Published in final edited form as:
*Biol Psychiatry*. 2012 February 1; 71(3): 254–261. doi:10.1016/j.biopsych.2011.06.024.

# Risk Factors for Illicit Anabolic-Androgenic Steroid Use in Male Weightlifters: A Cross-Sectional Cohort Study

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## Abstract

**Background—**Illicit anabolic-androgenic steroid (AAS) abuse, though an important public health problem, remains inadequately studied. Almost all AAS abusers are male and lift weights, but the risk factors for AAS use *among* male weightlifters remain poorly understood.

**Methods—**We recruited 233 experienced male weightlifters, of whom 102 (44%) reported lifetime AAS use, and assessed their childhood and adolescent attributes retrospectively using structured clinical interviews and computerized questionnaires. This "*cross-sectional cohort*" approach—a design that we have formally presented in the recent methodological literature—utilizes a study cohort, not selected for outcomes of interest, and assesses exposures and outcomes retrospectively. We hypothesized that *conduct disorder* and *body-image concerns* would be major risk factors for subsequent AAS use among male weightlifters.

**Results—**Within our study population, many attributes showed little association with AAS use, but conduct disorder and body-image concerns showed strong associations. For individuals with prior conduct disorder vs. those without, the hazard ratio [95% confidence interval] for subsequent AAS use was 2.2 [1.5, 3.4]. For individuals in the middle vs. lowest tertile of scores on a retrospective adolescent "muscle-dysmorphia" scale, the hazard ratio was 1.5 [0.84, 2.6]; for the highest vs. lowest tertile, the hazard ratio was 3.3 [2.0, 5.3]; and for the linear trend of hazard ratios, $P < 0.001$.

**Conclusions—**Conduct disorder and body-image concerns represent important risk factors for AAS use among male weightlifters. Thus, assessment of these attributes may help to identify individuals most likely to require interventions to discourage this form of substance abuse.

### Keywords

Anabolic-androgenic steroids; substance abuse; risk factors; body-image disorders; conduct disorder; men



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**Disclosures:**
Dr. Pope has served as an expert witness in legal cases involving anabolic-androgenic steroids. Dr. Pope reports no other biomedical financial interests or potential conflicts of interest. Drs. Kanayama and Hudson report no biomedical financial interests or potential conflicts of interest.

The anabolic-androgenic steroids (AAS) are a family of drugs that includes the male hormone, testosterone, together with its synthetic derivatives (1). Taken in supraphysiologic doses, AAS permit users to greatly increase their muscle mass, often well beyond natural limits (2). Elite athletes have used AAS since the 1950s (3), but it was not until the 1980s that illicit AAS use began to spread out of elite athletics and into the general population (4). Now, millions of individuals worldwide have used AAS (5–10)—and most are using these drugs not for athletic competition, but simply to become leaner and more muscular (11, 12). As many as 30% of these AAS users develop a dependence syndrome (13), potentially leading to adverse cardiovascular (14, 15), neuroendocrine (16), psychiatric (17), and other effects (4). However, despite growing evidence of AAS-induced morbidity and even mortality, these drugs remain among the least-studied of major drugs of abuse, and risk factors for AAS use are incompletely understood. In a recent study addressing this issue (18), we tentatively found that 1) childhood and adolescent conduct disorder and 2) adolescent concerns about muscularity and body image appeared to predict AAS use among male weightlifters. Given these preliminary findings, we initiated a larger study of 250 male weightlifters, using a “cross-sectional cohort design”—a type of cohort study design that we have formally presented in the recent methodological literature (19).

## Methods

### Participants

Since most illicit AAS users are male and lift weights (1, 20, 21), we chose this known high-risk population for study (see further discussion of this rationale below). Using methods developed in prior studies (18), we advertised in gymnasiums for men age 18–40 who could “bench-press 275 pounds for at least one repetition, now or in the past, for a psychological and medical evaluation.” As previously explained (18, 22), the bench-press requirement was simply a device to generate an unselected group of experienced weightlifters. To minimize selection bias, men were recruited *without inquiring about their AAS history or disclosing the study's focus on AAS*. Participants were recruited at three geographic sites in the United States—Boston, Massachusetts; Palm Beach, Florida; and Los Angeles, California—to minimize possible idiosyncrasies peculiar to one region. All participants were interviewed by the same two investigators (HGP and GK).

### Study Procedures

Upon arriving for evaluation, participants were first required to sign informed consent for the study, which was approved by the McLean Hospital Institutional Review Board. The consent form described all study procedures, but did not disclose the study's specific focus on AAS, in order to minimize response bias that might occur if participants knew their case status.

