Anabolic Androgenic Steroid Abuse in the United Kingdom; An Update

The increasing popularity of anabolic androgenic steroids.

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7,081 Words

Abstract

Anabolic androgenic steroids (AASs) are prescribed for medical conditions related to low testosterone. Abuse of AASs has surged as they become increasingly recognised as potent image enhancement drugs. The primary goal of most abusers is to obtain what they consider to be a more attractive outward appearance. Abuse is complex. There are a vast range of AAS substances available, although due to their illicit nature, the true composition of AAS substances is difficult to evaluate. Users follow dosing patterns which incorporate a number

This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1111/bph.14995

of different AASs, in addition to other pharmaceutical substances believed to complement the desired physical effects or manage unwanted effects.

Studies to support the harms of AASs are limited. Animal work and medical case reports suggest potential to cause serious hepatotoxicity, in addition to possible neurotoxicity, nephrotoxicity and damage to the cardiovascular and reproductive systems. As the long term AAS using cohort reach maturity, further controlled experimentation, with larger sample sizes, is required.

Data gathering should be directed towards establishing knowledge related to the most vulnerable AAS users, which is inclusive of females and adolescent boys.

1. Introduction

The increasingly lean and muscular body type now displayed by the mass media rarely exists in nature and could not be attained following the current recommendations on diet and exercise believed to constitute a 'healthy lifestyle'. In the hectic modern way of life, image and performance enhancing drugs (IPEDs) are becoming widely accepted as a convenient and readily obtainable means of attaining this widely advertised perfect body shape. Anabolic androgenic steroids (AASs) have become the IPEDs of choice (Bates & McVeigh 2016). With the popularity of AASs for enhancing purposes soring in recent years, the degree of AAS use, appears to have reached a level which poses a serious risk of harm, (Figure 1).

2. Anabolic Androgenic Steroids

Testosterone and dihydrotestosterone are endogenous male gonadal steroids (sex hormones). When present in excess, testosterone can increase muscle growth and strength (Bhasin et al. 1996). The term anabolic androgenic steroids (AASs) can include endogenously produced androgens but, for the purpose of this review, is intended to describe the wide range of synthetically manufactured abused derivatives of testosterone, Table 1. There is a large and increasing number of AASs available (Lood et al. 2012), few are legitimate, licensed medicinal products (only 9 types of AASs were prescribed in England and Wales in 2018-19, (NHS England 2019)). The majority are 'designer steroids' developed in research or

underground laboratories to optimise muscle growth while minimising unwanted androgenic effects.

The use of AASs outside of medicine was first exploited by professional athletes, most notably by competitive bodybuilders. With the impressive results of use clearly publicised in the media (Prendergast et al. 2003), dramatic effects are well recognised by the public and use for amateur/cosmetic purposes has largely been driven by availability. In the UK, AASs are most commonly derived from testosterone in various forms, predominantly testosterone enanthate (Table 2) (Bates & McVeigh 2016; Hildebrandt et al. 2010). This is reflective of male use however, females are thought to prefer the milder effects of stanozolol (Rexobol or Winstrol), Oxandrolone (Anavar) or Nandrolone (Deca) (Jespersen 2012).

3. AASs in the UK

AASs are controlled in the UK as Class C substances under Part III of Schedule 2 of the Misuse of Drugs Act, 1971, and Schedule 4 of the Misuse of Drugs Regulations, 2001. It is not an offence to possess AASs for personal use, or to import or export AASs if they are intended for personal use. Class C drugs represent the category of controlled drugs considered to present the least danger to the user/society and, as such are not the highest priority for policing or the focus of campaigns to raise awareness about harm, safe use or use prevention (House of Commons Science and Technology Comitty 2006). However, such classifications do not recognise the relationship between harm and user numbers. Tens of thousands of individuals using a slightly harmful drug could be more detrimental to society than a few dozen individuals using a very dangerous substance. Consistent with this fairly relaxed legislative classification however, users do not feel stigmatised as 'drug' users (Radcliffe & Stevens 2008; Parker et al. 2002). In fact, AAS users believe that by using AASs they adopt a 'lifestyle' incorporating wellbeing and exercise (Ravn & Coffey 2016; Dunn et al. 2014; Hildebrandt et al. 2012). The majority maintain order in their lives, with nice homes and employment, some even holding professional positions (Dunn et al. 2014). This is in stark contrast to *ad hoc* recreational drug use, aligned with deterioration of social circumstances, self-destructive and anti-social behaviour. User detachment of AAS use from psychoactive 'drug' abuse may be further encouraged by the substances being much more accessible (Brennan et al. 2016). Readily available on-line (Mcdonald et al. 2012) or

purchased from friends or gym associates (Ip et al. 2011) there is no perception of duplicitous dealing, violence or organised-crime .

The general public however, do not share this perception. Perhaps driven by the injectable nature of AASs, the general public are likely to stigmatise AAS users, as they would 'hard drug' users (Griffiths et al. 2016). This may be validated by AASs being rated more harmful than the Class A substances Ecstasy, LSD, buprenorphine and certain mushrooms (Nutt et al. 2010). Whilst this was almost entirely due to the harm posed to the user, the potential for social harm has been increasingly recognised through connections to binge drinking, use of psychoactive drugs (Lundholm et al. 2015; Darke et al. 2014; Ip et al. 2014; Hakansson et al. 2012; Lood et al. 2012; Leifman et al. 2011), spread of blood borne viruses (Hope et al. 2013), violence (Lundholm et al. 2015; Darke et al. 2014) criminal, aggressive and anti-social behaviour (Hallgren et al. 2015).

4. AAS User Demographics

AAS users are typically in their late 20s to early 30s and most often are heterosexual males (Hope et al. 2016; Ip et al. 2012; Ip et al. 2011), although gay/bisexual men are overrepresented in the AAS community (42%) (Van Beek & Chronister, 2015). Most users are single and participate in recreational exercise (Ip et al. 2012), largely comprising of weight training (Brennan et al. 2016). Gym users, especially private gym users, are more likely to be offered AASs (Leifman et al. 2011), which may partially contribute to popularity amongst this demographic. In the UK, AAS users display lower levels of education compared to non-users (Kanayama et al. 2013).

Females also use AASs, but to a much lesser extent (Börjesson et al. 2016), around 8 to 16 % of AAS users in the UK and Ireland are thought to be female (Mcveigh et al. 2015; Chandler & Mcveigh 2013)). Information related to female users is, therefore, limited. Most women are introduced to AASs by a male figure (Börjesson et al. 2016). Yet, the male dominated online community exhibit hostility towards female users, warning against masculinisation and infertility (Jespersen 2012). The online user community are also unwelcoming of adolescent males. Adult male users discourage younger men from AAS use until at least 21 years of age, when natural testosterone has peaked (Chandler & Mcveigh 2013).

The majority of UK users are aged over 18 at the time of first use (Chandler & Mcveigh 2013) but use amongst adolescents has been recognised (Brennan et al. 2016; Eisenberg et al. 2012; Ip et al. 2012). The last available figures for England and Wales, indicated use in males as young as eleven (Fuller et al. 2013), and in Scotland AAS use was recorded at 1% for 13 year old and 1% of 15 year old boys (over 2,200 respondents) (NHS Scotland 2014). This is low compared to findings elsewhere, where up to 5.9 % of teenage school boys were reported to have tried AASs (Heimly Jenssen & Johannessen 2015; Eisenberg et al. 2012).

