responsible for training NCUA employees in the obligations imposed by the Privacy Act and this subpart.

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DEPARTMENT OF JUSTICE

Drug Enforcement Administration

21 CFR Part 1300

[Docket No. DEA–285P]

RIN 1117–AB17

Classification of Three Steroids as Schedule III Anabolic Steroids Under the Controlled Substances Act

AGENCY: Drug Enforcement Administration (DEA), Department of Justice.

ACTION: Notice of proposed rulemaking.

SUMMARY: This Notice of Proposed Rulemaking (NPRM) proposes to classify the following three steroids as ''anabolic steroids'' under the Controlled Substances Act (CSA): boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione. The Drug Enforcement Administration (DEA) believes that this action is necessary in order to prevent the abuse and trafficking of these steroids. If the regulations are amended, these steroids will be listed as schedule III controlled substances subject to the regulatory control provisions of the CSA.

DATES: Written comments must be postmarked, and electronic comments must be sent on or before June 24, 2008.

ADDRESSES: To ensure proper handling of comments, please reference ''Docket No. DEA–285'' on all written and electronic correspondence. Written comments via regular mail should be sent to the Deputy Administrator, Drug Enforcement Administration, Washington, DC 20537, Attention: DEA Federal Register Representative/ODL. Written comments sent via express mail should be sent to DEA Headquarters, Attention: DEA Federal Register Representative/ODL, 8701 Morrissette Drive, Springfield, VA 22152. Comments may be sent directly to DEA electronically by sending an electronic message to *dea.diversion.policy@usdoj.gov.* Comments may also be sent electronically through *http://www.regulations.gov* using the electronic comment form provided on that site. An electronic copy of this document is also available at the *http://www.regulations.gov* Web site. DEA will accept attachments to electronic comments in Microsoft Word, WordPerfect, Adobe PDF, or Excel file formats. DEA will not accept any file format other than those specifically listed here.

Posting of Public Comments: Please note that all comments received are considered part of the public record and made available for public inspection online at *http://www.regulations.gov* and in the Drug Enforcement Administration's public docket. Such information includes personal identifying information (such as your name, address, etc.) voluntarily submitted by the commenter.

If you want to submit personal identifying information (such as your name, address, etc.) as part of your comment, but do not want it to be posted online or made available in the public docket, you must include the phrase ''PERSONAL IDENTIFYING INFORMATION'' in the first paragraph of your comment. You must also place all the personal identifying information you do not want posted online or made available in the public docket in the first paragraph of your comment and identify what information you want redacted.

If you want to submit confidential business information as part of your comment, but do not want it to be posted online or made available in the public docket, you must include the phrase ''CONFIDENTIAL BUSINESS INFORMATION'' in the first paragraph of your comment. You must also prominently identify confidential business information to be redacted within the comment. If a comment has so much confidential business information that it cannot be effectively redacted, all or part of that comment may not be posted online or made available in the public docket.

Personal identifying information and confidential business information identified and located as set forth above will be redacted and the comment, in redacted form, will be posted online and placed in the Drug Enforcement Administration's public docket file. If you wish to inspect the agency's public docket file in person, by appointment, please see the **FOR FURTHER INFORMATION CONTACT** paragraph.

FOR FURTHER INFORMATION CONTACT: Christine A. Sannerud, PhD, Chief, Drug and Chemical Evaluation Section, Office of Diversion Control, Drug Enforcement Administration, Washington, DC 20537 at (202) 307–7183.

SUPPLEMENTARY INFORMATION:

I. Background Information

On November 29, 1990, the President signed into law the Anabolic Steroids Control Act of 1990 (Title XIX of Pub. L. 101–647), which became effective February 27, 1991. This law established and regulated anabolic steroids as a class of drugs under schedule III of the Controlled Substances Act (CSA). As a result, a new anabolic steroid is not scheduled according to the procedures set out in 21 U.S.C. 811, but can be administratively classified as an anabolic steroid through the rulemaking process by adding the steroid to the regulatory definition of an anabolic steroid in 21 CFR 1300.01(b)(4).

On October 22, 2004, the President signed into law the Anabolic Steroid Control Act of 2004 (Pub. L. 108–358), which became effective on January 20, 2005. Section 2(a) of the Anabolic Steroid Control Act of 2004 amended 21 U.S.C. 802(41)(A) by replacing the existing definition of ''anabolic steroid.'' The Anabolic Steroid Control Act of 2004 classifies a drug or hormonal substance as an anabolic steroid if the following four criteria are met: (A) The substance is chemically related to testosterone; (B) the substance is pharmacologically related to testosterone; (C) the substance is not an estrogen, progestin, or a corticosteroid; and (D) the substance is not dehydroepiandrosterone (DHEA). Any substance that meets the criteria is considered an anabolic steroid and must be listed as a schedule III controlled substance. DEA believes that boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione meet this definition of anabolic steroid and is proposing that they be added to the list of anabolic steroids in 21 CFR 1300.01(b)(4).

Anabolic steroids are a class of drugs with a basic steroid ring structure that produces anabolic and androgenic effects. The prototypical anabolic steroid is testosterone. Anabolic effects include promoting the growth of muscle. The androgenic effects consist of promoting the development of male secondary sexual characteristics such as facial hair, deepening of the voice, and thickening of the skin.

