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Anabolic-Androgenic Steroids: A Series of Meta-Analyses

John Cochrane Spence

A Thesis

in

The Special Individualized

Program

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Abstract

Anabolic-androgenic steroids: A series of meta-analyses

John C. Spence, Ph.D.
Concordia University, 1997

The present study attempted to quantitatively summarize the psychological, physiological, and ergogenic effects of anabolic-androgenic steroids (AAS)s in healthy humans. Using the format of a meta-analysis, data from 127 studies and 35 outcomes was converted into 894 effect sizes and summarized across studies. In order to account for any significant variability within effect sizes, each finding was coded for 15 moderator variables. Unfortunately, due to a lack of adequate findings, no psychological outcome was included in the analysis.

As indicated by the magnitude of the average effect size, large negative treatment effects of AASs were observed for sperm concentration, testicle size, follicle-stimulating hormone, and luteinizing hormone. Moderate positive treatment effects were observed for lean body mass, thigh circumference, bench press, and squat; while moderate negative effects were found for Apo-AI, high-density lipoprotein, sex hormone binding-globulin, and albumin. Small positive treatment effects were observed for hemoglobin, hematocrit, body weight, biceps circumference, low-density lipoprotein, serum estrogen, and serum testosterone.

Significant heterogeneity among effect sizes for high-density lipoprotein (HDL), low-density lipoprotein (LDL), estrogen (E2), follicle-stimulating hormone (FSH),

luteinizing hormone (LH), and testosterone (T) indicated that these findings were not reliable and that further analysis of moderator variables was required. In general, type of drug and route of administration were identified as being the two most important moderators of variability in the six heterogeneous outcomes. Specifically, testosterone and its esters produced higher serum levels of HDL, E2, and T and lower levels of LDL and LH than other synthetic steroids. Also, when compared with oral compounds, the use of parenterally-administered steroids resulted in higher serum levels of T and lower levels of FSH and LH.

Models incorporating indicators of study quality and subjects' experience with AASs along with treatment variables (i.e., drug, form, dose, and cycle length) sufficiently accounted for the heterogeneity in HDL, LDL, and FSH. However, excess heterogeneity in effect sizes prevents conclusions of any certainty being made about the relationship between AASs and E2, LH, or T.

This work is dedicated to those who cared...

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The decision to pursue a graduate degree has led me down a long and winding road with many bumps, several road blocks, and a couple of valleys along the way. This is a trip that I would not have been able to make without the undying support and guidance of my supervisor and friend, Dr. Lise Gauvin. Thanks Lise, for helping to keep the buzzards at bay.

Finally, I will forever be indebted to my grandmother, Lillian Cochrane, who taught me to ask the reason why.

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I. Introduction

Because of their growth-promoting and masculinizing properties, anabolic-androgenic steroids (AAS)s[1] are used clinically to treat a variety of disorders, such as aplastic anemia, retarded growth, osteoporosis, and male hypogonadism. Also, testosterone esters have recently been used in clinical trials as possible contraceptives for males (World Health Organization Task Force on Methods for the Regulation of Male Fertility, 1996). However, AASs are probably more well known for their performance-enhancing (ergogenic) properties and, thus, for their use by various professional and Olympic athletes. The Ben Johnson affair, along with the subsequent Dubin Inquiry, revelations about ergogenic drug use by former East German athletes (see Franke & Berendonk, 1997), and recent drug scandals involving Chinese athletes have reinforced the perception that AAS use is rampant among elite athletes.

While prevalence data indicates that AAS use is not rampant in most sports, there is evidence to suggest that 40-70% of elite bodybuilders and weightlifters (Frankle, Cicero, & Payne, 1984; Yesalis et al., 1988), 30-70% of world class track & field athletes (Franke & Berendonk, 1997; Ljungqvist, 1975; Silvester, 1973), and 5-15% of US college athletes (Anderson, Albrecht, McKeag, Hough, & McGraw, 1991; Yesalis et al., 1990) have used steroids to enhance performance. A more disturbing finding is that

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Anabolic-androgenic steroids are derivatives of the hormone testosterone. However, for the sake of simplicity, the term anabolic-androgenic steroid (AAS) shall be used throughout the course of this paper as relating to exogenous testosterone and its synthetic derivatives.

somewhere between 3 to 12% of Canadian and American high school students, not necessarily athletes, are using AASs for the purposes of improving their appearance and body size (Melia, Pipe, & Greenberg, 1996; DuRant, Escobedo, & Heath, 1995).

AASs have been implicated in causing various physiological and psychological side-effects such as altered cholesterol and liver enzyme levels, liver tumors, muscle and tendon ruptures, infertility, and severe mood swings (Bahrke, Yesalis, & Wright, 1990; Wilson, 1988). The most detrimental side-effects may be those that AASs have on cholesterol sub-fraction (i.e., lipoproteins) levels. Whether young or old, male or female, AAS use results in significant decreases in high-density lipoprotein (HDL) levels (Glazer, 1991). These effects seem to be moderated by drug and route of administration, with synthetic oral compounds having the most dramatic negative effect on HDL levels. Also, there is a general consensus that AAS use results in significant increases in low-density lipoprotein (LDL) levels. Keeping in mind that HDL is the "good" cholesterol that aids in the metabolism of LDLs, the "bad" cholesterol, the above findings imply that AAS use may result in coronary heart disease (CHD) or other maladies related to abnormal cholesterol ratios.

However, in many areas the AAS research literature is characterized by controversy as to what effects these drugs have on humans. For example, there are just as many studies finding increases in aggression as a result of AAS use, as there are studies finding no changes in aggression (see Bahrke et al., 1990). Similarly, the literature on the ergogenic potential of AASs, specifically strength effects, is almost evenly split between studies finding benefits (e.g., Bowers & Reardon, 1977; Freed,

Banks, Longson & Burley, 1975) and studies finding no benefits (e.g., Fowler, Gardener & Egstrom, 1965; Golding, Freydinger & Fishel, 1974) of AAS use.

One outgrowth of this controversy is that athlete-users are skeptical of the research and medical communities. This skepticism seems to be fueled by the fact that several early studies (Fowler et al., 1965; Golding et al., 1974) found no significant ergogenic benefits of AAS use while regular users, especially athletes, were reporting definite performance benefits. Furthermore, in 1977, the American College of Sports Medicine (ACSM) issued a position statement on AASs in which they stated that "there is no conclusive scientific evidence that extremely large doses of anabolic steroids either aid or hinder athletic performance" (p. 12)[2]. Also, while the medical community was reporting quite severe physical side-effects associated with AAS consumption (e.g., peliosis hepatis), the athlete-users did not notice the same detrimental effects. Although it is probable that regular AAS users experience some physical maladies--for example there are reported cases of liver tumors (Creagh, Rubin & Evans, 1988), stroke (Frankle, Eichberg & Zachariah, 1988), heart problems (Dickerman, Schaller, Prather & McConathy, 1995), and muscle ruptures (Liow & Tavares, 1995) in previously healthy athletes who used AASs--the frequency of these disorders do not reach the magnitude predicted by the medical community (see Street, Antonio, & Cudlipp, 1996). Together,

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The ACSM has since modified their position statement on the ergogenic effects of AASs to one in which they state gains in muscular strength can occur in some individuals (American College of Sports Medicine, 1987).

these circumstances have led to distrust and skepticism of the medical research community on the part of athlete and recreational AAS-users.

Part of the confusion in the AAS literature is most likely due to one or a combination of the following factors: (a) while there are numerous AASs, researchers often lump these drugs together when discussing their effects and surreptitiously assume that 50 mg of steroid A will have the same effect as 50 mg of steroid B; (b) most of the early research on AASs and testosterone, and most of the research on hepatic effects of AASs to date, was done on hospitalized patients (e.g., post-surgical), institutionalized patients (e.g., schizophrenics), or people suffering from debilitating disorders (e.g., aplastic anemia, diabetes, cancer); (c) much of the AAS literature is characterized by studies of questionable research design, low internal validity, and incorrect statistical analyses; (d) ethically, most researchers have not been able to use the combination of drugs and administer equivalent doses as to what athletes and recreational users are consuming; and finally, (e) the topic of AAS use is quite volatile as these drugs are banned substances in most sporting venues and legally controlled substances in many countries, thus it may be difficult for researchers to avoid subjective bias when investigating the possible side-effects associated with AAS use. One other possible reason for equivocal findings in the literature, is that it may be impossible to use effective placebo control groups in AAS studies (Lombardo, 1990). Thus, some changes that have been attributed to AASs may actually be due to placebo effects. At least one group of researchers have been able to successfully use a placebo and demonstrate a placebo effect

for AASs (Ariel & Seville, 1972), however experienced AAS users may not be so easily duped (Lombardo, 1990).

The fact that such controversy exists in the AAS literature begs for the application of meta-analysis. That is, there is a lack of consensus as to what effects, if any, AASs have upon the human body and psyche. A quantitative review, of the scope and magnitude of the one proposed in this study, should provide a better understanding of the AAS literature. By focussing upon treatment effect and covariation between outcomes and study features, some of the confusion and controversy in the literature should be reduced, if not clarified. In particular, the objectivity of meta-analysis allows the reviewer to avoid some of the subjective pitfalls of a narrative review. Therefore, the purpose of this study was to resolve the confusion that exists about the various physiological, ergogenic, and behavioral effects of AASs by conducting a meta-analysis of the literature.

II. Review of Literature

This next section will serve as a review of the AAS literature. Since a meta-analysis is a quantitative review of the literature, the goal of this section is not to provide a comprehensive review of every study ever done on AAS but, rather, to serve as a conceptual backdrop for the meta-analysis performed herein. Specifically, there will be a brief description of what these drugs are and how they are consumed, a discussion of the prevalence of AAS use, a discussion of the adverse and ergogenic effects associated with AAS use, and a justification for the application of meta-analysis to the AAS literature.

Testosterone and its Synthetic Relatives

While the existence of a male hormone had been speculated about since the late 1800s, it was not until the late 1920s that testosterone was first described and isolated (see Kochakian, 1993). Soon after, a process for producing a synthetic form of the hormone was discovered (Ruzicka, Wettstein, & Kaegi, 1935 in Todd, 1987), which was followed closely by studies that documented positive protein-metabolism effects of testosterone (e.g., Kochakian & Murlin, 1935; 1936).

Testosterone functions can be categorized into androgenic and anabolic actions. The androgenic actions of steroids include sexual differentiation, spermatogenesis, the development and maintenance of secondary sexual characteristics in males, gene regulation, and male-pattern behavior. The anabolic actions of testosterone and its

derivatives include stimulation of red blood cell production, bone growth, and increased skeletal muscle mass.

When administered in a pure form, either orally or parenterally (i.e., injected), testosterone is rapidly degraded within the body--usually by the liver-- and very little actually enters into circulation. To overcome this problem, the testosterone molecule has been modified by scientists, most commonly, by alkylation at the 17α-position or esterification of the 17β-hydroxyl group (Wilson, 1988).

Alkylated compounds (e.g., methandrostenolone, stanozolol) are administered orally, as tablets, and either absorbed sublingually (under the tongue) from the buccal mucosa or by the gastrointestinal tract through swallowing. Alkylation prevents these compounds from being catabolized by the liver and thus increases the duration that the compound remains in the circulation system (Wilson, 1988).

Esterified compounds (e.g., testosterone enanthate, nandrolone decanoate) are usually administered parenterally and are suspended in either an aqueous or oil solution. Once administered, the use of oil solutions as a vehicle and esters with longer carbon chains slows the release of the steroid by making it more fat-soluble and thus prolongs its period of potency in the body (Wright, 1980). The aqueous-based steroids have similar molecular structures to the oil-based, but have much shorter half-lives and require more frequent injections.

The oral and parenteral compounds are often administered in quite different doses. Also, oral compounds usually have shorter half-lives than injectable steroids and thus must be taken more often. Oral AASs usually come in the form of 5, 10, or 15 mg

tablets and may be administered daily. Conversely, parenterally-administered steroids are usually given in 200 to 400 mg/ml solutions biweekly. The fact that oral AASs are administered in smaller doses is not indicative of the androgenic or anabolic strength of these drugs. Rather, oral compounds often have more immediate and potent effects than their parenteral cousins. Also, steroid abusers usually consume these drugs, both oral and parenteral compounds, in much larger doses than the recommended therapeutic amount.

In order for AASs to be effective treatment agents they usually have to be consumed over a period of weeks or months. When used as ergogenic aids by athletes and bodybuilders, AASs are consumed in cycles. That is, the users cycle on and off the drugs using them for periods of 8 to 16 weeks with a minimum break of at least 4 weeks in between cycles. The reason for using the drugs in these fluctuating patterns is to maximize the muscle-building effects of the drugs and to reduce the risk of negative side-effects associated with long term use (Wilson, 1988).

Prevalence of Non-Therapeutic AAS Abuse

While the use of AASs in elite sport has been taking place since the mid 1950's (Todd, 1987), it is only in the last twenty or so years that data have been available to document the extent of use. An unofficial poll of track & field athletes at the 1972 Olympic Games in Munich, found that 68% of the athletes had used AASs (Silvester, 1973). Sixty-one percent of the participants had used steroids in the previous 6 months. Ljungqvist (1975) surveyed a group of elite Swedish track & field athletes and found that 31% reported previous AAS use. According to Franke & Berendonk (1997), more than

2000 East German (GDR) athletes were treated annually with AAS in a state run program between the early 1970s and the late 1980s. The authors claim that the specific drug consumption of 400 GDR athletes has been documented in recently recovered government files. Among these athletes were many gold medal winners at international events, including all GDR gold medal winners in the throwing events at the 1988 Olympic Games (Franke & Berendonk, 1997). Furthermore, according to Manfred Höppner (in Franke & Berendonk, 1997), Deputy Director and Chief Physician of the Sports Medical Service of the GDR, as of 1977, AASs were used by GDR athletes in all Olympic sporting events, except sailing and gymnastics, and by all national teams. In 1979, GDR removed its AAS restriction on gymnastics and all national and Olympic female gymnasts, including many minors, were treated with mestanolone (Franke & Berendonk, 1997).

The 1976 Olympic Games in Montreal marked the first time that AAS-testing took place at the Olympics. However, only 8 of the 275 (3%) athletes screened were found to test positive for AASs (Todd, 1987). Suspicions that steroid-using athletes and their coaches may be using various masking drugs or resorting to the use of pure testosterone in order to avoid detection of synthetic steroid use led Donike and colleagues (see Donike, Barwald, Klosterman, Schnäzer, & Zimmerman, 1983; Todd, 1987) to develop a more sophisticated screening technique. In this procedure, an athlete cannot have more than a six to one (6:1) ratio of testosterone to epitestosterone in their urine sample. Such a ratio indicates that the athlete has six times more testosterone in their system than is considered "normal" and thus are most likely to be using exogenous

testosterone. Using this procedure, Donike performed an unofficial screening of all urine samples taken from the 1980 Olympics in Moscow. Even though no samples tested positive for synthetic steroids, 20% of the samples, including 16 gold medalists, had abnormal (i.e., greater than 6:1) testosterone ratios. Seven percent of all female urine samples were positive for testosterone doping (Franke & Berendonk, 1997).

The first official use of Donike's testosterone ratio was at the 1983 Pan American Games in Caracas where 15 male athletes tested positive for testosterone use (Bergman & Leach, 1985). More disturbing than the 15 positive tests was the fact that many athletes, including 12 US track & field members and 4 Canadian weightlifters, returned home before participating in their events. Such behavior leads one to believe that these athletes were concerned about testing positive for AAS use.

While there is speculation that almost 100% of elite weightlifters, bodybuilders, and professional wrestlers use AASs, the few surveys done with weightlifters and bodybuilders (Frankle et al., 1984; Lindstrom, Nilsson, Katzman, Janzon, & Dymling, 1990; McKillop, 1987; Tricker, O'Neill & Cook, 1989; Wagman, Curry, & Cook, 1995; Yesalis et al., 1988) do not support such allegations. However, there is enough evidence to suggest that a significant amount of these athletes are using AASs as ergogenic aids. For example, Frankle et al. (1984) reported 44% AAS use amongst 250 weightlifters from three gyms in Chicago. In studies by Lindstrom et al. (1990) and Tricker et al. (1989) similar usage rates of 53% and 54% were found amongst Swedish and midwestern US bodybuilders respectively. A survey of all the athletes (N = 61, with 45 responding) at the 1987 National Championship of the US Powerlifting Federation revealed that 33%

used AASs (Yesalis et al., 1988). However, in a follow-up phone interview, 55% of the athletes reported AAS use. This latter finding led Yesalis et al. (1988) to conclude that the levels of AAS use reported in surveys probably represents the lower bounds of such drug use.

It is evident that AAS consumption is not limited to the Olympic or professional sport ranks, since their use has become common at the high school and collegiate levels (e.g., Anderson et al., 1991; Buckley et al., 1988; DuRant et al., 1995; Gaa, Griffith, Cahill, & Tuttle, 1994; Pope, Katz, & Champoux, 1988; Scott, Wagner, & Barlow, 1996; Spence & Gauvin, 1996). Apart from the potential ergogenic benefits of these drugs, the most likely reason for their use amongst this population is for aesthetic purposes. Specifically, many adolescent males report using AASs in order to increase their muscle bulk (Wang, Yesalis, & Fitzhugh, 1994).

In the United States it is estimated that 3 to 15% of male high school students are users of AASs (see Yesalis & Bahrke, 1995). At a national level, these percentages would translate to approximately half a million to one million high school students using AASs. A Canadian survey of 16,169 high school students, ranging in age from 11 to 18 years, found that 2.8% of the students reported using AASs in the previous 12 months (Melia, Pipe, Greenberg, 1996). These results suggest that as many as 83,000 Canadian adolescents between the ages of 11 and 18 have used AASs in the previous year. In a national survey of alcohol and drug use by 3,264 US college student-athletes, Anderson et al. (1991) found that 5% of all athletes reported using AASs in the past 12 months. Conversely, a similar survey found less than 1% AAS use amongst 754 Canadian

university athletes (Spence & Gauvin, 1996). While it is possible that university athletes may be less likely to admit AAS-use than high school athletes and students, it is also possible that university athletes have more to lose (e.g., scholarship, lifetime ban, etc.) if caught using such substances, and thus, are more wary about using them.

In summary, prevalence studies reveal that AAS use is a fixture in our society and may even be rampant in certain professional sports. The numbers are more disturbing when one considers the potential negative side-effects associated with AAS use.

Adverse Effects Associated with AAS Use

Acne, weight gain, hirsutism, and deepening of the voice are examples of some of the so called secondary side-effects that AAS-users often report experiencing. However, the focus of this section will be on some of the more primary life threatening side-effects that may also occur in association with AAS use (cf. Street et al., 1996). Specifically, the possible detrimental effects of the drugs will be discussed in four major areas, namely cardiovascular, hepatic (i.e., liver), endocrinological/reproductive, and psychological.

Cardiovascular effects. It has been suggested that, potentially, the most adverse side-effects of AAS use are those related to heart structure, function and cholesterol levels (Lovejoy, 1995; Melchert & Welder, 1995). Some have gone so far as to relate AAS use to CHD (Glazer, 1991). Indeed, there are at least 12 reported cases of myocardial infarction in healthy males who were either using AASs at the time of their attack or had recently discontinued use of the drugs. Five of these cases resulted in death (Dickerman, Schaller, Prather, & McConathy, 1995; Kennedy, Corrigan, & Pilbeam,

1994; Kennedy & Lawrence, 1993; Luke, Farb, Virmani, & Sample, 1990; Lyndberg, 1991).

AAS use has led to changes in the structure and functioning of the heart muscle in healthy humans (Campbell, Farb, & Weber, 1993; Climstein, 1990; De Piccoli, Giada, Benettin, Sartori, & Piccolo, 1991; McKillop, Todd, & Ballantyne, 1986; Nieminen et al., 1996; Pearson, Schiff, Mrosek, Labovitz, & Williams, 1986; Sachtleben et al., 1993; Urhausen, Holpes, & Kindermann, 1989; Yeater, Reed, Ullrich, Morise, & Borsch, 1996). For example, in a study of 11 AAS-using weightlifters and 11 age-matched controls, Sachtleben et al. (1993) found significant increases in left ventricular (LV) mass, interventricular septum thickness (IVS), LV diameter, and LV posterior wall thickness as a result of AAS use. While some do not consider these changes in heart structure to be negative (e.g., Climstein, 1990), Sachtleben et al. (1993) concluded that such changes may cause the heart to be less efficient and effective as a pump over time. Alternatively, others have found no alterations in heart structure and function as a result of AAS use (Climstein, 1986; Palatini et al., 1996; Riebe & Fernhall, 1993; Salke, Rowland, & Burke, 1985; Scavone, 1986; Thompson et al., 1992; Zuliani et al., 1988).

There is less confusion when it comes to the relationship between AAS use and serum lipid concentrations. The majority of studies, on both diseased and healthy participants, have found that the use of synthetic androgens causes abrupt alterations in lipid and cholesterol levels (e.g., Anderson, Francis, & Faulkner, 1996; Cohen, Hartford, & Rogers, 1996; Hurley et al., 1984; Kluft et al., 1984; Sachtleben, Berg, Cheatham, Felix, & Hofschire, 1997; Taggert et al., 1982; Thompson et al., 1989). On the other

hand, while exogenous testosterone use may also lead to distorted cholesterol levels it appears that these changes are not as serious in terms of the magnitude of change and the specific cholesterol subfractions involved (Gillmer, 1992; Thompson et al., 1989).

In a quantitative review of 16 studies, Glazer (1991) analysed the effects of AASs on various lipids (i.e., cholesterol, HDL, HDL_2, HDL_3, apolipoprotein A-I [Apo-AI], & LDL). It was found that AAS use brought about significant decreases, as indicated by weighted mean percent change, in HDL (52%), HDL_2 (78%), HDL_3 (35%), and Apo-AI (35%) while significantly increasing LDL levels (36%). No change was found in overall cholesterol levels but this finding was attributed to the fact that changes in the various subcomponents of cholesterol (i.e., decreased HDL, increased LDL) were counteracting each other. Glazer also considered the effects of several moderating variables (i.e., dose, cycle, and route of administration) and found that there was no dose or time effect for most of the outcomes. It seemed that AAS effects occurred at therapeutic doses and were immediate (i.e., within the first week of use) for HDL and its subfractions. However, there was potentially a relationship between dose and LDL levels with the higher dose resulting in a greater increase in LDL levels. Route of administration was also suggested to be an important variable in that oral steroids seemed to have a more dramatic effect on HDL levels than parenteral steroids (see also Thompson et al., 1989). However, a general lack of studies hampered any further investigation of these relationships. Glazer concluded that CHD risk is increased three to six times the average by the effects of AASs on serum lipid levels in males and females of any age.

According to both Gillmer (1992) and Glazer (1991), the most likely mechanism for these AAS-induced cholesterol changes has to do with the direct effect that androgens and estrogens have on two hepatic enzymes, namely lipoprotein lipase (LPL) and hepatic triglyceride lipase (HTGL). Basically, LPL activity leads to the production of LDL particles while HTGL activity leads to the catabolism of HDL particles and in particular HDL_2. Both of these enzymes are positively related to androgen levels and negatively related to estradiol levels. Thus, the use of AASs that increase endogenous androgen levels and decrease estradiol levels (i.e., most synthetic steroids) will result in increased LPL and HTGL activity which in turn will lead to detrimental changes in cholesterol levels and more significantly, a distorted HDL:LDL ratio. On the other hand, AASs that have similar positive or negative effects on endogenous androgen and estradiol levels (i.e., testosterone) will likely not have as a dramatic effect on cholesterol levels. The previous discussion is supported by several studies in which it was found that the administration of stanozolol (a synthetic steroid), led to significant and large changes in LPL and HTGL activity (Applebaum-Bowden, Haffner, & Hazzard, 1987; Thompson et al., 1989) whereas the use of testosterone over a similar period had almost no effect on the activity of these enzymes (Thompson et al., 1989). As a result, those participants using stanozolol exhibited significant increases in LDL levels and significant decreases in HDL levels.

In summary, while there have been several cases of myocardial infarctions associated with AAS use, there seems to be some confusion in the literature as to what effects, if any, AASs have on the structure and functioning of the heart muscle itself.

However, there is little doubt that AASs, synthetic steroids in particular, can have detrimental effects on cholesterol subfraction levels which may in turn have negative arthogenic effects. The potential roles that factors such as dose and route of administration may play in this relationship still need to be clarified.

Hepatic effects. AAS use has been associated with liver tumors, peliosis hepatis and altered liver enzyme levels. While there are more than 40 reported cases of liver tumours in the AAS literature (see Falk, Thomas, Popper, & Ishak, 1979; Friedl, 1990; Hickson, Ball, & Falduto, 1989), specifically hepatocellular carcinoma, only three of these cases were in healthy individuals (Creagh, Rubin, & Evans, 1988; Goldman, 1985; Overly, Dankoff, Wang, & Singh, 1984). Two of the three cases resulted in death (Creagh et al., 1985; Overly et al., 1984).

Peliosis hepatis is a rare form of hepatitis that is characterized by blood filled cysts in the liver. It is a potentially life threatening disease that seems to occur at a higher than normal frequency in some AAS users. There are more than 70 reported cases of peliosis hepatis in the literature (see Boyer, 1979; Friedl, 1990; Ishak & Zimmerman, 1987). However, similar to the occurrence of liver tumours, this disorder has not been recorded very often in healthy AAS users (e.g., athletes). In fact, there is only one report of peliosis hepatis in a healthy athlete (Cabasso, 1994). However, long term therapeutic use of methyltestosterone in female-to-male transsexuals and otherwise healthy impotent males has resulted in prepeliotic symptoms (Westaby, Ogle, Paradinas, Randell, & Murray-Lyon, 1977).

Liver enzymes are measured by liver function tests (LFTs) and their levels are usually used as indicators of overall liver functioning. While not as much concern as peliosis hepatis or liver tumours, AAS use can have an effect upon various liver enzyme levels (Arias, 1962). For example, in an extensive review of the AAS literature, Haupt & Rovere (1984) found that approximately 50% of the athletes in studies displayed abnormal liver function tests in association with AAS use. Most of these athletes had greater than normal glutamic-oxalacetic transaminase (SGOT) and glutamic-pyruvic transaminase (SGPT) levels. However, the fact that weightlifting control participants in several studies (Hagerman, Jones-Witters, & Ranson, 1975; Strauss, Wright, Finerman, & Catlin, 1983) had equally as abnormal levels of SGOT and SGPT as AAS users indicates that heavy resistance training alone may result in alterations of these nonspecific liver enzymes. With regard to specific liver enzymes (i.e., lactate dehydrogenase, alkaline phosphatase), O'Shea and Winkler (1970) found abnormal lactate dehydrogenase levels in 11 competitive swimmers using methandrostenolone while Shephard, Killinger, & Fried (1977) identified two weightlifters with above normal levels of alkaline phosphatase.

Of all the GDR athletes who participated in a state sponsored AAS program, less than 1% manifested any functional or structural liver damage (Franke & Berendonk, 1997). This damage was found to be exacerbated by excessive alcohol consumption in association with AAS use. Also, in female athletes, the damaging effects of AASs was enhanced by contraceptives. The drug of choice for the East German athletes was Oral-Turinabol, a C-17 alkylated compound similar in structure to methandrostenolone. One

GDR athlete, Detlef Gerstenberg, a hammer thrower, is said to have died from extensive liver and bile duct damage related to his use of AASs (Höppner in Franke & Berendonk, 1997).

In almost all cases of peliosis hepatis, liver tumours or abnormal liver function tests, regardless of whether they are specific or nonspecific liver enzymes, the culprit was a C-17 alkylated compound. It seems that the C-17 alkylated oral drugs are much more hepatotoxic than testosterone or its C-17 esterified derivatives. In particular, methyltestosterone, oxymetholone, and oxandrolone are the least liver-friendly steroids (Haupt & Rovere, 1984).

