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Anabolic-Androgenic Steroid Use and Body Image in Men: A Growing Concern for Clinicians

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Recent decades have seen increasing attention to disorders of body image [1]. In the past, most body-image studies have focused on women [2], and especially women with eating disorders [3], but now a growing literature has also begun to address body image disorders in men [4]. In particular, it appears that today's men have become increasingly preoccupied with having a lean and muscular body, perhaps as a result of constant exposure to lean and muscular male images in movies, television, advertising, and elsewhere [1, 5]. Modern mental health professionals are very likely to encounter male patients who harbor such concerns. Importantly, many of these men use drugs (or "dietary supplements" containing drugs) in order to gain muscle or lose body fat. The use of these "body image drugs" had already surfaced as a clinical issue 20 years ago [6] and has generated increasing attention in recent years [7]. From a public health standpoint, the most concerning of these substances are the anabolic-androgenic steroids (AAS) – the family of hormones that includes testosterone and its synthetic derivatives. In this paper, we present a clinical update on AAS use.

Drugs in the AAS family all possess both *anabolic* (muscle-building) properties and *androgenic* (masculinizing) properties [8]. Soon after the identification of testosterone in the late 1930s, athletes discovered that AAS could allow them to greatly increase muscle mass, and attain levels of performance beyond that previously attained by "natural" athletes [9]. Consequently, AAS use spread rapidly through the elite athletic world from the 1950s through the 1970s, especially in sports requiring muscle strength, such as field events, weightlifting, and bodybuilding. However, it was not until the 1980s that AAS use began to spill out of the elite athletic world and into the general population. Nowadays, most AAS users are not competitive athletes, but simply men using these drugs primarily for personal appearance [10, 11]. About 98% of AAS users are male [12], in part because women rarely desire to be extremely muscular, and are also vulnerable to the androgenic effects of these drugs, such as beard growth, deepening of the voice, and masculinization of secondary sexual characteristics [13]. Consequently, the discussion below is focused on male AAS users.

AAS users frequently use several AAS simultaneously (a practice called "stacking"), often combining both injectable AAS and orally active AAS [8]. For example, a typical stack might include injected testosterone cypionate 400 mg per week plus injected nandrolone decanoate 400 mg per week plus oral methenolone 50 mg per day. Users may also add other appearance- performance-enhancing drugs to the stack (e.g., human growth hormone, clenbuterol, insulin, etc.), as well as drugs to counteract the side effects of AAS, such as anti-estrogens to prevent

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gynecomastia. Discussion of these other classes of drugs can be found elsewhere [8, 14–16]. Although AAS and these other drugs are illegal without a prescription in most developed Western countries, the drugs are readily available through local underground drug dealers and through numerous Internet sites. In addition, many "nutritional supplements," purchased over the counter or online [17], may contain surreptitious AAS or other ingredients of uncertain efficacy and toxicity [18].

In the 21st century, AAS use has continued to spread widely among men around the world, especially in Nordic countries, the United States, British Commonwealth countries, and Brazil, with many other Western countries following not far behind. AAS use remains rare in east Asia, however, apparently because the Confucian and related traditions in these societies place little value on musculature as a measure of masculinity [19, 20], whereas in the West, muscularity has been celebrated since ancient times, with a particular emphasis in the last several decades [21, 22]. At present, some tens of millions of men worldwide have used AAS, and thus AAS use arguably represents the youngest of the world's major substance use disorders.

Because AAS use is so new, science has only begun to appreciate the potential adverse effects of these substances. Other drugs, such as alcohol, cannabis, and opioids, have been used for thousands of years, and many decades of research have now evaluated the effects of these substances. By contrast, most of the world's older AAS users, those who first tried these drugs as youths in the 1980s or 1990s, are only now reaching middle age. Consequently, it is only in the last decade or two that it has become feasible to study the long-term effects of AAS [23]. For further discussion of the current state of knowledge, we refer the reader to our earlier comprehensive review [8]. Below, we briefly summarize this literature, with emphasis on effects most likely to be encountered by mental health clinicians.

