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# Counterfeiting in performance- and image-enhancing drugs

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The current drastic escalation in obesity may be contributing to the exponential rise in drugs used for image enhancement. Drugs such as anabolic-androgenic steroids (AAS) are perceived as a viable method of achieving a perfect physique. They are also the most widely abused drugs in sport. The Internet has encouraged the abuse of expensive drugs, particularly human growth hormone (hGH), resulting in increased importation for personal use. The substantial increase in this market has opened up avenues for counterfeiting, estimated as a multi-million pound business. The acute adverse effects from contaminated vials may result in a variety of pathologies including communicable diseases. In 2007, in the UK, a series of intramuscular abscesses, requiring surgical treatment, led us to study samples obtained from the underground market.

The analysis of 38 parenteral samples and 19 oral samples of tablets was performed by a World Anti-Doping Agency (WADA) accredited laboratory, in an attempt to establish the extent of available counterfeit products. Fifty-three per cent (20) of the injectable AAS esters and 21% (4) of the oral tablets were counterfeit. Culture and sensitivity revealed the presence of skin commensal organisms, which may have contributed to the development of the abscesses. Users of AAS and hGH for sport, including bodybuilding, are currently risking their health because of counterfeit and poorly controlled products. Copyright © 2009 John Wiley & Sons, Ltd.

Keywords: AAS; abscess; cosmesis; image; performance

# Introduction

Yearly reports from the World Anti-Doping Agency (WADA) demonstrate that anabolic-androgenic steroids (AAS) are the most abused group of drugs in competitive sport, despite enormous advances in detection techniques and severe penalties for positive test results.

Several countries, including Australia, Argentina, Brazil, Canada, the UK and the US have legislation to control AAS. Nevertheless, AAS are readily obtainable from many sources worldwide. Many countries permit the sale of AAS without a medical prescription [1,2] and sale of these products to overseas customers is also not restricted. This sale is made easier because of the Internet and email. Most AAS found in Europe originate not only from countries within the European Union and Russia but also from Thailand, Turkey, Egypt, India and Pakistan. [2] In the US, significant quantities of AAS emanate from Mexico, as well as from Russia, Romania, and Greece.[1] In the UK, AAS and recombinant human growth hormone (rhGH) are controlled under Schedule 4, Part 2 of the Misuse of Drugs Act 1971. There is an exemption from restriction on the possession of these substances, when they are in a medicinal product and are for self-administration. As a consequence of this legal control, AAS can only be obtained in the UK for non-medical use from sources of unknown provenance, such as the black market or the Internet. These products are not necessarily produced in accordance with Good Manufacturing Practice, as would be the case with licensed medicines obtained from legitimate sources. Of particular concern is the use of anabolic steroids as performanceand image-enhancing drugs by female athletes; this has led to an assessment of the type of drugs they use. [3]

A recent doping violation, contravening the WADA code, has illustrated the excessive "polypharmacy" in the athletic world, which is believed to be the tip of the iceberg.<sup>[4]</sup> A letter sent to *The Times* newspaper identified the doping regime of Mr Dwain Chambers, a disqualified international athlete, who admitted taking combinations of prohibited substances.<sup>[4,5]</sup>

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The use of performance-enhancing drugs is contrary to all Olympic values and creates negative role models for young athletes. Peer influence and the information that AAS administered by intra-muscular (IM) injection are 'less harmful' than oral products has increased the use of IM preparations. The escalation in the frequency of IM abscesses in a cohort of bodybuilders in 2007, seen by one of us (MRG), was considered to be as a result of the use of non-sterilized or contaminated counterfeit products obtained from the black market. These abscesses led to an analysis of the current injectable, transdermal and oral performance- and imageenhancing drugs (PIED) presently being used by competitive bodybuilders in the UK.