In the United States, only a small number of anabolic steroids are approved for either human or veterinary use. Approved medical uses for anabolic steroids include treatment of androgen deficiency in hypogonadal males, adjunctive therapy to offset protein catabolism associated with prolonged administration of corticosteroids, treatment of delayed puberty in boys, treatment of metastatic breast cancer in

women, and treatment of anemia associated with specific diseases (*e.g.*, anemia of chronic renal failure, Fanconi's anemia, and acquired aplastic anemia). However, with the exception of the treatment of male hypogonadism, anabolic steroids are not the first-line treatment due to the availability of other preferred treatment options. DEA is not aware of any legitimate medical use or New Drug Applications (NDA) for the three substances that DEA is proposing to classify by this NPRM as anabolic steroids under the definition set forth under 21 U.S.C. 802(41)(A). Moreover, DEA has not been able to identify any chemical manufacturers currently using these substances as intermediates in their manufacturing process(es).

Adverse effects are associated with the use or abuse of anabolic steroids. These effects depend on several factors (*e.g.*, age, sex, anabolic steroid used, the amount used, and the duration of use). In early adolescents, the use of testosterone and other anabolic steroids that have estrogenic effects can cause premature closure of the growth plates in long bones resulting in a permanently stunted growth. In adolescent boys, anabolic steroid use can cause precocious sexual development. In both girls and women, anabolic steroid use induces permanent physical changes such as deepening of the voice, increased facial and body hair growth, and the lengthening of the clitoris. In men, anabolic steroid use can cause shrinkage of the testicles, decreased sperm count, and sterility. Gynecomastia (*i.e.*, enlargement of the male breast tissue) can develop with the use of those anabolic steroids with estrogenic actions. In both men and women, anabolic steroid use can damage the liver and can cause high cholesterol levels, which may increase the risk of strokes and heart attacks. Furthermore, anabolic steroid use is purported to induce psychological effects such as aggression, increased feelings of hostility, and psychological dependence and addiction. Upon abrupt termination of long-term anabolic steroid use, a withdrawal syndrome may appear including severe depression.

II. Evaluation of Statutory Factors for Classification as an Anabolic Steroid

DEA is proposing by this NPRM to classify boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione as anabolic steroids under the definition set forth under 21 U.S.C. 802(41)(A). As noted previously, a drug or hormonal substance is classified as an anabolic steroid by meeting the following four definitional requirements: (A) The substance is chemically related to testosterone; (B) the substance is pharmacologically related to testosterone; (C) the substance is not an estrogen, progestin, or a corticosteroid; and (D) the substance is not DHEA.

A. Chemically Related to Testosterone

To classify a substance as an anabolic steroid, a substance must be chemically related to testosterone. A Structure Activity Relationship (SAR) evaluation for each of the substances compared the chemical structure of the steroid to that of testosterone, as substances with a structure similar to that of testosterone are predicted to possess comparable pharmacological and biological activity.

Boldione is also known by the following chemical name: androsta-1,4-diene-3,17-dione. DEA has determined that the chemical structure of boldione is chemically related to that of testosterone. The chemical structure of boldione differs from testosterone by only the following two chemical groups: A ketone group at carbon 17 and a double bond between the first and second carbon. The human body would be expected to metabolize the ketone group at carbon 17 into a hydroxyl group that is present on testosterone. Furthermore, the scientific literature reports that the additional double bond at carbon 1 in boldione does not significantly decrease the anabolic activity of the substance (Vida, 1969). Boldione is an anabolic steroid precursor, being metabolized by the body into boldenone (Galletti and Gardi, 1971), which is a schedule III anabolic steroid (21 U.S.C. 801(41)(A)(vi)).

Desoxymethyltestosterone (DMT) is also known by the following names: 17α-methyl-5a-androst-2-en-17β-ol; and madol. DEA has determined that the chemical structure of desoxymethyltestosterone is chemically related to testosterone. The chemical structure of desoxymethyltestosterone differs from testosterone by the following four chemical features: The lack of a ketone group at the third carbon, a double bond between the second and third carbon, the lack of a double bond between the fourth and fifth carbon, and a methyl group at carbon 17. Each of these four chemical features is known through the scientific literature not to eliminate the anabolic and androgenic activity of the substance (Brueggemeir *et al.*, 2002; Vida, 1969).

19-Nor-4,9(10)-androstadienedione is also known by the following chemical names: 19-norandrosta 4,9(10)-diene-3,17-dione; and estra-4,9(10)-diene-3,17-dione. DEA has determined that the chemical structure of 19-nor-4,9(10)-androstadienedione is chemically related to testosterone. The chemical structure of 19-nor-4,9(10)-androstadienedione differs from testosterone by the following three chemical groups: A ketone group at carbon 17, the absence of a methyl group at carbon 19, and a double-bond between the ninth and tenth carbon. The human body metabolizes the ketone group at carbon 17 into a hydroxyl group that is present on testosterone. Furthermore, the scientific literature reports that both the absence of the methyl group at carbon 19 and the additional double bond in 19-nor-4,9(10)-androstadienedione increase the anabolic activity of the substance (Vida, 1969).

B. Pharmacologically Related to Testosterone

A substance must also be pharmacologically related to testosterone (*i.e.*, produce similar biological effects) to be classified as a schedule III anabolic steroid. The pharmacology of a steroid, as related to testosterone, can be established by performing one or more of the following androgenic and anabolic activity assays: ventral prostate assay, seminal vesicle assay, levator ani assay, testicular atrophy assay, gonadotropin suppression assay, and androgen receptor binding and efficacy assays. These assays are described below.