Medical Effects

Cardiac Effects

The best-documented medical hazards of long-term AAS exposure involve the cardiovascular system. This is perhaps not surprising, since AAS affect muscles, and the heart is not only the strongest muscle in the body, but also the only muscle that never rests. Multiple studies have now shown that AAS use can lead to a cardiomyopathy, with compromised systolic function (e.g., an inadequate left ventricular ejection fraction) and diastolic function (e.g., impaired diastolic tissue velocity) [24, 25]. Tentative evidence, however, suggests that this cardiomyopathy may partially resolve after AAS are discontinued [24]. Perhaps more serious, however, is the association between long-term AAS exposure and atherosclerotic disease, which appears largely attributable to dyslipidemia caused by AAS [24, 26]. Multiple case reports and small case series have now documented myocardial infarctions and cerebrovascular accidents in young AAS users, and it seems likely that these events will become increasingly common as this population ages [8, 27].

Hypogonadism

Another ominous finding is that AAS users may develop prolonged hypogonadism after discontinuing these drugs [28–30]. In males, testosterone and other AAS all suppress natural production of testosterone and of spermatozoa via feedback inhibition. Thus, when a user discontinues AAS, especially if he has been using AAS for a prolonged period, his own testosterone level will often fall well below the lower limit of normal. This AAS withdrawal hypogonadism may persist for months, and recent evidence suggests that in some cases it may never become fully reversible. As a result, users may suffer prolonged intervals of decreased or absent libido, erectile dysfunction, infertility, and in some instances, major depressive disorder (discussed below). Faced with these effects, users may often resume taking AAS to self-treat the withdrawal symptoms and may ultimately develop an AAS dependence syndrome where they continue to use these drugs without interruption for years of time.

Other Organ Systems

Long-term AAS exposure may affect other organ systems, although in many cases our understanding of these effects remains limited. AAS may cause hepatotoxicity (although this is uncommon), nephrotoxicity, and possibly neurotoxicity, perhaps with a potential for causing early-onset dementia [31]. AAS users are also at a greater risk for some orthopedic problems, especially ruptured tendons, which can arise when the muscle grows too strong for its attached tendon. Lesser problems include truncal acne, gynecomastia caused by the aromatization of AAS into estrogenic compounds, and hair loss. Use of injections may lead to sepsis or to transmission of viral pathogens, although needle-sharing is rare among AAS users [10]. The reader is referred to recent reviews for more background on these issues [8, 32, 33].

Psychiatric Effects

Another category of AAS effects are the psychiatric effects. Among these are: (1) major mood disorders, which may be associated with aggression, violence, and sometimes criminal behavior; (2) muscle dysmorphia, which may be both a cause and an effect of AAS use; and (3) AAS dependence syndromes.

Major Mood Disorders

Starting in the 1980s, case reports and field studies began to appear, suggesting that AAS might cause some individuals to develop manic or hypomanic syndromes during AAS exposure and depressive symptoms, sometimes associated with suicidal ideation and, rarely, completed suicide, during AAS withdrawal [8, 34–39]. Manic or hypomanic reactions are idiosyncratic, affecting only a minority of AAS users, but may appear quickly, within days or weeks after AAS use is initiated. These reactions appear somewhat dose-related, occurring more commonly in individuals taking more than 1,000 mg of testosterone equivalent per week (which represents 15–20 times the natural male production of testosterone) [8, 34]. Depressive episodes typically arise within weeks of starting AAS withdrawal, but are also idiosyncratic, affecting only a minority of users [8, 40]. Importantly, several reports have described men with little or no history of psychiatric disorders or of criminal behavior prior to AAS use who committed murder or other violent crimes when exposed to AAS [8, 35, 39, 40–46]. In most of these reported cases, criminal behavior appeared associated with hypomanic symptoms, such as inflated self-esteem or grandiosity, hyperactivity, and involvement in other types of activities with a high potential for painful consequences.