We then administered verbal interviews covering 1) demographic indices; 2) history of weightlifting and other athletic activity; 3) history of psychiatric disorders in first-degree relatives, using methods previously described (23); 4) retrospective history of childhood conduct disorder and attention-deficit hyperactivity disorder (ADHD), using modules from the *Structured Clinical Interview for DSM-IV-Child Edition (KID-SCID)* (24); 5) lifetime history of DSM-IV Axis I disorders, using the *Structured Clinical Interview for DSM-IV* (*SCID*) (25); and 6) adolescent concerns about muscularity, using a "muscle dysmorphia" scale, previously developed at our center (26, 27) and available from the authors, adapted from the *Body Dysmorphic Disorder Modification of the Yale-Brown Obsessive Compulsive Scale (BDD-YBOCS*) (28).

Participants also completed a computerized battery of questionnaires covering various childhood and adolescent attributes previously shown to be associated with the development of adult substance abuse (18, 29–32). Instruments tapping pre-adolescent childhood and family history included 1) a Childhood and Family History Questionnaire, adapted from Finkelhor's Life Events Questionnaire (33); 2) the Parental Bonding Instrument (34); and 3) the 25-item Wender Utah Rating Scale (WURS) for childhood ADHD (35). Questionnaires tapping adolescent attributes (specified as age 13–16, "until your 17$^{th}$ birthday") included 1) the Impulsive Sensation-Seeking Scale (36, 37); 2) the Adolescent Risk-Taking Questionnaire (38); and 3) the Eating Disorders Inventory (EDI) (39), rephrased as a retrospective instrument with the "Body Dissatisfaction" items modified to focus on muscularity rather than obesity, as described in our pilot study (18) and studies of others (40, 41). For example, the item "I think my thighs are too big" was changed to "I felt that my legs were too small." We also administered 4) an Adolescent Experience Questionnaire of our own design, derived from the "gym questionnaire" used in our pilot study (18) and available from the authors, containing 23 Likert-type scales covering seven adolescent attributes: Physical Dominance, Physical Attractiveness, Athletic Status, Social Status, Vulnerability to Peer Pressure, Peer Drug Use, and Drug Availability.

We also assessed participants' height, weight, body fat, and fat-free mass index (an index of muscularity (2)), using methods previously described (18). Participants' urine samples were sent to Anti-Doping Research (Los Angeles, CA) to be tested for AAS; for opioids, cannabis, cocaine, amphetamines, and phencyclidine; and for the performance-enhancing drug clenbuterol. (Other performance-enhancing drugs were not assessed either because they were difficult to distinguish from the individual's endogenous hormones [e.g. human growth hormone, IGF-I, insulin] or too infrequently used to make testing cost-effective [e.g., human chorionic gonadotropin, triiodothyronine]). Samples of participants' head or axillary hair were tested at Psychemedics Corporation (Culver City, CA) for residues of opioids, cannabis, cocaine, amphetamines, and phencyclidine from the last 90 days. Note that hair could not be tested for AAS in this commercial laboratory, although such testing is currently under development (42–44).

All questions about participants' history of substance use, including AAS use, were placed at the end of the evaluation. Thus the investigators remained blinded to AAS history while eliciting all other information, minimizing potential observer bias that might arise from knowing a participant's case status in advance. However, blindness was necessarily imperfect, since some men were so muscular that they were visibly AAS users (12).

## Statistical Analyses

**Methods to minimize cases of false reporting—**We considered that some participants might not disclose their use of AAS or other drugs—a problem previously discussed (12, 18, 22). To minimize this source of bias, we excluded from analysis participants showing AAS or other drugs in their urine or hair inconsistent with their verbal reports. We also excluded men who denied AAS use, but showed body fat < 10% and fat-free mass index > 25.5 kg/m$^2$—since this combination of leanness and muscularity is rarely attained without AAS (2, 12). These criteria excluded 15 (6%) of the 250 men (6 with AAS in urine despite denial of use, 5 with other drugs in urine or hair despite denial, and 4 with implausibly high muscularity and low fat despite denial). Two other men were excluded for incomplete (N = 1) or internally inconsistent interviews (N = 1), leaving 233 evaluable cases.

**Cross-sectional cohort design—**To test the association between adolescent attributes and later AAS use, we used what we have termed a "cross-sectional cohort design." This

type of cohort study design has been used implicitly in previous major epidemiological studies of psychiatric disorders (45, 46), but to our knowledge, has not been described explicitly. In a recent paper (written in anticipation of the present study), we have formally presented the properties of this design, including the conditions required for its validity (19). Briefly, the cross-sectional cohort design samples a source population cross-sectionally, and then measures the association between exposures and outcomes assessed retrospectively. Unlike the more common case-control design, this method can 1) study multiple outcomes without separate sets of control participants for each outcome; 2) yield population-based measures of effect, such as risk ratios and hazard ratios; and 3) handle the effects of time more simply—all without entailing any threats to validity not already present in an equivalent case-control study.

