

# Modeling Population Heterogeneity in Appearance- and Performance-Enhancing Drug (APED) Use: Applications of Mixture Modeling in 400 Regular APED Users

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Appearance- and performance-enhancing drugs (APEDs) constitute a wide range of substances, including anabolic–androgenic steroids, nonsteroidal anabolics, and licit and illicit ergo/thermogenics. A great deal of heterogeneity exists in APED use patterns among weight-lifting men, and, consequently, little is known about how these patterns are related to side effect profiles or risk potential. In the current study, a sample of 400 adult men who were regular APED users completed an interactive Web-based instrument detailing information about APED use, side effects, and related indicators of risk. To explore the heterogeneity of APED use patterns, the authors subjected data on use patterns to (a) latent class analysis (LCA), (b) latent trait analysis (LTA), and (c) factor mixture analysis to determine the best model of APED use. Results indicated that a 4-class factor mixture model provided a better fit than LCA and LTA models. The authors also found that severity and latent class were uniquely associated with negative outcomes. Each of the 4 classes was associated with unique side effects, motivations, and participant use patterns. Implications for identifying pathological forms of APED use are discussed.

*Keywords:* anabolic–androgenic steroid, APED, weightlifting, men, mixture modeling

Appearance- and performance-enhancing drugs (APEDs) include a wide range of substances that affect physiological and psychological states relevant to both physical appearance and athletic performance (Cafri et al., 2005; Evans, 2004). The most widely discussed APEDs are anabolic–androgenic steroids (AASs), which include a range of testosterone and nortestosterone derivatives. All AASs have both androgenic and anabolic activity and exert the majority of their biological effects through binding to androgen receptors (Shahidi, 2001) or through biotransformation to other active compounds, such as dihydrotestosterone (Hartgens & Kuipers, 2004). Androgenic actions primarily include masculinization (e.g., male pattern hair growth, voice deepening), whereas anabolic (i.e., muscle building) actions affect protein synthesis or inhibit protein breakdown. Although most AASs are similar in chemical structure, all differ in the ratio of anabolic to androgenic activity and thus their anabolic potency and potential for androgenic side effects.

Although APEDs are often used for the purposes of increasing muscle mass and reducing body fat, many APEDs have legitimate

medical use. For example, AASs are indicated in the treatment of muscle wasting and catabolic disease (e.g., HIV-related muscle wasting), severe burn injuries, and alcoholic hepatitis (Basaria, Wahlstrom, & Dobs, 2001; Orr & Fiatarone Singh, 2004). Nonsteroidal anabolics, such as human growth hormone (HGH), insulin, and insulin-like growth factor (IGF-1), have also made their way into the APED pharmacopoeia because of their anabolic properties yet share a similar history to AASs in medical practice (Bowlby & Rapaport, 2004; Dills, 2001).

Not all APEDs, however, are used to build muscle; rather, people take a wide range of substances in attempts to reduce body fat or promote endurance. These substances include thyroid hormones (e.g., lithothyronine), beta-2 agonists (e.g., clenbuterol), red blood cell stimulants (e.g., erythropoietin), and central nervous system stimulants (e.g., ephedra). As with AASs and nonsteroidal anabolics, these substances have a history of use for a variety of medical conditions. For example, lithothyronine is used to treat hypothyroidism (Escobar-Morreale, Botella-Carretero, Escobar del Rey, & Morreale de Escobar, 2005), clenbuterol is used to treat asthma (Walters, Walters, & Gibson, 2003), and, until recently, ephedra was used for weight loss in obese individuals (Shekelle et al., 2003).

The physical effects of APEDs have been subject to considerable investigation (e.g., Evans, 2004; Friedl, 2000; Hartgens & Kuipers, 2004); in general, AASs have been shown to increase strength and muscle mass (Bhasin et al., 1996; Hartgens & Kuipers, 2004), with larger doses leading to greater physical gains (Woodhouse et al., 2003). Other APEDs also have documented performance- or appearance-enhancing effects. For example, HGH has been shown to increase protein synthesis in athletes (Godfrey, Madgwick, & Whyte, 2003), and ephedra has been shown to be effective at short-term weight loss (Shekelle et al., 2003).

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The negative effects of APEDs have also received considerable attention (Hartgens & Kuipers, 2004; Langenbucher, Hildebrandt, & Carr, in press). Evidence suggests that AASs have deleterious effects on reproductive functioning (e.g., Torres-Calleja, Gonzales-Unzaga, DeCelis-Carrollo, Calzada-Sanchez, & Pedron, 2001), blood pressure (e.g., Grace, Sculthorpe, Baker, & Davies, 2003), high-density lipoprotein (e.g., Hartgens, Rietjens, Keizer, Kuipers, & Wolffenbuttel, 2004), hepatic functioning (e.g., Soe, Soe, & Gluud, 1992), and, potentially, cardiac structure (e.g., Sachtleben et al., 1993). Over-the-counter (OTC) thermogenics have been linked to negative cardiac effects (Haller & Benowitz, 2000), and ergo/thermogenics have been associated with harmful central nervous system effects (Langenbucher et al., in press). Despite the extensive animal literature base and the modest human literature base on the negative physical effects of AASs and other APEDs, no studies have evaluated long-term effects of these substances in humans. Furthermore, several authors have noted that many of the negative effects of these substances are transitory and that the majority of risk with APED use is associated with long-term use (Evans, 2004; Hartgens & Kuipers, 2004; Pärssinen & Seppälä, 2002).

In addition to physical effects, APEDs have documented psychological effects. Hildebrandt, Langenbucher, Carr, Sanjuan, and Park (2006) described a range of these effects among APED users, the most common being enhanced self-esteem, perceived power over others, and better concentration. APEDs are also associated with negative psychological consequences, most notably with increases in depression, expansive mood, and aggression (Hall, Hall, & Chapman, 2005; Pope & Katz, 1994; Pope, Kouri, & Hudson, 2000; Pagonis, Angelopoulos, Koukoulis, & Hadjichristodoulou, 2006). Other variables associated with APED use include body image disturbance (Hildebrandt, Langenbucher, et al., 2006; Kanayama, Gruber, Pope, Borowiecki, & Hudson, 2001), personality changes (Lindstrom, Nilsson, Katzman, Janzon, & Dymling, 1990; Perry et al., 2003), and symptoms of abuse and dependence (e.g., Perry, Lund, Deninger, Kutscher, & Schneider, 2005). Other APEDs, such as the stimulants, are associated with similar psychological disturbances. In particular, stimulants are associated with anxiety, and, in rare cases, psychosis has been observed (Langenbucher et al., in press). Despite the knowledge of these negative effects, no animal or human models exist to test the polypharmacy found in APED use, and, consequently, very little is known about the potential interactions of these substances and the resulting impact on psychological functioning.

The most recent population-based prevalence estimates of AAS use among men and women indicate that about 2.7% to 2.9% of young Americans have taken AASs (an appropriate proxy for APED use) at least once in their life (National Institute on Drug Abuse, 2000). Field studies suggest greater use among adult gym attendees and athletes; prevalence reports vary from 11% to 67% for men (Bolding, Sherr, & Elford, 2002; Hildebrandt, Schlundt, Langenbucher, & Chung, 2006; Kanayama et al., 2001; Kersey, 1993; Green, Uryasz, Petr, & Bray, 2001; Lindstrom et al., 1990; Tricker, O'Neill, & Cook, 1989; Yesalis et al., 1988; Wagman, Curry, & Cook, 1995). There are no population-based estimates of other APED use; however, a number of authors have reported a wide range of APED use among AAS-using weight lifters and body builders (Copeland, Peters, & Dillon, 2000; Evans, 1997; Perry et al., 2005), noting the concurrent use of steroidal and

nonsteroidal anabolics, illicit ergo/thermogenics, OTC thermogenics, and ancillary agents aimed to reduce or prevent the impact of APED side effects. Thus, the use of AASs in the general population is likely a limited indicator of a more complex form of polysubstance use. This practice of polypharmacy has important implications for identifying pathological forms of APED use, as does expanding the focus from AASs to broader drug use patterns of APED users.

The polypharmacy inherent in APED use is likely to account for a significant amount of the heterogeneity among users. Among descriptive studies, the average number of APEDs used during a typical cycle<sup>1</sup> varies (from three to seven) significantly, which suggests that drugs are used in different combinations, perhaps for different outcomes and with different levels of relative risk (Evans, 1997; Pagonis et al., 2006; Perry et al., 2005). Other sources of variability related to the common types of APEDs used include cycle duration and the relative amount of APED used during a given cycle. Descriptive studies of APED use suggest that cycles generally last anywhere from 4 to 28 weeks and include multiple APEDs in the majority of cases (Copeland et al., 2000; Evans, 1997; Pope & Katz, 1994; Pagonis et al., 2006; Perry et al., 2005). The amount of AAS used in a typical cycle also suggests a great degree of APED heterogeneity. For instance, Evans (1997) reported a variation from 250 to 3,200 mg/week of AAS among 100 APED users. Some degree of this variability is likely attributable to differences in sampling methods (e.g., Pagonis et al., 2006, reported that 85.0% of their community sample typically used stanozolol, whereas Perry et al., 2005, found that only 22.0% used the same drug in their Internet sample), but it might also reflect sampling of different subtypes of APED users. The variability in type, amount, and duration of APED use raises the question of whether there are distinct subtypes of APED users who use APEDs for different reasons and with different levels of risk.

The search for typologies in substance-abusing populations has a long history, most prominently applied to alcohol use disorders (Babor et al., 1992; Cloniger, Bohman, & Sigvardsson, 1981) but also extended to other substance-abusing populations, such as cocaine users (Ball, Carroll, Babor, & Rounsaville, 1995). The identification of unique subgroups of substance abusers has clinical, diagnostic, and etiological implications, such as helping to more accurately identify genetic contributions to psychopathology (Johnson, van der Bree, & Pickens, 1996). The heterogeneity inherent in APED use, attributable to the complexity of drug regimens and regular practice of polypharmacy, suggests the possibility of distinct subgroups of APED users, with different levels of impairment and different risk profiles. Intrinsic to the search for these subtypes is the identification of pathological forms of APED use, which will inform clinical intervention and etiological theory of APED use.

Unlike other drugs of abuse, APEDs have the unique characteristic of long-lasting and desirable physical effects (i.e., muscle gain, fat reduction, and improved athletic performance). Such outcomes, however, are tempered by a range of potentially harmful

<sup>1</sup> APEDs are typically used in cycles, which consist of predetermined patterns of APED use with defined duration, amount, and types of APED. Cycles often include periods of discontinuation to allow for reregulation of the hypothalamic-pituitary-testicular axis.

psychological and physical effects. To accurately identify what forms of APED use are pathological, we must explore the relationship between types of APED use and relevant negative outcomes (e.g., mood disturbance, using high doses for long duration, and intended long-term use). The goals of such research are gaining a better understanding of what type of APED use is the best indicator of pathology, identifying potential predictors of pathological APED patterns, and recognizing relationships between specific types of APED use and unique risk profiles. These findings have implications for creating appropriate diagnostic criteria and identifying appropriate risk factors, and they have broader implications for understanding psychopathology. Most important, this research can contribute to the growing literature on whether drug use pathology is a unique phenomenon or occurs along a severity continuum (B. Muthén, 2006).