Despite the evidence from the observational studies above, questions remained as to whether the psychological effects observed might be attributable to factors other than AAS themselves – such as underlying personality factors, social effects from the AAS-using subculture, or expectational effects. To address these questions, several placebo-controlled double-blind studies have administered supraphysiologic doses of AAS to volunteers. There have now been four such studies using doses of at least 500 mg per week of testosterone or equivalent AAS [47–51]. Of the 109 men who received AAS at 500 mg per week or greater under blinded conditions in these four studies, there were 5 (4.6%) who displayed hypomanic or manic syndromes on AAS, as compared to no such cases on placebo. In one such study [49], a participant with no prior psychiatric history became so aggressive during blinded methyltestosterone administration that he requested to be placed in a seclusion room. In another study [48], a previously asymptomatic man became so aggressive during blinded testosterone administration that he frightened people at his workplace and needed to be withdrawn from the study for safety. In the course of this latter study, the investigators administered the Cherek Point Subtraction Aggression Paradigm, a protocol designed to elicit aggressive responses, to a subgroup of the study participants [52]. These participants displayed strikingly and significantly higher levels of aggression when they were receiving injections of testosterone as compared to placebo under double-blind conditions. In summary, therefore, it is now generally agreed that these AAS effects cannot be explained purely by environmental factors and likely have a biological basis that has yet to be fully elucidated.

Muscle Dysmorphia

Muscle dysmorphia is a form of body dysmorphic disorder in which an individual becomes concerned that he is not sufficiently muscular. Although they may in fact be very muscular, individuals with muscle dysmorphia may constantly inspect their body size in the mirror, avoid situations in which their bodies might be seen in public for fear that they would look "too small," compulsively lift weights to gain muscle, engage in rigorous high-protein low-fat diets, and frequently go on to use AAS. Although body dysmorphic disorder was recognized as early as the 19th century [53], the subtype of muscle dysmorphia was first described only about 20 years ago [54, 55]. In the last two decades, muscle dysmorphia has now become the subject of a growing literature, and has now been included in the fifth edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)* as a specific identified subtype of body dysmorphic disorder [56, 57]. The prevalence of the condition remains uncertain, but it is likely more common than generally believed, since victims rarely disclose their preoccupations to outsiders.

Not surprisingly, AAS use appears widespread among individuals with muscle dysmorphia [58, 59]. For example, in one study comparing 24 male weightlifters with muscle dysmorphia versus 30 normal comparison weightlifters, 46% of the men with muscle dysmorphia reported use of AAS as compared to only 7% of the comparison men [60]. Another study compared 28 bodybuilders to 30 men with eating disorders and 30 comparison men [61]. Although the bodybuilders had not been selected for the presence of muscle dysmorphia, they showed levels of

body dissatisfaction, comparable to the men with eating disorders and significantly greater than the comparison men; 93% of the bodybuilders, but none of the men in the other groups, admitted to use of AAS.

Paradoxically, however, when individuals with muscle dysmorphia use AAS and gain substantial amounts of muscle, they often experience little relief from their condition, and may instead become even more preoccupied with their perceived deficits in muscularity. This phenomenon may be partially attributable to mingling with other AAS users in the underground AAS subculture, where there is much discussion of body image and where users may potentiate each other's preoccupations. Unfortunately, individuals with muscle dysmorphia rarely seek psychiatric treatment, and we are not aware of any formal studies of therapeutic interventions for this specific form of body dysmorphic disorder, although serotonin reuptake inhibitors and cognitive behavioral therapy have been shown effective for other forms of body dysmorphic disorder [62, 63].

AAS Dependence

Although AAS are not addictive in the classical sense, a surprisingly high percentage of AAS users develop an AAS dependence syndrome. AAS dependence shares many features with dependence on classical drugs of abuse (e.g., tolerance, withdrawal, large amounts of time devoted to obtaining and using the drug, continued use despite adverse effects), but differ somewhat in that AAS do not deliver an immediate "reward" of intoxication in the manner of most classical drugs. For further discussion of AAS dependence, we refer the reader to our earlier review [64] and to a paper proposing criteria for diagnosing this syndrome [65]. Technically, in the terminology of DSM-5 [56], this syndrome would simply be labeled as an "AAS use disorder" of moderate or greater severity, but the term "AAS dependence" has been generally used in the literature. One analysis, examining pooled data from 10 studies worldwide that had assessed the prevalence of AAS dependence in various populations of users, found that 33% of users had experienced a dependence syndrome [12]. This high prevalence of dependence appears to arise via at least three separate pathways. First is the "body image pathway": men with muscle dysmorphia will often gravitate to AAS use, as just discussed, and then become reluctant to stop using AAS because they develop anxiety if they lose even a small amount of muscle mass [66]. Second is a "neuroendocrine pathway": as also mentioned above, men will frequently become hypogonadal after stopping a course of AAS and will then be tempted to resume using AAS after a short period in order to self-treat loss of libido, impaired erectile function, and possible depression [67]. Third, recent research has shown a "hedonic pathway," which has been well demonstrated in rodent models. For example, male hamsters will self-inject testosterone to the point of death, suggesting that they are experiencing a hedonic effect from the drug. This self-administration occurs even when the testosterone is administered directly into the cerebral ventricles, suggesting that dependence in hamsters cannot be attributed to a peripheral action of the drug [68]. Human AAS users do not uniformly describe a hedonic effect from AAS, but many describe feelings of greater self-confidence, or even a sense of being "invincible" when using the drugs – and these rewarding feelings may well contribute to the evolution of a dependence syndrome [66].