5. Popularity of AASs

Reliable data concerning AAS use is very difficult to obtain. The last estimate in England and Wales was that 411,000 adults had used an AAS (Home Office 2018). This represented substantial growth, an approximate doubling over a ten year period, Table 3. There are no national surveys which indicate AAS use in Scotland, but Needle and Syringe Programme (NSP) data also indicate substantial escalation. The number of attendees and needles dispensed reached an all-time high in 2014/15, showing a 45% and 15% rise on the previous year, respectively (NHS Scotland Information Services Division 2016). With no associated increase in supply of psychoactive drug paraphernalia, AASs were believed responsible for the upsurge. Provision of equipment for injection of IPEDs continued to grow in subsequent years (NHS Scotland 2018). Around two thirds of NSP new clients are AAS users (Whitfield et al. 2012) and up to 86% of clients overall (Kimergård & Mcveigh 2014). NSP data are expected to underestimate AAS use as needles can be obtained from other sources, and some users may embark on needle and/or syringe re-use and sharing (Bates & McVeigh 2016).

There is a high incidence of injection site infection and injury reported by ASS users (Hope et al. 2014), but many never seek specialist help with correct injection techniques. Relying instead on the guidance of other users or information from their supplier (Hanley Santos & Coomber 2017). The injection of AAS differs from psychoactive drugs which are typically injected intravenously. AAS are usually injected into a large muscle (buttocks or thigh) and require a longer, wider gauge needle than used for intravenous injections; a fact unknown to some users, and a method ignored by others as they find it technically difficult (Hope et al. 2014). Also, some users are known to perform ‘spot injections’. This is where AAS are injected into targeted, smaller muscle groups (e.g. biceps) (Hope et al. 2014; Kimergård & Mcveigh 2014; Bates & McVeigh 2016) in the belief that the anabolic action will be localised

(Evans 1997). However, this practice requires very good injection technique to avoid complications such as blood vessel breakage, muscle or nerve damage or even paralysis (Evans 1997). Spot injections are concomitant with injection site infections, abscesses and tumours (Hope et al. 2014; Weinreb et al. 2010), as are the re-use of needles and/or syringes and the use of multiple injection vials (Hope et al. 2014; Pai et al. 2013; Graham et al. 2009). Unlike most psychoactive drugs, AAS are usually purchased as a liquid, ready for injection. This represents a greater risk for bacterial contamination (Hope et al. 2014), especially as many AASs are produced without the necessary regard to quality and safety. AASs purchased online have been found to be contaminated with microorganisms which could result in abscess formation (Graham et al. 2009), serious infection, and in extreme cases could result in death (Russo et al. 2012; Perera et al. 2013).

AASs are usually sold by websites which either claim to specialise in medicines or dietary supplements (Cordaro et al. 2011). These are not pharmaceutical and the content of AASs purchased online cannot be guaranteed. Products have widely been identified as counterfeit, misleadingly named, and labelled with incorrect contents and dosages (Ribeiro et al. 2018; Weber et al. 2015; Abbate et al. 2014; Coomber et al. 2014; da Justa Neves et al. 2013; Cordaro et al. 2011; Graham et al. 2009). Thus, despite carefully planned administration regimes, users cannot be sure that what they purchase really contains the substance they need, at the correct dose. The unreliable nature of purchasing AAS products is well recognised within the user community, many of whom contribute to websites that rate online suppliers and their products. This can help users avoid suppliers who fail to deliver or supply poor products. The health risks of such uncertainty do not appear to trouble users, who are more concerned about being swindled or the product not being efficacious (Kimergård & McVeigh, 2014). Generally, the only way to know what a purchased vial contains, is to use it and experience the strength and/or bodily effects. Often the bodily gains do not manifest or there are unexpected side-effects, but this does not discourage use of AASs (Jespersen 2012).

6. Harms of AAS Use

In 2010, the UK Advisory Council on the Misuse of Drugs (ACMD) highlighted important gaps in available knowledge surrounding increasing AAS use (Advisory Council on the Misuse of Drugs 2010). The many obstacles to investigating the dangers of AAS use were well summarised, and these restraints have not eased with time. The study of AASs in adult

males remains limited due to experimental and ethical restrictions. The information that is available, generally comes from surveys or experiments in which AAS use is self-reported or from hospital/*post-mortem* case studies. Thus, the degree of AAS ingestion is never reliably known (even by those consuming them), and no account can be recorded of possible considerable polypharmacy, employed by most AAS users (Lusetti et al. 2015; Shamloul et al. 2014; Montisci et al. 2011).

As dominant users, most investigations of AAS toxicity focus on adult males. Little is known of the harmful effects of AAS use in females or adolescents; the groups in which they are expected to be most hazardous. Lacking in natural testosterone, females appear to be more susceptible to toxic effects (Börjesson et al. 2016) and represent a high proportion (82.5%) of hospitalisations (Henrique et al. 2013). This could however be related to the apparent preference amongst female users for oral AASs (Heimly Jenssen & Johannessen 2015), which have greater toxicity. Commonly recognised (irreversible) effects in females are those associated with masculinisation, for example deepening of the voice and abnormal hair growth (Jespersen 2012). Adverse effects typically experienced in males include acne, hair loss, painful gynecomastia and testicular atrophy. Side-effects which cannot be easily recognised by the user, however, include oligo-azospermia, damage to tendons and muscles, and more seriously, damage to the cardiovascular system and internal organs. Adverse effects of AAS use are largely dose dependent and reversible upon cessation of use, although there is some evidence to suggest that carcinogenesis may arise from even short-term use (Frankenfeld et al. 2014; Martins et al. 2010).

During AAS use natural testosterone secretion can be much reduced or even absent (Kanayama et al. 2015). This can continue into the off-cycle and periods of testosterone deprivation may result. Immediate effects of testosterone deficiency in adults include loss of libido, impaired erectile function and depression (Osta et al. 2016; Nieschlag & Vorona 2015). Symptoms can persist long after use has been discontinued and, in some cases, may not dissipate, even following medical intervention. This suggests permanent damage occurring to the testosterone producing leydig cells (Kanayama et al. 2015). This may explain why AAS use and resultant lack of natural testosterone production in adolescence, can lead to testicular atrophy and disrupted reproductive behaviours in adulthood (Olivares et al. 2014).

Damage to the cardiovascular system may also remain masked and may not manifest to a significant degree until later in life (Reza et al. 2012). Although, there is mounting evidence to link AAS associated cardiovascular damage and thrombosis with cerebrovascular accident in younger AAS users (Shimada et al. 2012; Cooper et al. 2011; Youssef et al. 2011). Accounts of alterations and effects on the cardiovascular system are somewhat inconsistent. Repeatedly reported during long term and/or high dose AAS use, are negative effects on lipid profiles, increased blood pressure, myocardial deformity and dysfunction (particularly of the left ventricle) and sudden death (Baggish et al. 2017; Alizade et al. 2015; Kaufman et al. 2015; Angell et al. 2012; Reza et al. 2012; Rothman et al. 2011; Achar et al. 2010; Baggish et al. 2010; Maior et al. 2010). The deformity to the myocardium and coronary arteries has been observed to be so significant that it may constitute a serious public health concern (Baggish et al. 2017).