Ventral Prostate Assay, Seminal Vesicle Assay, and Levator Ani Assay: The classic scientific procedure for examining the effects of a steroid as compared to testosterone is to perform the ventral prostate assay, seminal vesicles assay, and levator ani assay. Certain male accessory organs (*i.e.*, the ventral prostate, seminal vesicles, and levator ani muscle) specifically need testosterone to grow and remain healthy. Upon the removal of the testes (*i.e.*, castration), the primary endogenous source of testosterone is eliminated causing the atrophy of the ventral prostate, seminal vesicles, and levator ani muscle (Eisenberg *et al.*, 1949; Nelson *et al.*, 1940; Scow, 1952; Wainman and Shipoundoff, 1941). Numerous scientific studies have demonstrated the ability of exogenous testosterone administered to rats following castration to maintain the normal weight and size of all three testosterone sensitive organs (Biskind and Meyer, 1941; Dorfman and Dorfman, 1963; Kincl and Dorfman, 1964; Nelson *et al.*, 1940; Scow, 1952; Wainman and Shipoundoff, 1941). Thus, a steroid with testosterone-like activity will also prevent the atrophy of these three testosterone-dependent organs in castrated rats.

Testicular Atrophy Assay: Administering testosterone to non-castrated rats causes a decrease in serum levels of gonadotropins (*i.e.*, luteinizing hormone [LH] and follicle stimulating hormone [FSH]) from normal levels. Gonadotropins are pituitary hormones that affect the size and function of the testes. The suppression of these gonadotropins by excess testosterone results in a significant decrease in the size and weight of the testes (Boris *et al.*, 1970; McEuen *et al.*, 1937; Moore and Price, 1938). Accordingly, a steroid with testosterone-like activity will also significantly diminish the size and weight of the testes.

Gonadotropin Suppression Assay: The castration of rats causes a substantial increase in the serum levels of gonadotropins (*i.e.*, LH and FSH) above normal levels due to the removal of the principal source of endogenous testosterone (Gay and Bogdanove, 1969; Swerdloff *et al.*, 1972, 1973; Swerdloff and Walsh, 1973). The administration of testosterone to castrated animals suppresses the increase in the serum levels of gonadotropins (Gay and Bogdanove, 1969; Swerdloff *et al.*, 1972; Swerdloff and Walsh, 1973; Verjans *et al.*, 1974). The administration of anabolic steroids with testosterone-like activity will also prevent this increase in serum levels of LH and FSH.

Androgen Receptor Binding and Efficacy Assay: Androgen receptor binding and efficacy assays are also used to demonstrate that the activity of a steroid is similar to that of testosterone. Testosterone produces its anabolic effects subsequent to binding to and activating the androgen receptor. Different cell-based assays can compare candidate steroids to testosterone for their ability to bind to and activate androgen receptors.

There are several different types of assays used to establish androgen receptor binding and efficacy. In one assay, C3H10T1/2 stem cells express androgen receptors and are used to assess steroids for their ability to bind and activate the androgen receptor (Jasuja *et al.*, 2005a,b; Singh *et al.*, 2003). In these stem cells, the translocation of the androgen receptor to the nucleus of the cell in the presence of the ligand (*e.g.*, testosterone or its active metabolite dihydroxytestosterone) confirms that the ligand bound to the androgen receptor and activated the downstream signaling cascade. When activated, the C3H10T1/2 stem cells differentiate into skeletal muscle cells as demonstrated by the increase in the expression of muscle specific proteins (*i.e.*, myogenic determination transcription factor [MyoD] and myosin heavy chain [MHC]). Another assay uses human breast cancer cells genetically altered to contain a specific reporter gene (*e.g.*, luciferase gene) regulated by androgen receptor activation (Hartig *et al.*, 2002; Wilson *et al.*, 2002). The expression of a bioluminescent protein (*e.g.*, luciferase) signals both androgen receptor binding and activation.

Results of the Androgenic and Anabolic Activity Assays

In January 2006, DEA reviewed the published scientific literature for pharmacological data on the anabolic and androgenic activity of boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione using the assays described above. As discussed further below, there was sufficient information on the pharmacology of desoxymethyltestosterone in the reviewed scientific literature to determine that desoxymethyltestosterone is pharmacologically related to testosterone (*i.e.*, produces biological effects similar to those of testosterone). However, the published literature contained insufficient pharmacological data to determine whether boldione and 19-nor-4,9(10)-androstadienedione were pharmacologically related to testosterone. Consequently, as discussed further below, DEA sponsored pharmacological studies involving several different androgenic and anabolic activity assays to generate the data necessary to make this determination.

Androgenic and anabolic activity assay results indicate that boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione have similar pharmacological activity as testosterone.

Boldione

DEA sponsored a study [1] by the Veteran's Administration Puget Sound Health Care System to determine the anabolic and androgenic effects of boldione in intact and castrated rats (Matsumoto and Marck, 2006). The results of these studies were compared to the results of a study by the same laboratory using a similar protocol to characterize the androgenic and anabolic effects of testosterone (Marck *et al.*, 2003). Boldione administered to castrated male rats by silastic capsules implanted under the skin prevented atrophy of the ventral prostate, seminal vesicle, and levator ani, and the rise in serum gonadotropin (LH and FSH) associated with castration. Boldione administration also produced testicular atrophy in intact rats. Another DEA sponsored study [2] at a laboratory at Boston University examined the ability of boldione to bind to the androgen receptor and to cause the differentiation of C3H10T1/2 stem cells into muscle cells (Bhasin, 2005). All of these effects caused by boldione in C3H10T1/2 stem cells were comparable to those of testosterone as established in experiments using the same or similar methodology (Singh *et al.*, 2003). Collectively, the evidence indicates that the pharmacology of boldione is similar to testosterone.