Importantly, the cross-sectional cohort design is relatively resistant to selection bias, because the estimated hazard ratios will remain valid as long as the relative probability of selection into the study cohort for those with vs. those without the outcome remains constant across different levels of the exposure variables. Thus, the design is resistant to bias arising from either 1) factors influencing entry into the study cohort among individuals currently available from the underlying source population; or 2) factors rendering individuals currently unavailable for sampling due to early exiting from the source population. To illustrate the first of these considerations for the present study, suppose hypothetically that weightlifters who develop the outcome (AAS use) spend more time in gymnasiums than weightlifters lacking the outcome (nonusers)—so that the former group is more likely to see and respond to a study advertisement. Now suppose further that men with a given exposure (say, prior conduct disorder) are less likely to respond to an advertisement than those without that exposure. Even under these conditions, the study will still yield an unbiased estimate of the hazard ratio for development of AAS use among individuals with prior conduct disorder vs. those without—barring the implausible case that these two influences are not simply additive, but interact with each other, i.e., that a history of conduct disorder *selectively* decreases advertisement responding among AAS users, while not comparably decreasing advertisement responding among nonusers. Similar reasoning applies to early exiting from the source population: hazard ratios will remain unbiased, save for the unlikely case that a given exposure *selectively* accelerates exiting in one outcome group while not comparably accelerating exiting in the other. Our methodological paper (19) presents mathematical justification of these considerations.

**Statistical methods—**We used proportional hazards models, adjusted for study site (Massachusetts, Florida, or California), self-defined race/ethnicity (modeled as non-Hispanic white vs. all others), and birth cohort (1965–1973, 1974–1979, 1980–1983, and 1984–1990) to calculate hazard ratios for the associations between items in our battery of childhood and adolescent measures and risk of AAS use. Although AAS use was associated with duration of 8 weightlifting (see below), this association became non-significant after adjustment for age ($p = 0.31$), and age is already accounted for in the proportional hazards models. Thus, we did not adjust for duration of weightlifting. We performed univariate analyses for each individual measure and subsequently conducted bivariate analyses involving both conduct-disorder and body-image measures. For continuous exposure variables, we divided levels of exposure into tertiles, ranked in order of increasing pathology (e.g., impulsivity was ranked from low to high, whereas adolescent social status was ranked from high to low), and calculated hazard ratios for each of the two upper tertiles relative to the lowest (reference) tertile. We also calculated a *p* value for the linear trend of the hazard ratios by using the medians of the tertiles as a continuous variable. Throughout these analyses, no violations of the proportional hazards assumption were detected by inspection of log-log plots or by tests of weighted residuals (47). All initial analyses were univariate analyses involving individual measures from our assessment battery

All analyses were performed using Stata 9.2 (Stata Corporation, College Station, Texas), with alpha set at 0.05, 2-tailed. Note that the study generated multiple comparisons, increasing the likelihood of type I errors. However, Bonferroni corrections for these comparisons are too conservative and inflate type II error rates (48). Thus we present the results without correction, while noting that some differences, especially those of marginal statistical significance, might represent chance associations.

## Results

### Participants

We recruited 250 men at the three study sites, of whom 17 were excluded from analysis as described above, leaving 233 evaluable participants (Table 1). Of these, 102 (44%) reported lifetime AAS use (42 in Florida, 39 in Massachusetts, and 21 in California); their age at first AAS use ranged from 15 to 37 years, with a mean (SD) of 22.8 (5.1) years. Only six (6%) users reported onset of use prior to age 17. Users reported a mean of 110 (174) lifetime weeks of AAS use (range 1–900 weeks) at a mean weekly dose of 1117 (955) milligrams of testosterone equivalent (range 35–5000 mg), where weekly dose was estimated in the same manner as in our previous studies (18, 22, 49, 50).