Damage to the cardiovascular system could, however, be compounded by additional substances allied with AAS use. In particular, stimulants such as cocaine and 'fatburners', which are well documented to adversely affect the heart. Multi-drug use by AAS users could also be contributing to the implication of AASs in causing severe liver disease (Robles-Diaz et al. 2015; Simões Tanasov et al. 2014; Elsharkawy et al. 2012). 17α alkylation of steroids (e.g. methyltestosterone, methandrostenolone, oxymetholone, oxandrolone and stanozolol) to increase their oral bioavailability, slows their metabolism in the liver, meaning hepatocytes and cholangiocytes are exposed to the drug for longer, resulting in increased toxicity (Elsharkawy et al. 2012). Injectable AASs may still cause alterations in liver structure and function when consumed at high doses and for longer periods (Chandler & Mcveigh 2013). Such use can also contribute to kidney damage (Robles-Diaz et al. 2015), and potential to develop Wilm's tumour (Osta et al. 2016). The raised BMI and high protein diets of many AAS users increases susceptibility to nephrotoxicity (Harrington et al. 2011). At least one case is reported in the literature where kidney damage was so severe that transplantation was required (Harrington et al. 2011).

6.1. Blood Borne Viruses

In addition to the direct actions of AAS substances, their use in the form of injectables carries further potential for harm. Injected intramuscularly, rather than intravenously, users generally do not perceive themselves to be at high risk of contracting blood borne viruses

(BBV) (Van Beek & Chronister 2015). This could explain the much lower levels of hepatitis B vaccinations or hepatitis C testing seen in AAS users compared to other injecting drug users (Anon 2015). Although the rate of hepatitis B and C infection amongst AAS users is lower than in those injecting psychoactive substances, it is higher than the occurrence of these BBVs in the general UK population. AAS users also exhibit increased rates of HIV infection, which are equal to that of intravenous drug users (Hope et al. 2016; Anon 2015).

To reinforce the message that needles should never be reused or shared, and improve access to clean equipment, it was recommended that NSPs offer evening clinics specifically for AAS users (The Scottish Government 2010). AAS users had expressed unwillingness to attend NSPs, as they did not wish to be associated with 'problem' drug users, and many were unable to attend as they were in full time employment. Additionally, it was made possible for attendees to collect unlimited numbers of needles and syringes to allow for fewer visits. Two thirds of AAS users now obtain injection equipment from a NPS (Bates & McVeigh 2016), this is a change from 2013 when most users purchased equipment online (Chandler & Mcveigh 2013). The more challenging issue now is that attendees collect equipment for onward distribution (Van Beek & Chronister 2015). Around one fifth of users report collecting equipment to give to others (Bates & McVeigh 2016) and those in receipt will not benefit from the important advice on safe use, injection and disposal of needles. Neither will they receive sexual health advice, which appears to be a priority as precarious sexual conduct may be a more significant contributor to the spread of BBVs in this group. Only a small amount of needle sharing (1% (Bates & McVeigh 2016) to 6% (Hope et al. 2016)) and multiple-dose vial sharing (7% (Whitfield et al. 2012) to 12% (Bates & McVeigh 2016)) takes place.

7. Motivators for AAS use

Almost unanimously, the reason given for AAS use is to increase muscle mass (Hanley Santos & Coomber 2017; Bates & McVeigh 2016), commonly to improve physical appearance (Hanley Santos & Coomber 2017; Murray et al. 2016; Ip et al. 2014). The greatest motivator for this group is the rapid and convenient results of AAS use on muscle mass. Which, even without accompanying exercise, are greater than those that can be achieved by exercise alone (Bhasin et al. 1996). Use is encouraged through contact with other users (Hildebrandt et al. 2012), who can vouch for the outcome. A large proportion of

those wishing to improve their appearance are inspired to use AASs in pursuit of health and wellbeing. Many start out with use of health supplements and there are strong associations between supplement use and use of AASs (Heimly Jenssen & Johannessen 2015; Hildebrandt et al. 2012; Leifman et al. 2011).

Dietary supplements are often marketed as safer and legal alternatives to AASs. However, the reliability of certain 'specialist' supplements cannot be assured. Bulk products are imported to the UK, often from China (Advisory Council on the Misuse of Drugs 2010), and processed and packaged for sale in shops or online (Abbate et al. 2014). It is not unusual for these products to contain controlled AASs (Abbate et al. 2014; Chahla et al. 2014; El Sherrif et al. 2013; Cordaro et al. 2011) and around half of the dietary supplements available online contain undeclared AASs (Cordaro et al. 2011). Even where AASs are listed in the ingredients they may not be immediately recognisable as an AAS. Plus, ingredient lists cannot be trusted. Often an AAS may be listed as an ingredient of a supplement but it will not contain the AAS as advertised on the label. Rather, it will be found to contain a different AAS, usually present at a concentration in excess of that required to have an effect (Abbate et al. 2014). This could have serious implications, particularly in naïve users unaware that they are consuming what is considered to be a large dose. These individuals may be teenage boys, who have not yet reached sexual maturity and are therefore more susceptible to irreversible harm through AAS use (Ramos-Pratts et al. 2013; Clark et al. 2006; Henderson et al. 2006; Clark & Henderson 2003). Adolescent males have frequently reported behaviours such as those associated with the use of protein powders/shakes and other supplements to increase muscle size and tone (Eisenberg et al. 2012). In fact, a perception of being over or under weight as an adolescent has been linked to initiating AAS use (Heimly Jenssen & Johannessen 2015; Pope et al. 2012).

Concerns over body image and body dissatisfaction are common factors amongst those who use AASs (Jampel et al. 2016; Björk et al. 2013). Muscle dysmorphia (MD) is a psychiatric condition (classified under the conditions DSM-5 300.7 ICD-10 F22.8b (American Psychiatric Association 2013)), where the sufferer is obsessively and compulsively driven toward achieving a lean and muscular body. Also known as "reverse anorexia nervosa", it is a fear of being too small (Björk et al. 2013). It usually manifests in careful eating with excessive weightlifting, and sufferers often use AASs. In those suffering MD, body satisfaction is unlikely to improve with AAS use (Heimly Jenssen & Johannessen 2015), and

AASs have actually been implicated in the development and maintenance of the disorder (Björk et al. 2013).

A particularly muscular physique may be pursued for reasons other than to improve body image. For example, advantages in non-professional sports (especially weightlifting and powerlifting) or to increase strength, achievements or even intimidation in certain professions such as security for bars and clubs. Moreover, a minority of users have been reported as using AAS in order to appear threatening and/or increase strength to aid in the commission of crime (Lood et al. 2012). One such group of AAS users, familiar with violence, are heroin users. Increased size and strength from AASs prevents a heroin user from being physically intimidated in interactions with others involved in the illicit drug trade (Cornford et al. 2014). AASs can also provide a means to conceal current or past problem drug use by obscuring the excessive weight loss that is a recognisable indicator (Hanley Santos & Coomber 2017; Cornford et al. 2014; Nøkleby & Skårderud 2013). As heroin users are often stigmatised by society, a healthy body becomes an indicator of a 'good' person which can be used to support access to housing or employment (Nøkleby & Skårderud 2013). ASSs provide an easy means of appearing healthy and trustworthy (Cornford et al. 2014).