Desoxymethyltestosterone

Desoxymethyltestosterone was administered subcutaneously, orally, or intramuscularly to castrated rats (Dorfman and Kincl, 1963; Kincl and Dorfman, 1964; Nutting *et al.*, 1966). By all three routes of administration, desoxymethyltestosterone prevented the atrophy of ventral prostate, seminal vesicle, and levator ani. Desoxymethyltestosterone also induced the expression of the bioluminescent protein luciferase in CAMA–1 breast cancer cells signaling androgen receptor binding and activation (Ayotte *et al.*, 2006). Collectively, the evidence indicates that the pharmacology of desoxymethyltestosterone is similar to testosterone.

19-Nor-4,9(10)-Androstadienedione

DEA sponsored a study [3] by the Veteran's Administration Puget Sound Health Care System to determine the anabolic and androgenic effects of 19-nor-4,9(10)-androstadienedione in intact and castrated rats (Matsumoto and Marck, 2006). The results of these studies were compared to the results of a study by the same laboratory using a similar protocol to characterize the androgenic and anabolic effects of testosterone (Marck *et al.*, 2003). 19-nor-4,9(10)-androstadienedione administered to castrated male rats by silastic capsules implanted under the skin prevented the atrophy of the ventral prostate, seminal vesicle, levator ani, and the rise in serum gonadotropins (LH and FSH) associated castration. Another DEA sponsored study at a laboratory at Boston University [4]

[1] The study by the Veteran's Administration Puget Sound Health Care System may be found at *www.regulations.gov* in the electronic docket associated with this rulemaking.

[2] The study by Boston University may be found at *www.regulations.gov* in the electronic docket associated with this rulemaking.

[3] The study by the Veteran's Administration Puget Sound Health Care System may be found at *www.regulations.gov* in the electronic docket associated with this rulemaking.

[4] The study by Boston University may be found at *www.regulations.gov* in the electronic docket associated with this rulemaking.

examined the ability of 19-nor-4,9(10)-androstadienedione to bind to the androgen receptor and to cause the differentiation of C3H10T1/2 stem cells into muscle cells (Bhasin, 2005). All of these effects caused by 19-nor-4,9(10)-androstadienedione in C3H10T1/2 stem cells were comparable to those of testosterone as established in experiments using the same or similar methodology (Singh *et al.*, 2003). Collectively, the evidence indicates that the pharmacology of 19-nor-4,9(10)-androstadienedione is similar to testosterone.

C. Not Estrogens, Progestins, and Corticosteroids

DEA has determined that boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione are unrelated to estrogens, progestins, and corticosteroids. DEA evaluated the SAR for each of the substances. The chemical structure of each substance was compared to that of estrogens, progestins, and corticosteroids because the chemical structure can be related to its pharmacological and biological activity. DEA found that the three substances lacked the necessary chemical structures to impart significant estrogenic activity (*e.g.*, aromatic A ring) (Duax *et al.*, 1988; Jordan *et al.*, 1985; Williams and Stancel, 1996), progestational activity (*e.g.*, 17β-alkyl group) (Williams and Stancel, 1996), or corticosteroidal activity (*e.g.*, 17β-ketone group or 11β-hydroxyl group) (Miller *et al.*, 2002).

D. Not Dehydroepiandrosterone

Dehydroepiandrosterone, also known as DHEA, is exempt from control as an anabolic steroid by definition (21 U.S.C. 802(41)(A)). Boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione are not dehydroepiandrosterone and are therefore not exempted from control on this basis.

III. Conclusion

Therefore, based on the above, DEA concludes that boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione meet the CSA definition of ''anabolic steroid'' because each substance is: (A) Chemically related to testosterone; (B) pharmacologically related to testosterone; (C) not an estrogen, progestin, or a corticosteroid; and (D) not DHEA (21 U.S.C. 802(41)). All anabolic steroids are classified as schedule III controlled substances (21 U.S.C. 812). Once a substance is determined to be an anabolic steroid, DEA has no discretion regarding the scheduling of these substances. As discussed further below, all requirements pertaining to controlled substances in schedule III would pertain to these three substances.

IV. Impact of Proposed Rule

Effect of Classifying These Substances as Anabolic Steroids

If this rulemaking is finalized as proposed, DEA will classify boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione as schedule III anabolic steroids. If classified as schedule III anabolic steroids, any person who manufactures, distributes, dispenses, imports, or exports boldione, desoxymethyltestosterone, or 19-nor-4,9(10)-androstadienedione, or who engages in research or conducts instructional activities with respect to these three substances would be required to obtain a schedule III registration in accordance with the CSA and its implementing regulations. Manufacturers and importers of these three substances would be required to register with DEA and would be permitted to distribute these substances only to other DEA registrants. Only persons registered as dispensers would be allowed to dispense these three substances to end users. The CSA defines a practitioner as ''a physician, dentist, veterinarian, scientific investigator, pharmacy, hospital, or other person licensed, registered, or otherwise permitted, by the United States or the jurisdiction in which he practices or does research, to distribute, dispense, conduct research with respect to, administer, or use in teaching or chemical analysis, a controlled substance in the course of professional practice or research'' (21 U.S.C. 802(21)). At present, there are no approved medical uses for these three substances. Until a manufacturer applies to the Food and Drug Administration and gains approval for products containing these substances, no person may dispense them in response to a prescription.

Manufacture, import, export, distribution, or sale of boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione, except by DEA registrants, would become a violation of the CSA that may result in imprisonment and fines (21 U.S.C. 841 and 960). Possession of these three steroids, unless legally obtained, would also become subject to criminal penalties (21 U.S.C. 844).