#### **Materials**

Dodecane, potassium hydroxide (KOH), anhydrous sodium sulfate and hexane, all analytical reagent grade, and high-performance liquid cromatography (HPLC)-grade methanol and cyclohexane were purchased from Fisher Scientific (Loughborough, UK). Ammonium iodide (NH<sub>4</sub>I) and formic acid (Analar, BDH) were purchased from VWR International (Lutterworth, UK). N-Methyl-Ntrimethylsilyl-trifluoroacetamide (MSTFA), pyridine and methoxylamine hydrochloride, methyltestosterone, 19-nortestosterone, 19-norandrost-4-ene-3,17-dione, norethisterone, testosterone, androst-5-ene-3 $\beta$ ,17 $\beta$ -diol, androst-4-ene-3,17-dione and levothyroxine were purchased from Sigma-Aldrich (Poole, UK). Purified water was obtained from ELGA Purelab Maxima (Vivendi Water Systems, High Wycombe, UK). [16,16,17-<sup>2</sup>H<sub>3</sub>]-Testosterone (d<sub>3</sub>T) was purchased from LGC Promochem (Teddington, UK). Androst-5-ene-3 $\beta$ ,17 $\alpha$ -diol, 19-norandrost-4-ene-3 $\alpha$ ,17 $\beta$ -diol and 19-norandrost-4-ene-3 $\beta$ , 17 $\beta$ -diol were purchased from Steraloids (Newport, RI, USA). Mesterolone, methenolone and methenolone enanthate (Primobolan-Depot) were obtained from Schering Germany. DHEA, Sustanon 250 (testosterone propionate, testosterone isocaproate, testosterone phenylpropionate and testosterone decanoate), Durabolin (nandrolone phenylpropionate) and Deca-Durabolin (nandrolone decanoate) were obtained from Organon. Clenbuterol sulphate was obtained from K. Thomae. Clostebol acetate was obtained from Farmitalia. Methandienone was obtained from CIBA. Stanozolol was obtained from Sterling-Winthrop. Boldenone was kindly donated by Professor Don Catlin of UCLA.

#### **Methods**

The generic name of the drug was identified from the brand name, specified on the label. If more than one drug of the same generic formula was analysed, they were provided by different individuals and had different brand names and different packaging. To avoid cross-contamination all vials were analysed for culture and sensitivity (C & S) prior to analysis for chemical formulaic content.

#### Gas chromatography-mass spectrometry conditions

Gas chromatography-mass spectrometry (GC-MS) analysis was carried out on an Agilent 5973 mass selective detector coupled to an Agilent 6890 GC system with an Agilent 7683 autosampler. The GC was fitted with a cross-linked polymethylsiloxane capillary column (HP-1; length 25 m; internal diameter 0.2 mm; film thickness 0.11  $\mu$ m). The initial column temperature was 180 °C for 1 min; this was ramped to 220 °C at 8 °C/min, then from 220 to 250 °C at 3 °C/min and from 250 to 320 °C at 14 °C/min. The final

temperature, 320 °C, was held for 5 min. Injections (1  $\mu$ L) were made in the splitless mode; the injection port and transfer line temperatures were 250 and 280 °C, respectively. Helium was used as the carrier gas and the flow rate was 0.7 mL/min. For screening purposes, the mass spectrometer was operated in the selected ion monitoring mode (SIM) using an ion specific for the mono or bis-trimethylsilyl (TMS) derivative for each compound. The details are summarized in Table 1.

Full scan analysis (mass range m/z 80–650) was used for identification of the compounds detected. The chromatographic conditions are described above.

# LC-MS/MS analysis of thyroxine

A tablet from a bag labelled 'levothyroxine' was placed into a tube containing 10 mL of methanol. The contents were mixed thoroughly by sonication and centrifuged at 1320 g for 5 min. Then 0.8 mL of the supernatant was taken and diluted to 5 mL with methanol: water containing 0.1% formic acid (70:30). A solution containing 1 µg/mL of thyroxine was prepared in methanol: water containing 0.1% formic acid (70:30). A portion of these solution was transferred to autosampler vials and analysed by LC-MS/MS. Chromatographic separation was performed on an Waters Acquity UPLC system using a Zorbax Eclipse XDB-C18 column (50 × 2.1 mm,  $1.8 \, \mu m$  particle size) heated to 35 °C. The mobile phase consisted of 0.1% formic acid (v/v) (solvent A) and methanol containing 0.1% formic acid (v/v) (solvent B). A gradient was employed starting at 30% B, increasing to 55% B in 2 min and to 75% B in 8.66 min and returned to the initial conditions in 9.3 min and allowed to equilibrated for a further 0.7 min. The flow rate was 0.25 mL/min. The injection volume was 10 μL.