Clinical Evaluation

As increasing numbers of men use AAS, and as more of them move into middle age, mental health professionals will likely encounter more patients with AAS-related problems. However, AAS users are often skeptical of clinicians and reluctant to disclose their AAS use [69]. Thus, clinicians must be alert for signs of surreptitious AAS use, particularly in men who report weightlifting and display unusual muscularity. Other physical signs (e.g., truncal acne, gynecomastia, or prominence striae above the pectoralis muscles) and laboratory measures (e.g., abnormally high hematocrit, low HDL cholesterol, or high levels of testosterone along with suppressed luteinizing and follicle-stimulating hormones) may further increase a treater's index of suspicion (Table 1). Note that if an AAS user is taking other types of AAS but not testosterone, his testosterone level will be abnormally low, but LH and FSH will also be low. AAS users may also show elevated levels of alanine aminotransferase, aspartate aminotransferase, and creatine kinase, but these are nonspecific findings, since all of these enzymes are present in muscle, and may be substantially elevated from the trauma of weightlifting alone, even in the absence of AAS. In any case where the clinician has suspicions, a gentle and nonjudgmental inquiry may help to uncover an AAS history [70]. Upon obtaining such a history, clinicians should assess lifetime doses and durations of AAS use with particular attention to the possibility of AAS dependence. They should also inquire about comorbid conditions, including use of other appearance- and performance-enhancing drugs, classical substance use disorders (which are common among AAS users), muscle dysmorphia and other forms of body dysmorphic disorder, and

Table 1. Laboratory abnormalities in anabolic-androgenic steroid users

Blood chemistries	
Muscle enzymes	Increased CK, ALT, AST, and LDH
Liver function tests	Increased ALT, AST, LDH, GGT and total bilirubin (caution: increased ALT, AST, and LDH are often muscular in origin and do not indicate liver disease)
Lipids	Decreased HDL-C, increased LDL-C Increase or no change in total cholesterol and triglycerides
Hormonal levels	Increased testosterone and estradiol (with use of testosterone esters) Decreased testosterone (in individuals using other AAS but not testosterone) Decreased FSH and LH
Hematology	Increased RBC count, hemoglobin, and hematocrit
Urine testing	Positive for AAS in individuals currently or recently on AAS May be positive for other drugs of abuse as well
Electrocardiogram	Left ventricular hypertrophy (seen in intensive weight trainers also)
Echocardiogram	
Systolic function	Decreased left ventricular ejection fraction
Diastolic function	Decreased left ventricular relaxation velocity
Semen analysis	Decreased sperm count and motility, abnormal morphology

AAS, anabolic-androgenic steroids; CK, creatine kinase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; GGT, gamma-glutamyl transferase; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LH, luteinizing hormone; FSH, follicle stimulating hormone; RBC, red blood cell.

major mood disorders – especially hypomanic symptoms during AAS exposure and depressive symptoms during AAS withdrawal. A more detailed discussion of the suggested history, physical examination, and laboratory testing of AAS users can be found elsewhere [71].