In addition, under the CSA, these three substances could be imported only for medical, scientific, or other legitimate uses (21 U.S.C. 952(b)) under an import declaration filed with DEA (21 CFR 1312.18). Importation of these substances would be illegal unless the person importing these substances is registered with DEA as an importer or researcher and files the required declaration for each shipment. An individual who purchases any of these substances directly from foreign companies and has them shipped to the U.S. will be considered to be importing even if the steroids are intended for personal use. Illegal importation of these substances would be a violation of the CSA that may result in imprisonment and fines (21 U.S.C. 960).

Requirements for Handling Substances Defined as Anabolic Steroids

Upon consideration of public comments from this NPRM, DEA may issue a final rule classifying boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione as anabolic steroids. If classified as anabolic steroids, boldione, desoxymethyltestosterone, and 19-nor-4,9(10)-androstadienedione would become subject to CSA regulatory controls and administrative, civil, and criminal sanctions applicable to the manufacture, distribution, dispensing, importation, and exportation of a schedule III controlled substance, including the following:

Registration. Any person who manufactures, distributes, dispenses, imports, exports, or engages in research or conducts instructional activities with a substance defined as an anabolic steroid, or who desires to engage in such activities, would be required to be registered to conduct such activities with schedule III controlled substances in accordance with 21 CFR part 1301.

Security. Substances defined as anabolic steroids would be subject to schedule III–V security requirements and would be required to be manufactured, distributed, and stored in accordance with 21 CFR 1301.71, 1301.72(b), (c), and (d), 1301.73, 1301.74, 1301.75(b) and (c), 1301.76 and 1301.77.

Labeling and Packaging. All labels and labeling for commercial containers of substances defined as anabolic steroids would be required to comply with requirements of 21 CFR 302.03–1302.07.

Inventory. Every registrant required to keep records and who possesses any quantity of any substance defined as an anabolic steroid is required to keep an inventory of all stocks of the substances on hand pursuant to 21 CFR 1304.03, 1304.04 and 1304.11. Every registrant who desires registration in schedule III for any substance defined as an anabolic

steroid shall conduct an inventory of all stocks of the substances on hand at the time of registration.

Records. All registrants would be required to keep records pursuant to 21 CFR 1304.03, 1304.04, 1304.05, 1304.21, 1304.22, 1304.23 and 1304.26.

Prescriptions. All prescriptions for these schedule III substances or for products containing these schedule III substances would be required to be issued pursuant to 21 CFR 1306.03–1306.06 and §§ 1306.21–1306.27. All prescriptions for these schedule III compounds or for products containing these schedule III substances, if authorized for refilling, would be limited to five refills within six months of the date of issuance of the prescription.

Importation and Exportation. All importation and exportation of any substance defined as an anabolic steroid would be required to be in compliance with 21 CFR part 1312.

Criminal Liability. Any activity with any substance defined as an anabolic steroid not authorized by, or in violation of, the Controlled Substances Act or the Controlled Substances Import and Export Act is unlawful.

Disposal of Anabolic Steroids

If this regulation is finalized as proposed, persons who possess substances that become classified as anabolic steroids and who wish to dispose of them rather than becoming registered to handle them should contact their local DEA Diversion field office for assistance in disposing of these substances legally. DEA Diversion field office will provide the person with instructions regarding the disposal. A list of local DEA Diversion field offices may be found at *http://www.deadiversion.usdoj.gov.*

Regulatory Certifications

Regulatory Flexibility Act

The Deputy Administrator hereby certifies that this rulemaking has been drafted in accordance with the Regulatory Flexibility Act (5 U.S.C. 601–612). DEA is not able to determine whether this regulation will, if promulgated as a Final Rule, not have a significant economic impact on a substantial number of small entities. As of August 2007, DEA identified 22 dietary supplements promoted for building muscle and increasing strength that are purported to contain boldione, desoxymethyltestosterone, or 19-nor-4,9(10)-androstadienedione. Four dietary supplements purport to contain boldione; nine dietary supplements purport to contain desoxymethyltestosterone; and nine dietary supplements purport to contain 19-nor-4,9(10)-androstadienedione. All 22 dietary supplements are marketed and sold on the Internet.

The manufacturers and distributors of the 22 identified dietary supplements purported to contain boldione, desoxymethyltestosterone, or 19-nor-4,9(10)-androstadienedione also sell a variety of other dietary supplements. DEA has identified a substantial number of Internet distributors that sell these dietary supplements. However, these distributors also sell a variety of other nutritional products. Without information on the percentage of revenues derived from these dietary supplements, however, DEA is not able to determine the economic impact of the removal of these dietary supplements alone on the business of the firms. DEA has not been able to identify any chemical manufacturers that are currently using these substances as intermediates in their manufacturing process(es). DEA seeks comment on whether this regulation, if promulgated as a Final Rule, will have a significant economic impact on a substantial number of small entities.

As of August 2007, DEA identified 20 chemical manufacturers and distributors that sell at least one of the three substances addressed in this NPRM. Most of the companies are located in China and sell a variety of steroids. DEA notes that, as the vast majority of entities handling these substances are Internet based, it is virtually impossible to accurately quantify the number of persons handling these substances at any given time. Further, DEA has no information regarding the percentage of revenue these substances constitute for each handler.

DEA has identified one company based in the U.S. that is a DEA registrant that manufactures and distributes at least one of these substances as reference products for testing laboratories. DEA notes, upon placement into schedule III, these substances may be used for analytical purposes. This company is registered with DEA and is already in compliance with the CSA and DEA implementing regulations regarding the handling of schedule III substances.

Executive Order 12866

The Deputy Administrator hereby certifies that this rulemaking has been drafted in accordance with Executive Order 12866 section 1(b). It has been determined that this rule is a significant regulatory action. Therefore, this action has been reviewed by the Office of Management and Budget.