Endocrinological and reproductive effects. Because they mimic endogenous androgens, exogenous AASs can have very dramatic effects on the endocrinological and reproductive systems of humans. In general, the use of AASs results in decreased serum concentrations of luteinizing hormone (LH), follicle-stimulating hormone (FSH), and sex hormone binding-globulin (SHBG) (Alen, Reinila, & Vihko, 1985; Dehennin & Matsumoto, 1993; Friedl, Hannan, Jones, & Plymate, 1990; Jones, Fang, Landau, & Rosenfield, 1977; Thompson et al., 1989; Vigersky, Easley, & Loriaux, 1976). However, not all AASs have the same effects on endogenous hormones and reproductive functions. For example, while exogenous testosterone and its esters bring about dramatic and immediate increases in serum concentrations of testosterone (e.g., Bagatell et al., 1989; Cunningham, Silverman, Thornby, & Kohler, 1979), most synthetic AASs tend to decrease serum testosterone levels (Jones et al., 1977; Thompson et al., 1989; Vigersky et al., 1976). Similarly, while the administration of exogenous testosterone increases serum

estrogen levels (e.g., Bagatell et al., 1989; Hobbs, Plymate, Rosen, & Adler, 1993), most synthetic steroids have negative effects or no effect at all on serum estrogen (Aakvaag & Stromme, 1974; Friedl et al., 1990). This discrepancy can be explained by the fact that testosterone and some synthetics that share its chemical structure are readily metabolized in the body and, at an intermediary stage, turned into estrogen (i.e., aromatized). However, AASs such as fluoxymesterone, mesterlone, methyltestosterone, and stanozolol are lacking in the specific chemical structure to facilitate aromatization. This concomitant increase in estrogen levels with the use of some AASs can have feminizing effects in males. The most common of which is gynocomastia or "bitch tits" as referred to by bodybuilders (Franke & Berendonk, 1997; Friedl & Yesalis, 1989).

In healthy men, the majority of studies find that AASs cause significant and dramatic decreases in sperm counts (e.g., Anderson, & Wu, 1996; Arsyad, 1993; Heller, Morse, Su, & Rowley, 1970; Knuth, Behre, Belkien, Bents, & Nieschlag, 1985; Mauss et al., 1975; Palacios, McClure, Campfield, & Swerdloff, 1981; Reddy & Rao, 1972) and noticeable differences in sperm morphology and motility (Alen & Suominen, 1984; Knuth et al., 1985). In many cases, athletes and experimental participants experience shrinkage of the testes in association with AAS use (Palacios et al., 1981; Alen & Suominen, 1984), however these changes are often not noticeable to the AAS users (Knuth & Nieschlag, 1987). While both the disruption in sperm production and shrinkage of the testes seem to be reversible (Mauss, Börsch, Richter, & Bormacher, 1978; Steinberger, Smith, Rodriguez-Rigau, 1978), several studies have noted that sperm function may not return to normal for several months to a year after termination of AAS

use (Alen & Hakkinen, 1985; Ruokenen, Alen, Bolton, & Vihko, 1985). In a 10-year follow-up study of men who were treated with testosterone at puberty for excessively tall stature (Lemcke et al., 1996), sperm motility was significantly lower than when compared with normal men. Furthermore, a nonsignificant tendency towards lower sperm concentration, lower total sperm count, and reduced normal sperm morphology was observed in the treated men.

The above mentioned hormonal effects can be best understood if we first consider the role that endogenous hormones play in the reproductive system. That is, the male reproductive system is regulated by the hypothalamic-pituitary-testicular axis in which a negative feedback loop mechanism controls the production of testosterone and other hormones (Cumming & Wheeler, 1994). More specifically, gonadotropin-releasing hormone (GnRH) is released by the hypothalamus which results in the secretion of gonadotropins (i.e., LH and FSH) by the pituitary. LH then stimulates the production of testosterone in the Leydig cells of the testes while sperm production is stimulated by sufficient levels of this intratesticular testosterone and FSH. The release of GnRH and the gonadotropins is not continuous, rather, GnRH is released in a pulsating fashion, resulting in peak peripheral-blood testosterone levels approximately every 30 to 45 minutes. However, if plasma testosterone levels are raised above the normal physiological range (i.e., 5 - 10 mg/day), the hypothalamus will not release GnRH and the plasma levels of LH and FSH will decrease, resulting in a decline in sperm production (Wilson, 1988). The administration of exogenous testosterone will increase plasma testosterone levels while having no positive effect on the intratesticular

testosterone level and thus, through the negative feedback loop discussed above, result in decreased plasma gonadotropin levels and sperm production (Finkelstein et al., 1991). On the other hand, while most synthetic AASs have a negative effect on plasma testosterone levels they still bring about decreases in plasma gonadotropin levels (Vigersky et al., 1976; Jones et al., 1977). This action of exogenous testosterone and its synthetic derivatives is the basis for male contraceptive studies (see Knuth & Nieschlag, 1987; World Health Organization Task Force on Methods for the Regulation of Male Fertility, 1996).

In women, secondary sexual side-effects such as hirsutism, deepening of the voice, and clitoral enlargement have been reported in association with AAS use (see Franke & Berendonk, 1997; Strauss & Yesalis, 1993). Also, based upon what is known about the efficacy of female contraceptives and the fact that most AASs will alter serum testosterone, estrogen, and gonadotropin levels, it is safe to say that AAS use can disrupt the normal functioning of the female reproductive system (Cox et al., 1975; Davis & Burger, 1996; Franke & Berendonk, 1997; Henzl, Segre, & Nakamura, 1973; Klaiber, Henzl, Lloyd, & Segre, 1973). For example, in one study of 10 healthy females it was found that the administration of oxymetholone led to menstrual cycle changes such as shortening of the luteal phase and amenorrhea (Cox et al., 1975). Franke and Berendonk (1997) describe in detail the startling effects, including long-term amenorrhea, of androgen administration to young female East German athletes.

Psychological effects. Clinical studies on humans indicate that long term therapeutic doses of AASs can cause such affective and psychotic symptoms as mood

disorders, mania, and paranoia (Luisi & Franchi 1980; O'Carroll, Shapiro, & Bancroft, 1985; Wilson, Prange, & Lara, 1974). Anecdotal evidence from athletes using extraordinary doses of AASs reveals that psychological side-effects such as euphoria, delusions, anxiety, and rage can be experienced while on the steroids and depression and fatigue can occur when use is terminated (Annitto & Layman, 1980; Chaikin & Telander, 1987; Cowan, 1994). For example, using a structured diagnostic interview with 41 bodybuilders, Pope and Katz (1988) found self-reported psychotic symptoms (12%), manic episodes (12%), and full affective syndromes (22%) during AAS use. However, none of these symptoms were severe enough to warrant seeking medical attention.

Controversy exists in the data-based studies on athletes using AASs as to the generalizability of the effects of AAS use on mood and particularly aggression. Several studies report mood changes related to AAS use (Choi, Parrott, & Cowan, 1990; Humbert, 1990; Kouri, Lukas, Pope, & Oliva, 1995; Lefavi, Reeve, & Newland, 1990; Moss, Panzak, & Tartar, 1992; Parrott, Choi, & Davies, 1994; Rozenek, 1985; Spence, 1993; Yates, Perry, & Murray, 1992) while other studies report no fluctuations in mood parameters as a result of AAS use (Bahrke, Wright, Strauss, & Catlin, 1992; Bjorkqvist, Nygren, Bjorklund, & Bjorkqvist, 1994; Clark, 1991; Goudy, 1995; Swanson, 1989; Tricker et al., 1996).

Much of the confusion within the literature seems to be related to several factors. First, there is very little control of the drugs and doses being administered both within and between studies. Thus it is hard to make comparisons across studies and it is likely that the discrepancy in findings is related to differential effects of steroids on

psychological outcomes. Secondly, a major measurement issue exists in that there is much variability in the frequency of mood and/or aggression assessments across studies. For example, Bahrke et al. (1992) and Swanson (1989) measured mood only once in their studies, while Bjorkqvist et al. (1994), Humbert (1990), and Spence (1993) measured mood 3 or more times over a cycle. Third, in those studies that measure mood infrequently (i.e., less than three times), it is very unlikely that they would accurately reflect the mood changes that AAS users may experience across a cycle of steroids. Finally, recent reviews on the topic (Bahrke et al., 1990; Bahrke, Yesalis, & Wright, 1996; Riem & Hursey, 1995) have pointed out that many of these studies are characterized by methodological flaws and, in particular, have not controlled for expectancy effects. Indeed, many users have expectations about the ergogenic and psychological effects of AASs (Cicero & O'Connor, 1990; Riem, 1992).

In summary, while anecdotal evidence, mainly reports from athletes using large doses of AASs, suggests that AAS use can lead to mood swings and aggressive behavior, the research literature is much more equivocal. Furthermore, differences in drug, dosage, design, and measurement instruments make it hard to compare and summarize across studies in the behavioral area.

Ergogenic Effects Associated with AAS Use

While performance outcomes such as fusion frequency of flicker (Becker, Creuzfeldt, Schwibbe & Wuttke, 1980; Simonson, Kearns, & Enzer, 1944), reaction time (e.g., Maul, 1971), and broad jump (e.g., Win-May & Mya-Tu, 1975) have been

measured in association with AAS use, the majority of studies concerned with the ergogenic effects of AASs have focussed upon various tests of aerobic capacity and strength.

Aerobic capacity. Some may argue that aerobic capacity (i.e., VO_2 max, PWC_{170}) is a measure of cardiovascular fitness and not a performance outcome (Lombardo, 1990), however, most studies have treated this variable as an outcome measure for endurance. Because there is some evidence to suggest that AASs increase total blood volume, red blood cell count (RBC), and hemoglobin (Besa, Gorshein, & Gardner, 1974; Evens & Amerson, 1974), a pervasive assumption exists that aerobic capacity should, in turn, be enhanced. However, of the more than 15 studies that measured aerobic capacity, only two (Albrecht & Albrecht, 1969; Johnson & O'Shea, 1969) found significant increases in capacity as a result of AAS use.

Muscular strength. As mentioned previously, there has been some debate about the efficacy of AASs for improving muscular strength in humans. The literature is almost evenly split with some researchers finding increases in strength as a result of AAS consumption (e.g., Bowers & Reardon, 1972; Freed et al., 1975; Hervey et al., 1981; Stackpole, 1981; Tahmindjis, 1976; Urban et al., 1995; Ward, 1973), while others have found no changes in strength (e.g., Casner, Early, & Carlson, 1971; Fowler et al., 1965; Golding et al., 1974; Hervey et al., 1976; Young, Baker, Liu, & Seeman, 1993). Much of the heterogeneity in the ergogenic findings is most likely a result of several factors (e.g., drug, dose, weight-training experience, specificity of training, sample size). For example, in the majority of studies that didn't find significant ergogenic effects in

association with AAS use, the participants had very little or no previous weight-training experience. Conversely, most of the participants in studies that found significant ergogenic effects were experienced weight-trainers. Also, studies that used synthetic steroids in large doses (i.e., greater than the recommended therapeutic dosage) tended to find significant ergogenic effects as opposed to studies that used smaller doses or testosterone esters.

In order to resolve this debate, Elashoff, Jacknow, Shain, & Braunstein (1991) reviewed the effects of AASs on muscular strength (i.e., bench press, knee extension, hand grip, and squat) in a meta-analysis of 16 studies. After eliminating 13 studies, mostly because of concerns about faulty statistical analysis, Elashoff et al. found a rather large and significant positive effect across the three remaining studies (ES = 1.0, 95% CI, 0.49 to 1.5). These findings indicate that AAS use results in a mean increase of 1.0 standard deviation in muscular strength in healthy males. However, due to the small sample size (N = 3) and their general lack of confidence in the validity of the AAS studies, the authors concluded that AASs "may slightly enhance muscle strength in previously trained athletes" (p. 387).

Other reviews (Haupt & Rovere, 1984; Lombardo, 1990; Yesalis, Wright, & Lombardo, 1989) have also concluded that AAS use can improve muscular strength in humans and that, apart from factors related to the treatment (e.g., drug, dosage, etc.), other variables such as weight-training experience, previous experience with AASs and diet are possible moderating factors in this relationship. Similarly, the ACSM has changed their position stand on AASs from one in which they maintained that the drugs

had no effect on muscular strength (American College of Sports Medicine, 1977), to a more moderate view in which they state that gains in muscular strength can occur in some individuals (American College of Sports Medicine, 1987). These conclusions are supported by the fact that some sport performances, weightlifting in particular, have improved inordinately since the introduction of AASs into the athletic world (Bagiatis, 1993; Fair, 1988; Franke & Berendonk, 1997; Payne, 1975; Solberg, 1982; Virvidakis, Sideras, & Papadakis, 1987).

Several possible mechanisms have been proposed for the strength-enhancing effects of AASs. The first suggestion is that AASs enhance protein synthesis in the muscle thus providing more building blocks for the muscle (Rogozkin, 1979). Second, there is some evidence to suggest that AASs block the catabolic effect of glucocorticoids after exercise thus allowing for quicker recovery (see Hickson, Czerwinski, Falduto, & Young, 1990). Finally, some have suggested that AAS use leads to increased feelings of hostility and aggression which in turn facilitates the performance of more frequent weight-training sessions on the part of the user (Brooks, 1980). If AASs do indeed enhance muscular strength, these ergogenic effects most likely result from a combination of all three mechanisms discussed.

Recent revelations about systematic AAS use on the part of East German athletes shed some light on the ergogenic potential of these hormones (Franke & Berendonk, 1997). Based upon observations and experiences with hundreds of athletes using AASs, Manfred Höppner (in Franke & Berendonk, 1997), the former chief sports medical officer of East German, made the following comment:

> The positive value of anabolic steroids for the development of a top performance is undoubted. Here are a few examples...Performances could be improved with the support of these drugs within four years as follows: Shot-put (men) 2.5-4 m; Shot-put (women) 4.5-5 m; Discuss throw (men) 10-12 m; Discuss throw (women) 11-20 m; Hammer throw 6-10 m; Javelin throw (women) 8-15 m; 400 m (women) 4-5 sec; 800 m (women) 5-10 sec; 1500 m (women) 7-10 sec...Remarkable rates of increase in performances were also noted in the swimming events of women. (p. 1264)

In summary, the ergogenic benefits of AASs have been debated since the time of their discovery. The potential of these drugs has been tested most often with endurance and strength outcomes. There is very little experimental evidence to suggest that AASs will enhance endurance. Conversely, while there is confusion in the primary studies as to the strength-enhancing effects of AASs, there is a consensus in the quantitative and narrative reviews that AASs will improve strength. However, these endorsements of improvements in strength are predicated on the assumptions that experienced weight-trainers are being administered large doses of AASs and that weight-training will be taking place throughout the steroid cycle.

Efficacy of Narrative Reviews for AAS Research

While several excellent narrative reviews have been done in the area (e.g., Bahrke et al., 1990; Bahrke et al., 1996; Haupt & Rovere, 1984; Wilson, 1988; Wright, 1980), one is still left with unanswered questions about the various effects of AASs. For

example, factors such as diet, drug, and dosage, which tend to vary across studies, make it hard for reviewers to make definitive statements about steroids. Also, the narrative review does not provide insight as to the magnitude of the effect that AASs have. Specifically, due to the sensitive nature of the topic, studies in this area generally have small sample sizes and thus could be lacking sufficient power to detect a true treatment effect. Meta-analysis provides the tools to quantify and measure the treatment effect across studies and to examine covariation between study outcomes and study features, thereby allowing the reviewer to clarify some of the confusion that exists in narrative reviews.

Meta-Analysis: Rationale and Characteristics

In basic terms, meta-analysis is a set of rules and procedures for carrying out a quantitative review of a group of studies or research findings. The meta-analyst translates results from different studies to a common metric, usually the standardized mean difference (i.e., effect size), and statistically explores the relation between study characteristics and findings. A meta-analysis is most effective when there are apparent contradictions in the literature.

Since Glass (1976) first coined the term "meta-analysis" to describe his quantitative review techniques, it has become a widely accepted research tool encompassing a family of procedures in a variety of disciplines (see Louis, Fineberg, & Mosteller, 1985; Salazzar, Petruzzello, Landers, Etnier, & Kubitz, 1993; Suls & Swain, 1993). Meta-analysis typically follows the same steps as primary research, that is: (1) the

purpose is defined; (2) a sample is selected; (3) data are collected and transformed to a common metric (often effect size); (4) statistical procedures are used to investigate the relationships between study characteristics and findings; and, (5) data are interpreted and their relevance for advancing knowledge explained. Since Glass' pioneering work, many advances have been made in the original methodology (see Hedges & Olkin, 1985; Hunter, Schmidt & Jackson, 1983; Raudenbush, Becker & Kalaian, 1988; Rosenthal 1978), and meta-analyses have proliferated in the educational, psychological, and medical literature. A cursory review of the sports and exercise science literature reveals that the Glass approach (Glass, McGaw, & Smith, 1981) and the Hedges approach (Hedges & Olkin, 1985) are the two most popular meta-analyses used in the area to date (see also Salazar et al., 1993). However, conventional wisdom suggests that the Hedges approach is a more reliable and scientifically sound analysis (see Cooper & Hedges, 1994; Johnson, Mullen, & Salas, 1995).

The Glass meta-analysis consists of collecting all empirical studies relevant to a defined question using liberal inclusion criteria. The studies are then coded for certain study characteristics (e.g., age, gender, research design, etc.) and effect sizes are calculated for each dependent outcome. The unit of analysis in the Glass meta-analysis is the study finding, thus if a study has five different measures of anxiety it will be represented by five effect sizes in the meta-analysis. Parametric tests are then applied to identify relations between study outcomes and coded study characteristics.

The Hedges meta-analysis is similar to Glass' approach in many respects. However, the two approaches differ with regard to the unit of analysis, effect size

calculation, and statistical analysis of the effect sizes. Instead of reviewing a body of studies, the Hedges approach tries to approximate the pooling of all the participants from all the studies into one large comparison. To approximate this pooling of participants, the effect size is weighted by sample size. Thus, it could be said that the unit of analysis in the Hedges meta-analysis is the subject. While Glass uses the control group standard deviation in the effect size calculation, Hedges uses a pooled standard deviation. Because the Hedges meta-analysis consists of the pooling of data across studies consideration must not only be given to the variance between studies (cf. Glass), but also to the variance within studies. Thus, each effect size is calculated based upon the within study variance. Using a test of homogeneity, effect sizes can be grouped according to the size of their variance and if there are no differences the effect sizes are deemed to be homogeneous. Therefore, instead of first testing for differences between a priori categories (cf. Glass), the homogeneity approach tests whether such categorical tests are necessary at all. If a group of effect sizes is found homogeneous then the effect sizes can be combined and descriptive statistics calculated without any further testing.

Like Glass, Hedges advocates for the inclusion of all studies in a meta-analysis whether they are deemed methodologically strong or weak. Tests of homogeneity can then empirically detect whether this diversity produces heterogeneous outcomes (Hedges & Olkin, 1985). However, in contrast to Glass' use of broad categories when merging across dependent and independent variables, Hedges uses narrowly defined dependent measures that can be assumed to be linearly related (Bangert-Drowns, 1986). For example, within the Hedges approach one would not calculate an average effect size

across 14 different types of psychotherapy as Smith and Glass (1977) did with the Glass method.

Abrami, Cohen, and d'Apollonia (1988) suggest that the homogeneity approach provides more power than the techniques used in the classic Glass meta-analysis. This difference in power is due: First, to the larger sample size that occurs when using the subject as the unit of analysis as opposed to the study finding or the study itself; Second, in the Glassian approach the estimate of error variance usually contains both systematic and unsystematic components, while in the homogeneity approach the estimate of error variance is only the nonsystematic variance of the sampling errors. Thus, the Hedges meta-analysis provides more power by way of larger sample sizes and smaller error variance.

Meta-Analyses of the AAS Literature

While there exists numerous primary studies and narrative reviews of the various AAS effects, to date, only two meta-analyses (Elashoff et al., 1991; Glazer, 1991) have been done on the AAS literature. Elashoff et al. (1991) studied strength effects associated with AAS use in trained athletes and healthy young men, while Glazer (1991) analysed the arthogenic effects of AASs on serum lipid levels in males and females of all ages.

As discussed previously, Elashoff et al. (1991) found an average increase of 1.0 standard deviation in muscular strength as a result of AAS use in healthy males. However, this finding was based upon the data from three studies. One criticism of the

Elashoff et al. meta-analysis is the fact that the reviewers chose to use very restrictive inclusion criteria (i.e., use of a placebo control group, random assignment of participants to groups), which eliminated many of the studies from the start, instead of trying to control for some of these deficiencies by coding the studies for moderator variables (e.g., design, control group, assignment of participants to groups). Second, the inclusion of several different tests of muscle groups (e.g., bench press, squat, hand grip) in one analysis leads to a very confusing and most likely heterogeneous outcome. Interestingly enough, after dropping the majority of their studies, the bulk of Elashoff et al.'s analysis consisted of studies of bench press results.

Glazer (1991) found that AAS use significantly decreased HDL, HDL2, HDL3, and Apo-AI levels while causing LDL levels to rise in humans of all ages and gender. While this study was more inclusive than Elashoff et al. (1991), and provided an analysis of some moderator variables, it was not without its shortcomings. The major criticism being that, instead of effect size, percent change was used to quantify study treatment-effect. The problem with percent change is that it does not take within-study variability into account and thus does not provide a standardized measure that can be summarized across studies.

In summary, there is a need for more meta-analyses to quantify and summarize the various effects of AASs. Previous meta-analyses have provided some insight on the topic. However, these analyses either were limited in scope, did not thoroughly investigate the influence of moderator variables, or did not properly estimate study treatment effects.

Rationale for Study

Over the past decade, few drugs have received as much negative press as AASs. As a result, there exists a general perception that these are evil drugs that turn young gladiators into drooling muscle-bound monsters on our fields of play and in our arenas of entertainment. However, when not being abused by athletes or the Adonnises of the world, AASs are also used in the treatment of debilitating disorders such as aplastic anemia and osteoporosis and as possible birth control agents for males. Most of the early AAS-research on humans was done with participants who suffered from some type of chronic disease or illness. As a result, most of our early knowledge about AAS-effects was based upon findings that may have been clouded by the interaction between drug and the disease state. The fact that these drugs are now being used as contraceptive agents by healthy males and continuously abused by healthy athletes means that we need to reevaluate our knowledge of the potential side-effects associated with AAS use in healthy humans.

Current research indicates that the consumption of AASs can have negative consequences on cardiovascular and endocrinological functions in healthy humans. Conversely, the negative hepatic effects that have been witnessed in people receiving AASs as treatment for aplastic anemia and other illnesses have not generally been confirmed in healthy individuals. However, there are contradictions in the literature that need to be resolved. For example, in outcomes such as muscular strength and aggression the research literature is almost evenly split as to whether AASs have any treatment

effect or not. Also, across all outcomes, there is a need to identify and understand the factors, if any, that play a role in the treatment efficacy of AASs.

From a methodological standpoint, apart from the fact that too few studies use true experimental designs, the most glaring problem in the AAS literature is the general lack of statistical power. That is, many studies use sample sizes that are much too small to provide adequate power for the detection of all but the largest treatment effects. The inability to detect significant treatment effects contributes to the confusion in the literature and is not something that can be easily identified in a narrative review.

In summary, there is a need to effectively summarize the literature and elucidate on some of the important factors that may moderate AAS effects. However, the narrative review does not provide an adequate platform for carrying out such a task. The quantitative review or meta-analysis is the only form of literature review that provides the means by which the influence of moderator variables can be properly analysed while taking into account the diversity in sample size between studies.

Therefore, the general purpose of this study was to resolve the confusion that exists about the various effects of AASs by using the objective platform of a meta-analysis. A secondary interest of this study was to address some of the limitations of previous meta-analyses by using less stringent inclusion criteria, more specific categorizations of dependent variables, and better measures of treatment effect. More specifically, the purposes of this meta-analysis were to: (a) identify the most studied outcomes in the AAS literature, (b) determine the magnitude and direction of effect that AAS use has on these outcomes, and in the case of significant variability, (c) investigate

the relationship between various moderating variables (e.g., drug, dose, cycle length) and AAS effects.

The more extensive a man's knowledge of what has been done,

the greater will be his power of knowing what to do.

Benjamin Disraeli (1804-1881)

III. Method

Literature Search and Study Selection

Search strategy. Computer-based information searches were done on *Current Contents* (July 1993 - Dec. 1996), *Dissertation Abstracts* (1865 - Oct. 1996), *Medline* (1987 - Dec. 1996), and *Sports Discus* (1975 - Dec. 1996). Also manual searches were done through *Current Contents* (1987 - June 1993), *Physical Education Index* (1983 - 1993), and *Completed Research in Health, Physical Education, and Recreation* (1968 - 1995). Reference sections of all major reviews and primary studies were checked for unidentified references. To find relevant studies on the various side-effects associated with AASs, the keywords used in the searches included the following terms: *testosterone*, *androgenic*, *anabolic steroid(s)*, and *human*. Searches were also limited by language in that only studies published in English were included. Furthermore, in an attempt to obtain unpublished material, the 50 researchers with the largest number of publications in the area were contacted by letter and asked if they would share any unpublished manuscripts or data in their possession. The response rate to these inquiries was about 40% with five documents being retrieved. However, all of these manuscripts have since been published.

Inclusion/exclusion criteria. The criteria for including studies in the final sample were as follows:

1. Information was reported in a study format, either published or unpublished, as opposed to a case study or editorial.

2. Any study where steroids were used in combination with other drugs (e.g., human growth hormone, GnRH or LHRH tests, progestins) was excluded.

3. Studies had to include pretest data, either in a between- or within-groups design. Comparative designs (i.e., cross-sectional designs) were excluded because it was almost impossible to account for holdover effects from previous AAS-use. Within-group crossover designs were included as long as a minimum washout period of 4 weeks was allowed between drug use and placebo or baseline.

4. Data from "incomplete androgens" (e.g., Testolactone) was excluded.

5. Studies reported enough information to allow for effect size extraction.

6. Sample was composed of healthy human participants using exogenous testosterone or some synthetic derivative (anabolic-androgenic steroids).

7. Participants were aged between the range of 18 to 50 years-old.

8. Participants had not used AASs for a minimum of 4 weeks prior to commencement of the study.

9. Sample size was greater than 2 with a minimum of 4 participants in a between-groups design and 3 participants in a within-group design.

Coding of Study Characteristics

Nomological coding. To identify the study characteristics that could potentially explain the effects of AAS use, all of the studies included in the meta-analysis were nomologically coded (Abrami et al., 1988). Nomological coding is a procedure whereby one reads all, or a random sample, of the studies selected for a meta-analysis and identifies the various study characteristics of each study. The procedure involves not only identifying, but also tabulating the number of times a particular characteristic appears in the selected studies. In this way, a reviewer can identify the most frequently studied characteristics and be sure to include them in the meta-analysis coding scheme.

For the purposes of this meta-analysis, an additional feature was added to the nomological coding, namely the tabulation of the frequency of dependent variables or outcomes. Thus the outcomes that have been most studied in the selected studies were identified for inclusion in the meta-analysis. In order for an outcome to be considered for inclusion in the meta-analysis it had to have been studied at least 10 times in the literature while a study feature had to have been measured in at least 90% of the findings. The minimum limit for outcomes was arbitrary and designed to reduce information overload. The justification for the rather restrictive study-features criteria was twofold. First, Lipsey (1994) recommends that only those study features "that can be coded from primary studies in essentially complete and accurate form" (p. 120) should be included in the analysis. Second, when performing univariate model testing for effect-size heterogeneity, the inclusion of numerous moderator variables (i.e., study features) will lead to the inflation of Type I error (Harrell, Lee, & Mark, 1996).

Study features coding. The nomological coding yielded 35 outcomes (see Table 1 for list and acronyms, also Appendix A for a glossary of terms) and 15 study features (see below)[3,4]. Based upon the nomological coding, a codebook was developed (see Appendix

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Although there has been much discussion about the psychogenic effects of AAS use, the data in this area was either of insufficient quantity or quality to warrant inclusion in this analysis.

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Instead of including all measures of strength under one general strength outcome, separate strength outcomes were designated based upon the body part(s) and type of muscle contraction being tested. As a result, 35 different strength outcomes were identified (e.g., bench press - isotonic, elbow extension - isokinetic, elbow flexion - isometric, hand grip - isometric, knee flexion - isokinetic, squat - isotonic). However, only bench press (isotonic) and squat (isotonic) were measured often enough in the literature to warrant inclusion in this analysis. While some may argue that all strength measures should be included under one strength outcome, as was done in Elashoff et al. (1991), it is possible that such a procedure would contribute undue heterogeneity to the effect sizes. Due to a lack of specificity of training in many of the

B) and each relevant finding in a study was coded for the 15 study characteristics. Therefore, this meta-analysis is really a series of 35 meta-analyses with the following 15 study characteristics being measured in each:

Substantive Variables

Gender. Either male, female, or mixed sample.

AS Exp. Indicates whether the participants had reported any previous experience with AASs or not.

Publication. Whether the source of the finding was published or not.