This was coupled to an Applied Biosystems API 3200 triple quadrupole mass spectrometer with a Turbo lonspray source operating in the positive ion mode. The optimized source conditions were source heater probe temperature 500 °C, Turbo lonspray voltage 5500 V, curtain gas setting of 15 psi, collision gas setting of 6, ion source gas 1 setting of 45 psi, ion source gas 2 setting of 50 psi, declustering potential of 65 V, entrance potential of 10 V, cell exit potential of 51 V, and collision energy of 35 V. The mass spectrometric method was set up to detect the ions (mass range m/z 500–780) produced by after collision induced dissociation of m/z 777.2, the protonated molecule [M+H]<sup>+</sup>, (product ion scan).

The tablet extract gave a peak consistent with the retention times (4.68 min) and product ion spectrum for thyroxine. The protonated molecule was visible at m/z 777 and collision induced dissociation resulted in the formation of m/z 731, 634 and 605 are consistent with those reported previously.<sup>[6]</sup>

#### **Analysis of products**

A statistical sign test was applied to Tables 2 to 5.

Analysis of PIED tablets (Table 2)

Nineteen products were analysed. Oral products (tablets) were placed into tubes containing 10 mL of methanol. The tubes were mixed thoroughly by sonication then centrifuged at 1320 g for 5 min. The equivalent of 1–5  $\mu g$  of each substance was transferred to a glass tube, 0.1 mL of  $d_3$ -testosterone solution (50 ng), 5 ml of potassium hydroxide (0.1 M) and 5 mL of hexane, were added. The contents were mixed thoroughly and then centrifuged at

Compound (as trimethysilyl derivative; TMS)	Characteristic ions (m/z) and relative abundances (%)	<b>Retention</b> <b>time</b> (min)
clenbuterol bis TMS	<b>86</b> (100), 335(45), 337(30), 300(20)	5.8
19-norandrost-4-ene-3 $\alpha$ ,17 $\beta$ -diol bis TMS	<b>420</b> (100), 240(20), 182(16), 330(15)	10.4
19-norandrost-4-ene-3 $\beta$ ,17 $\beta$ -diol bis TMS	<b>420</b> (100), 240(19), 182(18), 330(15)	11.4
androst-5-ene-3 $\beta$ ,17 $\alpha$ -diol bis TMS	129(100), 239(90), <b>344</b> (80), 434(50)	11.6
19-norandrost-4-ene-3,17-dione bis TMS	<b>416</b> (100), 401(9), 194(8), 234(6)	12.0
dhea bis TMS	<b>432</b> (100), 417(70), 327(35), 303(15)	12.0
androst-5-ene-3 $eta$ ,17 $eta$ -diol bis TMS	129(100), 239(90), <b>344</b> (80), 434(50)	12.3
nandrolone bis TMS	<b>418</b> (100), 403(10), 194(7)	12.4
boldenone bis TMS	<b>206</b> (100), 430(70), 415(25), 325(20)	12.8
androst-4-ene-3,17-dione bis TMS	<b>430</b> (100), 415(10), 194(7), 234(7)	12.8
mesterolone bis TMS	<b>141</b> (100), 157(45), 448(30), 433(10)	12.9
testosterone bis TMS	<b>432</b> (100), 417(13), 208(8), 209(8)	13.1
d3-testosterone bis TMS	435	13.1
methenolone bis TMS	<b>195</b> (100), 446(25), 208(25), 179(20)	13.7
methandienone bis TMS	<b>206</b> (100), 444(60), 339(25), 429(12)	14.3
norethisterone bis TMS	<b>442</b> (100), 194(20), 302(15), 427(15)	14.3
methyl testosterone bis TMS	301(100), <b>446</b> (80), 356(15), 143(15)	14.7
testosterone propionate TMS	<b>416</b> (100), 401(12), 208(5)	15.8
clostebol acetate TMS	<b>436</b> (100), 438(40), 401(20), 421(8)	17.7
testosterone isocaproate TMS	<b>458</b> (100), 443(19), 99(8), 208(5)	19.2
stanozolol TMS	<b>143</b> (100), 385(20), 400(10),330(10)	19.3
testosterone enanthate TMS	<b>472</b> (100), 457(10), 113(10), 208(6)	20.4
methenolone enanthate TMS	<b>195</b> (100), 486(25), 208(25), 179(20)	20.7
testosterone cypionate TMS	<b>484</b> (100), 469 (8), 343 (5), 209 (10)	21.9
nandrolone decanoate TMS	<b>500</b> (100), 329(8), 155(5), 485(4)	22.2
nandrolone phenylpropionate TMS	<b>478</b> (100), 105(12), 194(10), 463(5)	22.3
testosterone decanoate TMS	<b>514</b> (100), 155(10), 343(8), 499(5)	22.6
testosterone phenylpropionate TMS	<b>492</b> (100), 477(10), 105(15), 343(3)	22.7
boldenone undecenoate TMS	<b>206</b> (100), 524(35), 325(20), 191(20)	23.1

