excitement and activation which, in turn, balanced out the activating placebo effect.

It is worth noting that our results are consistent with those of Ariel and Saville [1972], who also found a significant placebo effect, but no effect of steroid intake. However, unlike the present study, they examined the effect of steroids only on physical performance, and not on mood.

One has to remember that steroid dosage taken by athletes is considerably larger than that administered in the present experiment, or that which could be prescribed for therapeutic or experimental reasons. Athletes also continue steroid treatment for long periods and without medical supervision. The question whether steroid abuse causes uncontrollable mood change and aggression is still not settled: Hormonal variation may, or may not, be related to significant mood change. A parallel may be drawn to the debate about the premenstrual syndrome [Parlee, 1973], or mood change due to steroid based contraceptives. It is possible that sensitivity to intake of artificial steroids leading to unpleasant mood changes is subject to individual variation.

Two recent studies investigating the relationship between anabolic-androgenic steroid use and mood change [Anderson et al., 1992; Bahrke et al., 1992] yielded the same result as the present experiment: Steroid intake did not increase aggressiveness in human males. A third double-blind study [Hannan et al., 1991] did find increased aggressiveness, even more so in high-dose groups, with the "hostility" and "resentment and aggression" subscales of the MMPI as dependent measures. Two field studies [Brower et al., 1991; Perry et al., 1990] found that sportsmen abusing steroids reported increased aggressiveness. It should be borne in mind, however, that the last two studies were not double-blind experiments, and the reports may be explained as placebo effects. A review by Uzych [1992] does not exclude the possibility of increased aggressiveness after steroid abuse, and another by Bahrke et al. [1990] suggests that

psychological effects are variable, and related to type (17 alpha-alkalated rather than 17 beta-esterified), but not dose, of anabolic-androgenic steroids administered. The results of the present and other double-blind experiments suggest however that much of the effect of steroid intake is really placebo. To claim that aggression is caused by steroids, as in the news media, is misleading and dangerous for several reasons: 1) There is still no scientific evidence for such a relationship. 2) Even if a correlation were found between steroid abuse and aggressiveness, this would not prove a cause-effect relationship. Factors, such as unstable personality may be the source of willingness to abuse steroids as well as aggressiveness. Steroid abuse may, for some, be a mediating factor enhancing aggressive tendencies by producing states of elated emotionality. A parallel may be drawn to the complexity of problems encountered in establishing a causal link between alcohol intoxication and aggression [Brain, 1986; Evans, 1986]. 3) Dissemination of the myth of the steroid-aggressiveness connection may lead to anticipation (a placebo effect) of aggressiveness among steroid abusers and, in turn, to actual acts of violence. It may, in fact, work as an excuse for aggression.

Abuse of anabolic steroids may be addictive [Brower et al., 1991]. Abuse is dangerous for medical reasons, and increased vulnerability for cardiovascular disease has been observed [Cheever and House, 1992]. The problem of "increased aggressiveness" is really of minor importance. The disproportionate media coverage of the phenomenon appears to be a reflection of attitudes within our society, the cherished myth of the aggressive male, stuffed with androgens, and the submissive female.

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