Methodological Variables

Design. Denotes whether the design was within- or between-groups.

Control. Control condition, either no-use or placebo.

Subject Selection. Gives an indication of how the participants got into the experimental group, either by non-random or random assignment. In some instances, a subject selection may be designated as random even though the effect size has been coded as being from a within-groups design. This is because some findings come from between-groups studies where, due to incomplete data (e.g., no pre-post data for control group), it was impossible to estimate a between-groups effect size but enough data was available to calculate a within-groups effects size for the experimental group. Thus, even

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studies, it is unreasonable to expect similar improvements across strength measures. Also, the inclusion of several strength measures from one study, under one outcome, contributes multiple endpoints and violates the assumption of independence of effect sizes within an outcome. Elashoff et al. (1991) circumvented this problem by rank ordering strength measures according to the best measure of strength and then selecting the highest ranked measure available in each study. Instead of resorting to such a subjective procedure in this study, the decision was made to treat the strength measures as being independent and to include those outcomes that satisfied the minimum frequency requirement (i.e., >9).

though pre-post data was used for effect size calculation and the design was coded as being a within-groups design for these participants, the finding was coded according to the appropriate subject assignment so as to maintain the identity of being from a stronger study.

Blind. Subject and experimenter's awareness of conditions, either no blind, blind or double-blind. As discussed, there may be more findings coded as being blind or double-blind than there are between-groups studies (see Subject Selection for explanation).

Verify. Verification of AAS use, either subjective, experimenter-administered or tested for (i.e., urine sample, blood test).

Quality. A continuous indicator of study quality was estimated by calculating an average weighted composite across the 5 categorical methodological variables. In order to calculate this composite, the categorical variables were each weighted by their partial correlation (controlling for the other methods variables) with *d* so as to give a heavier weighting to those variables that had a higher association with the effect size. The coding for the categorical variables was such that, the lower the value the weaker the design. Therefore, the lower the value for quality, the weaker the design.

Treatment variables

Drug. Since numerous different AASs (N = 19, see codebook in Appendix B) were used or investigated across the studies, a classification scheme was adopted (Rogozkin, 1991) for purposes of organizing these steroids into common groupings based upon the pharmacological origins of the drugs. Thus, the AASs were classified as being

either testosterone, androstene-based (testosterone-related), androstane-based, estrane-based or a combination of several drugs (mix).

Structure. Most AASs can be categorized based upon a common molecular structure or alterations to this structure, either C-19 steroid (i.e., pure testosterone), C-17α alkylated, C-17β esterified, or mix.

Form. Route of administration, either intramuscular (IM), oral (PO), mix (both IM and PO), or other (e.g., peroral, infusion, percutaneous, pellets).

Stacking. Whether the participants used more than one AAS during their cycle. A discrepancy may exist between how findings are coded in Stacking, and the number of findings in the mixed classes within Drug and Structure and that is because the users may have been stacking several testosterone-based or C17-esterified drugs. Therefore, while several drugs were used and stacked, they would not have been classified in the mix classes.

Dose. Drug dosage, is coded as average mg/wk.

Cycle. Duration of drug use, measured in weeks.

At this point, it should be noted that while several reviewers (e.g., Haupt & Rovere, 1984; Lombardo, 1990; Wright, 1980; Yesalis & Bahrke, 1995) have discussed the importance of other moderator variables in understanding AASs-effects---such as diet, weight training experience, intensity of training, and the use of protein supplements---these variables have not been measured often enough in the literature, or do not pertain to the bulk of the studies, to warrant their inclusion in this meta-analysis.

Table 1

Outcomes in Meta-analysis

Blood Chemistry
Hemoglobin (Hb)
Platelet count (PC)
Hematocrit (Hct)
Red blood cell count (RBC)

Body Dimensions
Body fat% (BF%)
Body weight (BWt)
Biceps circumference - relaxed (Biceps)
Lean body mass (LBM)
Thigh circumference - relaxed (Thigh)

Cardiovascular Measures
Apolipoprotein AI (Apo-AI)
Diastolic blood pressure (DBP)
High density lipoprotein (HDL)
Low density lipoprotein (LDL)
Systolic blood pressure (SBP)
Total cholesterol (TC)
Triglycerides (TG)

Endocrine System
Estradiol (E2)
Follicle stimulating hormone (FSH)
Luteinizing hormone (LH)
Testosterone (T)

Liver Enzymes and Plasma Proteins
Alanine aminotransferase (ALAT)
Alkaline phosphatase (AP)
Aspartate aminotransferase (ASAT)
Albumin
Bilirubin (Bil)
Creatinine (Cr)
Gamma-glutamyltransferase (GGT)
Lactate dehydrogenase (LDH)
Sex hormone binding-globulin (SHBG)

Reproductive System
Semen volume (SV)
Sperm count (sperm)
Testicle size (Testes)

Performance
Bench press (Bench)
Squat
Cardiovascular capacity ($VO2_{max}$)

Reliability of Coding

One of the strengths of a meta-analysis can be determined by the reliability achieved in coding. The reliability between coders is usually gauged through computation of percent agreement or Cohen's kappa. However, in this case, only one coder was involved. Therefore a form of test-retest reliability called coder drift was used (Orwin, 1994). In applying this procedure, a random sample of thirty studies (approximately 25% of total sample) was selected for recoding of study features and effect sizes and per case agreement rate (the number of variables coded the same divided by the total number of variables coded) was calculated for each finding. A minimum mean agreement rate of .90 was required for acceptance. Rate of coder drift was satisfactory for both study-features (r = .94) and effect-size (r = .92) coding.

Design of Meta-Analysis

Effect size computation. For this meta-analysis, Hedges and Olkin's (1985) procedure to derive and analyse effect sizes was used. Specifically, the effect size, g, was defined as the difference between the means of the experimental group (AAS users) and the control group divided by the pooled within-group standard deviation. Similarly, for within-group designs, g was defined as the difference between the means of the posttest (experimental group) and the pretest (control group) divided by the pooled within-group standard deviation (Becker, 1988). The pooled standard deviation is considered to be a more representative indicator of within study variance (Hedges & Olkin, 1985). However, in extreme cases of unequal variances (i.e., value of control SD

was 3 times larger or 3 times smaller than experimental SD) the control group or pretest standard deviation was substituted for the pooled within-group standard deviation in the calculation of *g* (Thomas & French, 1986). If descriptive statistics were not available, then estimates of *g* were calculated from other statistics such as *t*, *F*, or *p* (see Dunlop, Cortina, Vaslow, & Burke, 1996; Glass et al., 1981; Ray & Shadish, 1996).

Effect sizes are positive if AAS users exhibited higher levels of the dependent variable than nonusers, and conversely, are negative if AAS users exhibited lower levels of the dependent variable than nonusers. The effect sizes were corrected, *d*, for the bias from *g*'s overestimate of the population effect size for small samples (Hedges, 1984). Statistical significance of *d* was determined by the absence or presence of 0 within the 99% CI for *d*. The absence of 0 from the CI indicates a significant effect size. The more conservative 99% CI was adopted so as to try and reduce the possibility of experiment-wise Type I error that may occur due to the multitude of comparisons being performed in this analysis (Hancock & Klockars, 1996)[5] .

Unit of analysis. The unit of analysis was the study finding. Therefore, for every different outcome within a study there was a specific effect size. Multiple effect sizes within outcomes were taken from studies only if different samples were used to produce these effect sizes (e.g., males vs. females). Otherwise, the most complete data with the

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An even more conservative approach to reducing Type I error would have been to adopt the Boole-Bonferroni Inequality, or Bonferroni Correction as it is also known, in which the minimum required α is corrected (α/p) for the number of variables in the overall analysis (p). However, in the present meta-analysis--where p was large--the resulting individual confidence intervals from such a correction would be very wide (e.g., $\alpha = .05$, $p = 35$, $\alpha/p = .001$). Thus, it was felt that the Bonferroni Correction would be too harsh for this analysis.

longest steroid cycle was used. The decision to code in this manner is supported by the fact that there is a dearth of studies reporting on prolonged AAS use (Lombardo, 1990).

Missing data. For a meta-analyst, there are two aspects related to missing data. First, there is the question of obtaining all of the studies on a specific topic. As can be seen in the description of the search strategy, every attempt was made to identify and retrieve relevant studies. However, the possibility remains that there are studies, published and unpublished, which were not identified. Secondly, once studies have been identified, there is the problem of missing data. That is, while an outcome may be measured in a study, there may not be enough statistical information (e.g., means, variances, etc.) to calculate an effect size. Bushman and Wang (1996) claim that missing data is the largest problem facing the meta-analyst. Indeed, they found that approximately 25% of studies in meta-analyses published in the past 5 years in Psychological Bulletin had missing data and that the most common method for dealing with this problem was to drop the studies involved. However, Bushman and Wang identified several problems with this practice, one of which is that "the variance of the effect-size estimates will be unnecessarily large when studies with missing effect-size estimates are omitted" (p. 67). Therefore, a series of procedures was adopted to estimate effect sizes when data were missing within studies.

In the case of missing variances, if means and *n*s were provided, an attempt was made to estimate an average variance for that outcome based upon the findings in other studies (Glass et al., 1981). An effect size of 0 was imputed if complete descriptive (i.e., means, variances, and sample size) and/or inferential statistics were unavailable and it

was clear that there was no treatment effect (i.e., $Mean_1$ - $Mean_2 = 0$). Also, if a result was only described as not significant and *n* and/or direction of change could not be determined then 0 was substituted for the effect size[6]. However, if a result was described as not significant, and *n* and direction of change could be determined then the effect size was calculated based upon the value of *t* for $p = .05$ and divided by a factor of 2 (Sedlmeier & Gigerenzer, 1989). The major assumption was that the population of nonsignificant findings is normally distributed.

Weighting of effect sizes. When calculating the overall average effect size for each outcome, d_+, each study effect size was weighted by the reciprocal of its variance before it was averaged with other effect sizes. This procedure gives additional weight to effect sizes that are more reliably estimated (Hedges & Olkin, 1985). In the case of within-group designs, before weighting of the effect sizes, the variance was corrected for the correlation between the posttest scores and the pretest scores (Becker, 1988). The effect of this correction is twofold. First, by removing the dependency between the observations, the pretest and posttest standard deviations become independent and thus on par with the control and experimental standard deviations from between-group designs. Second, as *r* increases, this correction reduces the effect-size variance, resulting in a heavier weighting of the more reliable effect sizes (Looney, Feltz, & VanVleet, 1994).

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Although it is possible that the inclusion of effect sizes estimated as 0 will lead to a more conservative estimate of treatment effect (Ray & Shadish, 1996), it was felt that erring on the side of conservatism was better than to ignore what may be a substantial body of nonsignificant findings. Also, the "0-effect" was tested in the meta-analyses.

Data Analysis

Univariate homogeneity analysis. To detect whether the studies shared a common effect size, the homogeneity of the set of effect sizes was tested with a homogeneity statistic, Q_r, which has an approximate chi-square distribution with k - 1 degrees of freedom, where k is the number of effect sizes (Hedges & Olkin, 1985). If the resulting chi-square was significant, the effect sizes were determined to be heterogeneous and categorical models (i.e., moderator variables) were tested to identify sources of heterogeneity. The techniques for calculating categorical models provide a between-classes effect and a test of the homogeneity of the effect sizes within each class. The between-classes effect is estimated by Q_b, which has an approximate chi-square distribution with p - 1 degrees of freedom, where p is the number of classes. A large value of Q_b indicates that there are significant differences among the classes of effect sizes. The homogeneity of the effect sizes within each class is estimated by Q_w, which has an approximate chi-square distribution with m - 1 degrees of freedom, where m is the number of effect sizes in the class.

Weighted correlations and weighted least squares (WLS) regressions were used to gauge the relationship between each continuous moderator variable (i.e., Quality, Cycle, and Dose) and d.

Regression analysis. A WLS regression procedure was also used to determine if the variability in heterogeneous outcomes could be explained by a multivariate model. The WLS regression is similar to univariate categorical model testing in that: d is weighted by the inverse of its variance; the total sums of squares for the regression is

equivalent to the homogeneity statistic Q_t; the sums of squares error provides a test of model specification (Q_E) which is tested as a chi-square with the same degrees of freedom as Q_w ; and, the sums of squares for regression (Q_R) gives a test of the regression which is tested as a chi-square with the same degrees of freedom as Q_b. However, the WLS regression is more advantageous than univariate categorical model testing in that it allows for the inclusion of multiple predictors and continuous variables in the search for an appropriate model while controlling for shared variance between the predictors.

Outlier analysis. Using a procedure suggested by Hedges & Olkin (1985), outliers were detected and removed from the analysis if a finding had a standardized residual value greater than +2.0 or less than -2.0.

Computer Software

Statistical analysis was completed through use of Quattro Pro 6.0 (Quattro Pro 6.0, 1994) and SPSS 6.1 (SPSS 6.1 for Windows, 1994). While there are several stand-alone meta-analyses programs available (e.g., DSTAT; Johnson, 1989), none of them have adopted recent well-founded suggestions for estimating effect sizes from within-group designs (Becker, 1988; Dunlop et al., 1996). Seeing as how within-group designs were included in these meta-analyses, the decision was made to do the analyses in-house, so to speak, incorporating the above mentioned suggestions.

IV. Results

Search Results

The literature search revealed 147 studies that met the inclusion criteria. However, 20 studies were dropped because they did not include one of the 35 outcomes identified through nomological coding. Thus, the final sample consisted of 127 studies (see References) and 894 effect sizes (see Appendix C for a breakdown of effect sizes by study) representing 12,566 participants.

Description of Studies

In almost all cases, the effect sizes were from studies using male participants only. In fact, less than 2% (N = 11) of the effect sizes were from female or mixed-gender samples. Only 20% of the participants had previous experience with AASs, while 67% reported never having used steroids before the current cycle. The within-group or pre-post design was the most frequently used design of researchers in the area with approximately 69% of the findings coming from such comparisons. Also, while every effort was made to obtain unpublished documentation of steroid-effects, including manuscripts and theses, less than 4% (N = 30) of the effect sizes in this series of meta-analyses were from unpublished sources. Unfortunately, the majority of retrieved unpublished studies either pertained to outcomes not included in the analyses or did not satisfy the inclusion/exclusion criteria. Finally, the most common treatment paradigm

used within the AAS literature involved testosterone esters administered parenterally at a dose of 200 mg/wk for a period of 6 to 12 weeks (see Table 2).

Overall Physiological and Ergogenic Effects of AAS Use

Mean effect sizes and homogeneity statistics are presented in Table 3 and discussed below. Unless otherwise stated, the reader can assume that the effect sizes for each outcome are homogeneous.

Blood Chemistry

Significant increases in Hb (d_+ = +0.22, SE = .06) and Hct (d_+ = +0.20, SE = .06) occurred as a result of AAS-use. No changes were observed for PC or RBC.

Body Dimensions

The use of AASs resulted in significant increases in BWt (d_+ = +0.32, SE = .04), biceps circumference (d_+ = +0.32, SE = .10), LBM (d_+ = +0.63, SE = .11), and thigh circumference (d_+ = +0.38, SE = .11). No significant changes were witnessed for BF%. As opposed to the general treatment paradigm observed in the AAS literature, the majority of body dimension findings came from studies using methandrostenolone administered orally at a dose of 70 mg/wk for a cycle of 6 to 12 weeks.

Cardiovascular Measures

AAS-use had no significant effect on DBP or SBP. However, AAS-use resulted in significant increases in LDL (d_+ = +0.15, SE = .05) and significant decreases in Apo-AI (d_+ = -0.47, SE = .09) and HDL (d_+ = -0.44, SE = .05) levels. No changes in TC or TG levels were apparent. The observed variances in study effect sizes for HDL and LDL

were significantly greater than what would be expected by chance as demonstrated by the values of Q_t for HDL (134.15, $df = 30$) and LDL (60.49, $df = 21$). Therefore, further analysis of moderator variables for HDL and LDL was warranted.

Endocrine System

AAS-use resulted in significant increases in serum concentrations of E2 (d_+ = +0.23, SE = .06) and T (d_+ = +0.32, SE = .04). Large decreases were observed in FSH (d_+ = -0.72, SE = .05) and LH (d_+ = -0.72, SE = .04) levels. However, significant effect-size heterogeneity for E2 (Q_t = 175.99, $df = 31$), FSH (Q_t = 127.66, $df = 59$), LH (Q_t = 169.11, $df = 68$), and T (Q_t = 557.61, $df = 77$) suggested that further analysis of moderator variables was necessary for these outcomes.

Liver Enzymes & Plasma Proteins

The use of AASs resulted in significant decreases in albumin (d_+ = -0.34, SE = .11) and SHBG (d_+ = -0.60, SE = .06). No changes were observed for ALAT, AP, ASAT, Bil, Cr, GGT or LDH.

Reproductive System

Significant decreases in sperm (d_+ = -0.99, SE = .08) and Testes (d_+ = -0.83, SE = .11) occurred as a result of AAS-use. No significant changes were observed for SV.

Table 2

The Most Common Treatment Paradigm Within Each Outcome

Outcomes	N	Most Common Treatment Paradigm			
		Drug	Form	Dose (mg/wk)	Cycle (wks)
Blood Chemistry					
Hb	°21	TE	parenteral	200	12
PC	13	TE	parenteral	200	12
Hct	°21	TE	parenteral	200	24
RBC	13	TE	parenteral	200	24
Body Dimensions					
BF%	19	methandrostenolone	parenteral	300	12
BWt	52	methandrostenolone	oral	70	6
Biceps	16	methandrostenolone	oral	70	8
LBM	14	methandrostenolone, TE	parenteral	*112.5	6
Thigh	14	methandrostenolone	oral	70	6
Cardiovascular Measures					
DBP	14	mix, TE	parenteral	*112.5	12
SBP	14	mix, TE	parenteral	112.5	12
Apo-AI	°11	TE	parenteral	*100	6
HDL	°30	mix	parenteral	594	6
LDL	°22	TE	parenteral	*200	6
TC	°35	mix, TE	parenteral	200	6
TG	°29	TE	parenteral	200	12
Endocrine System					
E2	°32	TE	parenteral	200	12

FSH	°60	TE	parenteral	200	1
LH	°69	TE	parenteral	200	1
T	°78	TE	parenteral	200	*1
Liver Enzymes & Plasma Proteins					
ALAT	°26	TE	parenteral	200	12
AP	30	TE	parenteral	200	24
ASAT	°34	TE	parenteral	200	12
Albumin	13	TE	parenteral	*70	12
Bil	23	TE	parenteral	200	*6
Cr	11	TE	parenteral	200	12
GGT	13	mix, TE	parenteral	200	12
LDH	15	TE	parenteral	200	6
SHBG	22	TE	parenteral	300	12
Performance					
Bench	18	methandrostenolone	oral	70	4
Squat	16	methandrostenolone	oral	70	*3
VO2	13	methandrostenolone	oral	70	3
Reproduction					
SV	14	TE	parenteral	200	6
Sperm	°27	TE	parenteral	200	24
Testes	°13	TE	parenteral	*100	24

Note. ° outlier(s) excluded. TE = testosterone enanthate. Mix = mixture of testosterone esters and synthetic steroids. *Multiple modes, the smallest value is shown.

Table 3

The Median, Mean, 99% Confidence Interval (CI), and Homogeneity Tests of Effect Sizes for All Outcomes

		Effect size estimates						
		Unweighted				99% CI for d_+		
Outcomes	N	median	d	d_+	SE	Lower	Upper	Q_t
Blood Chemistry								
Hb	°21	0.18	0.23	0.22	0.06	0.06	0.38	32.07
PC	13	0.00[a]	0.22	0.07	0.07	-.010	0.25	8.70
Hct	°21	0.00[a]	0.26	0.20	0.06	0.04	0.36	35.94
RBC	13	0.16	0.20	0.17	0.08	-0.04	0.38	25.72
Body Dimensions								
BF%	19	-0.02	-0.13	-0.08	0.06	-0.24	0.08	9.45
BWt	52	0.24	0.41	0.32	0.04	0.21	0.44	45.43
Biceps	16	0.39	0.43	0.32	0.10	0.06	0.57	19.20
LBM	14	0.78	0.80	0.63	0.11	0.35	0.91	16.26
Thigh	14	0.43	0.52	0.38	0.11	0.09	0.67	13.44
Cardiovascular Measures								
DBP	14	0.00	0.12	0.08	0.07	-0.09	0.26	12.90
SBP	14	0.00	0.14	0.06	0.07	-0.11	0.23	9.96
Apo-AI	°11	-0.67	-0.75	-0.47	0.09	-0.70	-0.25	22.45
HDL	°30	-0.59	-0.86	-0.44	0.05	-0.57	-0.32	**134.15
LDL	°22	0.09	0.20	0.15	0.05	0.03	0.27	**60.49
TC	°35	0.00	0.05	0.05	0.03	-0.03	0.14	52.00
TG	°29	0.00	0.09	0.08	0.03	0.00	0.17	40.41

Endocrine System								
E2	°32	0.24	0.23	0.23	0.06	0.08	0.37	**175.99
FSH	°60	-0.87	-0.98	-0.72	0.05	-0.84	-0.60	**127.66
LH	°69	-0.96	-1.05	-0.72	0.04	-0.84	-0.61	**169.11
T	°78	0.65	0.58	0.32	0.04	0.21	0.43	**557.61
Liver Enzymes & Plasma Proteins								
ALAT	°26	0.00	0.14	0.05	0.05	-0.08	0.19	25.98
AP	30	0.00	-0.01	0.01	0.04	-0.09	0.10	9.87
ASAT	°34	0.00[a]	0.22	0.12	0.05	0.00	0.25	37.28
Albumin	13	-0.32	-0.31	-0.40	0.07	-0.58	-0.22	21.38
Bil	23	0.00	-0.03	-0.01	0.04	-0.11	0.08	9.89
Cr	11	0.00	0.14	0.07	0.08	-0.15	0.28	13.91
GGT	13	0.00	-0.02	-0.04	0.07	-0.22	0.14	11.62
LDH	15	0.00	0.19	-0.04	0.05	-0.15	0.08	21.49
SHBG	22	-0.65	-0.73	-0.60	0.06	-0.75	-0.46	27.78
Performance								
Bench	18	0.46	0.70	0.59	0.12	0.28	0.90	15.02
Squat	16	0.49	0.96	0.69	0.14	0.32	1.06	17.68
VO2	13	0.00	0.11	0.10	0.12	-0.20	0.41	16.28
Reproduction								
SV	14	0.00	-0.14	-0.13	0.07	-0.32	0.05	19.57
Sperm	°27	-0.92	-1.15	-0.99	0.08	-1.21	-0.77	41.14
Testes	°13	-1.12	-1.11	-0.83	0.11	-1.11	-0.54	25.36

Note. ° outlier(s) excluded. Q_t is a test of homogeneity.
[a] indicates significant difference ($p < .01$) between those effect sizes estimated as 0 and those calculated by other methods.
** $p < .01$

Performance

Moderate increases in effect size were observed in bench (d_+ = +0.59, SE = .12) and squat (d_+ = +0.69, SE = .14) as a result of AAS use. No significant changes were observed in $VO2_{max}$. Similar to the body dimension outcomes, the bulk of the ergogenic findings was produced in studies using methandrostenolone, administered orally, at a dose of 70 mg/wk for a period of 3 to 4 weeks.

Testing for the 0-Effect

As is evident by the data in Table 3, a median *d* value of 0.00 occurred for 15 outcomes. In three of these cases (Hct, PC, and ASAT), a significant 0-effect was obtained. That is, a significant difference existed between the mean effect size of each outcome when zeros were imputed for nonsignificant nondirectional missing data, as opposed to when the 0-effects were excluded. Assuming that the true average effect sizes lie between the two "extreme" effect sizes presented for each outcome in Table 4, we would not see much of a change for Hct (d_+ = 0.20 vs. 0.30). However, the range for possible effect sizes is much wider for PC (d_+ = 0.07 vs. 0.53) and ASAT (d_+ = 0.12 vs. 38) and thus any conclusions drawn about these outcomes would be tenuous at best.

Table 4

Effects Sizes for Hct, PC, and ASAT

	0 Included				0 Excluded		
Outcomes	N	d_+	SE		N	d_+	SE
Hct	21	0.20*	0.06		12	0.30*	0.08
PC	13	0.07	0.07		4	0.53*	0.18
ASAT	34	0.12	0.05		14	0.38*	0.10

* $p < .01$

Moderator Variable Analysis for Heterogeneous Outcomes

It is likely that interesting relationships could be identified between the moderator variables and each one of the outcomes included in these meta-analyses. However, while some may disagree (cf. Rosenthal, 1996), a more traditional approach (Hedges & Olkin, 1985) was adhered to in this study. That is, moderator variables were only tested in outcomes that exhibited significant heterogeneity within effect sizes.

Almost all of the findings in the 6 heterogeneous outcomes (HDL, LDL, E2, FSH, LH, & T) were from published studies using male participants. In fact, females only accounted for one effect size in HDL (d_+ = -0.64 vs. -0.44 for males), one effect size in LDL (d_+ = -0.66 vs. 0.16 for males) and one effect size in T (d_+ = 0.54 vs. 0.31 for males). Similarly, unpublished studies contributed three effect sizes to HDL (d_+ = -0.79 vs. -0.41 for published studies) and three effect sizes to LDL (d_+ = 0.29 vs. 0.12 for published studies) only. Therefore, while Gender and Publication were intended to be included in any analysis of heterogeneous outcomes, these variables were dropped from further analysis because they did not apply to the following outcomes.

High-Density Lipoprotein

Methodological variables. As is evident by the findings presented in Table 5, Verify (Q_b = 38.64, df = 2) and Quality (r = 0.26, Q_R = 10.44, df = 1) were the only significant methodological moderators of effect size differences for HDL, with those findings based upon subjective verification having a significantly greater negative effect on serum HDL levels (d_+ = -1.14) than those in which the experimenter was in charge of administering the steroids (d_+ = -0.32) or where drug testing actually took place (d_+ = -0.35). These study features, along with the other methodological variables for HDL, indicated that weak internal validity may lead to larger negative effects of AASs.

Substantive variable. AS Exp (Q_b = 38.35, df = 1) served as a significant moderator of AASs effects on serum concentrations of HDL. Experienced AAS users (d_+ = -0.97) exhibited a much lower HDL concentration as a result of AAS use when compared to those users who had no previous experience with AASs (d_+ = -0.25).

Treatment variables. A significant value for Q_b or Q_R was found for Drug (Q_b = 67.39, df = 4), Structure (Q_b = 63.30, df = 2), Form (Q_b = 66.39, df = 3), Stacking (Q_b = 49.71, df = 1), and Cycle (r = 0.26, Q_R = 6.90, df = 1) within the HDL findings. Testosterone drugs (d_+ = -0.23), C-17 esterified drug structures (d_+ = -0.25), IM-administered drugs (d_+ = -0.27), the use of one AAS only (d_+ = -0.30), and longer cycles (r = 0.24) had the least negative effect upon HDL levels while mixed drugs (d_+ = -1.19), mixed-drug structures (d_+ = -1.22), PO-administered drugs (d_+ = -1.80), and the use of multiple AASs (d_+ = -1.22) brought about the greatest decrements. Apart from the

Stacking study-feature, the main cause of heterogeneity across these study features was the mixed classes.

Low-Density Lipoprotein

Methodological variables. Verify (Q_b = 18.46, *df* = 2) and Quality (Q_R = 8.38, *df* = 1) served as significant moderators of AASs on LDL levels (see Table 6). Subjective verification (d_+ = +0.48) and lower quality studies (r = -0.39) resulted in higher serum concentrations of LDL, suggesting that low internal validity is associated with larger positive increases in LDL within the AAS literature.

Substantive variable. AS Exp (Q_b = 10.89, *df* = 1) served as a significant moderator of AASs effects on LDL levels. As a result of AAS use, experienced AAS users (d_+ = +0.34) exhibited significantly higher LDL levels when compared to those users who had no previous experience with AASs (d_+ = -0.02).

Treatment variables. With regards to treatment variables, Drug (Q_b = 38.80, *df* = 4), Structure (Q_b = 35.34, *df* = 3), Form (Q_b = 30.79, *df* = 3), Stack (Q_b = 20.72, *df* = 1), and Cycle (Q_R = 14.35, *df* = 1) all had significant Q_b or Q_R values. Testosterone (d_+ = -0.13, *ns*), C-17 esterified drugs (d_+ = -0.08, *ns*), IM-administered drugs (d_+ = -0.02, *ns*), and the use of single drugs (d_+ = +0.01, *ns*) had little or no effect on serum LDL, while mixed drugs (d_+ = +0.48), C-17 alkylated drugs (d_+ = +0.56), PO-administered drugs (d_+ = +0.61), the use of multiple drugs (d_+ = +0.48), and shorter cycles (r = -0.53) led to significant positive increases in LDL concentrations.