1320 g for 5 min. The hexane layer was removed and added to 2 mL of 95% methanol, mixed thoroughly then centrifuged at 1320 g for 5 minutes. The hexane layer was removed and discarded. The methanolic layer was evaporated to dryness under a steady stream of nitrogen. The residues were derivatized to form ether-TMS and/or enol-TMS derivatives by heating at 60 °C for 15 min with 40  $\mu L$  of a mixture containing N-methyl-N-trimethylsilyltrifluoroacetamide (MSTFA), ammonium iodide and ethanethiol (1000/3/9 v/w/v). Following derivatization, the tubes were allowed to cool, 20  $\mu L$  of dodecane was added and the samples were analysed by SIM and full-scan GC-MS as described

Analysis of AAS vials and sachets (Tables 3, 4 and 5)

above.

The contents of the vials were intended for injection whereas the sachets were for topical application. There were 38 different products.

The contents were transferred to glass vials and a 200  $\mu$ L portion of each was taken and added to 10 mL of methanol in 20 mL tubes. The contents were mixed thoroughly and then centrifuged at 1320 g for 5 min. Methanol extracts (200  $\mu$ L) were taken and the solvent was evaporated to dryness under nitrogen at 60 °C. The residues were derivatized to form trimethylsilyl derivatives and analysed by full-scan GC-MS, as described above.

Steroids in which the tri-ene-one system is present, such as trenbolone acetate and tetrahydrogestrinone, do not derivatize well with the above procedure and a repeat analysis was performed using a different derivatizing technique to form a stable methyloxime trimethylsilyl derivative. Methanolic extracts (50  $\mu L$ ) were taken and evaporated to dryness under nitrogen at 60  $^{\circ}C$ . The residues were derivatized to form methyloximes by heating at 60  $^{\circ}C$  for 1 h with 50  $\mu L$  of methoxylamine hydrochloride (8% w/v in pyridine). Following derivatization, 2 mL of cyclohexane: dodecane (98:2 v/v) and 0.5 mL of water were added. The water layer was removed and anhydrous sodium sulphate was added to the organic layer, which was then decanted into a clean tube. The organic fraction was evaporated to dryness, the residue derivatized by trimethylsilylation and analysed by full-scan GC-MS, as described above.

Analysis of vials for human growth hormone (Tables 4 and 5)

Vials purported to contain hGH were subjected to direct analysis and following digestion with trypsin, using matrix-assisted laser desorption ionization time of flight, mass spectrometry (MALDITOF/MS) and peptide mass mapping, as described elsewhere.<sup>[7]</sup> The presence of recombinant hGH (22.1 kDa) was confirmed by peptide mass mapping (12 peptides identified).

#### Microbiological culture of drug samples

One hundred microlitres of each sample was inoculated onto two blood agar plates (Oxoid Ltd, Basingstoke, UK), one of which had been pre-reduced overnight. The inocula were spread over the plates using disposable plastic spreaders. The pre-reduced plates were incubated in 3.5 L anaerobic jars with an anaerobic atmosphere generated using the catalyst-free 'Anaerogen'® system (Oxoid). The second agar plate was incubated aerobically. All plates were incubated at 37 °C. Aerobic cultures were examined for bacterial growth after 24 and 48 h. Anaerobic cultures were examined after 48 h and again after 5 days.

# Results

Retention times and full scan spectra were compared to those obtained from analysis of standards and drugs supplied by licensed pharmaceutical companies. The results of tablet, sachet and vial analysis, obtained from the underground market, are displayed in Tables 2–5. Of the 57 samples analysed, 24 were counterfeit (42%). The statistical sign test applied to Tables 2–5 demonstrated significance in all groups (p < 0.05).