Briefly comparing current attributes of the groups, AAS users were older than nonusers (29.9 [6.1] versus 27.8 [5.8] years; $p = 0.007$ by t-test, 2-tailed), had lifted weights for longer (10.1 [5.8] versus 8.4 [5.0] years; $p = 0.017$), and were more muscular, as reflected by higher mean fat-free mass index (24.2 [2.8] versus 22.8 [1.9] $mg/kg^2$; $p < 0.001$). Users were more frequently non-Hispanic white (88 [87%] versus 89 [68%]; $p = 0.001$ by Fisher's exact test, two-tailed), and less likely to have graduated a two- or four-year college (32 [31%] versus 70 [53%]; $p < 0.001$). Both groups showed high rates of other illicit drug use, as shown by hair and urine analyses. Among 70 AAS users with sufficient hair for analysis, 37 (53%) showed no drug residues, while 18 (26%) showed cannabis, 24 (34%) cocaine, 3 (4%) opioids, and 2 (3%) amphetamines (total N is > 70 because some individuals showed > 1 drug). Among 87 evaluable nonusers, 55 (63%) were negative, while 26 (30%) showed cannabis, 16 (18%) cocaine, and one (1%) amphetamines. Looking at urine samples from the 102 AAS users, 50 (49%) were negative, 34 (33%) showed AAS, 2 (2%) clenbuterol, 15 (15%) cannabis, 8 (8%) cocaine, and one (1%) opioids. Of 128 nonusers with adequate urine samples, none showed AAS (by definition; see above), one (1%) showed clenbuterol, 29 (23%) cannabis, 7 (5%) cocaine, and one (1%) opioids.

Further demographic information on these men, including their use of other drugs and features associated with AAS dependence, is provided in prior papers describing portions of the group analyzed here (22, 51). Note, however, that these earlier papers reported merely *attributes* of various subgroups using simple comparative statistics, whereas the present paper addresses *risk factors* for overall AAS use, assessed by inferential methods using hazard ratios generated through time-to-onset data and the cross-sectional cohort design.

### Primary Analysis

Many of the various childhood attributes assessed, including family history of substance use, childhood ADHD, and childhood sexual abuse, were not significantly associated with increased risk for AAS use (see selected findings in Table 2 and detailed findings in Table S1 in the Supplement). However, men reporting negative childhood relationships with their fathers—both on the Childhood and Family Questionnaire and on the “Father Care” subscale of the Parental Bonding Instrument—showed a markedly increased risk for later AAS use. As hypothesized, conduct disorder was also strongly associated with risk for subsequent AAS use, as shown by a statistically significant 2.2-fold increase in hazard (see

Kaplan-Meier curves in Figure S1 in the Supplement). This finding was buttressed by participants' self-ratings on the Adolescent Risk-Taking Questionnaire, where Rebellious risk behaviors were associated with significantly increased risk for AAS use (Table 3).

Measures tapping adolescent body image and physical attractiveness also strongly predicted AAS use (Table 3 and Table S2 in the Supplement). These included several subscales of the modified EDI as well as EDI total score. On the Adolescent Experience Questionnaire, *lower* self-reported adolescent Physical Attractiveness and Athleticism predicted a *higher* risk of later AAS use, but the other five subscales showed no significant association. Scores on the "muscle dysmorphia" version of the BDD-YBOCS again strongly predicted AAS use, with participants in the upper tertile of scores (e.g., those with the greatest adolescent preoccupations and behaviors focused on a muscular body appearance) showing a 3.3-fold increased hazard for using these drugs (Figure S2 in the Supplement).

Notably, conduct disorder and body image disorder appeared to represent independent risk factors for AAS use, in that conduct disorder as assessed by the KID-SCID predicted AAS use even after adjustment for tertiles of BDD-YBOCS score (hazard ratio [95% confidence interval] 1.8 [1.1–2.7]; $p = 0.012$), and body image disorder predicted AAS use even after adjustment for conduct disorder (hazard ratio 1.7 [1.3–2.1] for each tertile of increase in BDD-YBOCS score; $p < 0.001$).

We also considered that six of the 102 AAS users initiated use prior to age 17—raising the possibility that adolescent attributes reported by these men might have been partially caused by AAS use, rather than representing pre-existing features. However, upon repeating the analyses involving adolescent attributes with these individuals deleted, all hazard ratios remained within 13% of their original values, and levels of statistical significance remained essentially unchanged. The only exception was the comparison of the highest versus lowest tertile of the Impulsive Sensation-Seeking Scale, where the hazard ratio rose from 1.4 [0.85–2.4] to 1.7 [1.01–3.0] with the six early-onset users deleted.

## Discussion

We explored risk factors for anabolic-androgenic steroid (AAS) use among 233 community-recruited male weightlifters, age 18–40, from Massachusetts, Florida, and California. The study used a "cross-sectional cohort" design (19), wherein the study population was recruited without selecting for the outcome variable (AAS use), and where the outcome and exposure variables were assessed retrospectively. As discussed above, this design entails no threats to validity not already present in an equivalent case-control design, and generates population-based measures of effect, such as hazard ratios, that are relatively resistant to selection bias. Our measured exposure variables, assessed via personal interviews and computerized questionnaires, included a range of childhood and adolescent characteristics spanning familial, individual, and community domains.