Table 5

Weighted Effect Sizes as a Function of Moderator Variables for High-density Lipoprotein (HDL)

Variable and Class	Q_b	k	d_+	99% CI for d_+ Lower	Upper	Q_w[a]
Methodological Variables						
Design	0.05					
Within		23	-0.44	-0.57	-0.31	**112.11
Between		7	-0.48	-0.92	-0.04	**21.99
Control Group	1.88					
No use		24	-0.46	-0.58	-0.33	**128.45
Placebo		6	-0.17	-0.70	0.35	3.81
Subject selection	2.49					
Non random		20	-0.48	-0.61	-0.34	**119.27
Random		10	-0.28	-0.57	0.01	12.39
Blind	6.75					
No blind		23	-0.48	-0.61	-0.35	**125.36
Blind		5	-0.07	-0.46	0.32	0.31
Double-blind		2	-0.59	-1.51	0.33	1.72
Verify	**38.64					
Subjective		9	-1.14	-1.46	-0.83	**45.39
Experimenter		17	-0.32	-0.46	-0.18	**44.25
Tested		4	-0.35	-0.75	0.05	5.86
Substantive Variable						
AS experience	**38.35					
Never used		13	-0.25	-0.40	-0.10	12.69
Experienced		14	-0.97	-1.22	-0.71	**54.69

Treatment Variables						
Drug	**67.39					
Testosterone		11	-0.23	-0.39	-0.08	4.05
Androstene		1	-1.66	-3.21	-0.12	0.00
Androstane		2	-0.96	-1.53	-0.39	4.92
Estrane		5	-0.25	-0.57	0.08	4.00
Mix		11	-1.19	-1.49	-0.90	**53.80
Structure	**63.30					
C-19		0	0.00	0.00	0.00	0.00
C-17 alkylated		3	-1.04	-1.58	-0.51	6.13
C-17 esterified		17	-0.25	-0.39	-0.11	11.39
Mix		10	-1.22	-1.53	-0.91	**53.32
Form	**66.39					
IM		17	-0.27	-0.41	-0.13	14.36
PO		2	-1.80	-2.75	-0.84	0.08
Mix		10	-1.22	-1.53	-0.91	**53.32
Other		1	-0.30	-0.93	0.34	0.00
Stack	**49.71					
Yes		10	-1.22	-1.53	-0.91	**53.32
No		20	-0.30	-0.43	-0.17	31.12
Continuous Variables	Q_R		r			
Quality	**10.44	30	0.26			
Dose	4.23	30	-0.17			
Cycle	**6.90	27	0.24			

Note. Q_b is a measure of between-classes effect. k = N-1. d_+ is the weighted mean effect size. Q_w is a measure of within-class effect. Q_R is a measure of regression effect. r = correlation between d and continuous variable.
* Significance indicates rejection of the hypothesis of homogeneity. ** $p < .01$

Table 6

Weighted Effect Sizes as a Function of Moderator Variables for Low-density Lipoprotein (LDL)

Variable and Class	Q_b	k	d_+	99% CI for d_+ Lower	99% CI for d_+ Upper	Q_w[a]
Methodological Variables						
Design	0.04					
Within		16	0.15	0.03	0.28	**57.95
Between		6	0.12	-0.32	0.56	2.51
Control Group	0.86					
No use		18	0.16	0.03	0.28	**59.55
Placebo		4	-0.08	-0.72	0.57	0.09
Subject selection	0.00					
Non random		14	0.15	0.02	0.29	**56.19
Random		8	0.14	-0.16	0.44	4.29
Blind	0.85					
No blind		17	0.17	0.04	0.29	**59.33
Blind		5	0.02	-0.37	0.41	0.31
Double-blind		0	0.00	0.00	0.00	0.00
Verify	**18.46					
Subjective		6	0.48	0.25	0.71	13.44
Experimenter		15	0.02	-0.13	0.17	28.59
Tested		1	0.07	-0.41	0.56	-0.00
Substantive Variable						
AS experience	**10.89					
Never used		11	-0.02	-0.19	0.15	14.33
Experienced		8	0.34	0.11	0.57	**26.00

Treatment Variables						
Drug	**38.80					
Testosterone		9	-0.13	-0.31	0.05	5.11
Androstene		1	0.83	-0.17	1.84	-0.00
Androstane		2	0.50	0.08	0.93	0.01
Estrane		3	0.13	-0.22	0.47	2.93
Mix		7	0.48	0.26	0.71	13.65
Structure	**35.34					
C-19		0	0.00	0.00	0.00	0.00
C-17 alkylated		3	0.56	0.16	0.95	0.60
C-17 esterified		12	-0.08	-0.23	0.08	10.90
Mix		7	0.48	0.26	0.71	13.65
Form	**30.79					
IM		12	-0.02	-0.18	0.13	15.59
PO		2	0.61	0.08	1.14	0.46
Mix		7	0.48	0.26	0.71	13.65
Other		1	-0.28	-0.91	0.35	-0.00
Stack	**20.72					
Yes		7	0.48	0.26	0.71	13.65
No		15	0.01	-0.14	0.16	26.12
Continuous Variables	Q_R		r			
Quality	**8.38	22	-0.39			
Dose	1.91	22	0.18			
Cycle	**14.35	19	-0.53			

Note. Q_b is a measure of between-classes effect. k = N-1. d_+ is the weighted mean effect size. Q_w is a measure of within-class effect. Q_R is a measure of regression effect. r = correlation between d and continuous variable.
[a] Significance indicates rejection of the hypothesis of homogeneity. ** $p < .01$

Table 7

Weighted Effect Sizes as a Function of Moderator Variables for Estrogen (E2)

Variable and Class	Q_b	k	d_+	99% CI for d_+ Lower	Upper	Q_w[a]
Methodological Variables						
Design	0.80					
Within		30	0.24	0.09	0.38	**163.59
Between		2	-0.12	-1.13	0.89	**11.60
Control Group	5.96					
No use		31	0.24	0.10	0.39	**170.03
Placebo		1	-0.86	-2.02	0.30	0.00
Subject selection	4.53					
Non random		22	0.29	0.13	0.46	**94.98
Random		10	0.02	-0.27	0.31	**76.49
Blind	**11.98			0.14		
No blind		23	0.31	0.14	0.47	**101.56
Blind		3	0.27	-0.17	0.72	**12.14
Double-blind		6	-0.26	-0.65	0.13	**50.31
Verify	**10.06					
Subjective		1	0.35	-0.39	1.09	0.00
Experimenter		29	0.18	0.03	0.33	**162.89
Tested		2	0.95	0.34	1.56	3.04
Substantive Variable						
AS experience	1.42					
Never used		28	0.24	0.09	0.39	**143.72
Experienced		2	0.56	-0.13	1.26	5.14

Treatment Variables						
Drug	**60.24					
Testosterone		19	0.47	0.30	0.65	**99.28
Androstene		3	0.00	-0.51	0.51	0.00
Androstane		5	-0.34	-0.73	0.04	5.13
Estrane		3	-0.90	-1.44	-0.37	6.21
Mix		2	0.56	-0.13	1.26	5.14
Structure	9.26					
C-19		8	-0.01	-0.29	0.28	**27.38
C-17 alkylated		3	0.00	-0.51	0.51	0.00
C-17 esterified		19	0.33	0.15	0.51	**134.22
Mix		2	0.56	-0.13	1.26	5.14
Form	3.36					
IM		18	0.27	0.08	0.45	**131.77
PO		5	0.08	-0.27	0.43	1.00
Mix		2	0.56	-0.13	1.26	5.14
Other		7	0.22	-0.21	0.65	**34.73
Stack						
Yes	1.63	2	0.56	-0.13	1.26	5.14
No		30	0.21	0.07	0.36	**169.23
Continuous Variables	Q_R		r			
Quality	4.91	32	-0.19			
Dose	1.89	32	-0.11			
Cycle	2.59	32	0.12			

Note. Q_b is a measure of between-classes effect. k = N-1. d_+ is the weighted mean effect size. Q_w is a measure of within-class effect. Q_R is a measure of regression effect. r = correlation between d and continuous variable.
* Significance indicates rejection of the hypothesis of homogeneity. ** $p < .01$

Estrogen

Methodological variables. As presented in Table 7, the computed Q_b value for the test of differences in effect size across the various methodological study-features for E2 were significant for Blind (Q_b = 11.98) and Verify (Q_b = 10.06). However, based upon the bimodal pattern of effect sizes for these particular variables, it is hard to interpret the significance of these findings.

Substantive variable. No substantive variable served as a significant moderator of AASs on E2 levels.

Treatment variables. Within the study features related to drug treatment, we see that Drug (Q_b = 65.33) is the only significant moderator of heterogeneity in the E2 findings. Apart from the mixed class, testosterone (d_+ = +0.47) has the largest positive effect upon serum E2 levels while estrane-based drugs have the largest negative effect on E2 (d_+ = -0.90). However, testosterone is also the main cause of within-class heterogeneity. Thus these findings should be interpreted with caution and further analysis is warranted.

Follicle-Stimulating Hormone

Methodological variables. No methodological variable served as a significant moderator of AASs on serum concentrations of FSH (see Table 8).

Substantive variable. No substantive variable served as a significant moderator of AASs on FSH levels.

Treatment variables. Drug (Q_b = 18.29, df = 4), Structure (Q_b = 31.17, df = 3), Form (Q_b = 28.73, df = 3), Dose (r = 0.26, Q_R = 9.26), and Cycle (r = -0.27, Q_R = 9.76) all

served as significant moderators of AAS effects on FSH. While all classes of drugs had significant negative effects on FSH levels, mixed (d_+ = -1.41) and estrane-based drugs had the greatest effect (d_+ = -0.90). An obvious cause of heterogeneity within FSH was testosterone as evidenced by the amount of within-group heterogeneity (Q_w = 83.01).

Luteinizing Hormone

Methodological variables. The only significant moderator of effect-size differences for LH was Blind (Q_b = 9.26, df = 2), revealing that larger treatment effects tend to occur in the more stringent designs (see Table 9). In fact, there is a general trend across the methodological variables for LH with greater effects coming from studies with higher internal validity.

Substantive variable. No substantive variable served as a significant moderator of AASs on LH levels.

Treatment variables. With regards to treatment variables, Drug (Q_b = 25.71, df = 4), Structure (Q_b = 30.88, df = 3), and Form (Q_b = 26.39, df = 3) all had significant values for Q_b. While all steroids have significant negative effects on LH, testosterone drugs (d_+ = -0.85) had significantly greater negative effects than did androstane-based drugs (d_+ = -0.37). Similarly, C-17 esterified drug structures (d_+ = -0.92) and IM-administered drugs (d_+ = -0.87) brought about greater decrements in LH levels than did C-17 alkylated drug structures (d_+ = -0.41) and PO-administered drugs (d_+ = -0.40) respectively.

Table 8

Weighted Effect Sizes as a Function of Moderator Variables for Follicle Stimulating Hormone (FSH)

Variable and Class	Q_b	k	d_+	99% CI for d_+ Lower	99% CI for d_+ Upper	Q_w[a]
Methodological Variables						
Design	0.53					
Within		48	-0.71	-0.83	-0.58	**96.51
Between		12	-0.82	-1.19	-0.44	**30.62
Control Group	0.15					
No use		49	-0.71	-0.84	-0.58	**99.49
Placebo		11	-0.77	-1.15	-0.39	**28.02
Subject selection	0.77					
Non random		37	-0.69	-0.83	-0.55	**67.68
Random		23	-0.78	-1.01	-0.55	**59.20
Blind	4.21					
No blind		36	-0.68	-0.82	-0.53	**66.08
Blind		14	-0.96	-1.29	-0.63	**35.92
Double-blind		6	-0.70	-1.13	-0.28	**18.07
Verify	4.50					
Subjective		2	-0.89	-1.53	-0.25	0.77
Experimenter		56	-0.69	-0.82	-0.57	**121.19
Tested		2	-1.25	-1.95	-0.54	1.21
Substantive Variable						
AS experience	1.94					
Never used		53	-0.69	-0.82	-0.57	**115.05
Experienced		6	-0.92	-1.33	-0.52	9.88

Treatment Variables						
Drug	**18.29					
Testosterone		40	-0.81	-0.96	-0.66	**83.01
Androstene		8	-0.38	-0.67	-0.10	10.61
Androstane		5	-0.49	-0.87	-0.11	7.33
Estrane		5	-0.92	-1.39	-0.45	7.53
Mix		2	-1.41	-2.40	-0.43	0.88
Structure	**31.17					
C-19		11	-0.43	-0.70	-0.16	12.25
C-17 alkylated		10	-0.43	-0.68	-0.19	17.15
C-17 esterified		34	-0.93	-1.10	-0.77	**66.20
Mix		1	-1.41	-2.40	-0.43	0.88
Form	**28.73					
IM		37	-0.89	-1.05	-0.73	**66.88
PO		13	-0.38	-0.59	-0.18	19.43
Mix		1	-1.41	-2.40	-0.43	0.88
Other		5	-0.74	-1.28	-0.21	11.74
Stack	3.37					
Yes		1	-1.41	-0.43	-0.43	0.88
No		57	-0.71	-0.58	-0.58	**123.42
Continuous Variables	Q_R		r			
Quality	0.81	60	0.02			
Dose	**9.26	60	0.26			
Cycle	**9.76	60	-0.27			

Note. Q_b is a measure of between-classes effect. k = N-1. d_+ is the weighted mean effect size. Q_w is a measure of within-class effect. Q_R is a measure of regression effect. r = correlation between d and continuous variable.
* Significance indicates rejection of the hypothesis of homogeneity. ** $p < .01$

Table 9

Weighted Effect Sizes as a Function of Moderator Variables for Luteinizing Hormone (LH)

Variable and Class	Q_b	k	d_+	99% CI for d_+ Lower	99% CI for d_+ Upper	Q_w[a]
Methodological Variables						
Design	4.33					
Within		57	-0.69	-0.81	-0.58	**132.35
Between		12	-1.02	-1.41	-0.63	**32.43
Control Group	2.89					
No use		58	-0.70	-0.82	-0.58	**137.55
Placebo		11	-0.97	-1.36	-0.58	**28.67
Subject selection	0.28					
Non random		44	-0.71	-0.84	-0.57	**97.27
Random		25	-0.76	-0.97	-0.55	**71.56
Blind	**9.26					
No blind		44	-0.66	-0.80	-0.53	**99.31
Blind		14	-1.08	-1.41	-0.75	28.04
Double-blind		7	-0.75	-1.15	-0.35	**29.12
Verify	3.72					
Subjective		2	-0.70	-1.29	-0.11	1.71
Experimenter		65	-0.71	-0.83	-0.59	**160.62
Tested		2	-1.24	-1.93	-0.54	3.06
Substantive Variable						
AS experience	1.90					
Never used		62	-0.74	-0.86	-0.62	**152.85
Experienced		6	-0.55	-0.89	-0.21	13.58

Treatment Variables						
Drug	**25.71					
Testosterone		43	-0.85	-1.00	-0.70	**82.44
Androstene		11	-0.45	-0.71	-0.19	**32.92
Androstane		8	-0.37	-0.68	-0.06	18.35
Estrane		5	-0.91	-1.38	-0.44	7.28
Mix		2	-1.48	-2.48	-0.48	2.41
Structure	**30.88					
C-19		15	-0.54	-0.79	-0.29	23.04
C-17 alkylated		13	-0.41	-0.64	-0.19	**38.86
C-17 esterified		38	-0.92	-1.08	-0.77	**73.91
Mix		2	-1.48	-2.48	-0.48	2.41
Form	**26.39					
IM		41	-0.87	-1.02	-0.72	**74.57
PO		16	-0.40	-0.61	-0.20	**40.14
Mix		2	-1.48	-2.48	-0.48	2.41
Other		10	-0.75	-1.12	-0.39	**25.60
Stack	3.82					
Yes		2	-1.48	-2.48	-0.48	2.41
No		67	-0.71	-0.83	-0.60	**162.88
Continuous Variables	Q_R		r			
Quality	1.44	69	-0.07			
Dose	0.94	69	0.03			
Cycle	1.70	69	-0.08			

Note. Q_b is a measure of between-classes effect. k = N-1. d_+ is the weighted mean effect size. Q_w is a measure of within-class effect. Q_R is a measure of regression effect. r = correlation between d and continuous variable.
* Significance indicates rejection of the hypothesis of homogeneity. ** $p < .01$

Table 10

Weighted Effect Sizes as a Function of Moderator Variables for Testosterone (T)

Variable and Class	Q_b	k	d_+	99% CI for d_+ Lower	99% CI for d_+ Upper	Q_w[a]
Methodological Variables						
Design	6.41					
Within		66	0.28	0.17	0.40	**488.20
Between		12	0.66	0.29	1.03	**63.00
Control Group	4.76					
No use		67	0.29	0.17	0.40	**491.93
Placebo		11	0.62	0.25	0.99	**60.92
Subject selection	0.61					
Non random		50	0.30	0.17	0.43	**336.65
Random		28	0.38	0.15	0.60	**220.35
Blind	**16.17					
No blind		50	0.30	0.17	0.43	**357.94
Blind		14	0.55	0.27	0.83	**80.12
Double-blind		10	-0.20	-0.59	0.19	**96.80
Verify	**14.63					
Subjective		5	0.09	-0.37	0.55	**27.54
Experimenter		71	0.31	0.19	0.42	**515.22
Tested		2	1.37	0.62	2.12	0.23
Substantive Variable						
AS experience	1.01					
Never used		66	0.36	0.24	0.48	**457.94
Experienced		9	0.21	-0.15	057	**61.22

Treatment Variables						
Drug	**228.96					
Testosterone		50	0.71	0.57	0.84	**220.24
Androstene		9	-0.76	-1.14	-0.38	**57.37
Androstane		8	-0.73	-1.09	-0.37	**25.24
Estrane		6	-0.93	-1.34	-0.52	10.04
Mix		5	0.56	0.05.	1.07	**15.75
Structure	**121.12					
C-19		19	0.21	0.00	0.43	**72.82
C-17 alkylated		13	-0.90	-1.23	-0.58	**64.81
C-17 esterified		42	0.59	0.44	0.74	**289.55
Mix		4	0.72	0.19	1.25	9.30
Form	**72.73					
IM		45	0.56	0.42	0.70	**296.16
PO		19	-0.28	-0.50	-0.06	**124.34
Mix		4	0.72	0.19	1.25	9.30
Other		10	0.20	-0.15	0.55	**55.08
Stack	3.98					
Yes		4	0.72	0.19	1.25	9.30
No		74	0.30	0.18	0.41	**544.33
Continuous Variables	Q_R		r			
Quality	1.01	78	0.04			
Dose	0.00	77	0.02			
Cycle	3.41	78	0.08			

Note. Q_b is a measure of between-classes effect. k = N-1. d_+ is the weighted mean effect size. Q_w is a measure of within-class effect. Q_R is a measure of regression effect. r = correlation between d and continuous variable.
[a] Significance indicates rejection of the hypothesis of homogeneity. ** $p < .01$

Testosterone

Methodological variables. Blind (Q_b = 16.17, df = 3) and Verify (Q_b = 14.63, df = 2) were significant methodological moderators of AAS-effects on T (see Table 10). A common methodological pattern generally applied to the T findings in that the stronger designs brought about larger effects. For example, those findings where drug testing took place (d_+ = +1.37) had a significantly larger effect than subjective verification (d_+ = +0.09). In all cases, the above-mentioned effect sizes were from heterogeneous subclasses as witnessed by the large values for Q_w. Thus caution must be exercised when evaluating these effects.

Substantive variables. No substantive variable served as a significant moderator of AASs on serum concentrations of T.

Treatment variables. Within the categorical study features related to treatment, we see that Drug (Q_b = 228.96, df = 4), Structure (Q_b = 121.12, df = 3), and Form (Q_b = 72.73, df =3) were significant moderators of AAS-effects on T. Not surprisingly, testosterone drugs had a significant positive effect (d_+ = +0.71) on T levels while androstene (d_+ = -0.76), androstane- (d_+ = -0.73), and estrane-based drugs (d_+ = -0.93) resulted in significant decrements in T levels. According to this study feature, the main source of heterogeneity for the T findings was testosterone drugs (Q_w = 220.24). In the case of Structure, C-17 alkylated drugs had a large significant negative effect (d_+ = -0.90) on T levels while all other structures had positive effects. Similarly, PO-administered drugs significantly reduced T levels (d_+ = -0.28) while IM- (d_+ = +0.56) and mix-

administered drugs ($d_+ = +0.72$) enhanced T levels. But the fact that most of these findings were from heterogeneous subclasses suggests that further analysis is required.

Regression Analyses for Heterogeneous Outcomes

Based upon the observation that there seems to be a predominant treatment paradigm across the heterogeneous outcomes (i.e., testosterone-based drug, C-17 esterified structure, IM-administered, no stacking), and in order to be used in regression analyses, the categorical study features were dichotomized and dummy coded to reflect this pattern[7]. However, colinearity led to the removal of Structure and Stacking from any further analysis. Structure was dropped due to high correlations with the variables Drug and Form, while Stacking was dropped due to high correlations with Dose. The decision of which correlated study features to drop was based upon their relative importance to the topic as discussed in the literature. Also, while Structure was correlated with Drug and Form the latter variables were not correlated with one another and thus were deemed to be better independent predictors of AAS-effects than Structure.

In order to present a clearer picture of the multitude of data generated in these meta-analyses, three WLS regressions were performed for each heterogeneous outcome.

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Because of concerns about limited degrees of freedom in the regression analyses, and thus low power, those variables that had more than two classes (i.e., Drug, Form, Structure) were dichotomized (0, 1) instead of creating numerous dummy variables. That is, in the dummy vector for DRUG, testosterone equaled 1 and all other drugs equaled 0. In the dummy vector for FORM, IM-administered drugs equaled 1 and all other forms equaled 0. Similarly, in the dummy vector for STRUCTURE, C-17 esterified drugs equaled 1 and all other structures equaled 0. There was no need to dichotimize AS EXP or STACKING since they only had two classes apiece. Thus, in the dummy vector for AS EXP, never used equaled 1 and experienced equaled 0. Finally, in the dummy vector for STACKING, no equaled 1 and yes equaled 0.

Although the primary objective of these regressions was to account for the heterogeneity in effect sizes for each outcome, other goals are also stated. The first WLS regression only included the significant univariate moderator variables for each outcome (as were identified in the previous analysis). The goal of this analysis was to determine the independent effects of each moderator, and to identify the most important predictors of AAS-effect for each outcome, by focussing upon the magnitude and significance of the unstandardized regression coefficients for each predictor[8].

A second WLS regression was done to determine if there were any significant treatment effects over and above those due to the actions of specific drugs as assessed by Drug. In this hierarchial analysis, Drug was entered on the first step and the other treatment variables (Form, Dose, Cycle) were entered stepwise as a block on the second step. Similar to regression #1, the focus of this analysis was upon the unstandardized regression coefficients of each predictor.

The third analysis was performed in order to develop a multivariate model of the variables tested univariately. All of the variables were entered in WLS regression using a hierarchial procedure. Quality was entered on the first step in order to account for any variability in the effect sizes due to the use of differential methodology between studies. Because of concerns about small sample size for some of the outcomes, a decision was made to use Quality as the sole methodological variable in the WLS regression since it

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For the purposes of a fixed-effects meta-analysis, such as the one conducted herein, SPSS provides the correct estimates of the unstandardized regression coefficients. However, the standard errors, standardized coefficients, and significance computed by the program are based on a slightly different model than that used for fixed-effects meta-analysis (see Hedges & Olkin, 1985, for further discussion and methods for calculating correct standard errors and significance tests for the regression coefficients).

was a composite of the other 5 categorical variables. AS Exp was entered on the second step, while the treatment variables (i.e., Drug, Form, Dose, Cycle) were entered as a block on the third and final step. Therefore, the purpose of this analysis was to see if AAS use has a significant effect upon HDL, LDL, E2, FSH, LH, and T levels and if these effects are best explained by a predominant treatment paradigm.

Regression #1: Significant Moderators

Apart from FSH, direct entry of the significant moderator variables into a multiple WLS regression indicated that Drug independently accounted for variation in all outcomes (see Table 11). Form independently accounted for variation in FSH, LH, and T. The methodological variables, Blind and Verify, served as significant predictors of effect-size variation for some combination of E2 (Blind & Verify), LH (Blind), and T (Verify). Of particular interest was the fact that, controlling for Drug and Form, experienced AAS users had higher serum concentrations of HDL (unstandardized beta = +0.43) after a cycle of AASs than those users who had no previous experience with AASs. Also of note, neither Dose nor Cycle served as independent predictors of variation for any of the heterogeneous outcomes.

Table 11

Weighted Effect Sizes as a Function of Specific Drugs for Heterogeneous Outcomes

	HDL		LDL		E2		FSH		LH		T	
Drug	N	d_+	N	d_+	N	d_+	N	d_+	N	d_+	N	d_+
DHT					5	-0.34	2	-0.35	5	-0.43	5	-0.49
Fluoxymesterone					2	0.00	4	-0.19	5	-0.42	3	-0.43
Methandienone							4	-0.53	5	-0.58	5	-1.27
Methyltestosterone	1	-1.66	1	0.83	1	0.00			1	0.00	1	2.30
Mix (T & synthetics)	11	-1.19	7	0.48	2	0.57	2	-1.41	2	-1.48	5	0.56
Mesterlone							1	0.36	1	0.00	1	-0.65
Nandrolone Decanoate	4	-0.04	2	0.16	3	-0.90	2	-1.80	2	-1.74	3	-1.29
19NT-HPP	1	0.00	1	0.00			3	-0.68	3	-0.68	3	-0.68
Stanozolol	2	-0.96	2	0.51			2	-0.77	2	-0.37	2	-1.70
Testosterone					1	1.51	7	-0.72	8	-0.74	10	0.62
TeCHB							1	-1.18	1	-1.18	1	0.00
Testosterone Cypionate	1	-0.54					1	-0.65	1	-0.65	1	0.65
Testosterone Enanthate	10	-0.22	9	-0.13	16	0.48	27	-0.95	30	-0.93	34	0.86
Testosterone Propionate							1	-0.74	1	-1.36		
Testosterone Undecanoate					2	0.16	3	-0.26	2	-0.33	4	0.19

Note. DHT = dihydrotestosterone, 19NT-HPP = 19 nortestosterone hexyloxyphenlpropionate, TeCHB = testosterone cyclohexanecarboxylate

Table 12

Multiple Weighted Least Squares Regressions for Heterogeneous Outcomes: Significant Univariate Moderator Variables as Predictors

Outcome	Moderator	*B*	Q_E
HDL			56.00***
	Quality	.660	
	AS Exp	.433**	
	Drug	.373*	
	Form	.355	
LDL			12.47
	Quality	.606	
	AS Exp	-.080	
	Drug	-.724***	
	Form	.078	
	Cycle	-.001	
E2			90.85***
	Blind	-.212**	
	Verify	.406*	
	Drug	.933***	
FSH			79.33*
	Drug	-.216	
	Form	-.280*	
	Dose	.000	
	Cycle	-.010	

LH			**86.73****
	Blind	-.323***	
	Drug	-.310**	
	Form	-.280*	
T			**284.56*****
	Blind	-.030	
	Verify	.324*	
	Drug	1.482***	
	Form	.248*	

Note. Significant univariate moderator variables were entered as one block. *B* denotes unstandardized regression coefficients. Q_E denotes test of model specification.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 13

Multiple Weighted Least Squares Regressions for Heterogeneous Outcomes: Significant Univariate Treatment Moderator Variables as Predictors

Outcome	Moderator	*B*	Q_E
HDL			42.06**
	Drug	.528***	
	Dose	-.002**	
	Form	.649***	
LDL			11.96
	Drug	-.557***	
	Dose	.001*	
E2			99.92***
	Drug	1.023***	
FSH			85.19**
	Drug	-.099	
	Form	-.470***	
LH			106.65***
	Drug	-.228*	
	Form	-.305**	
T			297.65***
	Drug	1.606***	

Note. Drug was entered on the first step of a hierarchial regression with three other treatment variables (FORM, DOSE, CYCLE) being entered stepwise on the second step. *B* denotes unstandardized regression coefficients. Q_E denotes test of model specification.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Regression #2: Treatment Effect Controlling for Drug

Based upon the results from the WLS stepwise regressions, Drug independently accounted for variation in effect sizes for 5 of the 6 heterogeneous outcomes (see Table 12). Similar to what was found in Regression #1, FSH was the only outcome where Drug did not serve as a significant predictor. In the cases of E2 and T, Drug was the only significant predictor of effect-size variation. Thus, it can be interpreted that when compared to other AASs, testosterone use leads to higher serum concentrations of HDL, E2, and T and lower levels of LDL and LH (see Table 13 for further analysis).