The utility of categorical distinctions in psychiatric research has been a topic of much theoretical debate, with some methodologists suggesting that categories may simply reflect ordered groups of different levels of severity (e.g., Kruger, Markon, Patrick, & Iacono, 2005). Whereas categorical distinctions may fit the data and identify groups with different clinical presentations, modeling of pathology along a continuum may provide a more parsimonious and clinically useful method of understanding population heterogeneity. Consistent with this critique, methodologists have begun to apply hybrid models to typology research in substance-abusing populations (Lubke & Muthén, 2005; B. Muthén, 2006; B. Muthén & Asparouhov, 2006). These models combine the search for unique user subtypes with the identification of a continuum of severity within these distinct groups. Such modeling allows for the potential to understand what characteristics are better indicators of severity as well as what different profiles of user characteristics are likely to exist within identified populations.

The purpose of the current study is to examine the heterogeneity in APED use. We have two specific aims: (a) to determine whether there are unique patterns of APED use within an APED-using population and (b) to examine the relationship between APED use patterns and outcomes. A Web-based approach, which has received support from methodologists (Gosling, Vazire, Srivastava, & John, 2004; McCabe, 2004), was selected because of the difficulty in accessing steroid users.

## Method

### Data Collection

Participants were recruited from Internet discussion boards between November 2003 and November 2005. Links posted to both moderated, unrestricted public message boards and selected membership boards devoted to performance-enhancing drugs, body building, power lifting, and physical fitness directed interested respondents to a Rutgers Web server where the data collection instrument resides. The instrument totaled 445 items and took an average of 20 to 30 min to complete. The measure may be viewed at <http://websurvey.rutgers.edu/steroids/>. The instrument was interactive, allowing individuals to skip out of sections not relevant to their use patterns (e.g., those who did not report OTC ergo/thermogenic use did not receive any questions about OTCs, which eliminated 96 items). Participants were assumed to have knowledge of the issues assessed, and efforts were made to use terms

with which APED users are comfortable. All participants gave informed consent and indicated that they were at least 18 years of age by clicking on the appropriate boxes on the Statement of Informed Consent that headed the instrument. All procedures were approved by the Rutgers University institutional review board.

To ensure data quality, we dropped from the final sample data submitted from identical IP addresses and participants who endorsed use of bogus APEDs or provided conflicting item endorsements. A total of 887 valid surveys were submitted, and 799 respondents identified themselves as men. Of these, 762 reported APED use. Duplicate IP addresses were identified in 54 cases, and 4 participants endorsed bogus drugs or conflicting items. Thus, the final sample consisted of 704 unique APED-using men. Descriptive information on the entire sample of 704 will be reported separately.

### Participants

Demographics for the sample are reported in Table 1. Participants were ages 18–72 ( $M = 29.19$ ,  $SD = 8.79$ ) and were mostly

Table 1  
Sample Demographics

Domain	Description	Regular users (> 2 cycles)	
		Freq.	%
Marital status	Married	157	39.3
	Living as married	32	8.0
	Divorced	17	4.3
	Widowed	0	0.0
	Single	193	48.4
Education level	Some high school	2	0.5
	High school	34	8.5
	Trade school	11	2.8
	Some college	147	36.8
	College degree	122	30.5
	Some graduate school	27	6.8
	Graduate degree	57	14.3
Employment	Full time	274	68.5
	Part time	39	9.8
	Homemaker	1	0.3
	Student	61	15.3
	Retired	3	0.8
	Unemployed	17	4.3
	Disabled	3	0.3
Income	Less than \$20,000	73	18.4
	\$20,000–\$29,999	49	12.3
	\$30,000–\$39,999	57	14.4
	\$40,000–\$49,999	52	13.1
	\$50,000–\$59,999	36	9.1
	\$60,000–\$74,999	50	12.6
	\$75,000–\$99,999	31	7.8
	\$100,000 or more	49	12.3
Race/ethnicity	White	345	87.6
	Hispanic/Latino	27	6.9
	African American	10	2.5
	Native American	7	1.8
	Asian	9	2.3
Sexual orientation	Heterosexual	384	96.2
	Homosexual	12	3.0
	Bisexual	3	0.8

Note.  $N = 400$ . Income is presented in thousands of dollars. Freq. = frequency.

from North America (United States = 81.0%, Canada = 8.0%); 6.8% were from Europe, 1.4% were from South America, and 1.3% were from Asia. Because of the significant heterogeneity in initial APED use, we selected regular users (defined as men who had completed more than two cycles) to examine the heterogeneity in APED use. Initiation of APED use is marked by a significant degree of heterogeneity (Evans, 1997) and is often influenced by a range of factors, including availability, novelty seeking, and experimentation. These patterns are unlikely to be stable or reflect true preferences for drug choice. For these reasons, we selected the lowest threshold at which true drug preferences could occur (i.e.,  $> 2$  cycles = 1 initial cycle + 2 standard cycles).

Of the total sample of 704, 400 men were regular users and were included in analyses. The regular users were similar in age to beginning users, but they were more educated, had a higher income, and were more likely to be married and to be employed full time.

### Measures

**Measure development.** The online instrument was developed to collect a range of APED user information, including demographic information, training and exercise history, motivations for APED use, prohormone use, steroidal and nonsteroidal anabolic use, OTC ergo/thermogenics use, illicit ergo/thermogenics use, ancillary drug use, and APED risk. Items were generated on the basis of the existing APED literature and reviews of the pharmacological, physical, and psychological effects of APED use. For instance, we pooled information on available drug choices from existing review and descriptive articles (e.g., Bolding et al., 2002; Evans, 1997) and several lay steroid sourcebooks, commonly referred to “steroid bibles” among users (see Llewellyn, 2005, for a recent example). We also consulted several databases, including the National Collegiate Athletic Association’s list of banned substances. We sent the complete list to several discussion board moderators, who provided feedback and street names for many of the drugs. The final lists were incorporated into the measure. We completed this process for all the sections used in the final assessment; to increase the quality of the item structure and content, we recruited a group of 12 APED users and asked them for specific feedback about the online instrument, which we incorporated into the instrument when possible. In particular, we gathered feedback about items related to drug use behavior (e.g., weeks between cycles, common language for describing amounts of different drugs) to create items with a high degree of face validity.

**Anabolic APEDs.** A total of 22 steroidal and nonsteroidal oral and injectible APEDs were queried. Participants were asked to

indicate which (if any) oral anabolics you have used. Check all that apply. If you have completed only one “cycle” of oral anabolics, check circles only in the column marked “1st Cycle.” If you have completed two or more cycles using oral anabolics, check circles in both columns. The column marked “Usual Use” here is meant to gather information on your usual pattern. Do your best with it, and indicate what is usual, typical, or characteristic for you.

Similar wording was used for injectible APEDs. Thus, participants indicated first cycle, usual cycle, and ever-in-lifetime use for all substances by clicking a box associated with each drug. Anabolics used during usual cycles reflect true drug preferences, whereas

ever-in-lifetime use may reflect overall experimentation with or access to anabolics. APED measurement included weekly milligrams of oral and injectible AASs (range = 0–3,000mg/week) at 250-mg/week intervals and standard international units (IUs) of insulin, HGH, and IGF-1 at 2-IU/day intervals.

**OTC ergo/thermogenics.** Twenty common ephedrine- and nonephedrine-based fat-burning supplements available over the counter were queried for first cycle use, usual use, and ever-in-lifetime use. A dichotomous variable (presence vs. absence) was created for current OTC use and subsequently used in analyses. Percentage of the most common OTC ingredients is reported for descriptive purposes.

**Illicit ergo/thermogenics.** Five illicit ergo/thermogenics were queried for first cycle use, usual use, and ever-in-lifetime use (clenbuterol, liothyronine, levothyroxine, erythropoiten, and dinitrophenol [DNP]). The amount per week of each substance was also queried, in intervals of 20 mg and 20 mcg, with the exception of DNP, which was measured in intervals of 100 mg/week. In addition, highest single dose amounts of each drug were recorded.

**APED risk.** Respondents were queried about side effects (see Bahrke & Yesalis, 2002; Langenbucher et al., in press, for comprehensive reviews), which in men are primarily feminization (water retention, fat deposition, and gynecomastia) from the aromatization into estrogen of androgens such as testosterone, suppression of the hypothalamic–pituitary–testicular axis, dysphoric mood (e.g., aggressiveness, depression), and symptoms related to central nervous system stimulation. Regarding anabolic APEDs, participants were asked, “What, if any, side-effects have you experienced during or immediately after a cycle of steroidal or nonsteroidal anabolics?” Similar language was used for OTC ergo/thermogenics and illicit ergo/thermogenics. All items were scored dichotomously; separate lists were provided for steroidal and nonsteroidal APEDs (21 items), OTC thermogenics (16 items), and illicit ergo/thermogenics (21 items). APED side-effect frequencies are reported in Appendix A.

Each set of side effects was subjected to exploratory factor analyses with Promax rotations via a mean- and variance-adjusted least squares estimator to determine whether there were meaningful side effect subscales. Solutions were evaluated for goodness of fit with chi-square tests and root-mean-square errors of approximation (RMSEAs). All factor loadings for final solutions are reported in Appendixes B, and D. The cutoff for acceptable RMSEA was  $< .05$ , and items with cross-loadings greater than  $|.40|$  were excluded.

A five-factor model provided the best solution for anabolic APED side effects,  $\chi^2(27, N = 400) = 27.04, p = .46$  (RMSEA = .002), and accounted for 64.27% of the variance. The factors were labeled Dysphoric Mood, Unwanted Tissue Growth, Sexual Dysfunction, Hyperarousal, and Androgenic Side Effects. A three-factor model provided the best solution for OTC thermogenics side effects,  $\chi^2(44, N = 305) = 53.04, p = .15$  (RMSEA = .027), and explained 57.23% of the variance. These factors were labeled Adrenaline-Related Side Effects, Sexual Dysfunction, and Dysphoric Mood. A five-factor solution provided the best fit for illicit ergo/thermogenics side effects,  $\chi^2(35, N = 262) = 38.28, p = .32$  (RMSEA = .019), and explained 62.43% of the variance. The factors were labeled Dysphoric Mood, Dehydration, Sexual Dysfunction, Vasoconstriction, and Autonomic Arousal. We summed side effect scales to create subscale scores. The Kuder



Richardson-20 (KR-20; Kuder & Richardson, 1937) formula was used as measure of internal consistency, which varied from .72 to .88 (see Appendixes A, B, and C for each subscale).

Additional consequence items included (a) "If you could meet all of your physical training goals very soon through the use of drugs or supplements, but knew that using these drugs would shorten your life, how many years of life would you be willing to sacrifice to meet all of your physical training goals very soon?" (range = 0–30), (b) whether the respondent would continue APED use with "absolute evidence that [APEDs] are likely to cause severe health problems," and (c) "For how long do you anticipate that you will be involved in the use of anabolics or ergogenic drugs, even if only on occasion" (5-point ordinal scale: *I will not use them again, 0–5 years, 6–10 years, more than 10 years but not forever, rest of my life*).

*Covariates.* The effects of several covariates were examined in the mixture models. We chose self-reported height, weight, and body fat percentage because of existing evidence that male weight-lifting subgroups significantly differ on these variables (Hildebrandt, Schlundt, et al., 2006). In addition, we hypothesized that the total number of years of APED use (current age – age of first APED use) would be related to the pattern of APED use because of increased exposure to and confidence with different APEDs. We also asked, "Is your livelihood (ability to make money) related at all to your physical strength, size or appearance? If so, how much of your livelihood (in percent) is dependent on strength, size or appearance?" We hypothesized that an individual's profession may promote different types of APED use, namely taking more risks with the types of APEDs used. For example, an individual who benefits from increased strength derived from APEDs may be willing to take more APEDs than someone whose livelihood is not strength or appearance based. Finally, we hypothesized that income would have an effect on the pattern of APED use because certain APEDs are particularly expensive (e.g., HGH) and financial resources may dictate the ability of an APED user to regularly add them to his or her usual APED pattern.

