

PSYCHOSOCIAL FACTORS FACILITATING ANABOLIC-ANDROGENIC STEROID
DEPENDENCE

by

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Abstract

The development of anabolic-androgenic steroid (AAS) dependence within the AAS using community is of some concern, due to the plethora of adverse health consequences it coincides with. To date, little is known about the factors aiding in the development of this condition. Utilising the current theory of AAS dependence and Bandura's (1991) theory of moral thought and action, this thesis aimed to investigate the psychosocial factors that facilitate the development of AAS dependence amongst recreational strength athletes.

Study 1 quantitatively explored the longitudinal relationship between AAS dependence and the experience of undesired physical and psychological effects via MD; in a population of male ($n = 118$) AAS users across three time points over a 12-month period. Mediation analysis revealed significant direct effects between AAS dependence, undesired physical effects ($\beta = 0.08$, 95% CI = 0.05 to 0.15), undesired psychological effects ($\beta = 0.10$, 95% CI = 0.07 to 0.13), and MD ($\beta = 0.10$, 95% CI = 0.07 - 0.13). However, no indirect effects were identified for the longitudinal relationship between AAS dependence on undesired physical and psychological effects via MD.

Attributed to the poor performance of AAS measures in Study 1, and the dearth of multidimensional AAS specific measures in the extant literature. Study 2 sought to develop and provide evidence of validity for measures of AAS dependence and AAS craving with two samples of male and female strength athletes who use AAS ($n_{\text{sample 1}} = 206$; $n_{\text{sample 2}} = 224$). Exploratory and Confirmatory Factor Analyses (CFA) with Sample 1 data were used to finalise the item sets for both measures and determine the factorial structures of the 15-item AAS Dependence Scale (AASDS) and the 16-item AAS Craving Scale (AASCS). Evidence supporting the concurrent, convergent and discriminant validity of scores obtained with both

scales was provided through their associations with the theoretically related variables. CFA with the data from Sample 2 confirmed the factor structures for both scales.

Study 3 aimed to observe the patterns in levels and trends of AAS craving, anticipated guilt, SRE, and affect across AAS administration; to determine if the observations were consistent with extant theory of craving. Through a naturalistic single case experimental investigation collating data across three independent 12-day periods from six participants, visual analysis identified patterns in level and trend for some, but not all, participants in particular phases of AAS use. However, no observed patterns in levels or trends were consistent across all participants. The absence of findings consistent with the current literature was attributed to the blast and cruise nature of AAS administration observed in each participant.

The findings of this thesis demonstrated the concerning longitudinal health effects attributed to AAS dependence, the requirement for appropriate measures to further explore the underlying dimensions of AAS dependence and craving amongst those who use AAS; and a degree of temporal patterns in craving across AAS administration. The findings from this thesis indicate that despite three decades of research in this area, there is still a need for a deeper understanding of the psychosocial factors that facilitate both AAS dependence and craving. By improving our knowledge and understanding, novel findings may aid in the development of harm reduction practices and interventions specific to the needs of AAS dependent individuals.

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“אין דבר העומד בפני הרצון”

“There is no such thing as can't” – Rachel Eleanor Zoob

Barnaby

Contents

Chapter 1: General Introduction	3
Summary and Aims of the Thesis	19
Chapter 2: The mediating role of moral disengagement in the longitudinal relationship between anabolic-androgenic steroid dependence and undesired health effects	21
Introduction	21
2.2 Methods	26
2.2.1 Participants	26
2.2.2 Measures	26
2.2.3 Procedures	28
2.2.4 Data Analysis	30
2.3 Results	31
2.3.1 Descriptive statistics and correlations.	31
2.4 Discussion	37
2.4.1 Limitations and Future Directions	41
2.4.2 Applied Implications	43
2.5 Conclusion	45
Chapter 3 - The Development and Validation of Dependence and Craving Measures Specific to Athletes Who Use Anabolic-Androgenic Steroids	47
3.1 Introduction	47
3.2 Methods	57
3.2.1 Item Development	57
3.2.2 Participants	59
3.2.5 Measures	60
3.2.5.1 Factorial, convergent, and discriminant validity, and internal consistency	60
3.2.6 Procedure	64
3.3 Results	65
3.3.1 Preliminary Analyses	65
3.3.2 Factorial Validity	65
3.3.3 Confirmation of Factor Structure	78
3.4 Discussion	78
3.4.1 Limitations and future directions	84
3.5 Conclusion	85