We chose to examine risk factors for AAS use only among male weightlifters, since the great majority of illicit AAS users are male and lift weights (1, 20, 21). Indeed no investigators, to our knowledge, have identified any large population of illicit AAS users who were not already male weightlifters before becoming AAS users. Thus, our study effectively assessed the second of two stages: once males have initiated weightlifting, why do some progress to AAS use and others not? Given this choice of study design, it should be recognized that we could not detect factors that might be predictive of AAS use simply by dint of the fact that they were risk factors for weightlifting in general.

Consistent with our hypotheses, two clusters of attributes emerged as strong and independent risk factors for subsequent AAS use: 1) conduct disorder and 2) adolescent

concern with body appearance and muscularity. These findings are particularly striking when it is considered that they were observed within a population of male weightlifters, since body-image concerns (and perhaps conduct disorder) are likely also risk factors for weightlifting in the first place. Thus the study may substantially underestimate the *total* contribution of these risk factors to AAS use. Interestingly, we also found that a poor childhood relationship with one's father was associated with AAS use among weightlifters, replicating our pilot observations (18). A further incidental finding was that all weightlifters, including even AAS non-users, frequently used classical illicit drugs such as cannabis and cocaine. Notably, participants were male, age 18–40, mostly unmarried, and often of lower educational or socioeconomic status—attributes all associated with greater illicit substance use (52)—but even with these considerations, rates of substance use were still high compared to national data (e.g., (52, 53)).

Several possible limitations of the study should be discussed. First, by restricting to male weightlifters, did the study miss a substantial population of illicit AAS users? Some anonymous school surveys have implied that many girls use AAS, but as we have detailed previously (21), these results appear largely due to false-positive survey responses, and such female users are likely rare. Recent population studies from Sweden (54) have reiterated that AAS use is very rare among women, including even among women who attend gymnasiums (55). Thus the restriction to males appears reasonable.

Another possibility is that the study missed certain male illicit AAS users, such as individuals who used AAS briefly for high school athletics, then ceased athletics and weightlifting after high school, and hence never encountered our advertisements. Such cases would represent early exiting from the underlying source population—but as noted earlier, hazard ratios generated in a cross-sectional cohort study are quite resistant to selection bias caused by early exiting. Further, we are not aware of studies that have actually exhibited or evaluated large numbers of such brief adolescent AAS users (again after correcting for potential false-positive responses in anonymous high-school surveys (1, 21)). Also, even if many such brief adolescent AAS users did exist, they would likely pose a much smaller public health threat than those continuing AAS use after age 18.

A second limitation is that our adolescent body-image measures (the "muscle dysmorphia" modifications of the EDI and BDD-YBOCS, as well as the Adolescent Experience Questionnaire) lacked formal psychometric documentation—although one can take some reassurance that such documentation exists for the original versions of the EDI (39) and BDD-YBOCS (28). Also, the convergent findings of these three instruments, together with the large effect sizes, suggest that the observed association of adolescent body-image concern and later AAS use is unlikely to be an artifact of psychometric deficiencies in these scales.

Third, one must consider risks from information bias. Specifically, *response bias* might have occurred if AAS users reported childhood or adolescent attributes differentially compared to nonusers. In retrospective studies, one cause of such differential recall is knowledge of one's case status (56). In the present study, however, this source of bias appears unlikely, since men were not informed of the outcome being studied (AAS use) and hence did not know their case status. Response bias might also have occurred if AAS users were more or less prone than nonusers to recall adverse childhood experiences. This source of bias also appears unlikely, however, since users and nonusers showed few differences on a wide range of retrospective measures other than conduct disorder and body image concerns (see Tables S1 and S2 in the Supplement)—arguing against systematic over- or under-reporting of adverse experiences by one of the groups.

Bias might have arisen if participants *failed to disclose* use of AAS or other drugs. However, we instituted several measures to identify such deception, resulting the exclusion of 17 (6.7%) of the participants. Furthermore, even if these measures were not fully successful, and the study included some non-disclosing AAS users misclassified as nonusers, such misclassification would likely only narrow the differences between groups on the various measures, leading to more conservative findings.

Finally, *observer bias* might have arisen if we knew in advance the group status of a participant being evaluated. However, we attempted to minimize this bias by structuring the evaluation so that questions about substance use were placed at the end, with questions about AAS last, so that we remained blinded to AAS history while eliciting all other information. Nevertheless, perfect blindness could not be achieved in practice, since some men were so muscular that they were visibly AAS users. Even in these cases, however, we were still blinded to the age of onset of the participant's AAS use until the end of the interview.

Fourth, it should be noted that our cross-sectional design limits inferences about causality. By way of illustration, consider the observed association between AAS use and a poor childhood relationship with one's father. This finding raises the intriguing possibility that experiences with one's father might perhaps contribute to AAS use by scripting attitudes towards male roles (57) and body image (58) in childhood. However, the present study does not permit conclusions about the temporal sequence of such events; for example, childhood attributes of future AAS users might influence their relationships with their fathers, rather than vice versa.