### Design and Analyses

*Model estimation.* To investigate patterns of APED use, we used latent class analysis (LCA). This methodology assumes that the relationship of given manifest (observed) variables within a population can be explained by a latent (unobserved) nominal variable (Vermunt & Magidson, 2002). Thus, population heterogeneity (in this case, pattern of APED use) can be reduced to a discrete set of homogeneous groups for which relationships between observed variables (type of APED) can be explained by membership in unobserved latent classes (subtypes of APED user). Fit statistics can then be used to compare different LCA models to determine the most appropriate model. Mplus Version 3.12 (L. K. Muthén & Muthén, 2004) was used to fit two- to five-class models to the data, and covariates were entered into the mixture model to improve the fit and estimate the effects of background variables on class membership. Each latent class represents a distinct profile of item endorsement probabilities (IEPs) that is theoretically the same for all members in the class. Participants can also be classified into subgroups on the basis of their posterior class probability. The Bayesian information criterion (BIC), an index that favors parsimony, with lower numbers indicating a better fit (Schwartz, 1978),

and the Akaike information criterion (AIC; Akaike, 1987) were used to measure goodness of fit. The best fitting model was chosen on the basis of consideration of AIC, BIC, classification quality (e.g., entropy value), and likelihood ratio chi-square. The adjusted Lo–Mendell–Rubin log-likelihood ratio test (Lo, Mendell, & Rubin, 2001) was used to compare successive models, with a significant log-likelihood ratio indicating that the model with a larger number of classes provides a better fit to the data.

The LCA models were compared with several competing models. First, a latent trait model (also referred to as item response theory or item response modeling) was fitted to the data (see Kruger et al., 2004). That is, a two-parameter logistic latent trait model was fitted to the data that assumed that a continuous latent variable explained the relationship between APEDs. This continuous latent variable was a measure of severity of APED use. Recent methodological work has combined latent class models with latent trait models to create hybrid models (termed *factor mixture analyses* [FMAs]; Lubke & Muthén, 2005; B. Muthén, 2006; B. Muthén & Asparouhov, 2006) in which both a continuous latent variable (e.g., severity) and a nominal latent variable (e.g., subtype of APED user) are estimated. Such hybrid models have been shown to be superior to LCA or two-parameter logistic item response theory models of substance abuse (e.g., Lubke & Muthén, 2005). Models were compared with BICs and AICs, with the lowest value indicating the best fitting model.

*Concurrent validity.* Standard regression analyses were used to evaluate the unique relationship of latent class and latent severity to measures of APED use, consequences, and risk. Bonferroni-corrected alpha levels were used because a large number of comparisons were made; the overall alpha level was set to  $p < .002$  ( $\alpha = .05/25 = .002$ ) to control for Type I error.

*Missing data.* There were very few missing data; the majority of variables were missing less than 5% of responses. Missing data were replaced via a missing-at-random function described by L. K. Muthén and Muthén (2004) that uses maximum likelihood estimation. This method allows for missing data to vary as a function of covariates but does not allow for covariates to have any missing data. Data were complete for the proposed covariates.

## Results

### Sample Description

Participants weighed, on average, 215.03 lbs ( $SD = 35.82$ ) and had a mean height of 71.09 in. ( $SD = 3.05$ ). Their average body mass index bordered on the clinical cutoff for obesity ( $M = 29.93$ ,  $SD = 7.79$ ; National Heart Lung and Blood Institute, 1998), but when we accounted for estimated body fat percentage ( $M = 13.09\%$ ,  $SD = 4.19\%$ ), we found that elevated body mass index was likely a function of increased lean mass. Fat-free mass index (Kouri, Pope, Katz, & Oliva, 1995) scores for participants averaged 26.97 ( $SD = 4.27$ ), the upper limit of lean mass attainable without APEDs. The participants were experienced users, having cycled an average of 5.84 ( $SD = 3.04$ ) times and having taken an average of between 250 and 500 mg/week of oral AAS and between 750 and 1,000 mg/week of injectible AAS for 7.50 ( $SD = 7.49$ ) and 13.90 ( $SD = 10.90$ ) weeks, respectively. Those using insulin ( $n = 29$ ) reported an average of 9.42 ( $SD = 4.44$ ) IUs per dose, and those using HGH or IGF-1 ( $n = 45$ ) reported 4.34 ( $SD =$

3.88) IUs per day. Participants exercised a median of 2–5 hr/day for a median of 5 days per week and had been exercising for an average of 10.61 ( $SD = 7.69$ ) years.

Use of OTC ergo/thermogenics was common ( $n = 305$ ; 76.3%), and illicit ergo/thermogenics were used by approximately one third of participants ( $n = 136$ ; 34.2%). OTC ergo/thermogenics were cycled for an average of 20.42 ( $SD = 15.61$ ) weeks, with the most common ingredients being caffeine ( $n = 135$ ; 44.26%), ephedra ( $n = 100$ ; 32.79%), mahuang ( $n = 99$ ; 32.46%), and yohimbine ( $n = 97$ ; 31.80%). Illicit ergo/thermogenics were cycled for an average of 10.73 ( $SD = 8.67$ ) weeks. Average doses were calculated for those who endorsed regular use of clenbuterol ( $M = 74.27$  mg,  $SD = 56.54$  mg), liothyronine ( $M = 50.76$  mcg,  $SD = 48.65$  mcg), and DNP ( $M = 468.19$  mg,  $SD = 236.93$  mg).

### Model Estimation

All APEDs were initially used in LCA models, including very rare ones; however, models including all APEDs were poorly fitted and difficult to interpret. Thus, APEDs used by less than 5% of the total sample (fluoxymestosterone, drostanolone propionate, IGF-1, methenolone enanthate, nandrolone laureate, parabolon acetate, testosterone suspension, and trenbolone hexahydrobenzylcarbonate) were subsequently dropped from model estimation. In addition, stanozolol injectible and oral administration as well as testosterone derivatives (single and multiple ester) were analyzed as single variables, respectively, because they are often used interchangeably.

Table 2 reports the AIC, BIC, and entropy for two- to five-class mixture models. The four-class FMA solution provided the best fit to the data on the basis of lowest AIC and lowest BIC, although entropy was similar to that in LCA models. The likelihood ratio chi-square test was also nonsignificant, indicating an appropriate fit to the data. The four-class FMA and LCA models also provided a better fit to the data than the two-parameter logistic item response theory model, which suggests that the population heterogeneity in APED use was better explained by unique subgroups of APED

users as opposed to a single dimension of severity. However, the superiority of the four-class FMA model indicated that there was additional heterogeneity within these unique classes that can be explained by a continuous dimension of severity.

We explored factorial invariance of the severity dimension by comparing a complete factorial-invariant model (i.e., intercept and slope equal across classes) with a relaxed model, in which parameter constraints were relaxed on intercept and loading parameters and a series of chi-square difference tests were used to evaluate improved fit with removal of parameter constraints. The specification search indicated that a model with partial invariance best fitted the data. Partial invariance holds when some but not all of the model parameters are invariant (Byrne, Shavelson, & Muthén, 1989). The slope parameters for nandrolone decanoate, oxymetholone, testosterone, OTC ergo/thermogenics, and OTC ergo/thermogenic intercept were found to be invariant. Freeing other parameters, however, provided significant improvement in goodness of fit; thus, these parameter estimates were retained in the final model (BIC = 4,721.588; AIC = 5,011.237; entropy = .0811).

IEPs for the four-class FMA model are reported in Figure 1. Class 1 (heavy polypharmacy;  $n = 43$ ; 10.75%)<sup>2</sup> was characterized by the highest probability of insulin and HGH use across classes as well as high probabilities of using most APEDs, including licit and illicit ergo/thermogenics. Class 2 (primarily fat burning;  $n = 67$ ; 16.75%) had the highest probability of using liothyronine, clenbuterol, and DNP as well as stanozolol, a steroid with a low androgenic-to-anabolic ratio that is often used to maintain leanness. Class 3 (primarily mass building;  $n = 83$ ; 20.75%) had the highest probability of using APEDs associated with adding muscle mass. IEPs were highest for methandrostenolone, boldenone undecylenate, and testosterone, agents that have a high androgen-to-anabolic ratio and thus are considered better for adding muscle mass. Finally, Class 4 (normative APED use;  $n = 207$ ; 51.75%) had the lowest probability of using all APEDs but generally had high probabilities of using testosterone, methandrostenolone, and OTC ergo/thermogenics.

Table 2

Summary or Fit Statistics and Classification Quality for Appearance- and Performance-Enhancing Drug Models

Model	AIC	BIC	Entropy
LCA			
Two-class	5,435.06	5,672.90	0.667*
Three-class	5,413.65	5,679.19	0.690*
<b>Four-class</b>	<b>5,397.49</b>	<b>5,570.77</b>	<b>0.761*</b>
Five-class	5,406.25	5,761.49	0.765
Latent trait			
2pl IRT	5,820.508	5,936.260	
Factor mixture			
Two-class	5,366.309	5,625.754	0.683*
Three-class	5,047.799	5,520.944	0.747*
<b>Four-class</b>	<b>5,019.432</b>	<b>5,489.934</b>	<b>0.782*</b>
Five-class	5,127.845	5,528.652	0.703

Note. Boldface type indicates best-fitting model. LCA = latent class analysis; AIC = Akaike information criterion; BIC = Bayesian information criterion; 2pl IRT = two-parameter logistic item response theory model.

\*  $p < .05$  (Lo–Mendell–Rubin test).

### Covariates

Table 3 summarizes the group means and effects of the covariates on APEDs, with Class 4 as the reference group. Results suggested that Class 1—s higher income and weight and greater experience with APEDs were associated with their unique APED use pattern. Class 2—s higher income, lower height, less experience with APEDs, and lower body fat percentage were associated with their unique APED use pattern. Finally, the degree to which Class 3—s livelihood depended on strength and appearance was significantly associated with its unique APED use pattern.

### Concurrent Validity

A chi-square analysis indicated that there were significant differences in training identity among groups,  $\chi^2(12, N = 400) = 32.36, p < .001$  ( $\phi = .303$ ), which suggests that the pattern of

<sup>2</sup> We calculated the size of latent classes by assigning members to a class on the basis of their highest posterior class probability.