Treatment

Unfortunately, because AAS use and dependence are relatively new, there is little research on the treatment of this condition. Moreover, substance use treatment facilities are often poorly informed about AAS use, and standard paradigms for treating classical substance use disorders may not be well suited for AAS users. Despite this paucity of data, we can offer some recommendations, summarized in Figure 1, which portrays the three pathways to AAS dependence enumerated above. As noted in the figure, the anabolic and androgenic effects of AAS result from entry of the AAS molecules into the nuclei of cells, where these molecules bind to intra-nuclear androgen receptors, which in turn generates messenger RNA that ultimately mediates these effects throughout the body. By contrast, the hedonic effects of AAS are believed to result from binding of AAS molecules to cell membranes, particularly in the nucleus accumbens. To effectively manage AAS dependence, it seems important to address all three pathways simultaneously when initiating treatment in order to maximize efficacy.

The Body Image Pathway

AAS users, even if they do not suffer from outright muscle dysmorphia, very often experience at least some pathology related to body image. Persuading an AAS user to stop using the drugs and potentially lose muscle is often as difficult as persuading patients with anorexia nervosa that they need to gain weight. Thus, clinicians must be sympathetic and sensitive to the unique concerns of these individuals. As noted above, there are little formal data on the treatment of muscle dysmorphia, although it appears reasonable to expect that this disorder may respond to treatments previously found effective for other forms of body dysmorphic disorder, including cognitive behavioral approaches that are specifically tailored to body dysmorphic disorder [72] and treatment with selective serotonin reuptake inhibitors [73].