Fifth, it should be noted that our analysis explores risk factors for AAS use as a whole, but does not differentiate between short-term "casual" AAS users and individuals with potentially more malignant AAS dependence. Since AAS-dependent men likely account for the majority of public health problems arising from AAS use, a logical follow-up to the present study would be to investigate risk factors for the progression from initial AAS use to later AAS dependence.

In conclusion, our findings support the study hypotheses that adolescent conduct disorder and adolescent concern about muscularity and body image represent major risk factors for illicit AAS use among male weightlifters. These findings may help professionals to identify boys and young men at highest risk for AAS use, allowing interventions to be targeted more narrowly at this population, leading to more effective prevention of this widespread, but still understudied form of substance abuse.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## Acknowledgments

Supported in part by grants DA016744 and DA029141 from the National Institute on Drug Abuse.

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## Table 1

Demographic Attributes of Study Participants

| Characteristic | N | % |
|---|---|---|
| **Site where recruited** | | |
| Florida | 95 | 41 |
| Massachusetts | 71 | 30 |
| California | 67 | 29 |
| **Age, y** | | |
| 18–23 | 51 | 22 |
| 24–27 | 64 | 27 |
| 28–33 | 59 | 25 |
| 34–40 | 59 | 25 |
| **Years of regular weightlifting**[a] | | |
| < 5 | 53 | 23 |
| 5–10 | 100 | 43 |
| > 10 | 80 | 34 |
| **Ethnicity/race**[b] | | |
| Non-Hispanic: | | |
| White | 177 | 76 |
| African-American | 30 | 13 |
| Asian/Pacific Islander | 6 | 3 |
| Hispanic: | | |
| White | 14 | 6 |
| African-American | 6 | 3 |

[a] Defined as cumulative lifetime years of lifting weights at least 3 days per week in a commercial gymnasium.

[b] Based on participant's self-definition

**Table 2**

Selected Childhood and Family Attributes of Study Participants

| | | AAS Users | | Hazard Ratio for AAS Use[g] | | | |
|---|---|---|---|---|---|---|---|
| **Attribute[a]** | **N[f]** | **N** | **%** | **Estimate** | **95% CI** | ***P* Value** | ***P* For Trend[h]** |
| **Single parent[b]** | | | | | | | |
| No | 153 | 66 | 43 | 1.0 | | | |
| Yes | 74 | 31 | 42 | 0.91 | 0.59–1.4 | 0.66 | |
| **Family history of substance abuse[c]** | | | | | | | |
| No | 154 | 58 | 38 | 1.0 | | | |
| Yes | 63 | 34 | 54 | 1.4 | 0.92–2.2 | 0.11 | |
| **Childhood sexual abuse[d]** | | | | | | | |
| No | 206 | 89 | 43 | 1.0 | | | |
| Yes | 22 | 10 | 45 | 0.90 | 0.46–1.8 | 0.77 | |
| **Childhood ADHD[e]** | | | | | | | |
| No | 193 | 79 | 41 | 1.0 | | | |
| Yes | 40 | 23 | 58 | 1.5 | 0.94–2.4 | 0.092 | |
| **Conduct disorder[e]** | | | | | | | |
| No | 168 | 61 | 36 | 1.0 | | | |
| Yes | 65 | 41 | 63 | 2.2 | 1.5–3.4 | < 0.001 | |
| **Childhood and Family Questionnaire:** | | | | | | | |
| **Relationship with father** | | | | | | | |
| "Excellent" | 59 | 17 | 29 | 1.0 | | | |
| "Good" | 78 | 30 | 38 | 1.5 | 0.78–2.7 | 0.24 | |
| "Fair," "Poor," or "Terrible" | 81 | 46 | 57 | 2.4 | 1.4–4.3 | 0.003 | 0.001 |
| **Relationship with mother** | | | | | | | |
| "Excellent" | 114 | 51 | 45 | 1.0 | | | |
| "Good" | 68 | 27 | 40 | 0.83 | 0.52–1.3 | 0.45 | |
| "Fair," "Poor," or "Terrible" | 39 | 19 | 49 | 1.2 | 0.67–2.0 | 0.60 | 0.68 |
| **Parental Bonding Instrument:** | | | | | | | |

| | | AAS Users | | Hazard Ratio for AAS Use[g] | | | |
|---|---|---|---|---|---|---|---|
| **Attribute[a]** | **N[f]** | **N** | **%** | **Estimate** | **95% CI** | ***P* Value** | ***P* For Trend[h]** |
| **Father Care** | | | | | | | |
| High (score 25 – 36) | 82 | 24 | 29 | 1.0 | | | |
| Medium (score 18 – 24) | 70 | 32 | 46 | 1.4 | 0.83–2.5 | 0.20 | |
| Low (score 0 – 17) | 66 | 37 | 56 | 1.9 | 1.1–3.2 | 0.025 | 0.023 |
| **Mother Care** | | | | | | | |
| High (score 32 – 36) | 78 | 29 | 37 | 1.0 | | | |
| Medium (score 24 – 31) | 75 | 33 | 44 | 1.1 | 0.64–1.8 | 0.80 | |
| Low (score 0 – 23) | 68 | 35 | 51 | 1.4 | 0.85–2.3 | 0.19 | 0.21 |