As discussed above, the effect of this rule would be to remove products containing these substances from the over-the-counter marketplace. DEA has no basis for estimating the size of the market for these products. DEA notes, however, that virtually all of the substances are imported. According to U.S. International Trade Commission data, the import value of all anabolic steroids in 2006 was $6 million. These three substances would be a subset of those imports. The value of anabolic steroid imports for the first six months of 2007 declined by 35 percent although the quantity imported increased. The total market for these products containing these substances, therefore, is probably quite small. Moreover, DEA believes that the importation of these three substances is for illegitimate purposes.

The benefit of controlling these substances is to remove from the marketplace substances that have dangerous side effects and no legitimate medical use in treatment in the United States. As discussed in detail above, these substances can produce serious health effects in adolescents and adults. If medical uses for these substances are developed and approved, the drugs would be available as schedule III controlled substances in response to a prescription issued by a medical professional for a legitimate medical purpose. Until that time, however, this action would bar the importation, exportation, and sale of these three substances except for legitimate research or industrial uses.

Executive Order 12988

This regulation meets the applicable standards set forth in Sections 3(a) and 3(b)(2) of Executive Order 12988 Civil Justice Reform.

Executive Order 13132

This rulemaking does not preempt or modify any provision of state law; nor does it impose enforcement responsibilities on any state; nor does it diminish the power of any state to enforce its own laws. Accordingly, this rulemaking does not have federalism implications warranting the application of Executive Order 13132.

Paperwork Reduction Act

This rule proposes to regulate three anabolic steroids, which are neither approved for medical use in humans nor approved for administration to cattle or other non-humans. Under this proposal, only chemical manufacturers who may use these substances as chemical intermediates for the synthesis of other steroids would be required to register

with DEA under the CSA. However, DEA has not been able to identify any chemical manufacturers that are currently using these substances as intermediates in their manufacturing process(es). Therefore, DEA is specifically seeking input from the chemical industry on any manufacturing process(es) that maybe impacted by this rulemaking. Thus, DEA does not expect this proposal to impose any additional paperwork burden on the regulated industry.

Unfunded Mandates Reform Act of 1995

This rule will not result in the expenditure by state, local, and tribal governments, in the aggregate or by the private sector, of $120,000,000 or more (adjusted for inflation) in any one year and will not significantly or uniquely affect small governments. Therefore, no actions were deemed necessary under the provisions of the Unfunded Mandates Reform Act of 1995.

Congressional Review Act

This rule is not a major rule as defined by Section 804 of the Small Business Regulatory Enforcement Fairness Act of 1996 (Congressional Review Act). This rule will not result in an annual effect on the economy of $100,000,000 or more; a major increase in cost or prices; or significant adverse effects on competition, employment, investment, productivity, innovation, or on the ability of United States-based companies to compete with foreign-based companies in domestic and export markets.

List of Subjects in 21 CFR Part 1300

Chemicals, Drug traffic control.

For the reasons set out above, 21 CFR part 1300 is proposed to be amended as follows:

PART 1300—DEFINITIONS

1. The authority citation for part 1300 continues to read as follows:

Authority: 21 U.S.C. 802, 871(b), 951, 958(f).

2. Section 1300.01 is amended in paragraph (b)(4) by:

A. Redesignating paragraphs (b)(4)(xiii) through (b)(4)(lx) as (b)(4)(xiv) through (b)(4)(lxi),

B. Adding a new paragraph (b)(4)(xiii),

C. Redesignating new paragraphs (b)(4)(xvii) through (b)(4)(lxi) as (b)(4)(xviii) through (b)(4)(lxii),

D. Adding new paragraph (b)(4)(xvii),

E. Redesignating new paragraphs (b)(4)(xlvii) through (b)(4)(lxii) as (b)(4)(xlviii) through (b)(4)(lxiii), and

F. Adding new paragraph (b)(4)(xlvii) to read as follows:

§ 1300.01 Definitions relating to controlled substances.

* * * * *

(b) * * *

(4) * * *

(xiii) boldione (androsta-1,4-diene-3,17-dione)

* * * * *

(xvii) desoxymethyltestosterone (17a-methyl-5a-androst-2-en-17-ol) (a.k.a., madol)

* * * * *

(xlvii) 19-nor-4,9(10)-androstadienedione (estra-4,9(10)-diene-3,17-dione)

* * * * *

Dated: April 11, 2008.

Michele M. Leonhart,

Deputy Administrator.

List of References

Ayotte, C., Goudreault, D., Gauthier, J., Ayotte, P., Larochelle, C., and Poirier, D. (2006). Characterization of chemical and hormonal properties of new steroid related to doping of athletes. Presented at the Cologne Workshop on Dope Analysis, June 2006.

Bhasin, S. (2005). [Pharmacological analysis of boldione and 19-nor-4,9(10)-androstadienedione for androgenic activity using C3H10T1/2 stem cells]. Unpublished report.

Biskind, G.R. and Meyer, M.A. (1941). The comparative androgenic potency of testosterone, methyltestosterone and testosterone propionate administered in pellet form. *Endocrinology,* 28(2): 217–221.

Boris, A., Stevenson, R.H., and Trmal, T. (1970). Comparative androgenic, myotrophic and antigonadotrophic properties of some anabolic steroids. *Steroids,* 15(1):61–71.

Brueggemeier, R.W., Miller, D.D., and Dalton, J.T. (2002). *Estrogen, Progestins and Androgens.* In D.A. Williams and T.L. Lemke *(Eds.) Foye's Principle of Medicinal Chemistry* (5th ed.). Philadelphia, Lippincott Williams and Wilkins.