7.1. Dependency

Upon commencing AAS use, it is possible that dependency becomes a motivator for continued use. Around one quarter (Ip et al. 2012) to one third (Hildebrandt et al. 2011) of AAS users claim to be dependent. "Androgen dependence syndrome" describes continued AAS use despite prominent adverse medical, psychological, or social effects (Kanayama et al. 2009). However, although AAS users may experience adverse effects during AAS use, these tend to increase during periods of abstinence. The severity of these negative side effects experienced during off-cycles may create difficulty for users to permanently stop, as they are eager to use again to ease their physical or emotional suffering (Kanayama et al. 2015). Additionally, continued use is sustained by the positive effects experienced by many users which not only include the desired increase in muscle mass but also feelings of wellbeing (Advisory Council on the Misuse of Drugs 2010), which have been likened to psychoactive drug use (Hanley Santos & Coomber 2017). Similar to psychoactive drug users, AAS users describe how AASs use can produce a mental 'high', by making them feel

more energetic and confident, from the belief that they have extreme strength (Hanley Santos & Coomber 2017; Cornford et al. 2014; Nøkleby & Skårderud 2013).

Thus, AAS dependency is not thought to be a physiological condition. Rather, it is a psychological addiction. The user is compelled to continue to use the drug but to stop would not result in a withdrawal syndrome (National Institute on Drug Abuse (NIDA) 2007). It is actually possible that AAS use creates a vulnerability in the user to develop a dependence syndrome. Animal studies have highlighted the importance of sex hormones as modulators of drug sensitivity (Struik et al. 2018; Marusich et al. 2015). Chronic AAS use is understood to suppress the endocannabinoid system (Struik et al. 2017), with consequent reduction in reward function (Seitz et al. 2017; Wallin et al. 2015). This manifests as a reduction in neurochemical and behavioural effects of a range of drugs of abuse (cannabinoids, Mhillaj et al., 2015; Dicky Struik et al., 2017) (cocaine, Kailanto, Kankaanpää, & Seppälä, 2011; Kurling-Kailanto, Kankaanpää, & Seppälä, 2010; Mhillaj et al., 2015) (amphetamines and alcohol, Mhillaj et al., 2015). In animals this was offset by increased drug (cannabis) administration (Struik et al. 2017). Thus, AAS users are considered to be at greater risk of initiating use, struggling with maintenance of use and development of addiction (Struik et al. 2018; Struik et al. 2017). Behaviours which continued beyond AAS elimination, suggesting changes to the central nervous system caused by AASs may be long-lasting (Struik et al. 2017; Kailanto et al. 2011; Kurling-Kailanto et al. 2010). Increased vulnerability to drug use in animals was reflected in surveys of human AAS users.

Frequency of substance misuse was demonstrated to be related to use of AASs (Lundholm et al. 2015; Sagoe et al. 2015). Substantial psychoactive drug use has been observed amongst AAS users (Hope et al. 2013; Lood et al. 2012; Lundholm et al. 2015; Hallgren et al. 2015; Molero et al. 2017). Almost 80% of UK AAS users have used an illegal drug (Chandler & Mcveigh 2013; Lorang et al. 2011)) and around one third (32%) reported recent (one year) use (Bates & McVeigh 2016; Mcveigh et al. 2015; Chandler & Mcveigh 2013), most commonly cannabis (24%) and cocaine (22%) (Bates & McVeigh 2016). In fact, cocaine is substantially more prevalent amongst AAS users than the general public (Hope et al. 2017; Kanayama et al. 2010). 46% of UK AAS users declared recent use (Hope et al. 2013; Ip et al. 2011) compared to 2.3 % of the UK general public (Home Office 2015).

As sex also stimulates reward centres in the brain, AAS induced dysfunction of reward circuits is postulated to influence sexual behaviour. AASs have been reported to increase

sexual arousal and desire in human subjects and exhibit dose-dependent stimulation of sexual behaviour in animals (Kim & Wood 2014). Rat models have shown, however, that there is no associated increase in willingness to work for sexual reward (Kim & Wood 2014). Rather, when sexual opportunities are presented they are more likely to be accepted. This combined with AAS induced increase in impulsivity and reduction in awareness of possible negative consequences (Hildebrandt et al. 2014), results in increased risky sexual behaviours (not using condoms and sex with multiple partners) (Begley et al. 2017; Hope et al. 2013). This is particularly true of homosexual AAS users who also reported unprotected sex with men of unknown HIV status (Bolding et al. 1999). Unsurprisingly, therefore, high testosterone levels have been linked with increased likelihood of contracting sexually transmitted disease (Booth et al. 1999).

8. How AASs are used

AAS user education and administration regimes tend to develop through imparted knowledge and experiences of other users (Chandler & Mcveigh, 2013) via on-line discussion forums, user produced websites and in gyms. Users share a strong sense of community which fosters an intense support network to encourage each other toward body image or performance goals (Hanley Santos & Coomber 2017). Members of the community can quickly learn and 'optimise' complex nutritional, exercise and AAS regimes (Hildebrandt et al. 2011), (Figure 2**Error! Reference source not found.**) as AAS use is not straightforward.
Steroids are typically utilised at steadily increasing doses, in the belief that ultimately much higher doses will be tolerated. 'Pyramiding' regimes are a continuous sequence of increasing and decreasing doses. 27% of UK users employ 'blast and cruise' regimes (Mcveigh et al. 2015; Sagoe et al. 2011); continuous 'blasts' of high dose AAS use interspaced with lower dose 'cruise' periods. 'Cruising' can still employ doses several times in excess of natural production (Bates & McVeigh 2016). To deal with the significant side effects exceptionally high AAS doses are likely to produce, most user regimes incorporate recovery periods (Ip et al. 2014; Jespersen 2012). Such regimes "cycle" through periods of administration and abstinence. The optimum cycle length is the subject of much user debate. The general guidelines suggest usage for 6 to 8 weeks (never more than 12) with an equal or longer off period (Llewellyn et al. 2011). Previously it was found that most users were adhering to this guideline (Chandler & Mcveigh 2013), but more recently the average cycle length of UK

AAS users was found to be 20 weeks (Bates & McVeigh 2016). This could perhaps indicate an increase of younger users who are generally more reckless in terms of cycle length and dosages (Brennan et al. 2016).

8.1. AAS Users and Polypharmacy

It is believed by users, but never scientifically demonstrated, that combining different AASs, or "stacking", will exert a synergistic effect on muscle growth i.e. the combined effects will be greater than the summed effects of each substance used individually. Stacking methods are increasing (Lood et al. 2012), with the vast majority of UK users combining both oral and injectable AASs (Chandler & Mcveigh 2013). Other potentially anabolic products, like human growth hormone or insulin may also be included (Brennan et al. 2016; Chandler & Mcveigh 2013; Jespersen 2012; Ip et al. 2011). Moreover, there are firm associations between AASs and use of other prescription or illicit drugs (Bates & McVeigh 2016; Hakansson et al. 2012; Leifman et al. 2011), with extensive polypharmacy practiced by many AAS users (Lundholm et al. 2015; Ip et al. 2014; Chandler & Mcveigh 2013; Ip et al. 2011).