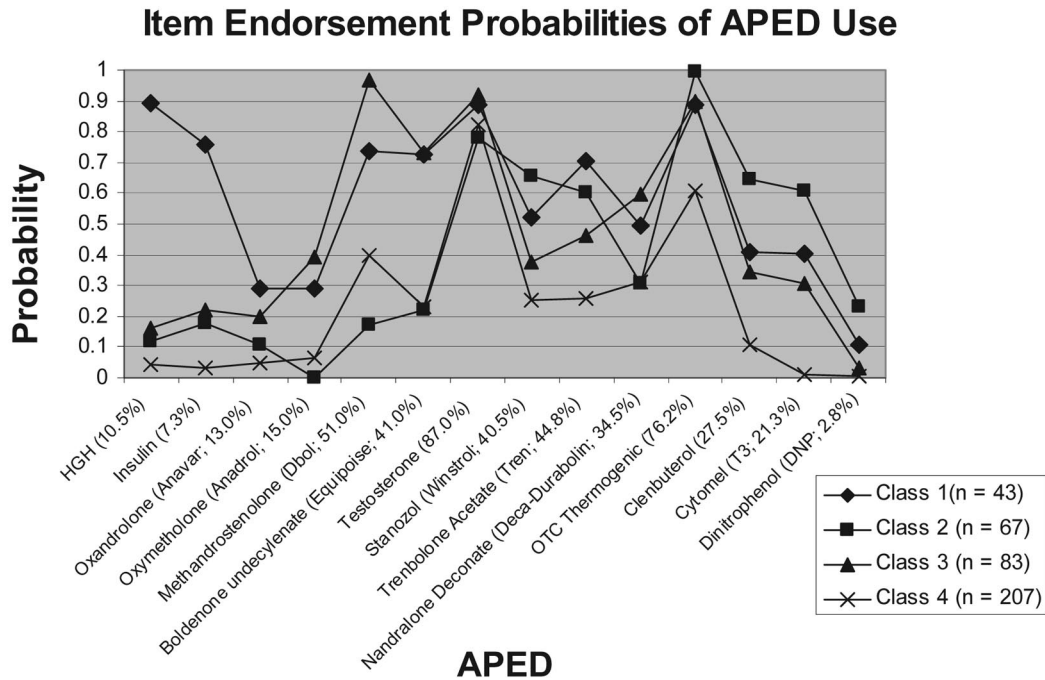


Figure 1. Item endorsement probabilities for appearance- and performance-enhancing drugs (APEDs) by latent class. Class 1 is the heavy polypharmacy group. Class 2 is the primarily fat-burning group. Class 3 is the primarily mass-building group. Class 4 is the normal APED group. HGH = human growth hormone; OTC = over-the-counter.

APED use was related to how participants identified their status as an athlete. Class 1 participants were more likely than all other groups (we calculated odds ratios [ORs] comparing individual class probability with the probability of all other classes) to identify themselves as body builders ( $n = 40$ ; 93.02%; OR = 2.07), and Class 4 participants were most likely to identify themselves as recreational weight lifters ( $n = 44$ ; 21.1%; OR = 2.18). Class 3 was characterized by the highest probability to identify as a power lifter ( $n = 18$ ; 21.69%; OR = 2.99) and Class 2 by the highest probability to identify as a fitness or endurance athlete ( $n = 7$ ; 10.44%; OR = 4.75). This pattern is consistent with Class 1 being

the heaviest APED-using group, with Class 2 using “cutting” agents, and with Class 3 using “bulking” agents. Similarly, there were significant differences in which goal, increasing size versus increasing leanness, was most important,  $\chi^2(3, N = 400) = 14.79$ ,  $p < .001$  ( $\phi = .201$ ). Class 2 ( $n = 51$ ; 76.12%; OR = 1.67) was 1.5 times as likely as other APED classes to desire increased leanness over size, whereas Class 3 ( $n = 60$ ; 72.29%; OR = 1.93) was nearly two times as likely to desire increases in strength and size over leanness. These class differences in training goals support the observed differences in both APED use patterns and athletic identities.

Table 3  
Results of Multinomial Logistic Regression of Latent Class on Background Variables

Variable	Class 1				Class 2				Class 3				Class 4 <sup>a</sup>	
	M	SD	(exp)β	SE	M	SD	(exp)β	SE	M	SD	(exp)β	SE	M	SD
Years of APED use	5.72	4.37	.060**	.014	4.17	4.19	-.067**	.015	5.68	5.84	.027	.029	4.98	5.23
Weight (kg)	104.63	21.77	.019**	.004	99.01	19.99	-.003	.005	94.94	26.12	-.002	.005	97.36	23.45
Height (cm)	180.87	8.43	-.024	.070	178.10	7.43	-.200**	.042	181.81	6.12	.011	.055	181.10	7.50
Body fat %	11.04	3.61	-.040	.046	9.51	4.89	-.430**	.050	12.45	3.43	-.033	.045	14.49	4.43
Income (\$)	42,670	24,060	.236*	.066	45,943	27,452	.158**	.031	31,953	28,798	-.160	.090	37,486	28,328
% livelihood	26.37	34.73	.006	.007	24.31	30.56	.004	.007	33.46	31.58	.015**	.003	23.44	29.07

Note. Results are drawn from the best fitting factor mixture model and thus reflect the simultaneous calculation of effects on latent class and latent severity. The means and standard deviations reflect those calculated from assigning participants to classes on the basis of highest posterior class probability. APED = appearance- and performance-enhancing drug; % livelihood = the percentage that an individual’s appearance is dependent on strength or appearance.

<sup>a</sup> Class 4 was used as the reference group in multinomial logistic regression.  
\*  $p < .05$ . \*\*  $p < .01$ .

Table 4 summarizes the relationship among severity, probability of latent class, and other pattern validation variables. Results are consistent with the probability of Class 1 membership being associated with heavy APED use, given that membership was associated with using APEDs for longer duration and higher doses than the other APED classes. Depending on the outcome variable, Class 2 or Class 3 membership was associated with more dangerous APED patterns. For example, probability of Class 3 membership had a stronger positive relationship with duration and amount of oral anabolic use than Class 2 membership. Conversely, probability of Class 2 membership had a stronger positive relationship with duration of ergo/thermogenic use. Overall, probability of Class 4 membership was associated with lower quantity and frequency of APED use, consistent with these individuals' higher likelihood of being recreational weight lifters.

### APED Severity

Table 5 results also suggest that severity was more strongly associated with certain APED pattern variables. In particular, the strongest associations were found between severity and number of cycles, weeks between APED cycles, weeks of illicit ergo/thermogenic use, and injectible anabolic quantity and frequency. The results also suggest that latent severity accounted for more unique variance in most pattern outcome variables than latent class membership.

Examining the relationship between individual APEDs and severity (see Table 5) suggests that certain APEDs were better indicators of severity within each latent class. As described, the model did not hold for strict measurement invariance, and the model used to estimate severity had constraints on slope parameters for nandrolone decanoate, oxymetholone, testosterone, OTC ergo/thermogenics, and the OTC ergo/thermogenic intercept. For instance, illicit thermogenics loaded heavily on severity for Classes 1 and 2 but not Classes 3 and 4. The reverse was true for oral anabolics; severity was best indicated by oral anabolics in Class 3 but not Classes 2 or 4. Other APEDs, such as testosterone, were not good indicators of severity in any class, which might have been in part because of common use. Within class, the amount of variance explained also differed between classes, with severity

explaining the most variability in Classes 1 and 2. Severity was also significantly related to the covariates (see Table 6). Years of APED use and percentage of livelihood dependent on strength and appearance had the strongest relationships with severity of APED use.

### Consequences and Risk

Regression analyses were used to examine the relationship among probability of latent class membership, latent severity, and APED consequences (see Table 7). Overall, severity and latent class accounted for a significant amount of variability in a range of different side effects but were the best predictors of dysphoric mood, hyperarousal, and androgenic- and adrenaline-related side effects. Conversely, these variables were poor predictors of sexual side effects and vasoconstriction. There were also differences in the parameter estimates for each latent class as well as differential effects of latent severity and latent class on these outcomes. Probability of Class 1 membership and latent severity had the strongest relationship with hyperarousal, dysphoric mood, and androgenic side effects related to anabolic APED use. However, probability of Class 2 membership had a stronger relationship than all other classes with adrenaline-related side effects. Furthermore, pairwise post hoc comparisons of regression coefficients indicated that probability of Class 1 membership consistently showed stronger correlations with negative side effects, but probability of Class 2 membership had significantly stronger correlations with OTC ergo/thermogenic side effects. Probability of Class 4 membership was weakly related to most outcomes, with the exception of strong negative relationships with illicit ergo/thermogenic side effects, which suggest that Class 4 membership may be protective of these types of side effects.

As with side effects, latent class membership and severity were significantly related to a range of risk factors (see Table 8). The strongest relationships were observed between latent severity and individual risk indicators, although probability of Class 1 membership was also a good predictor across APED risk indicators. In particular, probability of Class 1 membership was associated with total number of side effects, unwillingness to give up APED use, and willingness to sacrifice years of life to achieve performance or

Table 4  
Concurrent Validity of Severity and Probability of Latent Class on Drug Use Variables

Variable	Severity		Class 1		Class 2		Class 3		Class 4		<i>F</i> <sup>a</sup>	<i>p</i>	<i>R</i> <sup>2</sup>
	$\beta$	<i>SE</i>	$\beta$	<i>SE</i>	$\beta$	<i>SE</i>	$\beta$	<i>SE</i>	$\beta$	<i>SE</i>			
Weeks between cycles	-.303	.082*	-.101	.043*	.053	.012	.074	.096	.191*	.023*	4.65	<.001	.072
No. cycles <sup>b</sup>	.267	.073*	.049	.089	.117	.056*	.134	.052*	-.012	.112	13.16	<.001	.112
Milligrams oral anabolics/week <sup>b</sup>	.060	.041	.191	.095*	.123	.043*	.150	.023*	-.023	.043	7.02	<.001	.077
Cycle length of oral anabolics <sup>b</sup>	.122	.051*	.111	.032*	.032	.037	.189	.053*	.061	.044	7.69	<.001	.080
Milligrams injectible anabolics/week <sup>b</sup>	.240	.121*	.147	.057*	.140	.063*	.138	.054*	.002	.042	14.30	<.001	.131
Cycle length of injectible anabolics <sup>b</sup>	.231	.102*	.045	.037	.087	.040	.103	.038*	.087	.055	19.32	<.001	.227
Cost of APEDs/year	.233	.079*	.178	.055*	.062	.059	.165	.087*	-.109	.041*	16.32	<.001	.142
OTC ergo/thermogenics weeks/year ( <i>n</i> = 305)	.098	.056	.127	.043*	.260	.053*	.019	.034	-.113	.058*	7.12	<.001	.078
Illicit thermogenics weeks/year ( <i>n</i> = 262)	.255	.065*	.291	.101*	.131	.049*	.101	.032*	-.089	.065	10.55	<.001	.121

Note. Post hoc significance level was set at  $\alpha = .01$ . APEDs = appearance- and performance-enhancing drugs; OTC = over-the-counter.

<sup>a</sup> Between-subject and within-subject degrees of freedom are 5 and 394, respectively, for rows 1–7; 5 and 299, respectively, for row 8; and 5 and 256, respectively, for row 9. <sup>b</sup> Log transformations of dependent variables were calculated because of nonnormal distribution.

\* *p* < .05.