Over and above Drug-effects, Form was a significant predictor of variation for HDL, FSH, and LH. In fact, depending on how conservatively a level of significance one adopts, Form was the only significant predictor for FSH and LH. Indicating that IM-administered drugs had a more negative effect on serum gonadotropin levels than AASs administered in other forms. Dose served as an independent predictor for HDL and LDL, with higher doses of AASs bringing about greater decreases in HDL levels and greater increases in LDL levels.

Regression #3: Model Development

The results for this regression analysis are presented in Table 14.

High-density lipoprotein. The entry of Quality on the first step of the WLS regression accounted for 7% of the variance in HDL findings ($Q_R = 7.40$, $df = 1$, $p < .01$; $Q_E = 97.22$, $df = 23$, $p < .01$) . AS Exp further accounted for another 48% of the variance ($Q_R = 57.33$, $df = 2$, $p < .01$; $Q_E = 47.30$, $df = 22$, $p < .01$). With the block of treatment variables included, the model explained 75% of the variability ($Q_R = 78.38$, *df*

= 6, p < .01; Q_E = 26.25, *df* = 18, p > .01). While this model proves to be an adequate fit, as indicated by the nonsignificant Q_E on the last step, some caution should be exercised when interpreting these findings because of concerns about small sample size.

Low-density lipoprotein. At the first step, Quality served as a significant predictor of LDL-variability explaining 13% of the variance in the effect sizes (Q_R = 7.02, *df* = 1, p < .01; Q_E = 43.88, *df* = 15, p < .01). With the inclusion of AS Exp on the second step, another 18% of the effect-size variability in LDL was accounted for (Q_R = 15.94, *df* = 2, p < .01; Q_E = 34.95, *df* = 14, p < .01). Once the treatment variables were considered in the model, the amount of variance explained jumped to 82% (Q_R = 41.63, *df* = 6, p < .01; Q_E = 9.27, *df* = 10, p > .01). However, similar to the HDL analysis, the rather small sample size leads to an inadequate ratio of predictor variables to degrees of freedom, thus caution should also be exercised when interpreting these findings.

Estrogen. At the first step, Quality explained 4% of the variance in E2 findings (Q_R = 6.03, *df* = 1, p > .01; Q_E = 132.68, *df* = 27, p < .01). Apart from using up one degree of freedom, AS Exp contributed nothing (i.e., less than 0.1% variance explained) to this E2 analysis. With the inclusion of the treatment variables, the model explained 53% of the variability (Q_R = 73.65, *df* = 6, p < .01; Q_E = 65.07, *df* = 22, p < .01). However, while these findings were consistent with the univariate categorical analyses, a significant Q_E value indicated that there was still greater than expected residual variation, and thus, the model did not provide a good fit.

Follicle-stimulating hormone. The entry of Quality on the first step of the WLS regression accounted for less than 0.1% of the variance in FSH (Q_R = 0.71, *df* = 1, p >

.01; $Q_E = 112.03$, $df = 55$, $p < .01$). AS Exp also had a minimal effect, accounting for approximately 2% of the effect-size variability in FSH. The inclusion of treatment variables explained 35% of the variance, with the final model explaining 37% of the variability in E2 ($Q_R = 41.99$, $df = 6$, $p < .01$; $Q_E = 70.75$, $df = 50$, $p > .01$). The fact that Q_E was not significant after the third step indicated that the model provided a good fit and that the majority of variability was accounted for.

Luteinizing hormone. Quality was not a significant factor for LH and only explained 4% of the variability ($Q_R = 4.73$, $df = 1$, $p > .01$; $Q_E = 126.26$, $df = 64$, $p < .01$). On the second step, while not significant, the inclusion of AS Exp accounted for another 5% of the variability. Once treatment variables were considered in the model, the amount of variance explained jumped to 23% ($Q_R = 29.58$, $df = 6$, $p < .01$; $Q_E = 101.42$, $df = 57$, $p < .01$). However, a significant value for Q_E indicated that the model did not provide a good fit.

Testosterone. Quality accounted for less than 1% of effect-size variability when first entered in the regression for T ($Q_R = 2.26$, $df = 1$, $p > .01$; $Q_E = 511.31$, $df = 70$, $p < .01$). Similarly, AS Exp explained less than 1% of the effect-size variability. But with the block of treatment variables entered, the model was significant and accounted for 46% of the variance ($Q_R = 238.27$, $df = 6$, $p < .01$; $Q_E = 275.30$, $df = 65$, $p < .01$). However, this model did not provide a good fit as indicated by a very large and significant Q_E value.

Table 14

Multiple Weighted Least Squares Regressions for Heterogeneous Outcomes: Model Development

Outcome	$R^2_{Treatment}$	Overall R^2	Q_R	Q_E
HDL	.20	.75	78.38***	26.25
LDL	.50	.82	41.63***	9.27
E2	.49	.53	73.64***	65.07***
FSH	.35	.37	41.99***	70.74
LH	.18	.23	29.58***	101.42***
T	.46	.46	238.27***	275.30***

Note. In this set of hierarchial regressions, QUALITY was entered on the first step so as to account for any variability due to methodological differences. AS EXP was entered on the second step. On the third step, four variables related to drug effects (DRUG, FORM, DOSE, CYCLE) were entered as a treatment block. $R^2_{Treatment}$ relates to the four variables in the treatment block. Q_R is a measure of regression effect. Q_E denotes test of model specification.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Summary

The present study attempted quantitatively to summarize the psychological, physiological, and ergogenic effects of anabolic-androgenic steroids (AAS)s in healthy humans. Using the format of a meta-analysis, data from 127 studies and 35 outcomes was converted into 894 effect sizes and summarized across studies. To account for any significant variability within effect sizes, each finding was coded for 15 moderator variables. Unfortunately, due to a lack of adequate findings, no psychological outcome was included in the analysis.

As indicated by the magnitude of the average effect size, the use of AASs results in large treatment effects for sperm concentration (d_+ = -0.99), testicle size (d_+ = -0.83), follicle-stimulating hormone (d_+ = -0.72), and luteinizing hormone (d_+ = -0.72). Moderate treatment effects were observed for lean body mass (d_+ = 0.63), thigh circumference (d_+ = 0.38), Apo-AI (d_+ = -0.47), high-density lipoprotein (d_+ = -0.44), sex hormone binding-globulin (d_+ = -0.60), albumin (d_+ = -0.40), bench press (d_+ = 0.59), and squat (d_+ = 0.69). Small treatment effects were observed for hemoglobin (d_+ = 0.22), hematocrit (d_+ = 0.20), body weight (d_+ = 0.32), biceps circumference (d_+ = 0.32), low-density lipoprotein (d_+ = 0.15), serum estrogen (d_+ = 0.23), and serum testosterone (d_+ = 0.32).

However, significant heterogeneity among effect sizes for high-density lipoprotein (HDL), low-density lipoprotein (LDL), estrogen (E2), follicle-stimulating hormone (FSH), luteinizing hormone (LH), and testosterone (T) indicated that these findings were not reliable and that further analysis of moderator variables was required. Overall, type of drug and route of administration were identified as the two most important moderators of variability in the six heterogeneous outcomes. Specifically, testosterone and its esters produced higher serum concentrations of HDL, E2, and T and lower levels of LDL and LH than other synthetic steroids. Also, when compared with oral compounds, the use of parenterally-administered steroids resulted in higher serum concentrations of T and lower levels of FSH and LH.

Models incorporating indicators of study quality and subjects' experience with AASs along with treatment variables (i.e., drug, form, dose, and cycle length) sufficiently

accounted for the heterogeneity in HDL, LDL, and FSH. However, excess heterogeneity in effect sizes prevents conclusions of any certainty being made about the relationship between AASs and E2, LH, or T.

The findings from the present series of meta-analyses will be further summarized and interpreted within the following 7 broad areas: blood chemistry, cardiovascular measures, liver enzymes and plasma proteins, endocrine system, reproductive system, body dimensions, and performance. Also, for the purposes of this discussion and in keeping with the suggestions of Cohen (1988; 1992), a small effect size is anything smaller in absolute magnitude than 0.34, a medium effect size ranges from 0.35 to 0.70, and a large effect size is anything greater in absolute magnitude than 0.70. According to Cohen (1992), a medium effect size represents a change that is "likely to be visible to the naked eye of a careful observer" (p. 156)[9].

Based upon the suggestions of Eagley & Wood (1994), the findings will also be discussed in terms of how much certainty one may have in them. That is, high certainty findings are those where the relation between AAS use and outcome is consistent, as indicated by homogeneity of effect sizes, across many studies. A designation of moderate certainty will occur when fewer studies are involved or where heterogeneity in effect sizes was only understood once moderator variables were tested. Low certainty will be said to exist in those cases where heterogeneity remains unresolved even after

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While it is true that changes in many of the physiological outcomes in this analysis would not be visible to the naked eye (e.g., HDL, LDL, etc.), other changes such as in body composition or strength should be noticeable.

testing for the existence of moderator variables and/or where few studies have been done. The designation of high certainty implies that no further research needs to be done on the particular topic, while areas of moderate to low certainty require further research and even searches for new theoretical explanations.

Blood Chemistry

According to the results of this study, AAS use leads to small increases in Hct and Hb concentration while small nonsignificant increases are observed for PC and RBC. Though all outcomes are homogeneous, relatively small samples for PC and RBC, and significant 0-effects for PC and Hct, suggests that these findings be viewed with low to moderate certainty. As indicated by the data in Table 4, the PC findings are dominated by 0-effects and when these estimates are excluded the average weighted effect size increases dramatically. However, if one were to assume that the true treatment effect for PC lay between the effect size including 0-effects ($d_+ = 0.07$) and the effect size not including 0-effects ($d_+ = 0.53$), then it is very likely that there would still be a small treatment effect for PC. Similarly, while probably more reliable than the PC findings, the extreme range for Hct (i.e., 0.20 to 0.30) suggests that, at best, AAS use leads to a small treatment effect. Overall, in healthy humans, it appears that AASs will enhance the oxygen carrying capacity of the blood only in a small way. The clinical significance of the findings for blood chemistry needs to be determined to make any further judgements on the efficacy of AASs as a treatment for anemia and other blood disorders.

Cardiovascular System

While AAS use leads to a small increase in serum concentrations of LDL and

moderate decreases in Apo-AI and HDL levels, no treatment effect is observed for diastolic blood pressure, systolic blood pressure, total cholesterol, or triglycerides. In general, the lipid findings are stated with moderate certainty; however, a lack of sample size and over abundance of 0-effects lead one to make judgements of low certainty about the blood pressure findings. Interestingly, while AAS use leads to changes in serum concentrations of the various cholesterol subfractions, no change is observed in total cholesterol level. This suggests that the ratio of the various subfractions to one another (e.g., HDL:LDL), as opposed to overall cholesterol level, may be better indicators of AAS-effects. The lipid findings are consistent with those of Glazer (1991) and, in general, indicate that the use of AASs is a threat to a healthy cardiovascular profile. However, the effects of AASs on concentrations of HDL and LDL cannot be stated with any certainty unless one first takes into account several moderating factors. In particular, testosterone esters have a smaller negative effect on HDL than other synthetic steroids and may actually decrease LDL levels. Other factors such as route of administration and dose moderate AAS-effects on HDL; while LDL may be moderated by dose once drug is taken into account.

Liver Enzymes and Plasma Proteins

The liver outcomes are characterized mostly by nonsignificant treatment effects of low to moderate certainty. The only exceptions to this rule are the plasma proteins Albumin and SHBG, where medium negative AAS-effects are observed. The SHBG findings are viewed with high certainty, however, due to a small sample size (N=13), the Albumin findings can only be viewed with moderate certainty. Interestingly, these

decreases in Albumin and SHBG, which both bind with, and act as transports for, endogenous testosterone, will lead to an increased clearance rate for testosterone from the system.

Sample size is not an issue for ALAT, AP, ASAT, and Bil, however, an overabundance of 0-effects suggests that the effect sizes for these outcomes may be underestimated. The existence of a significant 0 effect for ASAT suggests that the true effect size probably lies somewhere between the two means in Table 4 ($d_+ = 0.12$ and $d_+ = 0.38$). Considering that the average effect size for ASAT is almost significant with the 0-effects included, as evidenced by the confidence intervals in Table 4, it is likely that a small significant treatment effect does exist for ASAT. However, increases in ASAT may not be a reflection of specific liver damage from steroid use but, rather, an indicator of skeletal muscle damage due to intense weight training (Friedl, 1993).

As for the other outcomes (i.e., Cr, GGT, and LDH), while all are homogeneous, small sample sizes and an abundance of 0-effects results in a vote of low confidence as to their reliability. Thus it would appear that the use of AASs by healthy individuals, typically male, does not result in severe damage to the liver as indicated by liver enzyme and protein levels. In particular, the liver-specific enzymes (GGT and LDH) are not affected adversely by AASs. However, these claims can only be made with low certainty due to insufficient evidence of dubious quality.

Endocrine System

Not surprisingly, AASs have very diverse effects on the hormonal outcomes included in these analyses. That is, small positive treatment effects are witnessed for E2

and T and large negative effects occur for the gonadotropins (FSH and LH). However, these outcomes exhibit significant heterogeneity among effect sizes. The FSH findings are viewed with moderate certainty once heterogeneity is resolved. But the fact that excess heterogeneity could not be resolved within E2, LH, and T suggests that the overall results for these outcomes can only be stated with low certainty.

Drug is the best predictor of AAS-effects for E2 and T due mainly to the fact that testosterone esters increase serum concentrations of E2 ($d_+ = 0.47$) and T ($d_+ = 0.71$); while other synthetic steroids have no effect or decrease the concentrations of these hormones. On the other hand, all AASs have a negative effect on serum concentrations of FSH and LH with the only discrepancy being the magnitude of effect that the different drugs have.

Reproductive System

The use of AASs does not affect semen volume; however, large negative treatment effects of moderate to high certainty are observed for testicle size and sperm concentration because of steroid use. Although there are insufficient findings to evaluate follow-up measurements, apparently these decreases in testicle size and sperm concentration are reversible (Wilson, 1988). The large decreases in sperm concentration are good evidence for the efficacy of these drugs, testosterone in particular, as male contraceptives. However, the fact that only 50-70% of Caucasian males administered exogenous testosterone achieve complete azoospermia (Anderson, Wallace, & Wu, 1996; Anderson & Wu, 1996), suggests that these effects are not uniform across all individuals and populations and that further research is required to identify moderating factors.

Body Dimensions

Overall, small to medium treatment effects of moderate to high certainty are witnessed in the body dimension outcomes. That is, AAS use leads to small increases in BWt and biceps circumference and moderate increases in thigh circumference and LBM. While these latter outcomes may suffer from small sample sizes, it does appear that the findings are consistent and moderately reliable. Interestingly, while almost a two-thirds of a standard deviation increase in LBM and only one-third of a standard deviation increase in BWt is found, there appears to be no changes in %BF because of AAS use.

Performance

Based upon the ergogenic findings in these meta-analyses, sufficient evidence exists to suggest that the use of AASs in association with a weight-training program will, at a minimum, lead to moderate improvements in strength performance as measured by bench press and squat press. Further support for strength improvement can be gleaned from the body dimensions outcomes where increases in biceps circumference, thigh circumference and LBM are indicative of muscle growth. The strength findings are stated with only moderate certainty because they generally originate from studies in which the most common dosages and cycle lengths are not representative of how these drugs are typically used as ergogenic aids. Thus, it is likely that larger dosages and longer cycle lengths will lead to even greater treatment effects in this area (cf. Bhasin et al., 1996; Forbes, 1985).

In sum, based upon the findings of this analysis, there should be no doubt about the fact AASs do indeed increase strength in association with a weight-training program.

While the basic strength question is no longer moot, there still can be some discussion about which drugs are more effective and at what dosages.

As for aerobic capacity, there may be almost no ergogenic benefit from using AASs. However, because of insufficient findings and because these few findings are dominated by effect sizes estimated as 0, a rating of low certainty would have to be accorded to the aerobic-capacity data in these meta-analyses. Based upon the blood chemistry outcomes, such as small increases in Hb and Hct and small though nonsignificant increases in RBC, there is reason to expect that AASs should influence aerobic capacity to some degree. However, the existence of a discrepancy between the most common cycle lengths in the aerobic-capacity studies (3 wks.) and the blood-chemistry studies (12 to 24 wks.) suggests that the lack of treatment effect for aerobic capacity may be due to insufficient exposure time to the steroids. Furthermore, though no significant difference exists between effect sizes estimated as 0 and other effect-size estimates, it is likely that the inclusion of these 0-effects has led to a more conservative estimate of aerobic capacity.

V. Discussion

This chapter will discuss the findings and implications of the present study. The focus of the chapter is on the following key areas: (1) discussion of findings, (2) conclusions, and (3) recommendations for future research.

Discussion of Findings

Within the series of meta-analyses presented in this paper, the 35 most studied dependent variables in the AAS literature were identified and analysed. In general, the findings support the consensus of previous reviews and meta-analyses that the largest treatment effects in healthy humans occur in the endocrinological and reproductive systems; while the most detrimental steroid-effects are likely related to distortions in cholesterol and lipid concentrations. Conversely, although much noise has been made about the possible dangers of AASs to hepatic functioning little evidence of any magnitude or consistency was found in these analyses to support or dispute such concerns[10]. Due to a lack of experimental or quasi-experimental studies no variable within the psychological domain was included in these analyses. Thus, questions still remain unanswered about the psychogenic effects of AASs and the validity of such

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Interpretation of hepatotoxicity of AASs from these analyses should be done with extreme caution. As indicated in the results section, most of the hepatic findings are viewed with low to moderate certainty. Also, an overabundance of findings from studies using parenteral compounds may have overshadowed the much discussed hepatotoxic effect of alkylated compounds. Finally, the common practice of dropping individuals from clinical AAS studies if they show distorted liver enzyme levels (e.g., Kluft et al., 1984) may result in an underestimation of hepatic effects.

conditions as 'roid rage'. However, the findings of these analyses should put to rest the controversy about the strength-enhancing properties of AASs. That is, AASs in association with a weight-training program do indeed increase strength as measured by bench press or squat press.

Unfortunately, due to the fact that very few AAS studies have been conducted with female participants, it was not feasible to assess gender as a moderator of AAS-effects. Thus, the findings are based upon healthy males and should only be generalized to healthy males.

Possible explanations for some of the significant changes in outcomes found in these analyses have been discussed in the literature. For example, mechanisms for the relationship between AASs and enhanced erythropoiesis (Evens & Amerson, 1974), changes in HDL and LDL concentrations (Gillmer, 1992; Melchart & Welder, 1995), enhanced muscular strength (Hickson et al., 1990), and decreased sperm concentration (Wilson, 1988) have been presented and debated. However, the extent to which such mechanisms do explain the findings in these analyses is only speculation seeing as how these mechanisms have not been tested enough in the literature. At some future point, if enough studies have been done, some of these mechanisms could be tested in a true multivariate meta-analysis (Shadish, 1996).

The Role of Moderator Variables

Of the 16 moderator variables identified and tested in this study, drug and route of

administration are the most important[11]. As both univariate categorical models and within the various regressions, these two variables consistently serve as significant predictors of AAS-effects and account for significant proportions of effect-size heterogeneity. Based upon the Q_bs of the univariate analyses and the regression analyses, it would appear that drug is the most important predictor of the two. However, once the variability due to drug is accounted for, route of administration often still serves as a significant predictor of AAS-effects (i.e., HDL, FSH, LH). The major dichotomies within the variables are between testosterone esters and other steroids and between parenteral and oral steroids. The testosterone esters increase serum concentrations of estradiol and HDL and decrease gonadotropin levels more than most other steroids. Similarly, parenterally administered AASs have a less detrimental effect on HDL concentrations and a more negative effect on gonadotropin levels than oral AASs.

Surprisingly, dose and cycle length do not serve as key independent moderators of AAS-effects in these analyses. While some significant correlations exist between dose and/or cycle length and AAS-effects in the univariate categorical models, these relationships tend to disappear once other predictors are taken into account. For instance, neither dose nor cycle length serve as significant predictors when all significant univariate moderators are entered in a regression (regression #1). Similarly, dose only

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One reviewer of this thesis suggested that the various analytical procedures used to determine levels of the outcome variables (e.g., hormone assays, strength measures) may serve as moderators of effect-size heterogeneity. For instance, they pointed out that recent laboratory advances have significantly improved the ability to assess hormone levels. This is a good point and while it is impossible to determine the role of analytical procedures at this time, future reviews should give consideration to analytical procedures as possible moderators of AAS effects.

appears as a significant treatment variable for HDL and LDL once the variation due to drug is removed (regression #2). It may be that the analyses are conducted at too broad a level for these particular indicators to have any bearing. That is, due to the differential dosages administered for many drugs, particularly the differences between parenteral (e.g., 200 mg/wk) and oral (e.g., 35 mg/wk) dosages, it may be that dose and even cycle length are irrelevant until the analysis gets to the level of specific drugs or families of drugs. Another possible explanation for the lack of dose effects is that androgen receptors in the body may become saturated at even moderate doses of some AASs.

Because most of the findings in these meta-analyses are from within-group designs, there was some concern that the overall average effect sizes would be biased by the abundance of these weaker designs. Typically, within-group designs result in larger treatment effects than between-groups designs (Ray & Shadish, 1996). Also, Shadish and Ragsdale (1996) have found that studies using random assignment of participants in controlled experiments yield larger effect sizes than those using nonrandom assignment. However, apart from HDL, neither design nor subject selection serve as significant univariate moderators of AAS-effects. This indicates that design and overall study quality are not significant moderators for 5 of the 6 heterogeneous outcomes. In the case of HDL, study quality did not serve as a significant independent predictor of AAS-effects once other moderators are taken into account. It is most likely that the statistical corrections made for effect sizes from within-group designs (Becker, 1988) reduced the overestimation of treatment effect in these analyses. Overall, study quality is not a major factor in these meta-analyses. However, because the relationship between design factors

and other variables is hard to tease out in meta-analyses (Shadish & Ragsdale, 1996), it would be nice to see more true experimental designs, including random assignment of participants, in the AAS literature.

In summary, when discussing the effects of AASs on serum concentrations of HDL, LDL, E2, LH, FSH, and T one must first consider the specific steroid or family of steroids involved and the route by which the drug is administered. In particular, injectable testosterone esters have quite different effects than oral steroids in these areas. Factors such as dose and cycle length are almost irrelevant until drug and route of administration are taken into account. However, the presence of unexplainable heterogeneity in some outcomes (i.e., E2, LH, & T), means that caution should be taken when interpreting those findings and that other sources of variability, not been assessed in this analysis, may exist.

Comparison of Findings with Other AAS Meta-Analyses

In comparing the findings of this study with previous meta-analyses on AASs (Elashoff et al., 1991; Glazer, 1991), it is encouraging to note that a similar pattern in effect sizes is observed. That is, AAS use leads to increased muscle strength and LDL concentrations and decreased HDL concentrations. However, differences in effect-size magnitude are to be noted. In particular, Elashoff et al. found a rather large treatment effect for AASs on strength (ES = 1.0) while more moderate effects were found in these meta-analyses ($d_+ = 0.59$ for bench press; $d_+ = 0.69$ for squat). These differences are most likely due to some combination of the following factors: differences in inclusion criteria, the presence of new studies since 1991, and the difference between using

specific categories of strength measures as opposed to grouping all measures under a general category.

Seeing as how Glazer (1991) used percent change as their measure of treatment effect, it is hard to compare the findings of this study with their findings. However, as was mentioned previously, similar patterns in treatment effect seem to emerge. Also, as was found in this study, Glazer surmised that route of administration and dose were important moderators of AAS-effects on HDL and LDL concentrations.

Taken together the findings of the present study in association with Elashoff et al. (1991) and Glazer (1991) provide a good window on the status of AAS research to date. However, meta-analyses are only as good as the studies they summarize and, in general, more studies of better quality are required to allow us to make claims with more certainty about the various effects of AASs. Specifically, studies testing mechanisms (e.g., HDL & HTLA), using true experimental designs, with varying dosages, and different drugs are required.

Sample Size and Power Issues

According to Cohen (1988), in order to detect a medium difference between two independent sample means ($d = .50$) at $\alpha = .05$ (two-tailed test) and power = .80 requires a sample of N = 64 in each group[12]. To detect a large treatment effect ($d = .80$), under similar conditions, a sample of N = 26 in each group would be required. Based upon the sample sizes of the studies in this meta-analysis it can be surmised that AAS studies are

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Overall & Doyle (1994) provide a good discussion on determining sample size for repeated measures designs.

generally undersized and underpowered. That is, researchers are not using samples of sufficient size to provide adequate power in their studies in order to detect significant findings for the majority of outcomes in the area.

For example, one of the most controversial topics in the AAS literature has to do with the strength-enhancing properties of the drugs. In most narrative reviews vote-counting procedures have been used to categorize strength studies by the significance of their findings. Thus treatment effect has rarely been discussed. In this meta-analysis, the unweighted median effect size ($M = 0.46$), the unweighted mean effect size ($d = 0.70$), and the weighted mean effect size ($d_+ = 0.59$) indicate at least a medium treatment effect for bench press across studies. However, in order for this treatment effect to be detected as being significant, the sample sizes of both control and experimental groups would have to range from 26 to 64 depending upon the size of the effect one chooses to use. Since the sample sizes in the bench press studies actually range from 3 (Ariel, 1973; Loughton & Ruhling, 1977; Maul, 1971) to 15 (Fahey & Brown, 1973) it is obvious that some decent sized treatment effects are going undetected and being classified as nonsignificant. A similar scenario occurs in other outcomes in the AAS literature. Thus, it is likely that lack of statistical power has contributed to some of the confusion surrounding AASs.

Implications of Findings for AAS Education Programs

Although there has been some discussion of AAS education/prevention programs in the literature (Goldberg, Bents, Bosworth, Trevisan, & Elliot, 1991; Goldberg, Bosworth, Bents, & Trevisan, 1990; Goldberg et al., 1996), it is hard to assess the content

of their message. That is, the discussion usually focuses upon the effectiveness of the program as opposed to the content of the message.

In light of the findings of this study, any future prevention or education program developed around AAS use should focus upon the detrimental effects to the cardiovascular, endocrinological, and reproductive systems (especially for males)[13]. At the same time, hyperbole about liver damage and psychological changes should be kept to a minimum. Also, the strength-enhancing properties of AASs should not be denied. From a public health standpoint we are most likely doing more harm than good by disputing the ergogenic potential of AASs while at the same time making grand claims about the psychogenic and addictive effects of the drugs. If the bulk of users do not experience such psychological effects then they are more likely to ignore what is said about other, more substantiated, dangers such as distorted serum lipids. Also, some of the more experienced users have a fairly sophisticated knowledge of the medical and clinical research (Goldman, Bush, & Klatz, 1987). As a result, anytime an unsubstantiated claim about AASs is made by an individual or organization in the health field they are often greeted by cynicism in the underground bodybuilding press. Thus, potential and novice users are exposed constantly to this war of words and probably become jaded about what those in authority have to say about the pros and cons of AAS use. Therefore, in order to foster the trust and respect of AAS users, it is important that

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The manner in which this message is presented may also need to be reconsidered. For example, Rothman and Salovey (1997) suggest that subsequent health behaviors are influenced by whether the message is framed in terms of benefits or costs associated with a behavior. Thus, instead of constantly talking about the negative effects associated with AAS use, it may be more effective to talk about the benefits or gains in health that may occur by avoiding AASs (e.g., proper sexual functioning).

any steroid campaign or education program be based upon sound scientific evidence that is relevant to the targeted population (i.e., don't use information based upon patient populations for adolescent recreational AAS users). While it is unlikely that education alone will change all user's and potential user's beliefs and attitudes about AAS use, it is imperative that the correct information be provided in an unbiased and non-judgemental manner in order to foster trust and understanding on the part of the users (Ardito, Goldstein, Bahrke, & Stattler, 1994).