Dorfman, R.I. and Dorfman, A.S. (1963). The assay of subcutaneously injected androgens in the castrated rat. *ACTA Endocrinologica,* 42: 245–253.

Dorfman, R.I. and Kincl, F.A. (1963). Relative potency of various steroids in an anabolic-androgenic assay using the castrated rat. *Endocrinology,* 72: 259–266.

Duax, W.L., Griffin, J.F., Weeks, C.M., and Wawrzak, Z. (1988). The mechanism of action of steroid antagonists: Insight from crystallographic studies. *Journal of Steroid Biochemistry and Molecular Biology*, 31: 481–492.

Eisenberg E, Gordan GS and Elliott HW (1949). Testosterone and tissue respiration of the castrate male rat with possible test for myotrophic activity. *Endocrinology,* 45(2): 113–119.

Galletti, F. and Gardi, R. (1971). Metabolism of 1-dehydroandrostanes in man. *Steroids*, 18(1): 39–50.

Gay, V.L. and Bogdanove, E.M. (1969). *Plasma and pituitary* LH and FSH in the castrated rat following short-term steroid treatment. *Endocrinology,* 84: 1132–1142.

Hartig, P.C., Bobseine, K.L., Britt, B.H., Cardon, M.C., Lambright, C.R., Wilson, V.S., and Gray, L.E. (2002). Development of two androgen receptor assays using adenoviral transduction of MMTV-Luc reporter and/or hAR for endocrine screening. *Toxicological Sciences, 66:* 82–90.

Jasuja, R., Catlin, D.H., Miller, A., Chang, Y.-C., Herbst, K.L., Starcevic, B., Artaza, J.N., Singh, R., Datta, G., Sarkissian, A., Chandsawangbhuwana, C., Baker, M., and Bhasin, S. (2005a). Tetrahydrogestrinone is an androgenic steroid that stimulates androgen receptor-mediated, myogenic differentiation in C3H10T1/2 multipotent mesenchymal cells and promotes muscle accretion in orchidectomized male rats. *Endocrinology, 146* (10): 4472–4478.

Jasuja, R., Ramaraj, P., Mac, R.P., Singh, A.B., Storer, T.W., Artaza, J., Miller, A., Singh, R., Taylor, W.E., Lee, M.L., Davidson, T., Sinha-Hikim, I., Gonzalez-Cadavid, N.F., and Bhasin, S. (2005b). (s-4-Androstene-3,17-dione binds androgen receptor, promotes myogenesis *in vitro,* and increases serum testosterone levels, fat-free mass, and muscle strength in hypogonadal men. *Journal of Clinical Endocrinology and Metabolism, 90(2):* 855–863.

Jordan, V.C., Mittal, S., Gosden, B., Koch, R., and Lieberman, M.E. (1985). Structure-activity relationships of estrogen. *Environmental Health Perspectives, 61:* 91–110.

Kincl, F.A. and Dorfman, R.I. (1964). Anabolic-androgenic potency of various steroids in a castrated rat assay. *Steroids, 3:* 109–122.

Marck, B.T., Wolden-Hanson, T., Tolliver, J.M., Matsumoto, A.M. (2003). Use of DEXA to assess the anabolic actions of androgens on relative lean body mass and bone mineral density in orchidectomized prepubertal rats. Unpublished manuscript, Veteran's Affairs Puget Sound Health Care System, Seattle, WA.

Matsumoto, A.M. and Marck, B.T. (2006). DEA Agreement No. DEA–04–P0007 Final Report [Analysis of the androgenic and anabolic activities of 1,4-androstadien-3,17-dione and 19-nor-4,9(10)-androstadienedione in male Sprague Dawley rats]. Unpublished report.

McEuen, C.S., Selye, H., and Collip, J.B. (1937). Effects of testosterone on somatic growth. *Proceedings of the Society for Experimental Biology and Medicine,* 36: 390–394.

Miller, D.D., Brueggemeier, R.W., and Dalton, J.T. (2002). Adrenocorticoids. In D.A. Williams and T.L. Lemke (Eds.) *Foye's Principle of Medicinal Chemistry* (5th ed.). Philadelphia, Lippincott Williams and Wilkins.

Moore, C.R. and Price, D. (1938). Some effects of testosterone and testosterone-propionate in the rat. *The Anatominal Record,* 71(1):59–78.

Nelson, D., Greene, R.R. and Wells, J.A. (1940). Variations in the effectiveness of percutaneously applied androgens in the rat. *Endocrinology,* 26: 651–655.

Nutting, E.F., Klimstra, P.D., and Counsell, R.E. (1966). Anabolic-androgenic activity of A-ring modified androstane derivatives. Part I: A comparison of parenteral activity. *ACTA Endocrinologica, 53:* 627–634.

Scow, R.O. (1952). Effect of testosterone on muscle and other tissues and on carcass composition in hypophysectomized, thyroidectomized, and gonadectomized male rats. *Endocrinology, 51:* 42–51.

Singh, R., Artaza, J.N., Taylor, W.E., Gonzalez-Cadavid, N.F., and Bhasin, S. (2003). Androgens stimulate myogenic differentiation and inhibit adipogenesis in C3H 10T1/2 pluripotent cells through an androgen receptor-mediated pathway. *Endocrinology, 144*(11): 5081–5088.

Swerdloff, R.S., Grover, P.K., Jacobs, H.S., and Bain, J. (1973). Search for a substance which selectively inhibits FSH—Effects of steroids and prostaglandins on serum FSH and LH levels. *Steroids,* 21(5): 703–722.

Swerdloff, R.S. and Walsh, P.C. (1973). Testosterone and oestradiol suppression of LH and FSH in adult male rats: Duration of castration, duration of treatment and combined treatment. ACTA Endocrinologica, 73: 11–21.