Table 5  
*Discrimination and Difficulty Parameter Estimates for Severity of Appearance- and Performance-Enhancing Drug Use Within Each Latent Class*

Drug	Class 1		Class 2		Class 3		Class 4	
	<i>a</i>	<i>b</i>	<i>a</i>	<i>b</i>	<i>a</i>	<i>b</i>	<i>a</i>	<i>b</i>
Oral anabolic								
Methandrostenolone								
Parameter	1.45	-1.68	3.35	3.55	4.35	-6.44	3.41	0.27
SE	0.39	0.77	0.73	0.33	1.10	0.85	0.73	0.21
Oxandrolone								
Parameter	0.95	1.74	0.45	5.81	0.29	3.28	2.65	7.44
SE	0.22	0.84	0.51	0.23	0.28	0.91	0.27	0.90
Oxymetholone								
Parameter	<b>1.97</b>	1.19	<b>1.97</b>	8.56	<b>1.97</b>	1.77	<b>1.97</b>	8.77
SE	<b>0.59</b>	0.34	<b>0.59</b>	0.03	<b>0.59</b>	0.68	<b>0.59</b>	0.46
Injectible anabolic								
Human growth hormone								
Parameter	5.35	-3.44	2.96	-6.55	3.29	4.89	5.22	8.81
SE	0.92	1.60	1.17	0.38	0.67	0.29	0.65	1.45
Insulin								
Parameter	4.58	-3.22	3.26	5.44	3.19	3.55	4.28	9.02
SE	0.77	1.18	1.01	0.29	1.01	0.39	0.93	1.72
Boldenone undecylenate								
Parameter	1.93	-2.17	3.33	2.27	2.69	-1.91	1.91	3.11
SE	0.56	0.29	0.57	0.72	0.80	0.49	0.29	0.27
Nandralone decanoate								
Parameter	<b>0.78</b>	0.22	<b>0.78</b>	-2.68	<b>0.78</b>	1.89	<b>0.78</b>	2.02
SE	<b>0.24</b>	0.15	<b>0.24</b>	0.30	<b>0.24</b>	0.65	<b>0.24</b>	0.24
Stanozolol								
Parameter	1.15	0.22	0.78	-2.57	1.25	2.18	1.89	2.18
SE	0.34	0.17	0.28	0.62	0.60	0.34	0.27	0.42
Testosterone (single and mixed ester)								
Parameter	<b>0.31</b>	-3.29	<b>0.31</b>	-2.83	<b>0.31</b>	-2.88	<b>0.31</b>	-2.62
SE	<b>0.13</b>	0.91	<b>0.13</b>	0.90	<b>0.13</b>	1.18	<b>0.13</b>	0.82
Trenbolone acetate								
Parameter	1.36	-1.92	0.89	-1.45	1.67	0.89	0.29	2.25
SE	0.84	0.37	0.29	0.89	0.23	0.25	0.19	1.21
Ergo/thermogenic								
OTC ergo/thermogenic								
Parameter	<b>0.41</b>	<b>-6.87</b>	<b>0.41</b>	<b>-6.87</b>	<b>0.41</b>	<b>-6.87</b>	<b>0.41</b>	<b>-6.87</b>
SE	<b>0.14</b>	<b>0.56</b>	<b>0.14</b>	<b>0.56</b>	<b>0.14</b>	<b>0.56</b>	<b>0.14</b>	<b>0.56</b>
Clenbuterol								
Parameter	2.30	-1.84	1.36	-4.12	2.53	1.28	3.14	5.91
SE	0.41	0.90	0.51	0.37	0.35	0.15	0.25	0.15
Liothyroxine								
Parameter	1.94	3.36	3.25	-2.59	1.27	1.41	1.25	8.29
SE	0.52	0.11	0.74	0.54	0.38	0.26	0.28	0.27
Dinitrophenol								
Parameter	1.29	6.77	2.94	4.29	0.19	9.55	0.89	9.54
SE	0.33	1.24	1.11	0.90	0.04	0.53	0.24	0.42
Within-class variance <sup>a</sup>								
Parameter	15.23		18.02		26.61		34.57	
SE	4.32		6.01		6.58		5.19	

Note. *a* is the discrimination or slope parameter, and *b* is the difficulty or location parameter in a standard two-parameter logistic item response theory model. Equality constraint was set for over-the-counter (OTC) ergo/thermogenics in the best fitting model. All parameters are significantly different than zero at *p* < .01. Parameters in boldface are constrained to be equal. Releasing parameter constraints did not significantly improve goodness of fit.

<sup>a</sup> To calculate within-class variance so that we could compare variance estimates across classes, we set parameter estimates for testosterone to 1.

Table 6  
Summary of Relationships Between Covariates of APED Use and Latent APED Severity

Covariate	$\beta$	SE	p
Years of APED use	.233	.028	<.01
Weight (kg)	.097	.025	<.05
Height (cm)	-.093	.028	<.05
Body fat	.069	.022	<.05
Income	-.043	.018	<.05
% livelihood	.177	.021	<.01

Note. APED = appearance- and performance-enhancing drug; % livelihood = the percentage that an individual's appearance is dependent on strength or appearance.

appearance goals. Probability of Class 2 membership appeared to be a better predictor of willingness to sacrifice years and number of side effects than unwillingness to give up APED use, whereas probability of Class 3 membership was negatively associated with unwillingness to give up APED use but positively associated with intentions for future use. Pairwise post hoc comparisons of regression coefficients indicated that severity had generally the strongest relationship with these risk factors, and, consistent with consequences, probability of Class 4 membership was the worst predictor of APED risk.

Table 7  
Results of Side Effects Regressed on Latent Severity and Probability of Latent Class Membership

Symptom cluster	Severity		Class 1		Class 2		Class 3		Class 4		F <sup>a</sup>	p	R <sup>2</sup>
	$\beta$	SE	$\beta$	SE	$\beta$	SE	$\beta$	SE	$\beta$	SE			
Anabolic													
Dysphoric mood	.335	.065 <sup>b***</sup>	.401	.053 <sup>b***</sup>	.102	.045 <sup>c**</sup>	.112	.065 <sup>**</sup>	-.052	.034	23.95	<.001	.308
Unwanted growth	.125	.035 <sup>**</sup>	.108	.078	.015	.044	.089	.078	-.032	.059	2.59	.036	.026
Sexual side effects	-.067	.069	-.013	.022	.019	.039	.096	.068	-.037	.044	0.84	.500	.008
Hyperarousal	.421	.066 <sup>d***</sup>	.244	.071 <sup>e**</sup>	.211	.075 <sup>c**</sup>	.156	.054 <sup>c**</sup>	-.091	.038 <sup>**</sup>	24.28	<.001	.315
Androgenic side effects	.183	.075 <sup>e**</sup>	.177	.045 <sup>**</sup>	.082	.061	.216	.073 <sup>**</sup>	-.056	.081	16.88	<.001	.217
OTC ergo/thermogenic													
Adrenaline-related side effects	.098	.088	.147	.069 <sup>c,f**</sup>	.230	.045 <sup>c,f**</sup>	.045	.061	.052	.066	15.98	<.001	.191
Sexual side effects	-.080	.074	.094	.077	.114	.078	.009	.091	-.004	.081	1.07	.373	.014
Dysphoric mood	.171	.045 <sup>e***</sup>	.101	.070 <sup>c</sup>	.149	.056 <sup>c**</sup>	.005	.097	-.023	.094	8.02	<.001	.087
Illicit ergo/thermogenic													
Dysphoric mood	.023	.059	.073	.062	.094	.071	-.015	.091	-.022	.055	1.91	.109	.029
Dehydration	.111	.082	-.009	.078	.201	.099 <sup>*</sup>	-.041	.055	-.331	.071 <sup>g**</sup>	9.98	<.001	.191
Sexual dysfunction	.044	.071	.058	.087	.077	.051	-.032	.064	-.004	.029	2.12	.079	.032
Vasoconstriction	.027	.055	.089	.059	.005	.072	-.049	.149	-.134	.112	1.93	.102	.030
Autonomic arousal	-.012	.068	.028	.088	.142	.068 <sup>c,f**</sup>	-.036	.101	-.229	.079 <sup>**</sup>	4.95	<.001	.078

Note. Dependent variables reflect factor scores of side effect subscales. Significance values indicate the probability that a parameter is different than zero. OTC = over-the-counter.

<sup>a</sup> Between-subjects and within-subject degrees of freedom are 5 and 394, respectively, for anabolics; 5 and 299, respectively, for OTC ergo/thermogenics; and 5 and 256, respectively, for illicit ergo/thermogenics. <sup>b</sup> Significantly greater than Classes 2–4. <sup>c</sup> Significantly greater than Class 4. <sup>d</sup> Significantly greater than Classes 1–4. <sup>e</sup> Significantly greater than Classes 3–4. <sup>f</sup> Significantly greater than severity. <sup>g</sup> Significantly greater than severity and Classes 2–4.

\*\*  $p < .01$ .

## Discussion

Three major findings arise from the current study. First, heterogeneity in APED use was explained both by unique patterns of APED use and by APED use severity. The four patterns of APED use were largely consistent with purposeful drug use, whereby APEDs were combined on the basis of desired appearance- and performance-based outcomes. Second, different APEDs were better indicators of latent APED severity than others, and the quality of each indicator varied by latent class. Finally, both latent severity and probability of latent class were related to negative APED outcomes, although differences in these relationships were observed across outcome variables. Most important, probability of membership in Class 1, labeled *heavy polypharmacy*, was associated with the greatest levels of risk and impairment.

The support for an FMA model in the current study lends utility to evidence that hybrid (combined continuous and categorical) models are generally useful for explaining population heterogeneity in substance-abusing populations (Lubke & Muthén, 2005; B. Muthén, 2006; B. Muthén & Asparouhov, 2006). Such models provide greater flexibility and coverage of the variability in different illicit drug use phenomena and are likely to influence further revision of existing diagnostic systems as well as provide better models to explore key aspects of psychopathology within certain populations.

Table 8  
Results of Drug Risk Indicators Regressed on Latent Severity and Probability of Latent Class Membership

Risk indicator	Severity		Class 1		Class 2		Class 3		Class 4		$F(5, 394)$ or $\chi^2(5, N = 400)$	$p$	$R^2$
	$\beta$	$SE$	$\beta$	$SE$	$\beta$	$SE$	$\beta$	$SE$	$\beta$	$SE$			
Anticipated future use	.111	.049**	.140	.068**	.121	.060**	.127	.062**	.102	.053**	6.92	<.001	.091
Unwillingness to stop using with knowledge of severe side effects <sup>a</sup>	.321	.034**	.510	.131**	.011	.092	-.231	.087**	.031	.089	14.30 <sup>b</sup>	.094	.510
No. years willing to sacrifice to achieve training goals	.421	.078 <sup>c**</sup>	.219	.048 <sup>d**</sup>	.149	.067 <sup>e**</sup>	.088	.054	.043	.055	9.55	<.001	.078
No. side effects	.449	.054 <sup>d**</sup>	.301	.071 <sup>e**</sup>	.277	.069 <sup>f**</sup>	.202	.081 <sup>f**</sup>	.152	.074 <sup>f**</sup>	22.90	<.001	.288

<sup>a</sup> Logistic regression results; parameter estimates are exp ( $\beta$ ), and Nagelkerke  $R^2$  reported. <sup>b</sup> Hosmer-Lemeshow chi-square test (8  $df$ ) of goodness of fit. <sup>c</sup> Significantly greater than Classes 1–4. <sup>d</sup> Significantly greater than Classes 2–4. <sup>e</sup> Significantly greater than Classes 3–4. <sup>f</sup> Significantly greater than Class 4.  
\*\*  $p < .01$ .

The current FMA model also has implications for understanding the presence of pathology among APED users. In particular, the results of the current study suggest that a majority of the negative outcomes associated with APEDs occur within the context of heavy polypharmacy, or combining steroid and nonsteroidal anabolics with both illicit and licit ergo/thermogenics. To date, the combination and interactions of these drugs have not been examined, although animal models of combined AAS use (i.e., stacking multiple AASs) have recently been developed (Wesson & McGinnis, 2006). On the basis of the existing data, it is clear that animal models that use both ergo/thermogenics and AASs are likely to have better external validity than simpler AAS-only models and provide a more clinically useful understanding of the mechanisms by which APED use leads to negative outcomes. Thus, if diagnostic items are to be developed for APED use, a key component will be the degree of polypharmacy used.