Implications of Findings for Testosterone as a Male Contraceptive

The evidence gleaned from these analyses would suggest that testosterone can serve as a relatively safe and effective contraceptive agent for males. Large decreases in sperm concentration with a small decrease in HDL levels are witnessed. While not significant, exogenous testosterone seems to also have a small negative effect on serum concentration of LDL. If this truly is the case, then the HDL:LDL ratio is showing almost no change with testosterone administration, suggesting that the cardiovascular risk due to distortions in lipids is minimal. While the treatment effect for sperm concentration is large, somewhere between 30-50% of Caucasian males do not achieve complete azoospermia after testosterone administration (Anderson & Wu, 1996). This suggests that further research is required to identify those individuals for which such therapy is effective and possible reasons for the lower efficacy of testosterone in others. Also, there is some concern that exogenous testosterone use will increase risk for prostate disease in healthy males (Bhasin & Bremner, 1997; Jin, Turner, Walters, & Handelsman, 1996). While the jury is still out in this regard, monitoring of prostate volume and serum

concentration of prostate-specific antigens should be performed regularly when androgens of any kind are being consumed by males. Distortions in either one of these indicators should lead to termination of the testosterone therapy.

Some Final Thoughts

Instead of conducting 35 univariate analyses, as was done in this study, some authors have suggested that multiple dependent outcomes in meta-analyses be analysed within a true multivariate meta-analysis (Gleser & Olkin, 1994; Raudenbush, Becker, & Kalaian, 1988; Shadish, 1996). In this procedure all 35 outcomes would have been considered together in an analysis akin to a MANOVA. The reason being that some of these outcome variables (e.g., total cholesterol and HDL) are most likely correlated and to ignore this dependency between variables may lead to the committing of statistical errors (Raudenbush, 1994). However, such a multivariate analysis requires the inclusion of the within-study correlations between outcomes. If these within-study or sample correlations are not available then values for population correlations are supposed to be imputed from other sources (e.g., other studies, test manuals, etc.). For this meta-analysis, because very few studies provide complete within-study correlations, it was felt that the combinations and permutations arising from 35 outcomes and 127 studies would lead to massive imputation of data that, in turn, would be no better than the univariate approach used herein. In the future, an effort should be made to identify the various correlations between outcomes and incorporate them in a true multivariate meta-analysis of the AAS literature.

Similarly, instead of using a fixed-effects model as was done throughout this analysis, heterogeneous outcomes should be analysed within a random-effects model (see Shadish & Haddock, 1994). Significant unresolved heterogeneity in effects sizes suggests the presence of random effects that no moderator variable or proposed model could account for. The random-effects model provides a more conservative estimate of treatment effect, and the associated confidence intervals, in the presence of unexplained heterogeneity. Thus, one can be more confident in the conclusions drawn from such data.

Finally, the findings of these meta-analyses should in no way be considered the last words on the physiological and ergogenic effects of AASs. Rather, they should be viewed as the first step in trying to provide an objective summary of the AAS literature. In this regard, it is the intention of this author to keep the meta-analyses presented in this dissertation alive. That is, as new studies bring relevant data to bear, they will be added to the existing database. Thus, these meta-analyses will be living entities that will grow with the AAS literature and provide an up-to-date gauge of treatment effect within many different outcomes. The idea of a "living meta-analysis" is important because it suggests the provision of a continuous and consistent assessment of the literature as opposed to the stop-and-go efforts that one typically sees in current research. It is hoped that the findings presented herein, and the resulting recommendations, will lead to more and better studies in the area that will in turn fuel the meta-analyses that have been started. Because of this process there may come a point, with the introduction of new and more methodologically sound studies, when psychological outcomes can be added to the list or when inconsistencies will be resolved within the heterogenous outcomes.

Conclusions

Within the limitations of the present series of meta-analyses, the following conclusions appear to be justified:

General Conclusions and Observations

- Overall, the AAS literature is populated by studies with weak internal validity and low power. That is, true experimental designs, including placebo controls and random assignment to groups, are used infrequently and more often than not convenient small samples are the focus of study.
- Apart from the effect on various endogenous hormone levels (i.e., testosterone, estradiol, follicle-stimulating hormone, etc.), exogenous testosterone tends to have less dramatic effect than synthetic steroids in most capacities.

Specific Conclusions

- The psychological area has been under researched and is particularly beset with studies of weak methodology to the extent that no psychological outcome is included in the meta-analyses discussed herein.
- A regimen of AASs will induce small increases in blood hemoglobin and hematocrit. However, these small changes in blood parameters do not seem to translate into enhanced aerobic capacity.
- True to their name, AASs promote tissue growth as evidenced by small to moderate increases in body weight, biceps circumference, thigh circumference, and lean body mass.
- Blood pressure is not affected by a cycle of AASs.

- While having almost no effect on overall serum triglyceride or cholesterol levels, AASs do have moderate negative effects on Apo-AI and HDL concentrations and a small positive effect on LDL levels. However, the effects for HDL and LDL are moderated by the following factors:

    1. Oral synthetic steroids cause larger changes in both HDL and LDL than do injected testosterone esters.

    2. Small dose effects are observed for both HDL and LDL. That is, the higher the dose the greater the decrement in HDL levels and greater the increase in LDL levels.

- Small to moderate increases in serum estradiol and testosterone occur from AAS use. However, these effects are moderated by the following factors:

    1. Testosterone esters cause large increases in both serum estradiol and testosterone levels while most other synthetic steroids actually decrease the concentrations of these hormones.

- The use of AASs causes large decreases in serum concentrations of follicle-stimulating hormone and luteinizing hormone. However, the gonadotropins are moderated by the following factors:

    1. Parenterally-administered steroids have larger negative effects on gonadotropin levels than oral steroids.

    2. Other than estrane-based drugs, testosterone esters have larger negative effects on luteinizing hormone levels than other synthetic steroids.

- Apart from albumin and sex hormone binding-globulin, where moderate decreases are observed, it is very hard to make conclusions with any certainty about the relationship between AAS-use and liver enzymes and plasma proteins. It seems that AASs cause small non-significant increases in alanine aminotransferase, aspartate aminotransferase, creatinine, and lactate dehydrogenase and do not affect alkaline phosphatase, bilirubin, and gamma-glutamyltransferase. However, until more studies start reporting means and standard deviations for non-significant findings it will be impossible to make any firmer conclusions about AASs and liver functions.
- Strength performance, as measured by bench press and squat, is enhanced moderately by AASs. Research in this area is dominated by studies using synthetic oral steroids (e.g., methandrostenolone).
- Regarding male reproductive functions, AASs have large negative effects on testicle size and sperm concentration. These decrements in sperm concentration are severe enough to render most males infertile. However, AASs have very little effect on semen volume.

Recommendations for Future Research

Based on the findings of this series of meta-analyses, the following recommendations for future research should be considered:

Methodological Considerations

1. In the future, more true experimental designs--including placebo control

groups and random assignment of participants to groups--should be used in AAS studies. For reasons most likely related to ethical considerations, most studies in the area use either cross-sectional or single-group pre-post designs. In fact, almost 70% of the findings across these meta-analyses are from within-group designs. Without true experimental designs it is very hard to assess treatment effects and very difficult to generalize the findings of a group of studies beyond the specific parameters of those studies. Also, since there is a suggestion that AAS-effects such as muscular strength and aggression may be due to expectancy effects (Cicero & O'Connor, 1990; Riem, 1992), there is a real need for placebo-control groups.

2. Sample size, or lack of, is another issue that should be of concern to AAS-researchers. Again, most likely a result of ethical concerns about administering AASs to healthy human participants, most studies suffer from low power due to insufficient sample size that may in turn lead to nonsignificant findings. In particular, studies investigating the ergogenic and psychological effects of AASs are plagued by small samples. It is only in the more recent contraceptive studies (e.g., World Health Organization Task Force on Methods for the Regulation of Male Fertility, 1996) that adequate samples are being utilized. Thus, it is highly recommended that researchers in the area use power analysis (Cohen, 1988) to estimate the minimum required sample size to detect any treatment effect. Cohen (1992) suggests that one possible reason why more researchers do not calculate power is that they have very little awareness of the "magnitude of the phenomena that they are investigating". In other words, they do not know what size of a treatment effect to expect from their intervention. If nothing else,

the effect sizes generated in this study should eliminate some of this ignorance.

3. Apart from the fact that means and standard deviations should be reported in every study, despite whether the findings are significant or not, researchers should also be encouraged to include some measure of treatment effect (e.g., effect size) in their table of results. This is even more critical to the AAS literature where, as mentioned above, studies quite often lack sufficient power to detect small to moderate treatment effects that may in turn have important clinical implications. Not one study in this meta-analysis included any measure of treatment effect and many were missing complete descriptive statistics.

4. More studies in the future should include multiple steroids and doses in their designs. That is, few studies have provided adequate comparisons between the various AASs and, more important, a serious lack of information exists on dose effects.

5. Overall, there is a need for long-term follow-up studies in the AAS literature. Most of the information available on outcome effects is based upon immediate post-test evaluations. While the possibility of long-term effects is often alluded to, they are rarely measured. For example, though we have an understanding about the acute lipoproteinemic effects of AASs, and that these effects are reversed once steroid treatment is terminated, we have very little insight on the chronic lipoproteinemic effects of cycling on and off these drugs.

Areas for Further Study

6. Without a doubt, there is a need for more well controlled studies investigating the psychological effects of AASs. The fact that no psychological outcomes are included

in these meta-analyses is a testament to this claim. Furthermore, if AASs are going to be considered as contraceptive agents for healthy males, and thus used on a much larger scale than is already the case, then there is a need to understand the potential psychogenic effects of these drugs better. As it stands now, most of the knowledge in this area is based on animal studies, anecdotal evidence, and retrospective studies.

7. Several recent studies (Baumstark, Schwarz, Huonker, Keul, & Berg, 1996; Cohen et al., 1996) have found that, while the HDL:LDL ratio is reduced, AAS use may have a positive arthogenic effect due to decreases in lipoprotein (a) [Lp(a)] concentrations in healthy males. It is understood that Lp(a) is associated with a twofold increase in the risk for coronary artery disease when serum concentrations are above 30 mg/dl^{-1} (Lobo et al., 1992), thus decreases in Lp(a) are beneficial for cardiovascular health. Moreover, another recent study (Karila, Laaksonen, Jokelainen, Himberg, & Seppälä, 1996) has found that AASs increase serum ubiquinones (mitochondrial coenzymes with antioxidant properties) which may in fact protect against atherosclerosis. These findings need to be confirmed in larger experimental studies along with further documentation of the mediating role that the enzymes HTGL and LPL play in the AAS-cholesterol relationship. In particular, identifying what effect exogenous testosterone has upon serum concentrations of Lp(a) and ubiquinones is important.

8. In keeping with the above recommendations in the cholesterol area, another suggestion would be that researchers should investigate the role that physical activity could play in reducing the possible arthogenic effects of AASs. For example, sufficient evidence exists to suggest that physical activity is inversely related to myocardial

infarction risk (O'Connor et al., 1995; Rauramaa & Leon, 1996; Tucker & Silvester, 1996). At least one study (Leon, 1991) has found that regular physical activity can increase HDL levels by as much as 15%. Thus, the small to moderate decreases in HDL associated with the administration of exogenous testosterone could be counteracted by a regimen of physical activity (see Berg, Frey, Baumstark, Halle, & Keul, 1994).

9. While a large negative treatment effect of AASs on sperm concentration was identified in this study, only 50-70% of Caucasian males administered exogenous testosterone achieve complete suppression of spermatogenesis to azoospermia (Anderson, Wallace, & Wu, 1996; Anderson & Wu, 1996). Thus, if testosterone or other AASs are to be adopted as efficient male contraceptives, then more studies are required to identify the populations and situations in which these drugs are most effective.

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Appendices

Appendix A

Glossary of Terms

Glossary

ALAT. *Alanine Aminotransferase*. Liver enzyme.

Albumin. *Albumin*. Plasma protein.

AP. *Alkaline Phosphatase*. Liver enzyme.

Apo-AI. *Apolipoprotein AI*. Cardiovascular measure.

ASAT. *Aspartate Aminotransferase*. Cardiovascular measure.

Bench. *Bench Press*. Performance.

BF%. *Percent Body Fat*. Body dimension.

Biceps circumference. *Biceps Circumference - relaxed*. Body dimension.

Bil. *Bilirubin*. Liver enzyme.

BWt. *Body Weight*. Body dimension

Cr. *Creatinine*. Liver enzyme.

d. *Unweighted effect size*.

$\mathbf{d_+}$. *Weighted effect size*. Weighted by inverse of variance.

DBP. *Diastolic Blood Pressure*. Cardiovascular measure.

E2. *Estradiol*. Endocrine system.

FSH. *Follicle-Stimulating Hormone*. Endocrine system.

GGT. *Gamma-glutamyltransferase*. Liver enzyme.

Hb. *Hemoglobin*. Blood chemistry.

Hct. *Hematocrit*. Blood chemistry.

HDL. *High Density Lipoprotein*. Cardiovascular measure.

LBM. *Lean Body Mass*. Body dimension.

LDH. *Lactate Dehydrogenase*. Liver enzyme.

LDL. *Low Density Lipoprotein*. Cardiovascular measure.

LH. *Luteinizing Hormone*. Endocrine system.

PC. *Platlet Count*. Blood chemistry.

Q_b. *Between-classes effect*. A measure of between group homogeneity in a univariate homogeneity analysis..

Q_E. Test of model specification in a weighted least squares regression.

Q_R. Is a measure of regression effect.

Q_t. *Total homogeneity*. A measure of overall homogeneity.

Q_w. *Within-class effect*. A measure of within group homogeneity in a univariate homogeneity analysis.

RBC. *Red Blood Cell*. Blood chemistry.

SBP. *Systolic Blood Pressure*. Cardiovascular measure.

SHBG. *Sex Hormone Binding-Globulin*. Plasma protein.

Sperm. *Sperm Concentration*. Reproductive system.

Squat. *Squat Press*. Performance.

SV. *Semen Volume*. Reproductive system

T. *Testosterone*. Endocrine system.

TC. *Total Cholesterol*. Cardiovascular measure.

Testes. *Testicles*. Reproductive system

TG. *Triglycerides*. Cardiovascular measure.

Thigh Circumference. *Thigh Circumference - relaxed*. Body dimension.

VO_2. *VO_2 max*. Performance

Appendix B

AAS Meta-Analysis Codebook

Rules and Regulations for SF Coding

Remember that in order to be included in the meta-analysis, a study must provide enough data to calculate an effect size. Secondly, we are most interested with between group studies of healthy humans. Finally, try to keep things simple (it helps to maintain sanity)!

1. **Cases must be independent of one another**. In other words, every effect size within a study must be distinguishable from each other by at least one category in the study features. We want to collapse cases as least as possible, however if no distinction can be made between two cases on the basis of our study features then they must be collapsed (e.g., two measures of aggression).

2. **Categories must match**. If a study is coded as a between-groups design, then the calculation of effect size can not be from within-group data. This is a critical fact because once the data are in the computer there will be much collapsing and recomputing and if the categories do not match then we have MEGA-problems.

3. **Record as much information as possible, especially when effect sizes have to be estimated**. It is very important to record sample sizes and means even when estimating from *t* or *p*, because we need to know the size of the sample and the direction of the change.

4. **Estimate effect size for non-significant findings by substituting a non-significant *t* value**. Based upon *n* we can estimate an effect size using the "Estimated *t* value". Such cases will be coded as 8 under *Quantification of Outcome*.

5. **As new studies are brought in, the nomological coding must be updated**. This is very important because new outcome categories may arise as a result of the incoming studies. Secondly, the nomological coding tells us a lot about what has not been done and we want our coding to be accurate in this respect.

6. **Do not be afraid to make up new categories or choices within the categories**. Do not feel restricted by the categories and choices that are provided. However, if you improvise be very careful in recording what was done and why.

7. **The number of studies included in the coding must match with Pro-Cite**. There should be no discrepancies between the two. If Pro-Cite has 25 studies for HDL then SPSS should also have 25 studies for HDL.

Author

Study ID#

Finding number (can have more than one finding per study)

Nature of Subjects

Gender (X+C) [assume male if not mentioned]
1. Male
2. Female
3. Mixed

Age AS (AAS users) _____

Age Ct (Control) _____

Sport or Activity of AAS users
1. Bodybuilding
2. Weightlifting
3. Mixed (bodybuilding and weightlifting)
4. Other sport
5. No sport

Sport or Activity of Controls
1. Bodybuilding
2. Weightlifting
3. Mixed (bodybuilding and weightlifting)
4. Other sport
5. No sport

Reason for AAS use
1. Experimental
2. Performance enhancement
3. Increase size/bulk

AS EXP: Previous AAS experience (assume no experience for experimental subjects)
1. Experienced (more than 70% of subjects have used AAS)
2. Some experience (less than 70% have used AAS)
3. No experience

AS USE: Previous non-use of AAS (assume never used for experimental subjects)

1. Never used
2. 6 or more weeks
3. 4 to 5 weeks

Supplements used by **AAS users**

1. Protein supplements (including amino acids)
2. Vitamin supplements (multivitamin, B-complex)
3. Wheat germ oil
4. Other
5. Mix
6. Not applicable

Supplements used by **controls**

1. Protein supplements (including amino acids)
2. Vitamin supplements (multivitamin, B-complex)
3. Wheat germ oil
4. Other
5. Mix
6. Not applicable

Diet of **AAS users**: Total caloric intake/day (avg.) _____

Diet of **controls**: Total caloric intake/day (avg.) _____

Diet of **AAS users**: Fat intake/day (avg.)

1. High (30% or more of calories)
2. Medium
3. Low (less than 20%)

Diet of **controls**: Fat intake/day (avg.)

1. High (30% or more of calories)
2. Medium
3. Low (less than 20%)

Diet of **AAS users**: Protein intake/day (avg.)

1. High (2.0 g/kg body wt or 20% of calories)
2. Medium (1.5 g/kg)
3. Low (.8 g/kg or less than 10% of calories)

Diet of **controls**: Protein intake/day (avg.)

1. High (2.0 g/kg body wt or 20% of calories)
2. Medium (1.5 g/kg)
3. Low (.8 g/kg or less than 10% of calories)

Weight training experience: **AAS users** (yrs.) _____

Weight training experience: **Controls** (yrs.) _____

Weight training experience: **AAS users** (weight training exp. > 1 month)

1. Yes
2. No

Weight training experience: **Controls** (weight training exp. > 1 month)

1. Yes
2. No

Training during cycle

1. Yes
2. No

Training regimen **AAS users**: Type of workout

1. Resistance exercise
2. Aerobic
3. Other
4. Not applicable

Training regimen **controls**: Type of workout

1. Resistance exercise
2. Aerobic
3. Other
4. Not applicable

Training regimen **AAS users**: # of sessions per week ____

Training regimen **controls**: # of sessions per week ____

Training regimen **AAS users**: # of minutes per week ____

Training regimen **controls**: # of minutes per week _____

Training regimen **AAS users**: Intensity

1. High
2. Medium
3. Low
4. Not applicable

Training regimen **controls**: Intensity

1. High
2. Medium
3. Low
4. Not applicable

Method Variables

COMP: Comparison

1. Within
2. Between (pre/post)
3. Between (posttest-only or cross-sectional)

DESIGN: Experimental Design

1. One group pretest-posttest (repeated measures)
2. Experimental vs. control group (pre/post)
3. Crossover design
4. Comparative study (posttest-only or cross-sectional)

*NAT.CT: Nature of control condition

1. No use (within group)
2. No use (between group)
3. Placebo
4. Other

CONTROL: Nature of control condition

1. No use
2. Placebo

*ASSIGN: Assignment of subjects to conditions (assume random assignment for placebo studies)

1. Non random
2. Non random matching
3. Random
4. Random after matching

ASSIGN2: Assignment of subjects to conditions (assume random assignment for placebo studies)

1. Non random
2. Random

BLIND : Subject's and experimenter's awareness of conditions (assume blind for placebo studies)

1. No blind
2. Blind
3. Double-blind

*VERIFY: Verification of AAS use (assume "subjective" unless otherwise stated)

1. Subjective
2. Urine test
3. Blood test
4. Other
5. Mix (urine and blood test)

VERIFY2: Verification of AAS use (assume "subjective" unless otherwise stated)

1. Subjective
2. Experimenter supplied drugs
3. Urine or blood test

QUALITY: Index of method quality (design + control + assign + blind + verify / 5)

Nature of Treatment

Type of drug (AAS): Generic name (assume: mix of testosterone and synthetics)

1. Testosterone
2. Methandienone
3. Nandrolone decanoate (ND)
4. Stanozolol
5. Methyltestosterone
6. Oxandrolone
7. Mix (synthetic steroids)
8. Mix (testosterone and synthetic steroids)
9. Methenolone
10. Mesterlone
11. Fluoxymesterone
12. DHT
13. Test. Enanthate (TE)
14. Test. Propionate (TP)
15. Test Cypionate (TC)

16. Test. Undecanoate (TU)
17. Norethandrolone
18. 19NT-HPP
19. TeCHB

CLASS: Type of drug (AAS): Classification by structure
1. Testosterone related
2. Androstane (DHT related)
3. Estrane (19-nortestosterone related)
4. Mix

*STRUC: Type of drug (AAS): Modification of structure
1. C-19 steroid - no alterations (i.e., testosterone)
2. Mostly C-17 alkylated (methyl - CH3 or ethyl - CH2)
3. Mostly C-17 esterified hydroxyl group
4. Other synthetics
5. Mix (synthetics)
6. Mix (testosterone and synthetics)
7. Mostly C-1 alkylated (e.g., Mesterlone, Methenolone)

STRUC2: Type of drug (AAS): Modification of structure
1. C-19 steroid - no alterations (i.e., testosterone)
2. Mostly C-17 alkylated (methyl - CH3 or ethyl - CH2)
3. Mostly C-17 esterified hydroxyl group
4. Mix (synthetics)
5. Mix (testosterone and synthetics)

*FORM: Route of administration
1. Intramuscular (IM)
2. Oral (PO)
3. Mix
4. Peroral (under tongue)
5. Infusion (IV)
6. Percutaneously (through skin)
7. Pellets (imbedded)

FORM2: Route of administration
1. IM
2. Other

STACK: Stacking?
1. Yes
2. No

CYC LTH: Length of cycle (wks.) _____

DOS.OR: Dosage (Oral): Oral drug x dosage/wk

DOS.INJ: Dosage (Parenteral): IM drug x dosage/wk

DOS.TOT: Dosage (Total): Total dosage/wk

TOT.OR: Total dosage per cycle (Oral)

TOT.INJ: Total dosage per cycle (IM)

TOT.DOS: Total dosage per cycle (Total)

TR.OR: Therapeutic ratio (Oral) - ratio of therapeutic dosage to actual dosage/wk

TR.INJ: Therapeutic ratio (Parenteral) - ratio of therapeutic dosage to actual dosage/wk

TR.TOT: Therapeutic ratio (Total) - ratio of therapeutic dosage to actual dosage/wk

Outcome

Outcome category
1. Physical
2. Psychological
3. Performance enhancement

Outcome (see attached sheet).

Outcome Type: Psychological
1. Standardized test (questionnaire)
2. Experimenter made test (non-standardized)
3. Observed (including DSM-III)
4. Self-report
5. Other

Outcome Type: Performance
1. Free weights
2. Dynamometer

3. Cable tensionometer
4. Other

Outcome Procedure: Performance
1. Maximal isometric force
2. 1 rep maximum
3. Other

Time of Measurement
1. 2 weeks or less into cycle
2. 3 - 6 weeks into cycle
3. 7 - 10 weeks into cycle
4. 10 <
5. Average across cycle (within subject)
6. Time not controlled

Source of effect size
1. Means and SDs (between groups)
2. Adjusted means and SDs (corrected for pretest - between groups)
3. F-test
4. t-test
5. Correlation
6. Significance level
7. Means and SDs (within groups)
8. Estimated missing or NS values
9. Other
10. Gain score (estimate SD)

Variance (SE or SD)
1. SD
2. SE
3. Does not apply (i.e., significance level)

Direction of change in NS findings
0. No change
1. Increase (+)
2. Decrease (-)

Study Context

PUBLISH: Publication Status (abstracts are unpublished)
1. Published
2. Unpublished

TYPE DOC: Type of document

1. Journal
2. Dissertation
3. Thesis
4. Report
5. Conference presentation
6. Book chapter
7. Abstract

YEAR: Year of Publication

1. < 1966
2. 1966-1976
3. 1977-1986
4. 1987-1996

Codes for Outcomes in AAS Meta-Analysis

1. ALAT. Alanine Aminotransferase. (#670)
2. Albumin (#680)
3. AP. Alkaline Phosphatase. (#20)
4. Apo-AI. Apolipoprotein AI. (#50)
5. ASAT. Aspartate Aminotransferase (#690)
6. Bench. Bench Press. (#90)
7. BF%. Body Fat. (#110)
8. Biceps. Biceps Circumference - relaxed. (#710)
9. Bil. Bilirubin. (#700)
10. BWt. Body Weight. (#560)
11. Cr9. Creatinine. (#730)
12. DBP. Diastolic Blood Pressure. (#750)
13. E2. Estradiol. (#160)
14. FSH. Follicle-Stimulating Hormone. (#180)
15. GGT (#770)
16. Hb. Hemoglobin. (#800)
17. Hct. Hematocrit. (#240)
18. HDL. HDL Cholesterol. (#210)
19. LBM. Lean Body Mass. (#830)
20. LDH. Lactate Dehydrogenase. (#840)

21. LDL. (#300)

22. LH. Luteinizing Hormone. (#320)

23. PC. Platlet Count. (#970)

24. RBC. Red Blood Cell. (#370)

25. SBP. Systolic Blood Pressure. (#890)

26. SHBG. Sex Hormone Binding-Globulin. (#410)

27. Sperm. Sperm Concentration. (#430)

28. Squat (#440)

29. SV. Semen Volume. (#650)

30. T. Testosterone. (#490)

31. TC. Total Cholesterol. (#500)

32. Testes. (#480)

33. TG. Triglycerides. (#520)

34. Thigh Circumference - relaxed (#920)

35. VO2 (#570)

Appendix C

Effect sizes broken down by study

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Anderson	6560.00	T	B	Male	TE	200.00	4.00	1.55
	12480.00	HDL	W	Male	TE	200.00	24.00	-.28
		LDL	W	Male	TE	200.00	24.00	-.15
		SHBG	W	Male	TE	200.00	24.00	-.63
		TC	W	Male	TE	200.00	24.00	-.19
		TG	W	Male	TE	200.00	24.00	.00
Ando	7540.00	E2	W	Male	DHT	350.00	.14	-.11
		FSH	W	Male	DHT	350.00	.14	-.53
		LH	W	Male	DHT	350.00	.14	-.39
		SHBG	W	Male	DHT	350.00	.14	-1.21
		T	W	Male	DHT	350.00	.14	-2.16
Ansell	210.00	AP	W	Male	Mix (T & AS)	.	.	.00
		TC	W	Male	Mix (T & AS)	.	.	-.35
		ALAT	W	Male	Mix (T & AS)	.	.	.00
		Albumin	W	Male	Mix (T & AS)	.	.	.00
		ASAT	W	Male	Mix (T & AS)	.	.	.00
		Bil	W	Male	Mix (T & AS)	.	.	.00
		PC	W	Male	Mix (T & AS)	.	.	.62
Ariel	240.00	Bench	W	Male	Methandrostenolone	70.00	4.00	1.81
		Squat	W	Male	Methandrostenolone	70.00	4.00	2.09
	260.00	Bench	W	Male	Methandrostenolone	105.00	4.00	2.09
		Squat	W	Male	Methandrostenolone	105.00	4.00	2.29
Arsyad	10100.00	AP	B	Male	TE	50.00	24.00	.00
		AP	B	Male	TE	100.00	24.00	.00
		FSH	B	Male	TE	50.00	24.00	-.86
		FSH	B	Male	TE	100.00	24.00	-1.80
		HDL	B	Male	TE	50.00	24.00	.00
		HDL	B	Male	TE	100.00	24.00	.00
		Hct	B	Male	TE	50.00	24.00	.00
		Hct	B	Male	TE	100.00	24.00	.00
		LDL	B	Male	TE	50.00	24.00	.00
		LDL	B	Male	TE	100.00	24.00	.00
		LH	B	Male	TE	50.00	24.00	-4.12
		LH	B	Male	TE	100.00	24.00	-2.78
		Sperm	B	Male	TE	50.00	24.00	-.76
		Sperm	B	Male	TE	100.00	24.00	-2.75
		Testes	B	Male	TE	50.00	24.00	-1.09
		Testes	B	Male	TE	100.00	24.00	-1.53