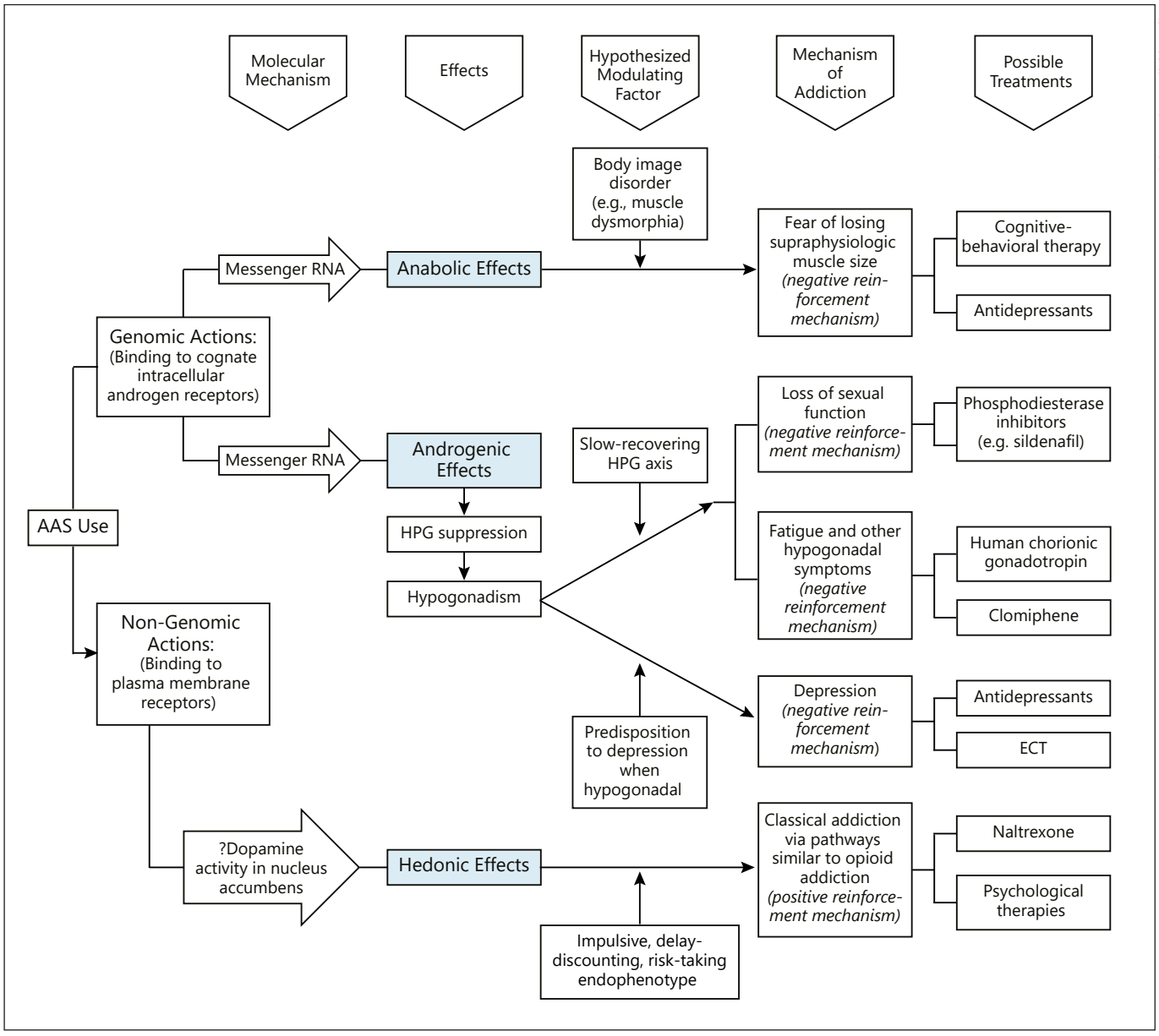

Fig. 1. A diagram of three pathways that may lead to anabolic-androgenic steroid dependence, together with possible therapeutic strategies to address each pathway. ECT, electroconvulsive therapy; HPG, hypothalamic-pituitary-gonadal; RNA, ribonucleic acid. Note that the term "anabolic effects" in the figure refers to the muscle-building effects of AAS, and "androgenic effects" refers to the masculinizing effects of these hormones. Note also that the types of antidepressants effective for body dysmorphic disorder are primarily serotonin reuptake inhibitors, whereas antidepressants from a wider range of chemical families may be effective for treating depression associated with hypogonadism. Reprinted from Kanayama et al. [66], with permission from Elsevier.

The Neuroendocrine Pathway

Dysphoria associated with AAS withdrawal hypogonadism may potentiate the urge to resume taking AAS, and thus hypogonadism deserves aggressive treatment. If hypogonadism is pronounced and prolonged, as may be the case during withdrawal from long-term AAS use [28–30], it is desirable to engage the services of an endocrinologist familiar with AAS withdrawal, although in our experience individuals with substantial expertise in this area are often hard to find. An endocrinologist might initially maintain the patient on a temporary modest dose of testosterone, while introducing human chorionic gonad-

otropin to restore pituitary production of luteinizing and follicle-stimulating hormones, and while also initiating clomiphene, often in conjunction with an aromatase inhibitor such as tamoxifen, in order to stimulate gonadotropin release from the pituitary [74]. Even with expert management of these endocrine parameters, treatment of hypogonadism may be prolonged and incomplete.

Meanwhile, psychiatric intervention may be necessary if the patient develops depression in association with AAS withdrawal. In studies of medically induced hypogonadism, it has been found that a majority of patients do not develop depression, but a minority, perhaps 10–20%, may develop serious depressive symptoms [75]. One small case series has described successful use of fluoxetine to treat such depression [76], and another individual case report described success with electroconvulsive therapy [77], but systematic studies are lacking. Thus, pending more definitive research, clinicians should approach AAS withdrawal depression in the same manner as an ordinary episode of major depressive illness. As noted above, treatment of the muscle dysmorphia form of body dysmorphic disorder, if present, with serotonin reuptake inhibitors may be efficacious for both body dysmorphic disorder and depressive symptoms [62].

The Hedonic Pathway

As mentioned above, male hamsters will self-inject testosterone to the point of death, but interestingly this behavior is blocked by the opioid antagonist naltrexone [78]. Indeed, several other reports have suggested parallels between the hedonic effects of opioids and those of AAS [8]. Thus, treatments found effective for opioid dependence might plausibly be helpful for AAS users. Such treatments might include motivational therapies to encourage commitment to treatment, contingency management, behavioral couple's therapy, and supportive-expressive therapy. Couple's treatment might be particularly promising with AAS users, since partners of AAS users are often eager for the AAS user to discontinue the drugs. We are not aware of any reports of naltrexone in the treatment of AAS dependence, although on theoretical grounds this drug might theoretically be effective.

In summary, then, at the present limited state of knowledge, much of the treatment of AAS users must be based on the clinician's assessment of each individual case. Now that AAS use has risen to become one of the world's major substance use disorders, it will become increasingly important for mental health professionals to stay abreast of evolving knowledge in this area.

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Author Contributions

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