Users incorporate, often numerous, substances into their regimes to prevent or self-treat a variety of side-effects (Sagoe et al. 2015; Ip et al. 2014; Ip et al. 2011). They may also utilise other IPEDs to better define and enhance the aesthetic appearance of the musculature achieved by AAS (Sagoe et al. 2015). For example, fat-loss agents are increasingly popular (Jespersen 2012). It is feared that the rapid results of combined AAS and fat-loss agent use are irresponsibly promoted within the AAS community. 85% of AAS users report incorporating a fat-loss agent (Hildebrandt et al. 2010), and 10% of UK AAS users declare use of life-threatening 2,4-Dinitrophenol (DNP) (Chandler & Mcveigh 2013; Bates & McVeigh 2016).

9. Psychiatric Effects

The complex AAS regimes and likely polypharmacy adopted by users, make studying and predicting behavioural changes in AAS users impossible. Administration of AAS compounds in animal studies and observations of human subjects displaying elevated levels of endogenous testosterone have predicted some of the behaviours expected in AAS abusers. Extreme anxiety, depression, irritability, increased aggression ("roid rage") (Lindqvist Bagge

et al. 2017; Rowe et al. 2017; Heimly Jenssen & Johannessen 2015), and violent behaviour (Lundholm et al. 2015; Advisory Council on the Misuse of Drugs 2010), have emerged as common responses.

These are all behaviours for which neural transmission mediated by GABA type A ($GABA_A$) receptors in various regions of the basal forebrain play a pivotal role. Chronic exposure to AASs has been shown to alter $GABA_A$ receptor subunit composition (Henderson et al. 2006) however, exploration of the effects of AASs on brain function is relatively new. The influence on pathways related to reproduction and sexual-behaviour are most well-known and studied (for review, Oberlander et al. 2012; Clark & Henderson 2003). Investigation of other characteristics are complicated by the impact of environment, in addition to age and sex (Oberlander et al. 2012; McIntyre et al. 2002). Shifts in emotion are more likely to be observed in response to specific environmental influences (threatening situations, availability of rewards like sex or drugs, etc.) (Hildebrandt et al. 2018). Expressions of aggression and violence have been shown to be unpredictable and context-dependent, and generally only encountered when triggered by a stimulus (Wallin et al. 2015; Kim & Wood 2014). Altered judgement pathways could make users' behaviours less flexible, and unable to adapt to changing situations (Wallin & Wood 2015). Similarly, naturally high testosterone levels have been correlated with reduced fear and diminished ability to empathise and make moral choices (Van Honk et al. 2005). The decision making process for affective behaviours are altered. These inabilities to modulate responses have serious consequences, such as increased incidence of violent offending (Lood et al. 2012) and intentional death (suicide/murder) (Thiblin et al. 2015; Darke et al. 2014). However, this may be compounded by co-administration of other psychoactive substances (Lundholm et al. 2015). Especially as animal studies suggest, that whilst AAS induced aggression may be a characteristic of adolescent AAS use, Anxiety is the more prominent attribute amongst adult users (Rowe et al. 2017; Olivares et al. 2014).

Anxiety has repeatedly been observed in animals exposed to AAS (Olivares et al. 2014; Onakomaiya et al. 2014; Oberlander & Henderson 2012; Ricci et al. 2012; Costine et al. 2010) and there is a disproportionate diagnoses of anxiety disorders amongst AAS users (Ip et al. 2011). It is therefore, surprising that long-term AAS use could actually have an anxiolytic effect in adults (Morrison et al. 2015). This suggests that the anxiety experienced in adulthood, could stem from an unrelated condition or it may be the result of AAS use in adolescence.

It is believed that adults experience aggression during AAS exposure and anxiety during withdrawal, relative to length of exposure (Lindqvist Bagge et al. 2017; Ricci et al. 2013). It is difficult to ascertain whether anxiety drives AAS use or is a result of use. Just as it is not clear whether heightened aggression may be an underlying personality trait of AAS users, rather than an outcome of use (Heimly Jenssen & Johannessen 2015). Aggression is only consistently observed with testosterone use and not significantly evidenced with other AASs (e.g. stanolozol may inhibit aggressive behaviours) (Tomlinson et al. 2016; Lumia & Mcginnis 2010). Testosterone however, is thought to be the most popular AAS in use (Mcveigh et al. 2015; Hildebrandt et al. 2010).

9.1. Female AAS Users

Initial work with female non-human subjects suggests that it is not possible to simply expect the same effects in female AAS users as male (Clark et al. 2006; Henderson et al. 2006). Hormone signalling pathways change naturally with age, sex and hormonal state. And there are sex-specific differences in endogenous hormones, hormone receptors and expression of hormone-metabolising enzymes. AAS treatment of mice indicated dose-dependent changes to the female brain that were not evident in males (Henderson et al. 2006).

Female subjects administered a relatively low dose of testosterone were found to be predisposed to antisocial behaviour (Van Honk & Schutter 2007). This was due to the AAS significantly reducing their ability to detect threat (Van Honk & Schutter 2007) and feel fear (Van Honk et al. 2005). This could provide some explanation as to why AAS use was found to be greater amongst females who had committed crime, than females in general (Lundholm et al. 2010). What is not clear however, is whether the increased criminal involvement could be a function of co-occurring poly-drug use (Lundholm et al. 2015).

9.2. Adolescent AAS Use

The adolescent brain, still in development, is more susceptible to negative effects of AAS use (Lumia & Mcginnis 2010) (for review, (Clark & Henderson 2003), which can change cell types and activity patterns within the hypothalamus (Morrison et al. 2016). The adolescent brain is primed for steroid dependent changes, thus changes may occur which would not be seen in adult users (Henderson et al. 2006). Many of these changes are expected to be

permanent (Clark et al. 2006). Even a single AAS administration in subjects so vulnerable to hormonal change, could adversely affect cognitive processes such as learning and memory (Ramos-Pratts et al. 2013).

Effects on social behaviours most often recognised in animals were aggressive actions (Rowe et al. 2017; Olivares et al. 2014). This translates well to human activity. Adolescent boys who use AASs were consistently found to be engaged in more serious acts of aggression (e.g. burglary, rape and/or use of weapons), and anti-social behaviour (e.g. criminality, bullying and/or truancy), compared to those who use other illegal drugs or who have no substance abuse history (Hallgren et al. 2015). These findings also reflect the greater impairment of inhibition in adolescents compared to adult users (Hildebrandt et al. 2014). Some of the repercussions on social behaviours may not even be evident until adulthood, when AAS use has been discontinued (Olivares et al. 2014; Salas-Ramirez et al. 2010). Short-term exposure of male rats to AASs during adolescence was discerned to promoted depressive or anxious-related behaviours in adulthood (Rainer et al. 2014).

AAS induced changes have been found to be greater, and more likely to be permanent, in female adolescents (Clark et al. 2006). In young female users AAS use can influence onset of puberty and expression of sexual behaviours (Clark et al. 2006).