Of the 19 orally active preparations (Table 2), four samples were labelled 'stanozolol' but only one contained this anabolic steroid; two of the remaining three contained other anabolic steroids and one contained no active ingredient. All the other products labelled contained the correct contents. From 38 vials or sachets for parenteral use, 18 samples contained what was described on the label (Table 3). Eight samples contained something other than what was on the label (Table 4). Twelve samples contained no active drug (Table 5).

Microbiological culture of the vials revealed the presence of contaminants, identified as skin commensals. Figure 1 shows a right biceps abscess and Figure 2 shows the microphotograph of an abscess wall scraping taken during surgery.



Figure 1. Abcess of the right biceps.

# **Discussion**

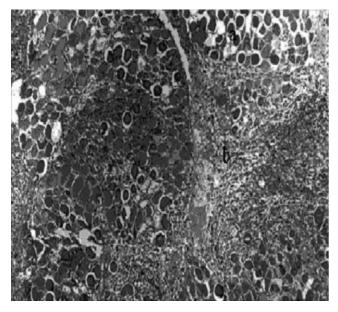
A number of tablets and a large proportion of the injectable AAS were found to be counterfeit, containing steroids other than those indicated or no steroid at all. The most 'sophisticated' example was a vial labelled 'Sustanon' (Table 4, sample 4), which was counterfeit as it contained testosterone enanthate, this ester not being an active ingredient amongst the testosterone esters listed in either Sustanon 250 or Sustanon 100 manufactured by Organon, the legitimate pharmaceutical company.

A comparison of the formal reference literature (Martindale: *The Complete Drug Reference*) and bodybuilding web sites, reveals that illegal laboratories are concocting their own names and product contents, as exemplified by sample 5, Trenbolone 80 (Table 4), which is not a recognized brand name in the reference literature (and the vial contained trenbolone esterified with acetate rather than the claimed enanthate, both of which are supplied by a

	Product label	Product claimed and Identified by analysis
	Deca durabolin (different manufacturer)	Nandrolone decanoate
2	Deca durabolin (different manufacturer)	Nandrolone decanoate
3	Equipoise	Boldenone undecylenate
4	Omnadren (different manufacturer)	T. Propionate, T. Phenylpropionate, T. Isocaproate, T. Decanoate
5	Sustanon (different manufacturer)	T. Propionate, T. Phenylpropionate, T. Isocaproate, T. Decanoate
6	Sustenon '250' (different manufacturer)	T. Propionate, T. Phenylpropionate, T. Isocaproate, T. Decanoate
7	Sustenon '250' (different manufacturer)	T. Propionate, T. Phenylpropionate, T. Isocaproate, T. Decanoate
8	Parabolan	Trenbolone acetate
9	Primobolan depot (different manufacturer)	Primobolan
10	Primobolan depot (different manufacturer)	Methenolone enanthate
11	Stanazol	Stanozolol
12	Testosterone enanthate (different manufacturer)	T. Enanthate
13	Testosterone enanthate (different manufacturer)	T. Enanthate
14	Testosterone enanthate (different manufacturer)	T. Enanthate
15	Testosterone propionate	T. Propionate
16	Trenbalone acitate	Trenbolone acetate
17	Zambon (winstrol depot)	Stanozolol
18	Norditropin simplexx	Growth hormone

	Product label	Product claimed	Product found
ı	Boldenona 50	Boldenone undecylenate	T. Propionate
2	Nandrolone decanoate	Nandrolone decanoate	T. Enanthate
3	Primobolan depot	Methenolone enanthate	Nandrolone phenylpropionate
4	Sustanon	T. Propionate, T. Phenylpropionate, T. Isocaproate, T. Decanoate	T. Propionate, T. Phenylpropionate T. Decanoate, T. Enanthate
5	Trenbolone 80	Trenbolone enanthate	Trenbolone acetate
5	Unlabelled	Growth hormone	rhGH material present (22 kda peak, only
7	Sachet 1	Www.821.in 'tep, 3 ml'	T. Propionate
3	Sachet 2	Www.821.in 'Indian aromatherapy oils, te3, 3 ml'	T. Propionate, t. Cypionate, T. Decanoate