An advantage of the FMA methodology is the simultaneous measurement of categorical and continuous latent variables. The groups identified in the current FMA model suggest that the pattern of APED use was associated with different training goals and identities. The heavy polypharmacy group, which made up about 10% of the sample, consisted primarily of body builders, who used the high doses for greater duration and also had the greatest amount of self-reported lean muscle mass. The members of the primarily bulking group, which made up about 20% of the sample, identified themselves primarily as power lifters, and their APED use was consistent with their desire to add muscle. In contrast to the bulking group, those defined by their ergo/thermogenic drug use, who made up about 15% of the sample, preferred leanness to muscle mass and had a higher likelihood of being fitness or endurance athletes. Finally, the majority of the sample, about 50%, used low to moderate amounts of AASs for shorter periods of time. They identified themselves as recreational weight lifters, and their pattern reflected less risk taking, with an even split in the desire to add muscle or increase leanness.

An important question that arises out of the application of hybrid modeling is to what degree latent severity versus latent class membership is important for assessing outcomes. For the current study, it appears that latent severity was a good general indicator of negative APED outcomes and risk. However, the addition of latent classes allows for better prediction of certain

types of risk, thus contributing useful clinical and diagnostic information. Most notable are the differences between the cutting drug group (Class 2, primarily interested in decreasing body fat) and the bulking group (Class 3, primarily interested in adding muscle size). Knowledge that an APED user is primarily attempting to reduce body fat and engages in an APED use pattern consistent with this goal by using more ergo/thermogenics and anabolics with a lower androgenic-anabolic ratio, such as stanozolol (Feldkoren & Andersson, 2005; Saartok, Dahlberg, & Gustafsson, 1984; Sundaram, Kumar, Monder, & Bardin, 1995), could help clinicians and researchers more accurately identify the probability of certain types of risks (e.g., cardiac symptoms). Thus, the current study indicates that identifying APED users by their pattern of APED use may be as important to assessing risk as measuring severity of APED use.

Consistent with the relative importance of both severity and latent class, a hybrid model with partial invariance (i.e., different severity parameter estimates within each class) was found to best fit the data. The implications for this finding are that different APEDs are likely to carry different degrees of risk within each subgroup of APED users. For instance, insulin and HGH use were both good indicators of severity of APED use within Classes 1 and 2, but the oral anabolics (e.g., methandrostenolone) were better indicators of severity in Classes 3 and 4. Researchers could extend such information to develop severity cutoffs for each class as well as identify the relative amount of information specific APEDs provide toward discrimination between pathological and non-pathological variants of APED use. Such application of these mathematical models has been suggested with other general systems of psychiatric diagnosis (Kruger et al., 2005).

In addition to examining outcomes, the current study explores the relationship between several covariates and APED use. Generally, findings indicate that APED users were attempting to increase body fat and reduce muscle mass, a goal consistent with achieving a masculine body ideal (Hildebrandt, Langenbucher, & Schlundt, 2004; Pope, Olivardia, Gruber, & Borowiecki, 1999). However, latent class differences suggest that individual patterns were aimed at reducing deficits in the self-ideal discrepancy (e.g., those with lower body fat used more mass-building drugs, and vice versa). Hildebrandt, Schlundt, et al. (2006) reported similar unique body image groups based on self-ideal discrepancies in a commu-

nity sample of weight lifters, suggesting that men may approach fitness as a method of compensation for perceived deficits in appearance. Thus, individual bodily characteristics may dispose individuals to certain patterns of APED use.

In addition, the positive relationship between the degree to which participants' livelihood was related to strength and appearance, on the one hand, and probability of Class 3 membership, on the other, suggests that those who derived income related to the effects of APEDs were more likely to be interested in bulking up or adding muscle. It is interesting that this group was also more willing to discontinue APED use with appropriate evidence that these substances cause harm. Such group attitudes suggest that certain at-risk professions (e.g., police officer, professional athlete) may be more responsive to personalized feedback interventions in which accurate risk assessment and feedback are provided. Unfortunately, long-term follow-up of APED users is essential before this type of feedback can be provided with confidence. Most APED users currently believe their knowledge of these drugs and their effects exceeds that of the medical community (Pope, Kanayama, Ionescu-Pioggia, & Hudson, 2004); thus, accurate feedback is essential to increasing motivation for behavior change.

Overall, side effects were common, with all users reporting at least one. For AASs, the most common side effects were excessive sex drive, acne, water retention, and testicular atrophy. Other descriptive reports of AAS use support these findings, suggesting that such side effects occur among 35%–65% of users (Bolding et al. 2002; Evans, 1997; Parkinson & Evans, 2006; Perry et al., 2005). More serious side effects, such as dysphoric mood (i.e., aggression, anxiety, irritability, and depression), were less common in the current sample (approximately 15%–25%). More impairing side effects, such as infertility, bone growth and/or intestinal growth related to HGH use, and abscesses at the injection site, were not common, occurring in less than 10% of regular users.

Among the side effects reported, dysphoric mood is one of considerable importance to defining pathological variants of APED use. Many of the concerns about the relationship between APED use and mood disturbances can be traced to case reports of body builders using AASs (Corrigan, 1996; Driessen, Muessigbrodt, Dilling, & Driessen, 1996; Pope & Katz, 1994). In fact, recent evidence suggests that AAS users are at greater risk for mortality related to violence or suicide than other substance abusers (Pettersson et al., 2006). However, experimental evidence supporting a direct causal link between AASs and mood is mixed (Bjorkqvist, Nygren, Bjorklund, & Bjorkqvist, 1994; Pope et al., 2000), and it is likely that the interaction of AASs and ergo/thermogenics is responsible for some degree of mood disturbance in APED users. Animal studies have suggested that AASs are likely to affect mood through effects on serotonin receptors in the anterior hypothalamus, ventrolateral hypothalamus, and medial amygdala (Grimes & Melloni, 2002). However, there are likely several neurochemical mechanisms that are responsible for increased aggression, mania, and depression among APED users. In particular, estrogen and dihydrotestosterone (testosterone metabolites) are also believed to effect mood states (Lynch & Story, 2000; McGinnis & Dreifuss, 1989) and are common byproducts of many AASs. Another APED pattern variable of importance is the particular drug or set of drugs being used; different AASs are likely to affect mood differently because of different degrees of androgen receptor binding. For example, stanozolol, an AAS common to the

cutting drug group, has been shown to inhibit aggressive responding in male rats and, accordingly, has low androgen receptor affinity (Farrell & McGinnis, 2003). Such findings may have important implications for predicting individual risk for APED-related mood disturbances.

Despite these important findings, the current study has a number of limitations. Recruitment over the Internet could have led to oversampling of knowledgeable APED users with greater income and more education and might have increased the chances of noise from random responding. There is very little methodological work on the directional biases attributable to Internet research; however, work by McCabe (2004) suggested that rates of illicit drug use via Internet report were comparable to rates as measured by other forms of anonymous self-report in undergraduate men and women. In response to these concerns, the current study relied on a conservative selection approach, using several validity and reliability checks to ensure data quality. Limiting the mixture model analyses to regular APED users might have also excluded participants with unique APED patterns; however, weight-lifting men who have not yet completed enough APED cycles to establish regular use are likely to have unstable APED patterns. Mixture analyses such as those conducted in the current study are also affected by the quality of measurement. Although the instrument used had strong evidence of content validity and internal consistency (see Appendixes B, C, and D), the accuracy of APED user self-report is currently unknown. The current study is also cross-sectional, which prevents any inferences about how APED patterns relate to side effects or risk over time and does not allow for inferences about causality. Finally, the adjustment of alpha levels might have limited the statistical power for some analyses. Given the exploratory nature of the study, however, controlling for Type I error was of greater concern.

The results of the current study raise many important questions for future research. Perhaps the most important determination is whether this model has any predictive validity. There are no current longitudinal studies of APED use, and many of the relationships (e.g., heavy polypharmacy and dysphoric mood) found in the current study should be evaluated in terms of long-term clinical outcomes. In addition, the current study provides evidence that diagnostic items may be generated on the basis of pattern as well as severity of APED use. Such applications may involve testing how well classifying users on the basis of the four groups maps onto long-term outcome or relates to psychiatric comorbidity. These investigations will likely yield more informed investigations of etiology, risk, and development of appropriate clinical prevention and intervention efforts.

## References

- Akaike, H. (1987). Factor analysis and AIC. *Psychometrika*, *52*, 317–332.
- Babor, T. F., Hofmann, M., Del Boca, F. K., Hesselbrock, V., Meyer, R. E., Dolinsky, Z. S., & Rounsaville, B. (1992). Types of alcoholics: I. Evidence for an empirically derived typology based on indicators of vulnerability and severity. *Archives of General Psychiatry*, *49*, 599–608.
- Bahrke, M. S., & Yesalis, C. E. (2002). *Performance enhancing substances in sport and exercise*. Champaign, IL: Human Kinetics.
- Ball, S. A., Carroll, K. M., Babor, T. F., & Rounsaville, B. J. (1995). Subtypes of cocaine abusers: Support for a Type A–Type B distinction. *Journal of Consulting and Clinical Psychology*, *63*, 115–124.