Abbreviations: AAS, anabolic-androgenic steroids; CI, confidence interval; ADHD, attention deficit hyperactivity disorder.

[a] This table is limited to attributes showing statistically significant associations with AAS use ($p < 0.05$), together with selected major negative findings. For a complete table presenting a full list of attributes assessed, please see Table 2 in supplemental materials.

[b] Lived with only one parent for at least some time prior to age 13 as a result of parental separation, divorce, or death.

[c] At least one first-degree relative judged to meet DSM-IV criteria for alcohol or illicit drug abuse or dependence, based on participant's report.

[d] Based on responses to the Childhood and Family Questionnaire (see text).

[e] Based on the Structured Clinical Interview for DSM-IV-Child Edition (see text).

[f] Total N is less than 233 for some items due to missing data (e.g., participants who lacked information about their biological parents).

[g] By proportional hazards model, with adjustment for race/ethnicity, study site, and birth cohort (see text).

[h] Calculated for the linear trend of the hazard ratios using the medians of the tertiles as a continuous variable.

**Table 3**

Selected Adolescent Attributes of Study Participants

| | | AAS Users | | Hazard Ratio for AAS Use[c] | | | |
|---|---|---|---|---|---|---|---|
| **Attribute[a]** | **N[b]** | **N** | **(%)** | **Estimate** | **95% CI** | **P Value** | ***P* For Trend[d]** |
| **Impulsive Sensation Seeking Scale** | | | | | | | |
| Low (score 0 – 9) | 72 | 27 | 38 | 1.0 | | | |
| Medium (score 10 – 14) | 72 | 30 | 42 | 1.0 | 0.62 – 1.8 | 0.86 | |
| High (score 15 – 19) | 85 | 42 | 49 | 1.4 | 0.85 – 2.4 | 0.18 | 0.18 |
| **Adolescent Risk-Taking Questionnaire:** | | | | | | | |
| **Thrill-Seeking Behaviors** | | | | | | | |
| Low (score 0 – 1.00) | 80 | 37 | 46 | 1.0 | | | |
| Medium (score 1.14 – 1.43) | 69 | 30 | 43 | 0.92 | 0.56 – 1.5 | 0.76 | |
| High (score 1.57 – 3.14) | 80 | 32 | 40 | 0.74 | 0.45 – 1.2 | 0.23 | 0.24 |
| **Rebellious Behaviors** | | | | | | | |
| Low (score 0 – 1.0) | 70 | 25 | 36 | 1.0 | | | |
| Medium (score 1.2 – 2.2) | 81 | 31 | 38 | 0.81 | 0.47 – 1.4 | 0.45 | |
| High (score 2.4 – 4.0) | 78 | 43 | 55 | 1.3 | 0.77 – 2.3 | 0.32 | 0.03 |
| **Reckless Behaviors** | | | | | | | |
| Low (score 0 – 0.6) | 79 | 25 | 32 | 1.0 | | | |
| Medium (score 0.8 – 1.4) | 74 | 30 | 41 | 1.3 | 0.74 – 2.2 | 0.38 | |
| High (score 1.6 – 3.2) | 76 | 44 | 58 | 1.8 | 1.1 – 3.1 | 0.025 | 0.078 |
| **Antisocial Behaviors** | | | | | | | |
| Low (score 0 – 0.8) | 70 | 26 | 37 | 1.0 | | | |
| Medium (score 1.0 – 1.4) | 93 | 37 | 40 | 1.1 | 0.67 – 1.9 | 0.65 | |
| High (score 1.6 – 3.4) | 66 | 36 | 55 | 1.5 | 0.89 – 2.6 | 0.12 | 0.094 |
| **Eating Disorders Inventory (modified for boys):** | | | | | | | |
| **Drive for Thinness** | | | | | | | |
| Low (score 0) | 114 | 43 | 38 | 1.0 | | | |
| Medium (score 1 – 2) | 50 | 19 | 38 | 1.3 | 0.74 – 2.2 | 0.38 | |