	Product label	Product claimed	Product found
1	Boldabol	Boldenone undecylenate	Nil
2	Boldebal-h	Boldenone undecylenate	Nil
3	Mastabol	Dromastanolone dipropionate	Nil
4	Primobolan depot	Methenolone enanthate	Nil
5	Spectriol	T. Esters	Nil
6	Testabol depot	T. Cypionate	Nil
7	Testex elmu prolangatum 250	T. Cypionate	Nil
8	Tesosterone cypionate injection (cypionax)	T. Cypionate	Nil
9	Trenbol 75-r	Trenbolone acetate	Nil
10	Youth gh	Growth hormone	Nil
11	Growth hormone	Somatotropin	Nil
12	Norditropin simplexx	Growth hormone	Nil



**Figure 2.** Microphotograph of abscess wall scraping taken during surgery shows muscle fascicles (a) and acute inflammatory cells with necrotic debris (b).

company in Poland). Trenbolone as the acetate and boldenone as the undecylenate have been used exclusively as veterinary anabolic agents and were confirmed to be present in the products with the legitimate brand names Parabolan and Equipoise. Within the European Union, the use of these veterinary drugs as livestock growth promoters is banned. Bodybuilders are known to use such veterinary preparations and athletes are still testing positive for these prohibited substances (WADA Laboratory Statistics, 2007). [8]

With respect to the non-steroidal drugs, three vials purported to be GH (Table 5, sample 10, 11, 12) contained no steroid or protein hormone (or steroid). It is not surprising that counterfeits will exist that contain no hGH, especially as pharmaceutical hGH is expensive compared with AAS. Conversely, thyroxine is relatively inexpensive compared to hGH and thyroxine was confirmed to be present in a tablet provided for this study. Thyroxine is a prohormone for the more active thyroid hormone tri-iodothyronine, which may be used to 'help accelerate the basic metabolic rate, increase

quickness and reduce sluggishness', as stated in a letter from Mr Victor Conte of BALCO, to the athlete Mr Dwain Chambers, to provide him with detailed information for UK Sport regarding the seven different types of drugs he was purported to be using (tetrahydrogestrinone, testosterone with epitestosterone, hGH, insulin, erythropoietin, modafinil and liothyronine). This extent of polypharmacy by an athlete is perhaps surprising but is very common amongst bodybuilders who use image-enhancing drugs. Drugs are not only used to enhance the body image but are taken to counteract the occurrence of any adverse side effects or potential side effects of other drugs. For example, males use tamoxifen to counteract the aromatization of excess testosterone, which can lead to gynaecomastia [9] but the rationale for its use by female bodybuilders may at first appear illogical, as anabolic steroid administration causes breast shrinkage and some competitors resort to implant surgery. It is reasoned that female bodybuilders have a much more difficult time reducing to the low levels of body fat needed for competition, which is why oestrogen antagonists such as tamoxifen or aromatase inhibitors such as anastrozole or letrozole are used. The oestrogen antagonists are thought to give the physique a more defined and vascular look for both men and women (personal communication between ATK and Mr Anthony Roberts, a consultant to professional bodybuilders).[10] Tamoxifen was included in the drug regimen to be used by a UK national competitive female bodybuilder (Table 6) (personal communication with MRG).

It has been estimated that one million individuals in the US, predominantly males under 25 years of age, are current or past users of AAS. Fifty per cent of these young adults administer their drugs intramuscularly, placing them at risk of infections related to injection. A survey on weightlifters in 2007, undertaken in the south Wales area of the UK, found that 70% of 146 respondents were regular AAS users and that 7% were females. Although the number of anabolic steroid users cannot readily be determined, it is clear that they are used throughout the UK, as discussed in depth by the report from the British Medical Association (BMA, 2002).