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Arsyad	10100.00	TC	B	Male	TE	50.00	24.00	.00
		TC	B	Male	TE	100.00	24.00	.00
		TG	B	Male	TE	50.00	24.00	.00
		TG	B	Male	TE	100.00	24.00	.00
		ALAT	B	Male	TE	50.00	24.00	.00
		ALAT	B	Male	TE	100.00	24.00	.00
		ASAT	B	Male	TE	50.00	24.00	.00
		ASAT	B	Male	TE	100.00	24.00	.00
		Cr9	B	Male	TE	50.00	24.00	.00
		Cr9	B	Male	TE	100.00	24.00	.00
		GGT	B	Male	TE	50.00	24.00	.00
		GGT	B	Male	TE	100.00	24.00	.00
		Hb	B	Male	TE	50.00	24.00	.00
		Hb	B	Male	TE	100.00	24.00	.00
		PC	B	Male	TE	50.00	24.00	.00
		PC	B	Male	TE	100.00	24.00	.00
Bagatell	6460.00	E2	W	Male	TE	200.00	1.29	1.02
		FSH	W	Male	TE	200.00	1.29	-3.49
		LH	W	Male	TE	200.00	1.29	-.74
		T	W	Male	TE	200.00	1.29	2.26
	6490.00	FSH	W	Male	TE	200.00	20.00	-3.42
		Hct	W	Male	TE	200.00	20.00	1.02
		LH	W	Male	TE	200.00	20.00	-4.72
		Sperm	W	Male	TE	200.00	20.00	-1.43
		T	W	Male	TE	200.00	20.00	2.00
		ASAT	W	Male	TE	200.00	20.00	.00
		Hb	W	Male	TE	200.00	20.00	.74
Baumstark	12590.00	Apo-AI	W	Male	Mix (T & AS)	2100.00	.	-.34
		HDL	W	Male	Mix (T & AS)	2100.00	.	-.34
		LDL	W	Male	Mix (T & AS)	2100.00	.	.00
Behre	380.00	FSH	W	Male	19NT-HPP	83.33	24.00	-.55
		LH	W	Male	19NT-HPP	83.33	24.00	-.55
		RBC	W	Male	19NT-HPP	83.33	24.00	.55
		Sperm	W	Male	19NT-HPP	83.33	24.00	-.78
		Testes	W	Male	19NT-HPP	83.33	24.00	-.55
		T	W	Male	19NT-HPP	83.33	24.00	-.55
Bhasin	12680.00	Bench	B	Male	TE	600.00	10.00	.41
		Bench	B	Male	TE	600.00	10.00	.43
		FSH	B	Male	TE	600.00	10.00	-2.85
		FSH	B	Male	TE	600.00	10.00	-1.78

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Bhasin	12680.00	HDL	B	Male	TE	600.00	10.00	.00
		HDL	B	Male	TE	600.00	10.00	.11
		Hct	B	Male	TE	600.00	10.00	.00
		Hct	B	Male	TE	600.00	10.00	.00
		LDL	B	Male	TE	600.00	10.00	-.09
		LDL	B	Male	TE	600.00	10.00	-.18
		LH	B	Male	TE	600.00	10.00	-2.20
		LH	B	Male	TE	600.00	10.00	-1.40
		RBC	B	Male	TE	600.00	10.00	.00
		RBC	B	Male	TE	600.00	10.00	.00
		SHBG	B	Male	TE	600.00	10.00	-.74
		SHBG	B	Male	TE	600.00	10.00	-.34
		Squat	B	Male	TE	600.00	10.00	.55
		Squat	B	Male	TE	600.00	10.00	.43
		T	B	Male	TE	600.00	10.00	2.45
		T	B	Male	TE	600.00	10.00	3.28
		TC	B	Male	TE	600.00	10.00	.00
		TC	B	Male	TE	600.00	10.00	.00
		TG	B	Male	TE	600.00	10.00	-.30
		TG	B	Male	TE	600.00	10.00	-.36
		BWt	B	Male	TE	600.00	10.00	.20
		BWt	B	Male	TE	600.00	10.00	.54
		Cr9	B	Male	TE	600.00	10.00	.00
		Cr9	B	Male	TE	600.00	10.00	.39
		Hb	B	Male	TE	600.00	10.00	.36
		Hb	B	Male	TE	600.00	10.00	.61
		LBM	B	Male	TE	600.00	10.00	.30
		LBM	B	Male	TE	600.00	10.00	.63
Bjorkqvist	7010.00	T	B	Male	TU	280.00	1.00	.39
Bowers	460.00	Bench	B	Male	Methandrostenolone	70.00	3.00	.95
		Squat	B	Male	Methandrostenolone	70.00	3.00	.95
		BWt	B	Male	Methandrostenolone	70.00	3.00	.95
		VO2	B	Male	Methandrostenolone	70.00	3.00	.00
		Bicepl	B	Male	Methandrostenolone	70.00	3.00	.95
Capell	9530.00	FSH	W	Male	TP	400.00	1.00	-.74
		LH	W	Male	TP	400.00	1.00	-1.36
Carlstrom	710.00	E2	W	Male	TE	250.00	1.00	.24
		LH	W	Male	TE	250.00	1.00	-.47
		T	W	Male	TE	250.00	1.00	.47
Casner	720.00	BWt	B	Male	Stanozolol	42.00	8.00	.20

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Clerico	880.00	FSH	W	Male	Methandrostenolone	200.00	2.00	-.74
		LH	W	Male	Methandrostenolone	200.00	2.00	-.51
		T	W	Male	Methandrostenolone	200.00	2.00	-1.12
Climstein	5430.00	HDL	B	Male	Mix (T & AS)	594.00	.	-.72
		LDL	B	Male	Mix (T & AS)	594.00	.	.11
		TC	B	Male	Mix (T & AS)	594.00	.	-.12
		Cr9	B	Male	Mix (T & AS)	594.00	.	-.14
Cohen	930.00	TC	W	Male	Mix (T & AS)	.	8.00	1.03
		TC	W	Male	Mix (T & AS)	.	12.00	2.46
Crist	1060.00	HDL	W	Male	TC	400.00	3.00	-.54
		TC	W	Male	TC	400.00	3.00	-.90
		TG	W	Male	TC	400.00	3.00	-.38
Cunningham	9930.00	E2	W	Male	TE	200.00	12.00	1.23
		FSH	W	Male	TE	200.00	12.00	-1.63
		LH	W	Male	TE	200.00	12.00	-1.72
		Sperm	W	Male	TE	200.00	12.00	-.69
		Testes	W	Male	TE	200.00	12.00	.00
		T	W	Male	TE	200.00	12.00	.67
		TC	W	Male	TE	200.00	12.00	.00
		TG	W	Male	TE	200.00	12.00	.00
		BWt	W	Male	TE	200.00	12.00	.67
		SV	W	Male	TE	200.00	12.00	-.49
		ALAT	W	Male	TE	200.00	12.00	.00
		ASAT	W	Male	TE	200.00	12.00	.49
		DBP	W	Male	TE	200.00	12.00	.00
		Hb	W	Male	TE	200.00	12.00	.49
		SBP	W	Male	TE	200.00	12.00	.00
		PC	W	Male	TE	200.00	12.00	.00
	10340.00	FSH	W	Male	TE	200.00	12.00	-1.80
		LH	W	Male	TE	200.00	12.00	-2.22
		Sperm	W	Male	TE	200.00	12.00	-1.57
		T	W	Male	TE	200.00	12.00	2.79
		SV	W	Male	TE	200.00	12.00	-.32
Dickinson	10000.00	Albumin	W	Male	TC	150.00	2.00	-.45
Dowben	11440.00	ASAT	W	Male	Norethandrolone	.	6.00	.85
		LDH	W	Male	Norethandrolone	210.00	6.00	2.07

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Era	1390.00	VO2	B	Male	Mix (T & AS)	.	26.00	.06
Fahey	1410.00	Bench	B	Male	Nandrolone	25.00	9.00	.14
		BF%	B	Male	Nandrolone	25.00	9.00	.08
		BWt	B	Male	Nandrolone	25.00	9.00	.14
		VO2	B	Male	Nandrolone	25.00	9.00	.61
		Bicep1	B	Male	Nandrolone	25.00	9.00	-.15
		LBM	B	Male	Nandrolone	25.00	9.00	.13
		Thigh1	B	Male	Nandrolone	25.00	9.00	.17
Finkelstein	6500.00	E2	W	Male	TE	105.00	.57	.69
		FSH	W	Male	TE	105.00	.57	-1.15
		LH	W	Male	TE	105.00	.57	-1.85
		T	W	Male	TE	105.00	.57	1.54
Forbes	10640.00	LBM	W	Male	TE	260.00	12.00	1.45
Fowler	1530.00	BWt	B	Male	Methenolone	140.00	16.00	-.04
		BWt	B	Male	Methenolone	140.00	16.00	.00
		BWt	B	Male	Methenolone	140.00	16.00	.02
		ALAT	B	Male	Methenolone	140.00	16.00	-.35
		ALAT	B	Male	Methenolone	140.00	16.00	.00
		ALAT	B	Male	Methenolone	140.00	16.00	.00
		ASAT	B	Male	Methenolone	140.00	16.00	.00
		ASAT	B	Male	Methenolone	140.00	16.00	.53
		ASAT	B	Male	Methenolone	140.00	16.00	.65
Friedl	1640.00	Apo-AI	W	Male	Nandrolone	100.00	6.00	.05
		Apo-AI	W	Male	Nandrolone	300.00	6.00	-.83
		Apo-AI	W	Male	TE	100.00	6.00	-.46
		Apo-AI	W	Male	TE	300.00	6.00	-.86
		BF%	W	Male	Nandrolone	100.00	6.00	-.02
		BF%	W	Male	Nandrolone	300.00	6.00	-.04
		BF%	W	Male	TE	100.00	6.00	.06
		BF%	W	Male	TE	300.00	6.00	.05
		E2	W	Male	Nandrolone	100.00	6.00	-2.01
		E2	W	Male	Nandrolone	300.00	6.00	-1.39
		E2	W	Male	TE	100.00	6.00	-.10
		E2	W	Male	TE	300.00	6.00	2.05
		FSH	W	Male	Nandrolone	100.00	6.00	-2.00
		FSH	W	Male	Nandrolone	300.00	6.00	-1.63
		FSH	W	Male	TE	100.00	6.00	-1.00
		FSH	W	Male	TE	300.00	6.00	-1.20
		LH	W	Male	Nandrolone	100.00	6.00	-1.56

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Friedl	1640.00	LH	W	Male	Nandrolone	300.00	6.00	-2.07
		LH	W	Male	TE	100.00	6.00	-2.00
		LH	W	Male	TE	300.00	6.00	-2.55
		SHBG	W	Male	Nandrolone	100.00	6.00	.12
		SHBG	W	Male	Nandrolone	300.00	6.00	-.53
		SHBG	W	Male	TE	100.00	6.00	-.46
		SHBG	W	Male	TE	300.00	6.00	-.42
		Sperm	W	Male	Nandrolone	100.00	6.00	-1.75
		Sperm	W	Male	TE	100.00	6.00	-4.22
		Sperm	W	Male	TE	300.00	6.00	-3.05
		Testes	W	Male	Nandrolone	100.00	6.00	.10
		Testes	W	Male	Nandrolone	300.00	6.00	-1.17
		Testes	W	Male	TE	100.00	6.00	-1.13
		Testes	W	Male	TE	300.00	6.00	-1.49
		T	W	Male	Nandrolone	100.00	6.00	-2.53
		T	W	Male	Nandrolone	300.00	6.00	-1.91
		T	W	Male	TE	100.00	6.00	.92
		T	W	Male	TE	300.00	6.00	3.34
		BWt	W	Male	Nandrolone	100.00	6.00	.42
		BWt	W	Male	Nandrolone	300.00	6.00	.42
		BWt	W	Male	TE	100.00	6.00	-.01
		BWt	W	Male	TE	300.00	6.00	.16
		SV	W	Male	Nandrolone	100.00	6.00	.00
		SV	W	Male	Nandrolone	300.00	6.00	.00
		SV	W	Male	TE	100.00	6.00	.00
		SV	W	Male	TE	300.00	6.00	.00
	1650.00	AP	W	Male	Methyltestosterone	280.00	12.00	.00
		AP	W	Male	TE	280.00	12.00	.00
		Apo-AI	W	Male	Methyltestosterone	280.00	12.00	-1.48
		Apo-AI	W	Male	TE	280.00	12.00	.00
		BF%	W	Male	Methyltestosterone	280.00	12.00	.00
		BF%	W	Male	TE	280.00	12.00	.00
		E2	W	Male	Methyltestosterone	280.00	12.00	.00
		E2	W	Male	TE	280.00	12.00	2.33
		HDL	W	Male	Methyltestosterone	280.00	8.00	-1.66
		HDL	W	Male	TE	280.00	8.00	-.11
		Hct	W	Male	Methyltestosterone	280.00	12.00	.00
		Hct	W	Male	TE	280.00	12.00	.00
		LDL	W	Male	Methyltestosterone	280.00	12.00	.83
		LDL	W	Male	TE	280.00	12.00	.27
		LH	W	Male	Methyltestosterone	280.00	12.00	.00
		LH	W	Male	TE	280.00	12.00	-.65
		SHBG	W	Male	Methyltestosterone	280.00	12.00	-.65
		SHBG	W	Male	TE	280.00	12.00	-.65

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Friedl	1650.00	T	W	Male	Methyltestosterone	280.00	12.00	2.30
		T	W	Male	TE	280.00	12.00	4.03
		TC	W	Male	Methyltestosterone	280.00	12.00	.00
		TC	W	Male	TE	280.00	12.00	.00
		TG	W	Male	Methyltestosterone	280.00	12.00	.00
		TG	W	Male	TE	280.00	12.00	.00
		Albumin	W	Male	Methyltestosterone	280.00	12.00	.00
		Albumin	W	Male	TE	280.00	12.00	.00
		ASAT	W	Male	Methyltestosterone	280.00	12.00	.00
		ASAT	W	Male	TE	280.00	12.00	.00
		Bil	W	Male	Methyltestosterone	280.00	12.00	.00
		Bil	W	Male	TE	280.00	12.00	.00
		DBP	W	Male	Methyltestosterone	280.00	12.00	.00
		DBP	W	Male	TE	280.00	12.00	.00
		Hb	W	Male	Methyltestosterone	280.00	12.00	.00
		Hb	W	Male	TE	280.00	12.00	.00
		LDH	W	Male	Methyltestosterone	280.00	12.00	.00
		LDH	W	Male	TE	280.00	12.00	.00
		SBP	W	Male	Methyltestosterone	280.00	12.00	.00
		SBP	W	Male	TE	280.00	12.00	.00
		PC	W	Male	Methyltestosterone	280.00	12.00	.00
		PC	W	Male	TE	280.00	12.00	.00
Glazer	7220.00	HDL	W	Male	Nandrolone	100.00	6.00	-.22
		HDL	W	Female	Nandrolone	100.00	6.00	-.64
		LDL	W	Male	Nandrolone	100.00	6.00	.24
		LDL	W	Female	Nandrolone	100.00	6.00	-.66
		TC	W	Male	Nandrolone	100.00	6.00	.09
		TC	W	Female	Nandrolone	100.00	6.00	-.83
		TG	W	Male	Nandrolone	100.00	6.00	-.05
		TG	W	Female	Nandrolone	100.00	6.00	.65
		BWt	W	3	Nandrolone	100.00	6.00	.13
		DBP	W	3	Nandrolone	100.00	6.00	.00
		SBP	W	3	Nandrolone	100.00	6.00	-.12
Golding	1810.00	Bench	B	Male	Methandrostenolone	70.00	12.00	.00
		Bench	B	Male	Methandrostenolone	70.00	12.00	.33
		BF%	W	Male	Methandrostenolone	70.00	12.00	-.20
		BF%	W	Male	Methandrostenolone	70.00	12.00	.00
		BWt	B	Male	Methandrostenolone	70.00	12.00	-.04
		BWt	B	Male	Methandrostenolone	70.00	12.00	.15
		Bicep1	W	Male	Methandrostenolone	70.00	12.00	.00
		Bicep1	B	Male	Methandrostenolone	70.00	12.00	.00
		Thigh1	W	Male	Methandrostenolone	70.00	12.00	.00
		Thigh1	B	Male	Methandrostenolone	70.00	12.00	.00

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Griggs	1880.00	FSH	W	Male	TE	220.50	12.00	-2.50
		Hct	W	Male	TE	220.50	12.00	1.09
		LH	W	Male	TE	220.50	12.00	-1.60
		T	W	Male	TE	220.50	12.00	1.44
		TC	W	Male	TE	220.50	12.00	.00
		TG	W	Male	TE	220.50	12.00	.00
		BWt	W	Male	TE	220.50	12.00	.52
		Cr9	W	Male	TE	220.50	12.00	.67
Hagerman	1910.00	AP	B	Male	Methandrostenolone	35.00	12.00	.20
		ALAT	B	Male	Methandrostenolone	35.00	12.00	.60
		ASAT	B	Male	Methandrostenolone	35.00	12.00	.20
		LDH	B	Male	Methandrostenolone	35.00	12.00	.18
Handelsman	10500.00	AP	W	Male	TE	63.00	12.00	.00
		AP	W	Male	TE	200.00	12.00	.00
		E2	W	Male	TE	63.00	12.00	.56
		E2	W	Male	TE	200.00	12.00	1.16
		FSH	W	Male	TE	63.00	12.00	-2.27
		FSH	W	Male	TE	200.00	12.00	-1.74
		HDL	W	Male	TE	63.00	12.00	-.30
		HDL	W	Male	TE	200.00	12.00	-.16
		LDL	W	Male	TE	63.00	12.00	-.28
		LDL	W	Male	TE	200.00	12.00	-.49
		LH	W	Male	TE	63.00	12.00	-2.31
		LH	W	Male	TE	200.00	12.00	-3.38
		SHBG	W	Male	TE	63.00	12.00	-.17
		SHBG	W	Male	TE	200.00	12.00	-.77
		Sperm	W	Male	TE	63.00	12.00	-1.93
		Sperm	W	Male	TE	200.00	12.00	-1.75
		T	W	Male	TE	63.00	12.00	2.08
		T	W	Male	TE	200.00	12.00	2.76
		TC	W	Male	TE	63.00	12.00	-.58
		TC	W	Male	TE	200.00	12.00	-.65
		TG	W	Male	TE	63.00	12.00	-.74
		TG	W	Male	TE	200.00	12.00	.32
		ALAT	W	Male	TE	63.00	12.00	.00
		ALAT	W	Male	TE	200.00	12.00	.00
		Albumin	W	Male	TE	63.00	12.00	-.41
		Albumin	W	Male	TE	200.00	12.00	-.81
		ASAT	W	Male	TE	63.00	12.00	.00
		ASAT	W	Male	TE	200.00	12.00	.00
		Bil	W	Male	TE	63.00	12.00	.00
		Bil	W	Male	TE	200.00	12.00	.00

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Handelsman	10500.00	Cr9	W	Male	TE	63.00	12.00	.00
		Cr9	W	Male	TE	200.00	12.00	.00
		GGT	W	Male	TE	63.00	12.00	.00
		GGT	W	Male	TE	200.00	12.00	.00
		Hb	W	Male	TE	63.00	12.00	.70
		Hb	W	Male	TE	200.00	12.00	1.79
		PC	W	Male	TE	63.00	12.00	.00
		PC	W	Male	TE	200.00	12.00	.00
Hartgens	11620.00	Hct	B	Male	Mix (T & AS)	.	8.00	.00
		RBC	B	Male	Mix (T & AS)	.	8.00	-.15
		Hb	B	Male	Mix (T & AS)	.	8.00	.47
		PC	B	Male	Mix (T & AS)	.	8.00	.82
	12410.00	Bicep1	B	Male	Mix (T & AS)	.	8.00	.24
		Thigh1	B	Male	Mix (T & AS)	.	8.00	.80
Heller	7410.00	Sperm	W	Male	TP	200.00	16.00	-1.74
	9750.00	Sperm	W	Male	Norethandrolone	210.00	8.00	-.84
Hervey	2060.00	AP	B	Male	Methandrostenolone	700.00	6.00	.00
		FSH	B	Male	Methandrostenolone	700.00	6.00	-.42
		LH	B	Male	Methandrostenolone	700.00	6.00	-.42
		T	B	Male	Methandrostenolone	700.00	6.00	1.02
		BWt	B	Male	Methandrostenolone	700.00	6.00	1.17
		VO2	B	Male	Methandrostenolone	700.00	6.00	.00
		ALAT	B	Male	Methandrostenolone	700.00	6.00	.00
		ASAT	B	Male	Methandrostenolone	700.00	6.00	.00
		Bil	B	Male	Methandrostenolone	700.00	6.00	.00
		Bicep1	B	Male	Methandrostenolone	700.00	6.00	.86
		DBP	B	Male	Methandrostenolone	700.00	6.00	.00
		LBM	B	Male	Methandrostenolone	700.00	6.00	1.17
		SBP	B	Male	Methandrostenolone	700.00	6.00	.00
		Thigh1	B	Male	Methandrostenolone	700.00	6.00	.86
	2070.00	Bench	B	Male	Methandrostenolone	700.00	6.00	1.09
		Squat	B	Male	Methandrostenolone	700.00	6.00	1.53
		BWt	B	Male	Methandrostenolone	700.00	6.00	1.53
		Bicep1	B	Male	Methandrostenolone	700.00	6.00	1.09
		LBM	B	Male	Methandrostenolone	700.00	6.00	2.16
		Thigh1	B	Male	Methandrostenolone	700.00	6.00	1.53
Hobbs	5590.00	BF%	W	Male	Nandrolone	300.00	6.00	.13
		BF%	W	Male	TE	300.00	6.00	-.19

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Hobbs	5590.00	E2	W	Male	Nandrolone	300.00	6.00	-.58
		E2	W	Male	TE	300.00	6.00	2.79
		SHBG	W	Male	Nandrolone	300.00	6.00	-.74
		SHBG	W	Male	TE	300.00	6.00	-.72
		T	W	Male	Nandrolone	300.00	6.00	-.93
		T	W	Male	TE	300.00	6.00	1.98
	12530.00	BWt	W	Male	Nandrolone	300.00	6.00	.51
		BWt	W	Male	TE	300.00	6.00	.29
Holma	2140.00	BWt	W	Male	Methandrostenolone	105.00	8.00	.23
	2150.00	Sperm	W	Male	Methandrostenolone	105.00	8.00	-.88
	2160.00	FSH	W	Male	Methandrostenolone	105.00	8.00	-.89
		LH	W	Male	Methandrostenolone	105.00	8.00	-2.11
		T	W	Male	Methandrostenolone	105.00	8.00	-1.96
Hurley	2200.00	BF%	W	Male	Mix (T & AS)	878.52	4.50	.00
		HDL	W	Male	Mix (T & AS)	878.52	4.50	-2.36
		LDL	W	Male	Mix (T & AS)	878.52	4.50	1.13
		T	W	Male	Mix (AS)	173.02	4.50	-1.01
		T	W	Male	Mix (T & AS)	878.52	4.50	1.83
		TC	W	Male	Mix (T & AS)	878.52	4.50	.73
		TG	W	Male	Mix (T & AS)	878.52	4.50	.65
		BWt	W	Male	Mix (T & AS)	878.52	4.50	.22
Johnsen	9970.00	AP	W	Male	Testosterone	2800.00	3.00	.00
		T	W	Male	Testosterone	2800.00	3.00	1.37
		ALAT	W	Male	Testosterone	2800.00	3.00	-.48
		Albumin	W	Male	Testosterone	2800.00	3.00	-.76
		Bil	W	Male	Testosterone	2800.00	3.00	-.05
Johnson	2320.00	Bench	B	Male	Methandrostenolone	70.00	3.00	1.11
		Sperm	B	Male	Methandrostenolone	70.00	3.00	.00
		Squat	B	Male	Methandrostenolone	70.00	3.00	1.11
		BWt	B	Male	Methandrostenolone	70.00	3.00	.82
		VO2	B	Male	Methandrostenolone	70.00	3.00	.00
		Bicepl	B	Male	Methandrostenolone	70.00	3.00	.34
		Thighl	B	Male	Methandrostenolone	70.00	3.00	.34
	2330.00	BWt	B	Male	Stanozolol	42.00	3.00	.00
		VO2	B	Male	Stanozolol	42.00	3.00	-.49
	2340.00	AP	W	Male	Methandrostenolone	70.00	3.00	-.35

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Johnson	2340.00	Bench	B	Male	Methandrostenolone	70.00	3.00	1.11
		Squat	B	Male	Methandrostenolone	70.00	3.00	1.11
		TC	W	Male	Methandrostenolone	70.00	3.00	.18
		BWt	B	Male	Methandrostenolone	70.00	3.00	1.11
		VO2	B	Male	Methandrostenolone	70.00	3.00	1.11
		ASAT	W	Male	Methandrostenolone	70.00	3.00	.18
		Bil	W	Male	Methandrostenolone	70.00	3.00	-.43
		Bicepl	B	Male	Methandrostenolone	70.00	3.00	1.11
		LDH	W	Male	Methandrostenolone	70.00	3.00	.18
		Thighl	B	Male	Methandrostenolone	70.00	3.00	.34
Jones	6980.00	E2	W	Male	Fluoxymesterone	70.00	12.00	.00
		E2	W	Male	Fluoxymesterone	140.00	12.00	.00
		FSH	W	Male	Fluoxymesterone	70.00	12.00	-.27
		FSH	W	Male	Fluoxymesterone	140.00	12.00	-.42
		LH	W	Male	Fluoxymesterone	70.00	12.00	.00
		LH	W	Male	Fluoxymesterone	140.00	12.00	.42
		SHBG	W	Male	Fluoxymesterone	70.00	12.00	-.27
		SHBG	W	Male	Fluoxymesterone	140.00	12.00	-.42
		Sperm	W	Male	Fluoxymesterone	70.00	12.00	-.39
		Sperm	W	Male	Fluoxymesterone	140.00	12.00	-.59
		T	W	Male	Fluoxymesterone	140.00	12.00	-.21
Kicovic	6930.00	E2	W	Male	TU	840.00	.86	.31
		FSH	W	Male	TU	840.00	.86	-.54
		LH	W	Male	TU	840.00	.86	-.16
		T	W	Male	TU	840.00	.86	.54
Kiraly	2490.00	HDL	W	Male	Mix (T & AS)	350.00	12.00	-1.45
		Hct	W	Male	Mix (T & AS)	350.00	12.00	.24
		RBC	W	Male	Mix (T & AS)	350.00	12.00	.60
		ALAT	W	Male	Mix (T & AS)	350.00	12.00	.60
		ASAT	W	Male	Mix (T & AS)	350.00	12.00	.60
		GGT	W	Male	Mix (T & AS)	350.00	12.00	-.24
		Hb	W	Male	Mix (T & AS)	350.00	12.00	.60
	2500.00	Testes	W	Male	Mix (T & AS)	350.00	12.00	-1.32
Kirschner	10260.00	SV	W	Male	Fluoxymesterone	280.00	.71	-1.22
		SV	W	Male	Fluoxymesterone	280.00	.71	-.75
	10470.00	T	W	Male	Fluoxymesterone	70.00	.43	-1.16
Kleiner	2550.00	Apo-AI	B	Male	Mix (T & AS)	594.00	5.90	-1.73
		HDL	B	Male	Mix (T & AS)	594.00	5.90	-2.77