| | | AAS Users | | Hazard Ratio for AAS Use[c] | | | |
|---|---|---|---|---|---|---|---|
| **Attribute[a]** | **N[b]** | **N** | **(%)** | **Estimate** | **95% CI** | **P Value** | ***P* For Trend[d]** |
| High (score 3 – 20) | 65 | 37 | 57 | 2.0 | 1.3 – 3.1 | 0.003 | 0.002 |
| **Bulimia** | | | | | | | |
| Low (score 0) | 155 | 62 | 40 | 1.0 | | | |
| Medium (score 1 – 2) | 39 | 15 | 39 | 1.1 | 0.60 – 2.0 | 0.76 | |
| High (score 3 – 20) | 35 | 22 | 63 | 1.9 | 1.2 – 3.1 | 0.011 | 0.009 |
| **Interoceptive Awareness** | | | | | | | |
| Low (score 0) | 81 | 28 | 35 | 1.0 | | | |
| Medium (score 1 – 2) | 65 | 28 | 43 | 1.4 | 0.80 – 2.3 | 0.26 | |
| High (score 3 – 19) | 83 | 43 | 52 | 2.0 | 1.2 – 3.5 | 0.009 | 0.016 |
| **Body Dissatisfaction** | | | | | | | |
| Low (score 0 – 2) | 72 | 22 | 31 | 1.0 | | | |
| Medium (score 3 – 8) | 85 | 35 | 41 | 1.3 | 0.76 – 2.3 | 0.33 | |
| High (score 9 – 24) | 72 | 42 | 58 | 1.9 | 1.1 – 3.3 | 0.014 | 0.003 |
| **Ineffectiveness** | | | | | | | |
| Low (score 0) | 70 | 18 | 26 | 1.0 | | | |
| Medium (score 1 – 5) | 89 | 37 | 42 | 1.7 | 0.93 – 3.1 | 0.082 | |
| High (score 6 – 26) | 70 | 44 | 63 | 3.0 | 1.7 – 5.4 | < 0.001 | < 0.001 |
| **Total score** | | | | | | | |
| Low (score 0 – 20) | 76 | 25 | 33 | 1.0 | | | |
| Medium (score 21 – 33) | 78 | 32 | 41 | 1.4 | 0.83–2.5 | 0.19 | |
| High (score 34 – 109) | 75 | 42 | 56 | 2.0 | 1.2 – 3.3 | 0.01 | 0.004 |
| **Adolescent Experience Questionnaire:** | | | | | | | |
| **Physical Attractiveness** | | | | | | | |
| High (score 21.5 – 30) | 81 | 26 | 32 | 1.0 | | | |
| Medium (score 16.5 – 21) | 74 | 34 | 46 | 1.4 | 0.81 – 2.4 | 0.23 | |
| Low (score 0 – 16) | 74 | 39 | 53 | 2.0 | 1.2 – 3.4 | 0.008 | 0.015 |
| **Athleticism** | | | | | | | |
| High (score 27.5 – 30) | 73 | 24 | 33 | 1.0 | | | |



*Biol Psychiatry*. Author manuscript; available in PMC 2013 February 1.

| | | AAS Users | | Hazard Ratio for AAS Use[c] | | | |
|---|---|---|---|---|---|---|---|
| **Attribute[a]** | **N[b]** | **N** | **(%)** | **Estimate** | **95% CI** | **P Value** | ***P* For Trend[d]** |
| Medium (score 21.5 – 27) | 78 | 32 | 41 | 1.4 | 0.79 – 2.3 | 0.27 | |
| Low (score 0 – 21) | 78 | 43 | 55 | 2.2 | 1.3 – 3.6 | 0.004 | 0.003 |
| **BDD - YBOCS** | | | | | | | |
| Low (score 0 – 1) | 90 | 27 | 30 | 1.0 | | | |
| Medium (score 2 – 4) | 69 | 27 | 39 | 1.5 | 0.84 – 2.6 | 0.17 | |
| High (score 5 – 34) | 74 | 48 | 65 | 3.3 | 2.0 – 5.3 | < 0.001 | < 0.001 |

Abbreviations: AAS, anabolic-androgenic steroids; CI, confidence interval; BDD-YBOCS, retrospective "muscle dysmorphia" version of the Body Dysmorphic Disorder Modification of the Yale Brown Obsessive Compulsive Scale.

[a] This table is limited to attributes showing statistically significant associations with AAS use ($p < 0.05$), together with selected negative findings of interest. For a complete table presenting a full list of attributes assessed, please see Table 3 in supplemental materials.

[b] Total N is less than 233 for some items due to missing data.

[c] By proportional hazards model, with adjustment for race/ethnicity, study site, and birth cohort (see text).

[d] Calculated for the linear trend of the hazard ratios using the medians of the tertiles as a continuous variable.