- Basaria, S., Wahlstrom, J. T., & Dobs, A. S. (2001). Clinical review 138: Anabolic-androgenic steroid therapy in the treatment of chronic disease. *Journal of Clinical Endocrinology and Metabolism*, *86*, 5108–5117.
- Bhasin, S., Storer, T. W., Berman, N., Callgari, C., Clevenger, B., Phillips, J., et al. (1996). The effects of supraphysiological doses of testosterone on muscle size and strength in normal men. *New England Journal of Medicine*, *335*, 1–7.
- Bjorkqvist, K., Nygren, T., Bjorklund, A.-C., & Bjorkqvist, S.-E. (1994). Testosterone intake and aggressiveness: Real effect or anticipation. *Aggressive Behavior*, *20*, 17–26.
- Bolding, G., Sherr, L., & Elford, J. (2002). Use of anabolic steroids and associated health risks among gay men attending London gyms. *Addiction*, *97*, 195–203.
- Bowlby, D. A., & Rapaport, R. (2004). Safety and efficacy of growth hormone in childhood. *Pediatric Endocrinology Review*, *2*(Suppl. 1), 68–77.
- Byrne, M. W., Shavelson, R. J., & Muthén, B. (1989). Testing for equivalence of factor covariance and mean structures: The issue of partial measurement invariance. *Psychological Bulletin*, *105*, 456–466.
- Cafri, G., Thompson, J. K., Ricciardelli, L., McCabe, M., Smolak, L., & Yesalis, C. (2005). Pursuit of the muscular ideal: Physical and psychological consequences and putative risk factors. *Clinical Psychology Review*, *25*, 215–239.
- Cloniger, C. R., Bohman, M., & Sigvardsson, S. (1981). Inheritance of alcohol abuse: Cross-fostering analysis of adopted men. *Archives of General Psychiatry*, *38*, 861–868.
- Copeland, J., Peters, R., & Dillon, P. (2000). Anabolic-androgenic steroid use disorders among a sample of Australian competitive and recreational users. *Drug and Alcohol Dependence*, *60*, 91–96.
- Corrigan, B. (1996). Anabolic steroids and the mind. *Medical Journal of Australia*, *165*, 222–226.
- Dills, D. G. (2001). New aspects of insulin therapy in Type 1 and Type 2 diabetes. *Endocrinology and Metabolism Clinics of North America*, *30*, 935–982.
- Driessen, M., Muessigbrodt, H., Dilling, H., & Driessen, B. (1996). Child sexual abuse associated with anabolic androgenic steroid use. *American Journal of Psychiatry*, *153*, 1369.
- Escobar-Morreale, H. F., Botella-Carretero, J. I., Escobar del Rey, F., & Morreale de Escobar, G. (2005). Treatment of hypothyroidism with combinations of levothyroxine plus liothyronine. *Journal of Clinical Endocrinology and Metabolism*, *90*, 4946–4954.
- Evans, N. A. (1997). Gym and tonic: A profile of 100 male steroid users. *British Journal of Sports Medicine*, *31*, 54–58.
- Evans, N. A. (2004). Current concepts in anabolic-androgenic steroids. *American Journal of Sports Medicine*, *32*, 534–542.
- Farrell, S. F., & McGinnis, M. Y. (2003). Effects of pubertal anabolic-androgenic steroid (AAS) administration exposure on reproductive and aggressive behavior in male rats. *Behavioral Neuroscience*, *117*, 904–911.
- Feldkoren, B. I., & Andersson, S. (2005). Anabolic-androgenic steroid interaction with rat androgen receptor in vivo and in vitro: A comparative study. *Journal of Steroid Biochemistry and Molecular Biology*, *94*, 481–487.
- Friedl, K. E. (2000). Effect of anabolic steroid use on body composition and physical performance. In C. E. Yesalis (Ed.), *Anabolic steroids in sport and exercise* (2nd ed., pp. 139–165). Champaign, IL: Human Kinetics.
- Godfrey, R. J., Madgwick, Z., & Whyte, G. P. (2003). The exercise-induced growth hormone response in athletes. *Sports Medicine*, *33*, 599–613.
- Gosling, S. D., Vazire, S., Srivastava, S., & John, O. P. (2004). Should we trust Web-based studies? A comparative analysis of six preconceptions about Internet questionnaires. *American Psychologist*, *59*, 93–104.
- Grace, F., Sculthorpe, N., Baker, J., & Davies, B. (2003). Blood pressure and rate pressure product response in males using high-dose anabolic androgenic steroids (AAS). *Journal of Science, Medicine, and Sport*, *6*, 307–312.
- Green, G. A., Uryasz, F. D., Petr, T. A., & Bray, C. D. (2001). NCAA study of substance use and abuse habits of college student-athletes. *Clinical Journal of Sport Medicine*, *11*, 51–56.
- Grimes, J. M., & Melloni, R. H., Jr. (2002). Serotonin modulates aggressive attack in anabolic steroid-treated hamsters. *Pharmacology, Biochemistry, and Behavior*, *73*, 713–721.
- Hall, R. C. W., Hall, R. C. W., & Chapman, M. J. (2005). Psychiatric complications of anabolic steroid abuse. *Psychosomatics*, *46*, 285–290.
- Haller, C. A., & Benowitz, N. A. (2000). Adverse cardiovascular and central nervous system events associated with dietary supplements containing ephedra alkaloids. *New England Journal of Medicine*, *343*, 1833–1838.
- Hartgens, F., & Kuipers, H. (2004). Effects of androgenic-anabolic steroids in athletes. *Sports Medicine*, *34*, 513–554.
- Hartgens, F., Rietjens, G., Keizer, H. A., Kuipers, H., & Wolffenbuttel, B. H. (2004). Effects of androgenic-anabolic steroids on apolipoproteins and lipoprotein (a). *Sports Medicine*, *38*, 253–259.
- Hildebrandt, T., Langenbucher, J., Carr, S., Sanjuan, P., & Park, S. (2006). Predicting intentions for long-term anabolic-androgenic steroid use among males: A covariance structure model. *Psychology of Addictive Behaviors*, *20*, 234–240.
- Hildebrandt, T., Langenbucher, J., & Schlundt, D. G. (2004). Muscularity concerns among men: Development of attitudinal and perceptual measures. *Body Image*, *1*, 169–181.
- Hildebrandt, T., Schlundt, D. G., Langenbucher, J., & Chung, T. (2006). Presence of muscle dysmorphia symptomatology among male weightlifters. *Comprehensive Psychiatry*, *47*, 127–135.
- Johnson, E. O., van der Bree, M. B., & Picken, R. W. (1996). Subtypes of alcohol-dependent men: A typology based on relative genetic and environmental loading. *Alcoholism, Clinical and Experimental Research*, *20*, 1472–1480.
- Kanayama, G., Gruber, A. J., Pope, H. G., Jr., Borowiecki, J. J., & Hudson, J. I. (2001). Over-the-counter drug use in gymnasiums: An underrecognized substance abuse problem? *Psychotherapy and Psychosomatics*, *70*, 137–140.
- Kersey, R. (1993). Anabolic-androgenic steroid use by private health club/gym athletes. *Journal of Strength and Conditioning Research*, *7*, 118–126.
- Kouri, E. M., Pope, H. G., Jr., Katz, D. L., & Oliva, P. S. (1995). Fat-free mass index in users and nonusers of anabolic-androgenic steroids. *Clinical Journal of Sport Medicine*, *5*, 223–228.
- Kruger, R. F., Markon, K. E., Patrick, C. J., & Iacono, W. G. (2005). Externalizing psychopathology in adulthood: A dimensional-spectrum conceptualization and its implications for DSM-IV. *Journal of Abnormal Psychology*, *114*, 537–550.
- Kruger, R. F., Nichol, P. E., Hicks, B. M., Markon, K. E., Patrick, C. J., Iacono, W. G., & McGue, M. (2004). Using latent trait modeling to conceptualize an alcohol problems continuum. *Psychological Assessment*, *16*, 107–119.
- Kuder, G., & Richardson, M. (1937). The theory of estimation of test reliability. *Psychometrika*, *2*, 151–160.
- Langenbucher, J. W., Hildebrandt, T., & Carr, S. (in press). Effects of steroidal and nonsteroidal anabolics and ergogenic drugs. In J. Brick (Ed.), *Handbook of medical consequences of drug abuse*. Binghamton, New York: Hawthorn Press.
- Lindstrom, M., Nilsson, A., Katzman, P., Janzon, L., & Dymling, J. (1990). Use of anabolic-androgenic steroids among body builders—Frequency and attitudes. *Journal of Internal Medicine*, *227*, 407–411.
- Llewellyn, W. (2005). *Anabolics 2005*. Jupiter, FL: Body of Science.
- Lo, Y., Mendell, N., & Rubin, D. B. (2001). Testing the number of components in a normal mixture. *Biometrika*, *88*, 767–778.

- Lubke, G. H., & Muthén, B. (2005). Investigation population heterogeneity with factor mixture models. *Psychological Methods, 10*, 21–39.
- Lynch, C. S., & Story, A. J. (2000). Dihydrotestosterone and estrogen regulation of androgen-receptor immunoreactivity. *Physiology and Behavior, 69*, 445–453.
- McCabe, S. E. (2004). Comparison of web and mail surveys in collecting illicit drug use data: A randomized experiment. *Journal of Drug Education, 34*, 61–72.
- McGinnis, M. Y., & Dreifuss, R. M. (1989). Evidence for the role of testosterone androgen receptor interactions in mediating masculine behavior in rats. *Endocrinology, 124*, 618–626.
- Muthén, B. (2006). Should substance abuse disorders be considered categorical or dimensional? *Addiction, 101*(Suppl. 1), 6–16.
- Muthén, B., & Asparouhov, T. (2006). Item response mixture modeling: Application to tobacco dependence criteria. *Addictive Behavior, 31*, 1050–1066.
- Muthén, L. K., & Muthén, B. (2004). *Mplus user's guide*. Los Angeles: Muthén & Muthén.
- National Heart Lung and Blood Institute. (1998). *Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: The evidence report* (Rep. No. 98–4083). Bethesda, MD: National Institutes of Health.
- National Institute on Drug Abuse. (2000). About anabolic steroid abuse. *NIDA Notes, 15*(3).
- Orr, R., & Fiatarone Singh, M. (2004). The anabolic androgenic steroid oxandralone in the treatment of wasting and catabolic disorders: Review of efficacy and safety. *Drugs, 64*, 725–750.
- Pagonis, P., Angelopoulos, N., Koukoulis, G. N., & Hadjichristodoulou, C. S. (2006). Psychiatric side effects induced by supraphysiological doses of combinations of anabolic steroids correlate to the severity of abuse. *European Psychiatry, 21*, 551–562.
- Parkinson, A. B., & Evans, N. A. (2006). Anabolic androgenic steroids: A survey in 500 users. *Medicine, Science, Sports and Exercise, 38*, 644–651.
- Pärssinen, M., & Seppälä, T. (2002). Steroid use and long-term health risks in former athletes. *Sports Medicine, 32*, 83–94.
- Perry, P. J., Kutscher, E. C., Lund, B. C., Yates, W. R., Holman, T. L., & Demers, L. (2003). Measures of aggression and mood changes in male weightlifters with and without androgenic anabolic steroid use. *Journal of Forensic Science, 48*, 646–651.
- Perry, P. J., Lund, B. C., Deninger, M. J., Kutscher, E. C., & Schneider, J. (2005). Anabolic steroid use in weightlifters and bodybuilders: An internet survey of drug utilization. *Clinical Journal of Sport Medicine, 15*, 326–330.
- Petersson, A., Garle, M., Holmgren, P., Druid, H., Krantz, P., & Thiblin, I. (2006). Toxicological findings and manner of death in autopsied users of anabolic androgenic steroids. *Drug and Alcohol Dependence, 81*, 241–249.
- Pope, H. G., Jr., Kanayama, G., Ionescu-Pioggia, M., & Hudson, J. I. (2004). Anabolic steroid users' attitudes towards physicians. *Addiction, 99*, 1189–1194.
- Pope, H. G., Jr., & Katz, D. L. (1994). Psychiatric and medical effects of anabolic-androgenic steroid use. A controlled study of 160 athletes. *Archives of General Psychiatry, 51*, 375–382.
- Pope, H. G., Jr., Kouri, E. M., & Hudson, J. I. (2000). Effects of supra-physiologic doses of testosterone on mood and aggression in normal men: A randomized controlled trial. *Archives of General Psychiatry, 57*, 133–140.
- Pope, H. G., Jr., Olivardia, R., Gruber, A., & Borowiecki, J. (1999). Evolving ideals of male body image as seen through action toys. *International Journal of Eating Disorders, 26*, 65–72.
- Saartok, T., Dahlberg, E., & Gustafsson, J. A. (1984). Relative binding affinity of anabolic-androgenic steroids: Comparison of the binding to the androgen receptors in skeletal muscle and in prostate, as well as to sex hormone-binding globulin. *Endocrinology, 114*, 2100–2106.
- Sachtleben, T. R., Berg, K. E., Elias, B. A., Cheatham, J. P., Felix, G. L., & Hofschire, P. J. (1993). The effects of anabolic steroids on myocardial structure and cardiovascular fitness. *Medicine and Science in Sports and Exercise, 25*, 1240–1245.
- Schwartz, G. (1978). Estimating the dimension of a model. *Annals of Statistics, 6*, 461–464.
- Shahidi, N. T. (2001). A review of the chemistry, biological action, and clinical applications of anabolic-androgenic steroids. *Clinical Therapeutics, 23*, 1355–1390.
- Shekelle, P. G., Hardy, M. L., Morton, S. C., Maglione, M., Mojica, W. A., Sutton, M. J., et al. (2003). Efficacy and safety of ephedra and ephedrine for weight loss and athletic performance: A meta-analysis. *Journal of the American Medical Association, 289*, 1537–1545.
- Soe, K. L., Soe, M., & Gluud, C. (1992). Liver pathology associated with the use of anabolic-androgenic steroids. *Liver, 12*, 73–79.
- Sundaram, K., Kumar, N., Monder, C., & Bardin, C. W. (1995). Different patterns of metabolism determine the relative anabolic activity of 19-norandrogens. *Journal of Steroid Biochemistry and Molecular Biology, 53*, 253–257.
- Torres-Calleja, J., Gonzales-Unzaga, M., DeCelis-Carrollo, R., Calzada-Sanchez, L., & Pedron, N. (2001). Effect of androgenic anabolic steroids on sperm quality and serum hormone levels in adult male bodybuilders. *Life Sciences, 68*, 1769–1774.
- Tricker, R., O'Neill, M. R., & Cook, D. (1989). The incidence of anabolic steroid use among competitive bodybuilders. *Journal of Drug Education, 19*, 313–325.
- Vermunt, J. K., & Magidson, J. (2002). Latent class cluster analysis. In J. A. Hagenaars & A. L. McCutcheon (Eds.), *Applied latent class analysis* (pp. 89–106). Cambridge, England: Cambridge University Press.
- Wagman, D., Curry, L., & Cook, D. (1995). An investigation into anabolic androgenic steroid use by elite U.S. powerlifters. *Journal of Strength and Conditioning Research, 9*, 149–154.
- Walters, E. H., Walters, J. A., & Gibson, M. D. (2003). Inhaled long acting beta agonists for stable chronic asthma. *Cochrane Database Systematic Database Reviews, 3*, CD001385.
- Wesson, D. W., & McGinnis, M. Y. (2006). Stacking anabolic androgenic steroids (AAS) during puberty in rats: A neuroendocrine and behavioral assessment. *Pharmacology, Biochemistry, and Behavior, 83*, 410–419.
- Woodhouse, L. J., Reisz-Porszasz, S., Javanbakht, M., Storer, T. W., Lee, M., Zerounian, H., et al. (2003). Development of models to predict anabolic response to testosterone administration in healthy young men. *American Journal of Physiology, Endocrinology, and Metabolism, 284*, E1009–E1017.
- Yesalis, C. E., Herrick, R., Buckley, W., Friedl, K., Brannon, D., & Wright, J. (1988). Self-reported use of anabolic-androgenic steroids by elite power lifters. *Physician and Sportsmedicine, 16*, 91–100.