9.3. AAS Use and Cognitive Impairment

There is very little data concerning active AAS users and cognitive effects. AAS use only became reasonably widespread throughout the late eighties and early nineties and the long term effects are therefore only now becoming discernible (Kanayama et al. 2013). One emerging complication is neurotoxicity. Experimental evidence supports AASs permanently alter brain structure and function (Bjørnebekk et al. 2017; Seitz et al. 2017; Westlye et al. 2016; Kanayama et al. 2013; Caraci et al. 2011) causing changes related to mental health and cognitive deficits (Westlye et al. 2016). The pathway of the neurodegeneration which results from AASs use is complex and little understood. What is known is that apoptotic mechanisms contribute, at least in part, to the pathophysiology (Pomara et al. 2015). Long term high dosages of AASs may be linked with the onset of Alzheimer's disease (Kaufman et al. 2015; Kanayama et al. 2013). Long term AAS users are not expected to have used the excessive doses in use today, in their early AAS careers. Yet, a degree of brain damage has been demonstrated as reduced visuospatial learning and memory (Kanayama et al. 2013). As

a ‘new’ drug, most long term users are not of an age at which cognitive dysfunction is perceptible.

10. Limits of this review

Whilst there are many accounts of the extreme popularity of AASs for image enhancement purposes (from users, medical professionals, needle exchanges, etc), published research in support of such anecdotes is lacking. It is incredibly difficult to predict physiological and behavioural consequences of AAS abuse as these are now recognised to vary with AAS structure, metabolism and administration pattern (Clark et al. 2006; Henderson et al. 2006). The metabolites of designer AASs are not the same as the products that result from endogenous androgenic compounds. The interaction of such compounds with androgen and estrogen receptors are not well documented, particularly at the concentrations associated with abuse (Henderson et al. 2006). Differing abundances and chemical structures may result in varied interactions with the neuroendocrine systems AASs have been shown to influence. This may help to explain some of the contradictory information arising from animal studies of behaviours related to depression and anxiety. Thus, the effects of each AAS must be studied individually. What is presented here can only provide a general overview of the more common AASs.

Furthermore, the majority of studies, both human and animal, focus on post adolescent males. This has resulted in significant dearth of information and understanding of the physical and psychological ramifications of steroid use in adolescent and in particular female users. In fact, the lack of studies on AASs, in comparison to other psychoactive drugs of abuse, may perpetuate the apparently false belief of ‘safeness’ amongst users and policy makers.

Understanding how to tackle the problem of increasing AAS use is extremely difficult. There are many complex psychological and social routes to AAS use, many of which are not fully understood. There have been no studies to evidence successful interventions in relation to anabolic steroids. Currently, there are no formal academic evaluations of harm reduction, treatment or prevention interventions in the United Kingdom or elsewhere (Petróczi et al., 2014). However, usage patterns would suggest that interventions which focus on recreational gym users and target on-line supply, might have greatest success.

11. Conclusion

The true extent of AAS use in the UK is unknown but expanding. Data gathering on AAS use within the UK largely targets those with an on-line presence. Improvements are required to try and better represent others within the AAS using community, particularly those most at risk, females and adolescent boys. The specific AASs in common circulation are unknown but rely on user reports.

AASs are very easily obtained, usually from online sources. As the use of health supplements has been strongly associated with AAS use, and these have been found to contain AAS substances, better regulation of this market could be beneficial. Particularly as secondary school children are amongst the consumers. Further research is required in order to establish other potential triggers for AAS use in order to guide policy makers and public health initiatives to those most at risk.

The complexity of different usage patterns makes it extremely difficult to evaluate the negative effects of AAS use. This is further complicated by the strong association between AAS use and other drug misuse. AASs alter the behavioural effects and the rewarding properties of drugs of abuse and appear to be linked with addition. The extent of polypharmacy now observed is troubling, as the toxicity of AASs when taken in combination with other substances is not known. The immediate dangers of AAS use appear to be the unreliability of composition and sterility of injectable products. The susceptibility of AAS users to BBVs needs to be addressed by targeting improved sexual behaviours.

Harm posed by AASs still cannot be fully assessed, although they do appear to be commensurate with aggression, violence and criminality. As the long term AAS using cohort reach maturity, further studies, with larger sample sizes, are required to investigate the potential for the severe negative health effects associated with their use. Particularly to the cardiovascular system, cerebrovascular, renal and hepatic systems and associated decreases in cognitive function.

12. Competing Interests

None

13. Declaration of transparency and scientific rigour

This Declaration acknowledges that this paper adheres to the principles for transparent reporting and scientific rigour of preclinical research as stated in the BJP guidelines for Design & Analysis, and as recommended by funding agencies, publishers and other organisations engaged with supporting research.

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Table 1: Anabolic Androgenic Steroids available from on-line sources. Presented are some of the many AAS substances which are available to buy on-line. Those which are believed to be most popular amongst certain user groups have been highlighted (ł popular amongst those purchasing on-line (Dynamic Sports Nutrition 2016), ⁱ popular amongst female users (Jespersen 2012), * popular amongst UK users (Bates & McVeigh 2016)). The information presented comes from on-line user reports and supplier marketing information.

Chemical Name	Commercial Name	Formulation	Pharmacology	Recommended effective dose (on-line community)		Popularity
				Male	Female	
androisoxazol	Neo-Ponden	oral	Cutting (fat loss)	15-40 mg/day	5-10 mg/day	
bolasterone	Myagen	oral	Bulking (muscle gain)	50-100 mg		
bolazine caproate	Roxilon Inject	Injectable	Cutting	100-500 mg/week	50-100 mg/week	
boldenone blend	Equilon 100	injectable	All purpose	400 - 600 mg/week		
boldenone undecylenate	Equipoise®	injectable	All purpose	200-600 mg/week	50-100 mg/week	ł
boldenone/methylandrostenediol blend	Drive®	injectable	All purpose	300-600 mg/week		
calusterone	Methosarb	oral	Cutting	200 mg/day	200 mg/day	
chlorodehydromethylandrostenediol	Halodrol	oral	Bulking and strength	100-150 mg/day	Not recommended	
4-chlorodehydromethyltestosterone	Oral Turinabol	oral	Cutting	15-40mg/day	2.5-5mg/day	
chloromethylandrostenediol	Promagnon	oral	All purpose	50-100 mg	Not recommended	
clostebol acetate	Megagrisevit-Mono®	oral	Bulking	100-200 mg/day		
danazol	Danocrine®	oral	Androgenic	400 mg/day		
demethylstanozolol tetrahydropyranyl	Prostanozol	oral	Cutting	100-150 mg/day	25 mg/day	
desoxymethyltestosterone	Madol	oral	Bulking and strength	40-60 mg/day		
dihydroboldenone	1-Testosterone, DHB	oral	Bulking	50-75 mg/day		
dihydrotestosterone	Andractim®	transdermal	Reduce gynecomastia	25 mg x2/day		
dihydrotestosterone	Neodrol	injectable	All purpose	Generally advised against use		
dimethazine	Roxilon	oral	All purpose	10-20 mg/day	2.5 mg/day	