Swerdloff, R.S., Walsh, P.C., and Odell, W.D. (1972). Control of LH and FSH secretion in the male: Evidence that aromatization of androgens to estradiol is not required for inhibition of gonadotropin secretion. *Steroids,* 20(1): 13–22.

Verjans, H.L., Eik-Nes, K.B., Aafjes, J.H., Vels, F.J.M., and van der Molen, H.J. (1974). Effects of testosterone propionate, 5alpha-dihydrotestosterone propionate and oestradiol benzoate on serum levels of LH and FSH in the castrated adult male rat. *ACTA Endocrinologica, 77: 643–654.*

Vida, J.A. (1969). *Androgens and Anabolic Agents: Chemistry and Pharmacology.* New York: Academic Press.

Wainman, P. and Shipounoff, G.C. (1941). The effects of castration and testosterone propionate on the striated perineal musculature in the rat. *Endocrinology,* 29(6): 975–978.

Williams, C.L. and Stancel, G.M (1996). Estrogens and Progestins. In J.G. Hardman, L.E. Limbird, P.B. Molinoff, R.W. Ruddon, A. Goodman Gilman (Eds.) *Goodman and Gilman's The Pharmacological Basis of Therapeutics* (9th ed.). New York: McGraw-Hill, 1411–1440.

Wilson, V.S., Bobseine, K., Lambright, C.R., and Gray, L.E. (2002). A novel cell line, MDA-kb2, that stably expresses an androgen- and glucocorticoid-responsive reporter for the detection of hormone receptor agonists and antagonists. *Toxicological Sciences,* 66: 69–81.

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DEPARTMENT OF THE TREASURY

Internal Revenue Service

26 CFR Part 1

[REG–104946–07]

RIN 1545–BG36

Hybrid Retirement Plans; Correction

AGENCY: Internal Revenue Service (IRS), Treasury.

ACTION: Correction to notice of proposed rulemaking.

SUMMARY: This document contains corrections to a notice of proposed rulemaking (REG–104946–07) that was published in the **Federal Register** on Friday, December 28, 2007 (72 FR 73680) providing guidance relating to sections 411(a)(13) and 411(b)(5) of the Internal Revenue Code concerning certain hybrid defined benefit plans.

FOR FURTHER INFORMATION CONTACT: Lauson C. Green or Linda S. F. Marshall at (202) 622–6090 (not a toll-free number).

SUPPLEMENTARY INFORMATION:

Background

The correction notice that is the subject of this document is under section 411 of the Internal Revenue Code.

Need for Correction

As published, the notice of proposed rulemaking (REG–104946–07) contains errors that may prove to be misleading and are in need of clarification.

Correction of Publication

Accordingly, the publication of the notice of proposed rulemaking (REG–104946–07), which was the subject of FR Doc. E7–25025, is corrected as follows:

1. On page 73683, column 3, in the preamble, first paragraph of the column, line 15, the language ''reasonably expected to result in a larger'' is corrected to read ''reasonably expected to result in a smaller''.

2. On page 73685, column 1, third paragraph of the column, line 8, the language '' 'capital' rule of section 411(b)(5)(b)(i)(II)'' is corrected to read '' 'capital' rule of section 411(b)(5)(B)(i)(II)''.

3. On page 73689, column 2, line 3 from the bottom of the fifth paragraph of the column, the language ''section 411(d)(6) is available for the'' is corrected to read ''section 411(d)(6) relief is available for the''.

PART 1—[CORRECTED]

§ 1.411(a)(13)–1 [Corrected]

4. On page 73691, column 1, § 1.411(a)(13)–1(d)(3)(ii), line 18, the language ''larger annual benefit at normal'' is corrected to read ''smaller annual benefit at normal''.

5. On page 73691, column 2, § 1.411(a)(13)–1(d)(3)(iii)(B), line 9, the language ''reasonably expected to result in a larger'' is corrected to read ''reasonably expected to result in a smaller''.

§ 1.411(b)(5)–1 [Corrected]

6. On page 73693, column 3, § 1.411(b)(5)–1(c)(3)(ii)(A), line 17, the language ''participant under the lump sum-based'' is corrected to read ''participant under the lump sum-based benefit''.

7. On page 73695, column 1, § 1.411(b)(5)–1(c)(5) *Example 1.* (ii), line 17, the language ''permitted to elect (with spousal consent)'' is corrected to read ''permitted to elect (with spousal consent if applicable)''.

8. On page 73695, column 2, § 1.411(b)(5)–1(c)(5) *Example 2.* (iii), line 5, the language ''consent) payment in the same generalized'' is corrected to read ''consent if applicable) payment in the same generalized''.

9. On page 73695, column 3, § 1.411(b)(5)–1(c)(5) *Example 2.* (v), line 12, the language ''of 5.5 percent. Thereafter, Participant's A's'' is corrected to read ''of 5.5 percent. Thereafter, Participant A's''.

LaNita Van Dyke,

Chief, Publications and Regulations Branch, Legal Processing Division, Associate Chief Counsel (Procedure and Administration).

[FR Doc. E8–9026 Filed 4–24–08; 8:45 am]

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DEPARTMENT OF THE TREASURY

Internal Revenue Service

26 CFR Part 20

[REG–112196–07]

RIN 1545–BH64

Gross Estate; Election to Value on Alternate Valuation Date

AGENCY: Internal Revenue Service (IRS), Treasury.

ACTION: Notice of proposed rulemaking.

SUMMARY: This document contains proposed regulations that provide guidance relating to the availability of the election to use the alternate valuation method under section 2032 of