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Kleiner	2550.00	LDL	B	Male	Mix (T & AS)	594.00	5.90	.67
		TC	B	Male	Mix (T & AS)	594.00	5.90	.18
		TG	B	Male	Mix (T & AS)	594.00	5.90	-.05
		DBP	B	Male	Mix (T & AS)	594.00	5.90	.48
		SBP	B	Male	Mix (T & AS)	594.00	5.90	.17
Knuth	10060.00	AP	W	Male	19NT-HPP	200.00	13.00	.00
		FSH	W	Male	19NT-HPP	200.00	13.00	-.65
		HDL	W	Male	19NT-HPP	200.00	13.00	.00
		Hct	W	Male	19NT-HPP	200.00	13.00	.65
		LDL	W	Male	19NT-HPP	200.00	13.00	.00
		LH	W	Male	19NT-HPP	200.00	13.00	-.65
		RBC	W	Male	19NT-HPP	200.00	13.00	.65
		Sperm	W	Male	19NT-HPP	200.00	13.00	-.92
		Testes	W	Male	19NT-HPP	200.00	13.00	-.65
		T	W	Male	19NT-HPP	200.00	13.00	-.65
		TC	W	Male	19NT-HPP	200.00	13.00	.00
		TG	W	Male	19NT-HPP	200.00	13.00	.00
		BWt	W	Male	19NT-HPP	200.00	13.00	.00
		SV	W	Male	19NT-HPP	200.00	13.00	.00
		ALAT	W	Male	19NT-HPP	200.00	13.00	.00
		ASAT	W	Male	19NT-HPP	200.00	13.00	.00
		Bil	W	Male	19NT-HPP	200.00	13.00	.00
		Cr9	W	Male	19NT-HPP	200.00	13.00	-.65
		GGT	W	Male	19NT-HPP	200.00	13.00	.00
		Hb	W	Male	19NT-HPP	200.00	13.00	.65
		PC	W	Male	19NT-HPP	200.00	13.00	.00
Kuhn	7530.00	E2	B	Male	DHT	1750.00	1.50	-.86
		FSH	B	Male	DHT	1750.00	1.50	.00
		LH	B	Male	DHT	1750.00	1.50	-.86
		SHBG	W	Male	DHT	1750.00	1.50	-.38
		T	B	Male	DHT	1750.00	1.50	-1.59
Kuipers	2600.00	AP	W	Male	Mix (T & AS)	207.10	9.00	-.36
		AP	B	Male	Nandrolone	112.50	8.00	.08
		AP	W	Male	Nandrolone	112.50	8.00	-.34
		HDL	W	Male	Mix (T & AS)	207.10	9.00	-.94
		HDL	B	Male	Nandrolone	112.50	8.00	-.06
		HDL	W	Male	Nandrolone	207.10	10.00	-1.01
		TC	W	Male	Mix (T & AS)	207.10	9.00	.40
		TC	B	Male	Nandrolone	112.50	8.00	.17
		TC	W	Male	Nandrolone	112.50	8.00	.11
		TG	W	Male	Mix (T & AS)	207.10	9.00	.38
		TG	B	Male	Nandrolone	112.50	8.00	.38

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Kuipers	2600.00	TG	W	Male	Nandrolone	112.50	8.00	.48
		BWt	W	Male	Mix (T & AS)	207.10	9.00	.90
		BWt	B	Male	Nandrolone	112.50	8.00	1.10
		BWt	W	Male	Nandrolone	112.50	8.00	.59
		ALAT	W	Male	Mix (T & AS)	207.10	9.00	.06
		ALAT	B	Male	Nandrolone	112.50	8.00	.51
		ALAT	W	Male	Nandrolone	112.50	8.00	1.09
		Bicepl	W	Male	Mix (T & AS)	207.10	9.00	.90
		Bicepl	B	Male	Nandrolone	112.50	8.00	.45
		Bicepl	W	Male	Nandrolone	112.50	8.00	.00
		DBP	W	Male	Mix (T & AS)	207.10	9.00	.89
		DBP	B	Male	Nandrolone	112.50	8.00	.29
		DBP	W	Male	Nandrolone	112.50	8.00	-.48
		GGT	W	Male	Mix (T & AS)	207.10	9.00	-.32
		GGT	B	Male	Nandrolone	112.50	8.00	.95
		GGT	W	Male	Nandrolone	112.50	8.00	.31
		LBM	W	Male	Mix (T & AS)	207.10	9.00	.90
		LBM	B	Male	Nandrolone	112.50	8.00	1.10
		LBM	W	Male	Nandrolone	112.50	8.00	.24
		SBP	W	Male	Mix (T & AS)	207.10	9.00	.87
		SBP	B	Male	Nandrolone	112.50	8.00	.58
		SBP	W	Male	Nandrolone	112.50	8.00	.07
Lee	7490.00	FSH	W	Male	Testosterone	7.00	.43	-.27
		FSH	W	Male	Testosterone	35.00	.43	-.71
		FSH	W	Male	Testosterone	87.50	.43	-1.18
		FSH	W	Male	Testosterone	175.00	.43	-1.18
		LH	W	Male	Testosterone	7.00	.43	-.27
		LH	W	Male	Testosterone	35.00	.43	-.71
		LH	W	Male	Testosterone	87.50	.43	-1.18
		LH	W	Male	Testosterone	175.00	.43	-1.18
		T	W	Male	Testosterone	7.00	.43	.27
		T	W	Male	Testosterone	35.00	.43	.71
		T	W	Male	Testosterone	87.50	.43	1.18
		T	W	Male	Testosterone	175.00	.43	1.18
Lenders	2700.00	HDL	W	Male	Mix (T & AS)	594.00	8.90	-2.06
		LDL	W	Male	Mix (T & AS)	594.00	8.90	.73
		TC	W	Male	Mix (T & AS)	594.00	8.90	.12
		TG	W	Male	Mix (T & AS)	594.00	8.90	.02
		BWt	W	Male	Mix (T & AS)	594.00	8.90	.07
		ALAT	W	Male	Mix (T & AS)	594.00	8.90	.58
		ASAT	W	Male	Mix (T & AS)	594.00	8.90	.83
		DBP	W	Male	Mix (T & AS)	594.00	8.90	.45
		GGT	W	Male	Mix (T & AS)	594.00	8.90	.14

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Lenders	2700.00	SBP	W	Male	Mix (T & AS)	594.00	8.90	.33
Loughton	2830.00	Bench	B	Male	Methandrostenolone	70.00	6.00	.10
		Bench	B	Male	Methandrostenolone	70.00	6.00	.23
		Squat	B	Male	Methandrostenolone	70.00	6.00	-.17
		Squat	B	Male	Methandrostenolone	70.00	6.00	-.35
		BWt	B	Male	Methandrostenolone	70.00	6.00	.17
		BWt	B	Male	Methandrostenolone	70.00	6.00	.76
		VO2	B	Male	Methandrostenolone	70.00	6.00	1.45
		VO2	B	Male	Methandrostenolone	70.00	6.00	-.83
		Thigh1	B	Male	Methandrostenolone	70.00	6.00	.09
		Thigh1	B	Male	Methandrostenolone	70.00	6.00	.53
Lowe	7390.00	Hct	W	Male	Mesterlone	700.00	3.00	-.12
		PC	W	Male	Mesterlone	700.00	3.00	.34
Marynick	10220.00	E2	W	Male	TE	105.00	.57	1.51
		FSH	W	Male	TE	105.00	.57	-.65
		LH	W	Male	TE	105.00	.57	-1.76
		T	W	Male	TE	105.00	.57	5.78
Matsumoto	5620.00	AP	B	Male	TE	25.00	24.00	.00
		AP	B	Male	TE	50.00	24.00	.00
		AP	B	Male	TE	100.00	24.00	.00
		AP	B	Male	TE	300.00	24.00	.00
		FSH	B	Male	TE	25.00	24.00	-.36
		FSH	B	Male	TE	50.00	24.00	-.88
		FSH	B	Male	TE	100.00	24.00	-.99
		FSH	B	Male	TE	300.00	24.00	-.99
		Hct	W	Male	TE	25.00	24.00	.00
		Hct	W	Male	TE	50.00	24.00	.00
		Hct	W	Male	TE	100.00	24.00	.56
		Hct	W	Male	TE	300.00	24.00	1.09
		LH	B	Male	TE	25.00	24.00	-.36
		LH	B	Male	TE	50.00	24.00	-.88
		LH	B	Male	TE	100.00	24.00	-.99
		LH	B	Male	TE	300.00	24.00	-.99
		Sperm	B	Male	TE	25.00	24.00	-.36
		Sperm	B	Male	TE	50.00	24.00	-.88
		Sperm	B	Male	TE	100.00	24.00	-.99
		Sperm	B	Male	TE	300.00	24.00	-.99
		T	B	Male	TE	25.00	24.00	.00
		T	B	Male	TE	50.00	24.00	.37
		T	B	Male	TE	100.00	24.00	.86
		T	B	Male	TE	300.00	24.00	1.17

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Matsumoto	5620.00	BWt	B	Male	TE	25.00	24.00	.00
		BWt	B	Male	TE	50.00	24.00	.00
		BWt	B	Male	TE	100.00	24.00	.86
		BWt	B	Male	TE	300.00	24.00	.86
		ASAT	B	Male	TE	25.00	24.00	.00
		ASAT	B	Male	TE	50.00	24.00	.00
		ASAT	B	Male	TE	100.00	24.00	.00
		ASAT	B	Male	TE	300.00	24.00	.00
		Bil	B	Male	TE	25.00	24.00	.00
		Bil	B	Male	TE	50.00	24.00	.00
		Bil	B	Male	TE	100.00	24.00	.00
		Bil	B	Male	TE	300.00	24.00	.00
		LDH	B	Male	TE	25.00	24.00	.00
		LDH	B	Male	TE	50.00	24.00	.00
		LDH	B	Male	TE	100.00	24.00	.00
		LDH	B	Male	TE	300.00	24.00	.00
	9550.00	SV	B	Male	TE	166.67	21.00	-.07
Maul	2990.00	Bench	W	Male	Methandrostenolone	35.00	12.00	.45
		BF%	B	Male	Methandrostenolone	35.00	12.00	.16
		Squat	B	Male	Methandrostenolone	35.00	12.00	.39
		BWt	B	Male	Methandrostenolone	35.00	12.00	.09
		VO2	B	Male	Methandrostenolone	35.00	12.00	.10
Mauss	5880.00	AP	W	Male	TE	250.00	21.00	.00
		FSH	W	Male	TE	250.00	21.00	-.90
		LH	W	Male	TE	250.00	21.00	-.90
		RBC	W	Male	TE	250.00	21.00	.00
		T	W	Male	TE	250.00	21.00	.24
		BWt	W	Male	TE	250.00	21.00	.24
		ALAT	W	Male	TE	250.00	21.00	.00
		ASAT	W	Male	TE	250.00	21.00	.00
		DBP	W	Male	TE	250.00	21.00	.00
		Hb	W	Male	TE	250.00	21.00	.00
		SBP	W	Male	TE	250.00	21.00	.00
	9660.00	Sperm	W	Male	TE	250.00	21.00	-.80
		Testes	W	Male	TE	250.00	21.00	-.84
		SV	W	Male	TE	250.00	21.00	.00
McKillop	9460.00	Albumin	W	Male	Mix (AS)	2175.00	12.00	-.54
		ASAT	W	Male	Mix (AS)	2175.00	12.00	1.64
		Cr9	W	Male	Mix (AS)	2175.00	12.00	.27

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Mies	10860.00	FSH	W	Male	TU	1120.00	2.00	-.11
		LH	W	Male	TU	1120.00	2.00	.09
		T	W	Male	TU	1120.00	2.00	.33
Millar	7840.00	AP	W	Male	Methenolone	140.00	7.00	.05
		HDL	W	Male	Methenolone	140.00	7.00	-.58
		TC	W	Male	Methenolone	140.00	7.00	.18
		TG	W	Male	Methenolone	140.00	7.00	.11
		ALAT	W	Male	Methenolone	140.00	7.00	.40
		ASAT	W	Male	Methenolone	140.00	7.00	.45
		Bil	W	Male	Methenolone	140.00	7.00	.04
		LDH	W	Male	Methenolone	140.00	7.00	-.07
Muller	3210.00	HDL	W	Male	Mix (T & AS)	594.00	.	-2.40
		LDL	W	Male	Mix (T & AS)	594.00	.	.56
Munson	3220.00	BF%	B	Male	Oxandrolone	140.00	4.00	-.70
		BWt	B	Male	Oxandrolone	140.00	4.00	.16
		VO2	B	Male	Oxandrolone	140.00	4.00	.29
		Bicep1	B	Male	Oxandrolone	140.00	4.00	-.14
		Thigh1	B	Male	Oxandrolone	140.00	4.00	.17
Nieschlag	9790.00	E2	W	Male	TU	1680.00	12.00	.00
		FSH	W	Male	TU	1680.00	12.00	-.24
		LH	W	Male	TU	1680.00	12.00	-.60
		Sperm	W	Male	TU	1680.00	12.00	-.33
		Testes	W	Male	TU	1680.00	12.00	.00
		T	W	Male	TU	1680.00	12.00	-.60
		BWt	W	Male	TU	1680.00	12.00	.00
		SV	W	Male	TU	1680.00	12.00	.00
		DBP	W	Male	TU	1680.00	12.00	.00
		SBP	W	Male	TU	1680.00	12.00	.00
	10960.00	T	W	Male	Testosterone	441.00	.14	.00
		T	W	Female	Testosterone	441.00	.14	.54
O'Shea	3390.00	AP	B	Male	Stanozolol	56.00	5.00	-.39
		Bench	B	Male	Stanozolol	56.00	5.00	.24
		Squat	B	Male	Stanozolol	56.00	5.00	.21
		BWt	B	Male	Stanozolol	56.00	5.00	.12
		ASAT	B	Male	Stanozolol	56.00	5.00	-.39
		Bil	B	Male	Stanozolol	56.00	5.00	.00
	3410.00	Bench	B	Male	Methandrostenolone	70.00	4.00	.47

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
O'Shea	3410.00	Squat	B	Male	Methandrostenolone	70.00	4.00	.29
		BWt	B	Male	Methandrostenolone	70.00	4.00	.41
	3420.00	AP	W	Male	Oxandrolone	70.00	6.00	-.22
		Squat	W	Male	Oxandrolone	70.00	3.00	4.41
		TC	W	Male	Oxandrolone	70.00	6.00	-.22
		BWt	W	Male	Oxandrolone	70.00	6.00	.39
		Albumin	W	Male	Oxandrolone	70.00	6.00	-.22
		ASAT	W	Male	Oxandrolone	70.00	6.00	.22
		Bil	W	Male	Oxandrolone	70.00	6.00	-.22
		LDH	W	Male	Oxandrolone	70.00	6.00	-.81
Palacios	3460.00	Testes	W	Male	TE	200.00	16.00	-1.03
	3470.00	Hct	W	Male	TE	200.00	16.00	.35
		Hct	W	Male	TE	200.00	16.00	.77
		RBC	W	Male	TE	200.00	16.00	.18
		RBC	W	Male	TE	200.00	16.00	.88
		Hb	W	Male	TE	100.00	16.00	.29
		Hb	W	Male	TE	200.00	16.00	.68
Palonek	12300.00	E2	W	Male	TE	200.00	36.00	2.26
		LH	W	Male	TE	200.00	36.00	-.57
		SHBG	W	Male	TE	200.00	36.00	-.76
		T	W	Male	TE	200.00	36.00	1.45
Peterson	3090.00	FSH	W	Male	TE	75.00	1.00	-.42
		LH	W	Male	TE	75.00	1.00	-1.23
	3660.00	HDL	W	Male	Mix (T & AS)	594.00	6.00	-1.14
		TC	W	Male	Mix (T & AS)	594.00	6.00	.38
Preston	7380.00	Albumin	W	3	Stanozolol	70.00	6.00	.00
		Hb	W	3	Stanozolol	70.00	6.00	.00
		PC	W	3	Stanozolol	70.00	6.00	.00
Reddy	3800.00	Sperm	W	Male	TP	175.00	6.43	-4.10
		SV	W	Male	TP	175.00	8.57	.94
Remes	3840.00	FSH	W	Male	Methandrostenolone	35.00	4.00	.10
		FSH	W	Male	Methandrostenolone	70.00	4.00	.06
		Hct	W	Male	Methandrostenolone	35.00	4.00	-.27
		Hct	W	Male	Methandrostenolone	70.00	4.00	-.32
		LH	W	Male	Methandrostenolone	35.00	4.00	-.82
		LH	W	Male	Methandrostenolone	70.00	4.00	.20

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Remes	3840.00	RBC	W	Male	Methandrostenolone	35.00	4.00	-.47
		RBC	W	Male	Methandrostenolone	70.00	4.00	-.60
		T	W	Male	Methandrostenolone	35.00	4.00	-1.75
		T	W	Male	Methandrostenolone	70.00	4.00	-3.85
		Hb	W	Male	Methandrostenolone	35.00	4.00	-.53
		Hb	W	Male	Methandrostenolone	70.00	4.00	-.23
Ruokonen	4010.00	T	W	Male	Mix (T & AS)	.	26.00	.74
Sachtleben	5660.00	BF%	W	Male	Mix (T & AS)	.	.	-.08
		BWt	W	Male	Mix (T & AS)	.	.	.28
		VO2	W	Male	Mix (T & AS)	.	.	.00
		LBM	W	Male	Mix (T & AS)	.	.	.39
Santen	6520.00	E2	W	Male	Testosterone	105.00	.14	1.51
		E2	W	Male	DHT	105.00	.14	-.76
		LH	W	Male	Testosterone	105.00	.14	-.61
		LH	W	Male	DHT	105.00	.14	-1.18
		T	W	Male	Testosterone	105.00	.14	1.48
		T	W	Male	DHT	105.00	.14	-.33
Schaison	10520.00	E2	W	Male	DHT	87.50	12.00	-.62
		LH	W	Male	DHT	87.50	12.00	.33
		SHBG	W	Male	DHT	87.50	12.00	-1.24
		T	W	Male	DHT	87.50	12.00	-.42
Schulte-B.	10280.00	FSH	W	Male	TE	194.00	1.00	-.65
		FSH	W	Male	TC	200.00	1.00	-.65
		LH	W	Male	TE	194.00	1.00	-.65
		LH	W	Male	TC	200.00	1.00	-.65
		T	W	Male	TE	194.00	1.00	.65
		T	W	Male	TC	200.00	1.00	.65
Schurmeyer	7850.00	AP	W	Male	19NT-HPP	200.00	11.00	.33
		FSH	W	Male	19NT-HPP	200.00	13.00	-1.18
		Hct	W	Male	19NT-HPP	200.00	11.00	.16
		LH	W	Male	19NT-HPP	200.00	13.00	-1.18
		RBC	W	Male	19NT-HPP	200.00	11.00	.16
		Sperm	W	Male	19NT-HPP	200.00	13.00	-1.67
		Testes	W	Male	19NT-HPP	200.00	13.00	-1.95
		T	W	Male	19NT-HPP	200.00	13.00	-1.18
		TC	W	Male	19NT-HPP	200.00	11.00	.29
		BWt	W	Male	19NT-HPP	200.00	13.00	1.42
		SV	W	Male	19NT-HPP	200.00	13.00	.00
		ALAT	W	Male	19NT-HPP	200.00	11.00	1.19

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Schurmeyer	7850.00	ASAT	W	Male	19NT-HPP	200.00	11.00	.78
		Bil	W	Male	19NT-HPP	200.00	11.00	.00
		Cr9	W	Male	19NT-HPP	200.00	11.00	.98
		GGT	W	Male	19NT-HPP	200.00	11.00	-.86
		Hb	W	Male	19NT-HPP	200.00	11.00	.18
		LDH	W	Male	19NT-HPP	200.00	11.00	1.27
	11120.00	FSH	W	Male	TE	200.00	1.00	-1.18
		FSH	W	Male	TeCHB	200.00	1.00	-1.18
		LH	W	Male	TE	200.00	1.00	-1.18
		LH	W	Male	TeCHB	200.00	1.00	-1.18
		T	W	Male	TE	200.00	1.00	.27
		T	W	Male	TeCHB	200.00	1.00	.00
Sherins	6530.00	FSH	W	Male	Testosterone	115.00	1.00	-1.98
		LH	W	Male	Testosterone	115.00	1.00	-1.72
Small	7290.00	AP	W	Male	Stanozolol	25.00	2.00	.03
		HDL	W	Male	Stanozolol	12.50	4.00	-.69
		Hct	W	Male	Stanozolol	25.00	2.00	-.25
		LDL	W	Male	Stanozolol	12.50	4.00	.49
		TC	W	Male	Stanozolol	12.50	4.00	.36
		TG	W	Male	Stanozolol	12.50	4.00	-.22
		ALAT	W	Male	Stanozolol	25.00	2.00	-.28
		Albumin	W	Male	Stanozolol	25.00	2.00	-.73
		ASAT	W	Male	Stanozolol	25.00	2.00	-.20
		Bil	W	Male	Stanozolol	25.00	2.00	-.52
		GGT	W	Male	Stanozolol	25.00	2.00	-.29
	9740.00	FSH	W	Male	Stanozolol	70.00	2.00	-.94
		LH	W	Male	Stanozolol	70.00	2.00	-1.02
		SHBG	W	Male	Stanozolol	70.00	2.00	-.87
		T	W	Male	Stanozolol	70.00	2.00	-1.85
Sokol	10180.00	E2	W	Male	TE	200.00	1.00	.00
		E2	W	Male	TE	200.00	1.00	.24
		FSH	W	Male	TE	200.00	1.00	-.60
		FSH	W	Male	TE	200.00	1.00	-.60
		LH	W	Male	TE	200.00	1.00	-.60
		LH	W	Male	TE	200.00	1.00	-.60
		T	W	Male	TE	200.00	1.00	.00
		T	W	Male	TE	200.00	1.00	.78
Stamford	4440.00	Bench	B	Male	Methandrostenolone	100.00	4.00	1.19
		BWt	B	Male	Methandrostenolone	100.00	4.00	.24

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Steinberger	7360.00	FSH	W	Male	TE	200.00	8.00	-.37
		LH	W	Male	TE	200.00	8.00	-.37
	7420.00	Sperm	W	Male	TE	300.00	4.00	-1.08
		T	W	Male	TE	300.00	4.00	1.28
Stewart	7470.00	FSH	W	Male	Testosterone	49.00	1.00	-.39
		FSH	W	Male	Testosterone	245.00	1.00	-1.19
		LH	W	Male	Testosterone	49.00	1.00	-1.32
		LH	W	Male	Testosterone	245.00	1.00	-1.22
		T	W	Male	Testosterone	49.00	1.00	.68
		T	W	Male	Testosterone	245.00	1.00	2.66
Stromme	4550.00	BWt	B	Male	Mesterlone	788.00	8.00	.36
		VO2	B	Male	Mesterlone	788.00	8.00	-.88
		Bicep1	B	Male	Mesterlone	788.00	8.00	.00
		Thigh1	B	Male	Mesterlone	788.00	8.00	1.20
Swerdloff	7350.00	E2	W	Male	TE	100.00	16.00	-.24
		E2	W	Male	TE	200.00	16.00	1.28
		FSH	W	Male	TE	100.00	16.00	-.78
		FSH	W	Male	TE	200.00	16.00	-1.19
		LH	W	Male	TE	100.00	16.00	-.96
		LH	W	Male	TE	200.00	16.00	-1.24
		Sperm	W	Male	TE	100.00	16.00	-1.19
		Sperm	W	Male	TE	200.00	16.00	-2.10
		T	W	Male	TE	100.00	16.00	.00
		T	W	Male	TE	200.00	16.00	1.88
	9680.00	FSH	W	Male	Fluoxymesterone	140.00	.57	-.25
		FSH	W	Male	Fluoxymesterone	140.00	.57	.00
		LH	W	Male	Fluoxymesterone	140.00	.57	-.74
		LH	W	Male	Fluoxymesterone	140.00	.57	-.71
Tahmindjas	4600.00	ALAT	W	Male	Mix (AS)	.	5.00	.00
		ASAT	W	Male	Mix (AS)	.	5.00	.00
		Bil	W	Male	Mix (AS)	.	5.00	.00
		Hb	W	Male	Mix (AS)	.	5.00	.00
Thompson	4690.00	AP	W	Male	Stanozolol	42.00	6.00	.00
		AP	W	Male	TE	200.00	6.00	.00
		Apo-AI	W	Male	Stanozolol	42.00	6.00	-2.28
		Apo-AI	W	Male	TE	200.00	6.00	-.35
		FSH	W	Male	Stanozolol	42.00	6.00	-.66

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Thompson	4690.00	FSH	W	Male	TE	200.00	6.00	-2.06
		HDL	W	Male	Stanozolol	42.00	6.00	-1.88
		HDL	W	Male	TE	200.00	6.00	-.47
		LDL	W	Male	Stanozolol	42.00	6.00	.52
		LDL	W	Male	TE	200.00	6.00	-.43
		LH	W	Male	Stanozolol	42.00	6.00	-.12
		LH	W	Male	TE	200.00	6.00	-.90
		T	W	Male	Stanozolol	42.00	6.00	-1.61
		T	W	Male	TE	200.00	6.00	1.15
		TC	W	Male	Stanozolol	42.00	6.00	.15
		TC	W	Male	TE	200.00	6.00	-.38
		TG	W	Male	Stanozolol	42.00	6.00	-.29
		TG	W	Male	TE	200.00	6.00	.22
		ALAT	W	Male	Stanozolol	42.00	6.00	.00
		ALAT	W	Male	TE	200.00	6.00	.00
		ASAT	W	Male	Stanozolol	42.00	6.00	.00
		ASAT	W	Male	TE	200.00	6.00	.00
		Bil	W	Male	Stanozolol	42.00	6.00	.00
		Bil	W	Male	TE	200.00	6.00	.00
		LDH	W	Male	Stanozolol	42.00	6.00	.00
		LDH	W	Male	TE	200.00	6.00	.00
Veldhuis	6540.00	E2	W	Male	DHT	49.00	.64	.00
		LH	W	Male	DHT	49.00	.64	-1.46
		T	W	Male	DHT	49.00	.64	.00
Vigersky	6970.00	LH	W	Male	Fluoxymesterone	280.00	.43	-1.01
		T	W	Male	Fluoxymesterone	280.00	.43	-2.52
Wallace	6700.00	E2	W	Male	TE	200.00	24.00	1.44
		T	W	Male	TE	200.00	24.00	2.57
Wang	10290.00	T	W	Male	TE	200.00	1.00	2.03
Ward	4920.00	Bench	B	Male	Methandrostenolone	70.00	4.00	.49
		BF%	B	Male	Methandrostenolone	70.00	4.00	-.26
		Squat	B	Male	Methandrostenolone	70.00	4.00	.32
		BWt	B	Male	Methandrostenolone	70.00	4.00	.02
		LBM	B	Male	Methandrostenolone	70.00	4.00	.18
Webb	4950.00	HDL	W	Male	Mix (T & AS)	825.00	8.00	-2.63
		LDL	W	Male	Mix (T & AS)	825.00	8.00	.57
		TC	W	Male	Mix (T & AS)	825.00	8.00	-.02
		TG	W	Male	Mix (T & AS)	825.00	8.00	.37

Author	Study#	Outcome	Design	Gender	Drug	Dose	Cycle	d
Welle	10880.00	T	W	Male	TE	219.00	12.00	2.20
Win-May	5130.00	BF%	B	Male	Methandrostenolone	35.00	12.00	-.73
		BWt	B	Male	Methandrostenolone	35.00	12.00	.73
		Bicep1	B	Male	Methandrostenolone	35.00	12.00	.73
		LBM	B	Male	Methandrostenolone	35.00	12.00	.73
		Thigh1	B	Male	Methandrostenolone	35.00	12.00	.73
Young	5640.00	AP	W	Male	TE	200.00	24.00	.26
		Testes	W	Male	TE	200.00	24.00	-1.24
		T	W	Male	TE	200.00	24.00	3.71
		Albumin	W	Male	TE	200.00	24.00	-.60
		LBM	B	Male	TE	200.00	24.00	.83
Zmuda	5650.00	AP	W	Male	TE	200.00	3.00	.00
		Apo-AI	W	Male	TE	200.00	3.00	-.67
		E2	W	Male	TE	200.00	3.00	.82
		FSH	W	Male	TE	200.00	3.00	-1.13
		HDL	W	Male	TE	200.00	3.00	-.13
		LDL	W	Male	TE	200.00	3.00	.07
		LH	W	Male	TE	200.00	3.00	-1.08
		T	W	Male	TE	200.00	3.00	1.30
		TC	W	Male	TE	200.00	3.00	-.10
		TG	W	Male	TE	200.00	3.00	.34
		ALAT	W	Male	TE	200.00	3.00	.00
		ASAT	W	Male	TE	200.00	3.00	.00
		Bil	W	Male	TE	200.00	3.00	.00
		DBP	W	Male	TE	200.00	3.00	.00
		LDH	W	Male	TE	200.00	3.00	.00
		SBP	W	Male	TE	200.00	3.00	.00
Zuliani	5320.00	Apo-AI	W	Male	Mix (T & AS)	594.00	6.00	-1.60
		HDL	W	Male	Mix (T & AS)	594.00	6.00	-.98
		TC	W	Male	Mix (T & AS)	594.00	6.00	-.48
		TG	W	Male	Mix (T & AS)	594.00	6.00	-.23