dimethyltrienolone	Dimethyltrienolone	oral	All purpose	20-40 mg/day	Not recommended	
drostanolone enanthate	Masteron®	injectable	Cutting	300-400 mg/week		ǂ
drostanolone propionate	Masteron®	injectable	Cutting	100 mg/2 days	50-100 mg/week	ǂ *
ethylestrenol	Orabolin®	oral	Bulking	40 mg	10 mg	
fluoxymesterone	Halotestin®	oral	Cutting	10-40 mg/day		ǂ
formebolone, formyldienolone	Esiclene®	Injecting (oral available)Injecting (oral available)	Bulking	As required (e.g. pre competition)		Irritant at injection site
furazabol	Miotolan®	oral	Bulking/ Reduce colesterolBulking/ Reduce colesterol	50-100 mg/day		
hydroxytestosterone	Hydroxytest	injectable	Bulking	300-2000 mg/week	Not recommended	
mepitiostane	Thioderon	oral	Cutting	25-50 mg/day	20 mg/day	
mestanolone	Ermalone	oral	Cutting	10-20 mg/day	Not recommended	
mesterolone	Proviron®	oral	Cutting	25-200 mg/day		
methandrostenolone, methandienone	Dianabol®, Dbol	oral	Bulking	15-50 mg/day		ǂ
methenolone acetate	Primobolan®	oral	Cutting	50-150 mg/day	25-75 mg/day	ǂ
methenolone enanthate	Primobolan® Depot	Injectable	Cutting	350-600 mg/week	100 mg/week	ǂ
methepitiostane	Havoc	oral	Cutting	10-20 mg/day	5 mg/day	
methylandrostenediol	Methandriol	Oral or injectable	Bulking	30-50 mg/day (o) 300-500 mg/week (i)		
Methyldienolone	Methyl-D	oral	Bulking	2-10 mg/day	Not recommended	
methyldihydroboldenone	Methyl-1-testosterone	oral	All purpose	10-50 mg/day	2.5 mg/day	
methyldrostanolone	Superdrol	oral	Cutting	10-30 mg/day		
methylhydroxynandrolone	MOHN	oral	All purpose	10-30 mg/day	Not recommended	
methylnortestosterone acetate	MENT	oral	Bulking	10 mg/day	Not recommended	

methyltestosterone	Metandren	oral	Bulking	25-100 mg/day		
methyltrienolone	Metribolone	oral	Cutting	500-750 mg/day		
mibolerone	Cheque Drops®	oral	Strength	200-500 mg/day		
nandrolone blend	Dinandrol	injectable	All purpose	200-600 mg/week	50 mg/week	
nandrolone cyclohexylpropionate	Fherbolico	injectable	Bulking	200-400 mg/week	50-100 mg/week	
nandrolone cypionate	Dynabol®	injectable	Bulking	200-400 mg/week	50-100 mg/week	
nandrolone decanoate	Deca-Durabolin®	injectable	Bulking	200-600 mg/week	50-100 mg/week	ǂ *
nandrolone hexyloxyphenylpropionate	Anadur®	injectable	Bulking	200-600 mg/week	50-100 mg/week	
nandrolone laurate	Laurabolin®	injectable	Bulking	200-600 mg/week	50-100 mg/week	
nandrolone phenylpropionate	Durabolin®, NPP	injectable	All purpose	300-400 mg/week	50 mg/week	
nandrolone undecanoate	Dynabolon®	injectable	All purpose	160-600 mg/week		
nandrolone/methandriol blend	Libriol	injectable	Bulking	200-600 mg/week	50-100 mg/week	
nandrolone/methandriol blend	Tribolin	injectable	Bulking (weak)	200-400mg/week	50-100 mg/week	
nandrolone/methandriol blend	Nandrabolin	injectable	Bulking (weak)	200-400mg/week	50-100 mg/week	
norbolethone	Genabol	oral	Bulking (weak)	10-15 mg/day	5 mg/day	
norclostebol acetate	Anabol 4-19	injectable	Bulking/cutting	200-600 mg/week	50-100 mg/week	
norethandrolone	Nilevar®	oral	Bulking/cutting		20-40 mg/day	
normethandrolone	Orgasteron	oral	Bulking	10-40 mg/day	2.5-10mg/day	
oxabolone cypionate	Steranabol Ritardo	injectable	Bulking	300-800 mg/week	40-150 mg/week	
oxandrolone	Anavar, Oxandrin, LonovarAnavar, Oxandrin, Lonovar	oral	Cutting	50-80 mg/day	10-20 mg/day	ǂ
oxymesterone	Oranabol	oral	Cutting	20-40 mg/day	10 mg/day	
oxymetholone	Anadrol®- 50	oral	Bulking	100mg/day		ǂ
quinbolone	Anabolicum Vister	oral	All purpose		50-100 mg/day	
stanozolol	Winstrol®	Oral	Cutting	25-50 mg/day	5-10 mg/day	ǂ

stanozolol	Winstrol® Depot	Injectable	Cutting	50 mg/day	20mg/4 days	
stenbolone acetate	Anatrofin	Injectable	Cutting	350-700 mg/week		
testosterone	Androderm®	transdermal	All purpose	2.5-5mg/day		
testosterone	AndroGel®	transdermal	All purpose	5-10g/day		
testosterone	Striant®	Sublingual	All purpose	30mg 2x/day		
testosterone	Testoderm®	transdermal	All purpose	300-2000mg/week	Not recommended	
testosterone	Testopel®	Subcutaneous implant	All purpose	150-450 mg/3-6 months		
testosterone blend	Deposterona	injectable	All purpose	400-600 mg/week		
testosterone blend	Equitest 200	Injectable	All purpose	200-600 mg/week		
testosterone blend	Omnadren® 250 Testosterone-Propionate, Testosterone-Phenylpropionate, Testosterone-Isocaproate, Testosterone-Caproate, Primobolan	injectable	All purpose	250-1000 mg/week	Not recommended	
testosterone blend	Sustanon® 100 Propionate, Phenylpropionate, Isocaproate, Decanoate	injectable	All purpose	500-2000 mg/week	Not recommended	
testosterone blend	Sustanon® 250	injectable	All purpose	500-2000 mg/week	Not recommended	ǂ *
testosterone blend	Triolandren	injectable	All purpose	200-400mg/week	25mg/week	
testosterone buciclate	20 AET-1	injectable	All purpose	1000mg/month	Not recommended	
testosterone cyclohexylpropionate	Andromar Retard	injectable	All purpose	300-600mg/week		
testosterone cypionate & propionate	Sten	injectable	All purpose	200-2000mg/week	Not recommended	

testosterone cypionate	Depo®-Testosterone	injectable	All purpose	300-2000mg/week	Not recommended	ǂ *
testosterone decanoate	Neotest 250	injectable	All purpose	300-2000mg/week	Not recommended	
testosterone enanthate	Delatestryl® or Primosteston	injectable	All purpose	300-2000mg/week	Not recommended	ǂ *, Extremely popular on-line, and most common in UK
testosterone hexahydrobenzoate	Sterandryl Retard	injectable	All purpose	300-2000mg/week	Not recommended	
testosterone isobutyrate	Agovirin Depot	injectable	All purpose	300-2000mg/week	Not recommended	
testosterone nicotinate	Bolfortan	injectable	All purpose	300-2000mg/week	Not recommended	
testosterone phenylacetate	Perandren	injectable	All purpose	300-2000mg/week	Not recommended	
testosterone phenylpropionate	Testolent	injectable	All purpose	35-1000 mg/week		
testosterone propionate & estradiol	Synovex®	injectable	All purpose	300-2000mg/week	Not recommended	
testosterone propionate	Oreton	injectable	All purpose	200-400mg/week	25mg/week	ǂ *
testosterone propionate/enanthate blend	Testoviron®	injectable	All purpose	100-1000mg/week		ǂ
testosterone suspension	Andronaq	injectable	All purpose	50-200mg/day	Not recommended	
testosterone undecanoate	Andriol®	oral	All purpose	120-500mg/day		
testosterone undecanoate	Nebido	injectable	All purpose	1000mg/month	Not recommended	
testosterone/estrogen blend	Estandron	Injectable	All purpose	1000-1500mg/week		
testosterone/nandrolone/methandriol blend	Spectriol	Injectable	All purpose	200-2000mg/week	Not recommended	
tetrahydrogestrinone	THG	oral	Bulking	2-5mg/day	Not recommended	