The demand for AAS for performance and image enhancement has led to an expanding counterfeit market – a market that has existed at a sophisticated level for more than a decade. In 1997, an investigation demonstrated that 15 (36%) of 42 AAS analysed by GC-MS did not contain the expected ingredients. [14] Much more recently, Thevis *et al.* [15] investigated the contents of 70 confiscated products, where 17 (35%) of 48 compounds labelled as anabolic

Table 6. AAS Regime of a current female UK bodybuilding champion					
Week	5	4	3	2	1
Drug					
Clenbuterol (Spiropent) (oral) (20 µg.tablet <sup>-1</sup> .d <sup>-1</sup> )	6	6	6	6	6
Growth hormone (Somatropin) (Jintropin) (SC) ( $IU.d^{-1}$ , 4 days.week $^{-1}$ )	2	2	2	2	2
Oxandrolone (Anavar) (oral) (10 mg.tablet $^{-1}$ .d $^{-1}$ , 5 days.week $^{-1}$ )	1	1	1	1	1
Stanozolol (Winstrol) tablets (oral) (2 mg.tablet <sup>-1</sup> .d <sup>-1</sup> , 5 days.week <sup>-1</sup> )	4	5	6	5	4
Stanozolol (Winstrol) suspension (IM) (50 mg.ml $^{-1}$ .week $^{-1}$ )	1	2	2	2	1
Tamoxifen (Nolvadex) (oral) (10 mg.tablet <sup>-1</sup> .d <sup>-1</sup> , alternate days)	1	1	1	1	1
Testosterone propionate (IM) (150 mg.2ml <sup>-1</sup> .week <sup>-1</sup> )	1	1	1	1	1

 $IM = Intramuscular \, (parenteral) \, administration \,$ 

SC = Subcutaneous (parenteral) administration

Oral = Enteral administration

 $d^{-1} = daily dose$ 

 $week^{-1} = weekly dose$ 

steroids did not contain or did not only contain the declared ingredients. Even the counterfeit packaging examined in this study were hardly distinguishable or not distinguishable from authentic boxes. Moreover, a study performed in 2001 and 2002 on 634 nutritional supplements, purchased in 13 different countries, showed that apart from low-level contamination with androgens in a significant proportion of non-hormonal supplements, others that were actually declared by the suppliers as prohormones were counterfeit as they contained high amounts (>1 mg.g<sup>-1</sup>) of 'classic' anabolic steroids. [16] This unregulated manufacture may produce preparations that are contaminated with infectious agents and are of poor quality.<sup>[17]</sup> Due consideration should also be given to possible adverse reactions to the vehicle used, as well as the risk of particulates. Individuals who inject AAS are also known to develop complications from contaminated syringes or non-sterile injection techniques. The sharing of AAS 'multi-dose' vials, whether the vials are from a legitimate source or otherwise, is commonplace, exposing individuals to the risk of intramuscular abscesses. Reported infections associated with AAS injection include abscesses attributable to Mycobacterium smegmatis, Staphylococcus, Streptococcus, Pseudomonas, hepatitis B, hepatitis C and human immunodeficiency virus. [18-20] Thigh abscesses, [11,20] pectoral and deltoid abscess have been reported in bodybuilders using 'spot shots' or 'site locations', which are local injections into a specific muscle, believed to increase isolated muscle growth. [21,22] Gluteal abscesses have also occurred in contaminated products.<sup>[15]</sup> Administration of large volumes of testosterone esters in one injection (up to 5 mL) is common, exposing an individual to sterile abscess formation, where a pathogenic organism cannot be found.<sup>[23]</sup>

Contamination of vials with used needles would be an effective means of transmitting blood-borne pathogens. [24,25] This is comparable to the sharing of spoons among intravenous drug users, who inject street drugs. [26] Education and the unquestioned provision of disposable of sterile needles remains a practical way to prevent abscesses and blood-borne infections between such individuals. Needle-exchange programmes currently provide AAS users with clean, sterile needles and may be a unique opportunity

for intervention and education to this population. [27] Despite the provision of sterile needles and syringes to user groups, there has been a pandemic increase in hepatitis C from recreational drug abuse and currently 50% of all recreational drug abusers are suffering with the virus. [28] There is a direct relationship between the increase in performance-enhancing drug use and infections and communicable diseases, which are reaching pandemic proportions. [29] Contemporary research is providing evidence to educate and prevent the catastrophic effects of doping. [30] There is a need for the unlimited provision of sterile equipment with the assurance of anonymity, embracing the fact that all previous methods have failed. Despite the information being promoted that the administration of AAS by parenteral injection is less harmful than oral administration, deaths are still occurring from chronic high dose usage, irrespective of the mode of administration. [31]

# Conclusion

The risk of counterfeits and poorly controlled products for performance and image enhancement, including AAS and hGH, exposes users to enormous health risks, especially when these products are injected.

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