Appendix A

Self-Reported Side Effects of Anabolic Appearance- and Performance-Enhancing Drugs (APEDs)

Anabolic APEDs			OTC ergo/thermogenics			Illicit ergo/thermogenics		
Side effect	Frequency	%	Side effect	Frequency	%	Side effect	Frequency	%
Abscess	33	8.3	Anxiety	98	32.1	Anxiety	52	19.8
Acne	231	57.8	Dehydration	91	29.8	Dehydration	72	27.5
Aggressiveness	132	33.0	Erectile dysfunction	36	11.8	Erectile dysfunction	8	3.1
Anxiety	84	21.0	Digestive trouble	23	5.8	Diarrhea	17	16.5
Bone growth	15	3.8	Excessive sweating	123	40.3	Excessive sweating	128	48.9
Depression	83	20.8	Easily frustrated	42	13.8	Easily frustrated	25	9.5
Sleep difficulty	189	47.3	Headaches	85	27.9	Headaches	86	32.8
Erectile dysfunction	91	22.8	Heart palpitations	83	27.2	Heart palpitations	58	22.1
Excessive sex drive	241	60.3	Heart skips beats	26	8.5	Heart skips beats	13	5.0
Flulike symptoms	95	23.8	Inability to orgasm	15	4.9	Inability to orgasm	2	0.8
Gynecomastia	86	21.5	Increased BP	64	21.0	Increased BP	45	17.2
Hair loss	75	18.8	Irritability	64	21.0	Irritability	40	15.3
Increased BP	162	40.5	Prostate trouble	15	4.9	Prostate trouble	4	1.5
Increased LFT	48	12.0	Nosebleeds	8	2.0	Nosebleeds	5	1.9
Infertility	10	2.5	Tremor (shakiness)	114	28.5	Tremor (shakiness)	125	47.7
Intestinal growth	5	1.3	Sleep disturbance	171	42.8	Sleep disturbance	126	48.1
Irritability	100	25.0				Cannot stop moving	31	11.8
Loss of sex drive	107	26.8				Stomach pain	14	5.3
Muscle cramps or spasms	90	22.5				Heat intolerance	47	17.9
Testicular shrinkage	223	55.8				Nausea	19	7.3
Too much energy	48	12.0				Loss of appetite	42	16.0
Water retention	239	59.8						

Note. For anabolic APEDs,  $n = 400$ ; for over-the-counter (OTC) ergo/thermogenics,  $n = 305$ ; for illicit ergo/thermogenics,  $n = 262$ . BP = blood pressure; LFT = liver function test.

Appendix B

Exploratory Factor Analyses for Self-Reported Side Effects of Anabolic Appearance- and Performance-Enhancing Drugs

Side effect	Factor				
	1	2	3	4	5
Abscess <sup>a</sup>	0.467	0.129	-0.048	-0.094	-0.088
Acne <sup>b</sup>	0.284	0.111	-0.017	-0.107	0.287
Aggressiveness	<b>0.538 (0.583)</b>	-0.116	0.107	0.128	0.205
Anxiety	<b>0.713 (0.672)</b>	0.041	-0.091	0.068	0.115
Bone growth	0.076	<b>0.690 (0.545)</b>	0.020	0.020	0.089
Depression	<b>0.739 (0.871)</b>	-0.247	0.178	-0.113	0.058
Sleep difficulty	0.207	0.012	-0.093	<b>0.287 (0.457)</b>	0.404
Erectile dysfunction	0.073	0.044	<b>0.912 (1.064)</b>	-0.030	-0.055
Excessive sex drive	0.088	-0.008	0.028	<b>0.466 (0.612)</b>	0.198
Flulike symptoms	<b>0.432 (0.453)</b>	-0.017	-0.032	0.037	0.150
Gynecomastia	0.076	<b>0.576 (0.464)</b>	0.183	-0.165	0.085
Hair loss	-0.068	0.215	0.372	-0.018	<b>0.237 (0.430)</b>
Increased blood pressure	-0.147	0.080	-0.070	0.046	<b>0.703 (0.681)</b>
Increased liver function tests <sup>b</sup>	-0.049	0.247	0.005	0.095	0.310
Infertility	0.096	0.0198	<b>0.297 (0.457)</b>	-0.088	-0.075
Intestinal growth	-0.034	<b>1.011 (1.383)</b>	-0.022	0.368	-0.079
Irritability	<b>0.903 (0.919)</b>	0.089	0.072	0.109	-0.210
Loss of sex drive	0.089	0.022	<b>0.818 (0.643)</b>	0.015	-0.069
Muscle cramps or spasms <sup>b</sup>	0.146	0.091	0.152	0.350	0.057
Testicular shrinkage	-0.147	-0.219	0.191	-0.093	<b>0.638 (0.676)</b>
Too much energy	0.027	0.211	-0.056	<b>0.720 (0.589)</b>	-0.086
Water retention	0.188	0.087	-0.171	-0.191	<b>0.670 (0.624)</b>
KR-20	0.88	0.80	0.79	0.77	0.75

Note. All loadings are expressed in regression coefficients. Final model regression coefficients are in parentheses. KR-20 = Kuder Richardson-20. Boldface type indicates |factor loading| > .4.

<sup>a</sup> Item was removed because it is a side effect of administration method and not drug action. <sup>b</sup> Item was removed because of overloading on multiple factors or insufficient loadings.

(Appendixes continue)

Appendix C  
*Exploratory Factor Analyses of Self-Reported Side Effects of  
 Over-the-Counter Ergo/Thermogenic Drugs*

Side effect	Factor		
	1	2	3
Anxiety <sup>a</sup>	0.346	-0.027	0.396
Dehydration	<b>0.666</b> <b>(0.465)</b>	-0.047	0.193
Erectile dysfunction	0.013	<b>0.897</b> <b>(0.876)</b>	-0.133
Digestive trouble	<b>0.616</b> <b>(0.530)</b>	0.181	0.101
Excessive sweating	<b>0.641</b> <b>(0.541)</b>	-0.111	0.253
Easily frustrated	0.149	-0.118	<b>0.856</b> <b>(0.909)</b>
Headaches	0.185	0.101	<b>0.444</b> <b>(0.534)</b>
Heart palpitations	<b>0.705</b> <b>(0.909)</b>	0.019	-0.175
Heart skips beats	1.032 (0.704)	-0.033	0.053
Inability to orgasm	0.073	<b>0.978</b> <b>(0.872)</b>	0.001
Increased BP	0.065	<b>0.497</b> <b>(0.534)</b>	0.203
Irritability	-0.032	0.122	<b>0.807</b> <b>(0.831)</b>
Prostate trouble	0.164	<b>0.727</b> <b>(0.763)</b>	-0.025
Tremor	<b>0.622</b> <b>(0.612)</b>	-0.019	0.027
KR-20	.86	.79	.78

*Note.* All loadings are expressed in regression coefficients. Final model regression coefficients are in parentheses. BP = blood pressure; KR-20 = Kuder Richardson-20. Boldface type indicates |factor loading| > .4.

<sup>a</sup> Item was removed because of overloading on multiple factors.



## Appendix D

*Exploratory Factor Analyses of Self-Reported Side Effects of Illicit Fat-Burning Drugs*

Side effect	Factor				
	1	2	3	4	5
Anxiety	<b>0.475 (0.565)</b>	0.266	0.137	0.246	0.084
Dehydration	0.035	<b>0.731 (0.770)</b>	0.098	0.008	0.210
Diarrhea	-0.027	<b>0.597 (0.789)</b>	0.121	0.115	0.027
Erectile dysfunction	-0.029	0.057	<b>0.782 (1.010)</b>	-0.135	0.209
Sleep disturbance	0.136	<b>0.607 (0.614)</b>	0.135	0.186	0.292
Loss of appetite	0.219	<b>0.454 (0.521)</b>	0.254	0.097	0.277
Excessive sweating	-0.079	0.146	-0.121	0.205	<b>0.883 (0.556)</b>
Cannot stop moving	0.232	0.208	0.139	0.123	<b>0.489 (0.453)</b>
Easily frustrated	<b>0.567 (0.651)</b>	0.290	0.149	0.191	0.147
Headaches	0.055	<b>0.558 (0.600)</b>	0.273	0.120	0.209
Heart palpitations	-0.127	0.078	0.086	<b>0.903 (0.682)</b>	0.202
Heart skipping beats	0.188	0.242	0.186	0.817 (0.904)	0.148
Heat intolerance	0.187	-0.263	0.204	0.132	<b>0.594 (0.631)</b>
Inability to orgasm	0.057	0.127	<b>0.724 (0.704)</b>	0.288	0.211
Increased BP	0.023	0.122	0.222	<b>0.449 (0.463)</b>	0.243
Irritability	<b>0.516 (0.775)</b>	0.048	0.101	0.129	-0.228
Nausea	-0.047	0.238	<b>0.776 (0.656)</b>	-0.147	-0.189
Nosebleeds	-0.155	-0.026	0.287	<b>0.423 (0.753)</b>	0.073
Prostate trouble	0.289	0.142	<b>0.606 (0.653)</b>	0.180	0.083
Stomach pain <sup>a</sup>	-0.204	0.447	0.671	-0.195	-0.332
Tremor	0.201	<b>0.533 (0.595)</b>	-0.248	-0.124	0.018
KR-20	.87	.84	.81	.75	.72

*Note.* All loadings are expressed in regression coefficients. Final model regression coefficients are in parentheses. BP = blood pressure; KR-20 = Kuder Richardson-20. Boldface type indicates |factor loading| > .4.

<sup>a</sup> Item was removed because of overloading on multiple factors or insufficient loadings.

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