thiomesterone	Emdabol	oral	Bulking	15-25mg/day	Not recommended	
trenbolone acetate	Finajet, Fina	injectable	All purpose	50-100mg/2nd day	Not recommended	ǂ *
trenbolone enanthate	Trenabol	injectable	All purpose	150-450mg/week		*
trenbolone hexahydrobenzylcarbonate	Parabolan®	injectable	All purpose	300-500mg/week	Not recommended	

Table 2: AASs in use in the UK (Bates & McVeigh 2016)

Anabolic Androgenic Steroid	Percentage of IPED users using in past year
Testosterone enanthate	60
Sustanon	43
Testosterone propionate	38
Nandrolone Decanoate	36
Underground lab blend	34
Trenbolone acetate	33
Testosterone cypionate	30
Drostanolone propionate	27
Trenbolone enanthate	25
Boldenone (Equipoise)	25
Stanozolol (oral AAS)	11
Testosterone Suspension	9

Table 3: Estimated AAS use in the UK (Home Office 2018)

	Recent Drug Use	Drug Use in Last Year		
	16-59 year olds	16-59 year olds		16-24 year olds
Year	Users (up to)	Percentage	Users (up to)	Percentage
2017/18	361,000	0.2	84,000	0.3
2016/17	411,000	0.2	83,000	0.4
2015/16	320,000	0.2	75,000	0.1
2014/15	340,000	0.2	98,000	0.5
2013/14	317,000	0.2	91,000	0.5
2012/13	311,000	0.2	79,000	0.2
2011/12	262,000	0.2	89,000	0.3
2010/11	243,000	0.2	68,000	0.3
2009/10	259,000	0.1	65,000	0.4
2008/09	212,000	0.1	61,000	0.3
2007/08	215,000	0.1	28,000	0.1

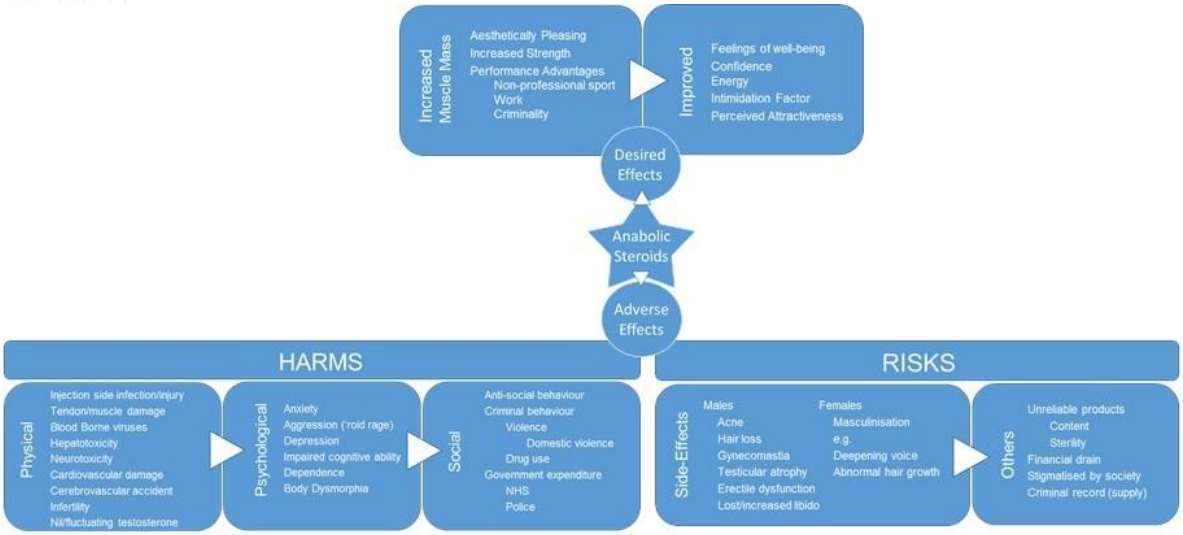

Figure 1: The Effects of Anabolic Androgenic Steroid Use; the physical and psychological motivators for AAS use, contrasted with the numerous negative consequences of AAS use. The adverse effects range from minor side-effects to serious physical and psychological harms.

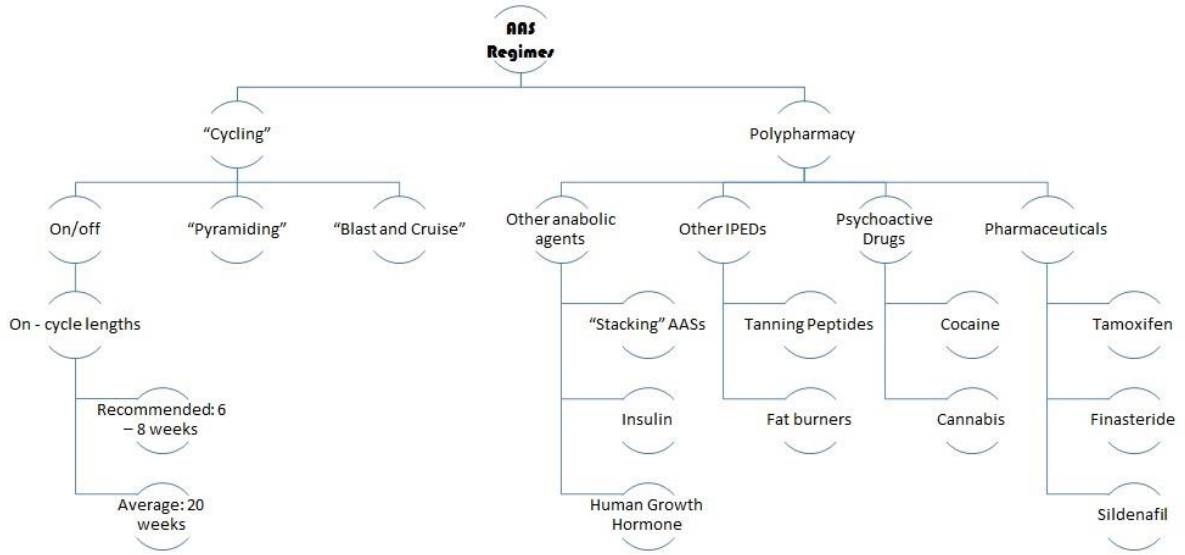

Figure 2: Anabolic Androgenic Steroid usage regimes with some examples of common polypharmacy