Chapter 4 - Temporal Changes In AAS Craving, Anticipated Guilt, Self-Regulatory Efficacy and Mood in Anabolic-Androgenic Steroid Use: A Naturalistic Single Case Experimental Investigation.....	87
4.1 Introduction.....	87
4.2 Methods.....	87
4.2.1 Measures	93
4.2.2 Procedures.....	94
4.2.3 Data Analysis	96
4.3.1 Participant 1	97
4.3.2 Participant 2.....	100
4.3.3 Participant 3.....	102
4.3.4 Participant 4.....	104
4.3.5 Participant 5.....	106
4.3.6 Participant 6.....	108
4.4 Discussion.....	119
4.4.1 Limitations and Future Directions	119
4.5 Conclusion.....	127
Chapter 5 – General Discussion.....	129
5.1 Thesis Summary	129
5.2 Discussion and Findings	131
5.3 Limitations and Future Directions	142
5.4 Applied Implications.....	146
5.5 Conclusion.....	148
Chapter 6 – References.....	150
Chapter 7 – Appendices.....	192
7.1 Appendix A	192
7.2 Appendix B	194
7.3 Appendix C	196

List of Tables and Figures

Tables

Table 2.1 Descriptive statistics and correlations of doping MD, AAS dependence, and number of self-reported undesired effects associated with AAS from T1, T2, and T3.....	p34
Table 2.2 Direct and indirect effects for AAS dependence on both undesired physical and undesired psychological effects via doping MD.....	p35
Table 3.1 Model fit indices for each CFA model run for AAS dependence and craving measures for the first and second samples.....	p67
Table 3.2 Internal consistency and correlations between factors from final AAS dependence scale models (M1c and M1b 15-item five factor models), DMDS, DSRES and DSMV from Sample 1 and Sample 2.....	p69
Table 3.3 M1c Items, standardized factor loadings and error variances for the AAS dependence scale (AASDS) from Sample 1 ($N = 206$) and Sample 2.....	p69
Table 3.4 Internal consistency and correlations between factors in the final craving model (Sample 1 M4c and Sample 2 M2b 4 factor models), Doping MD, Doping SRE and AAS dependence.....	p72
Table 3.5 Items, standardized factor loadings and error variances for the AAS craving scale (AASCS) from Sample 1 and Sample 2.....	p73
Table 3.6 Descriptive statistics, internal consistencies, and correlations between AASDS, number of self-reported undesired effects, AASCS, DSMV, AAS-WSCS, DMDS, and DSRE from Sample 1.....	p77
Table 4.1 Within-condition analysis for AAS craving during phase A (end of blast), B (middle of a cruise), and C (end of a cruise) identifying descriptive statistics, trend, level, and stability of responses.....	p111
Table 4.2 Within-condition analysis for anticipated guilt during phase A (end of blast), B (middle of a cruise), and C (end of a cruise) identifying descriptive statistics, trend, level, and stability of responses.....	p112
Table 4.3 Within-condition analysis for SRE during phase A (end of blast), B (middle of a cruise), and C (end of a cruise) identifying descriptive statistics, trend, level, and stability of responses.....	p113
Table 4.4 Within-condition analysis for affect during phase A (end of blast), B (middle of a cruise), and C (end of a cruise) identifying descriptive statistics, trend, level, and stability of responses.....	p114
Table 4.5 Between-condition analysis for each participant's response to single-item of AAS craving.....	p115

Table 4.6 Between-condition analysis for each participant’s response to single-item of anticipated guilt.....	p116
Table 4.7 Between-condition analysis for each participant’s response to single-item of SRE.....	p117
Table 4.8 Between-condition analysis for each participant’s response to single-item of affect.....	p118

Figures

Figure 2.1 The effects of AAS dependence on the undesired physical effects of AAS, and the mediating role of doping MD.....	p36
Figure 2.2 The effects of AAS dependence on the undesired psychological effects of AAS, and the mediating role of doping MD.....	p36
Figure 4.1 Participant 6 daily notification data taken at the end of a blast (phase A), during the middle of a cruise (phase B), and at the end of a cruise period (phase C) for single items measuring craving, doping MD, doping SRE, and Affect.....	p99
Figure 4.2 Participant 6 daily notification data taken at the end of a blast (phase A), during the middle of a cruise (phase B), and at the end of a cruise period (phase C) for single items measuring craving, doping MD, doping SRE, and Affect.....	p101
Figure 4.3 Participant 6 daily notification data taken at the end of a blast (phase A), during the middle of a cruise (phase B), and at the end of a cruise period (phase C) for single items measuring craving, doping MD, doping SRE, and Affect.....	p103
Figure 4.4 Participant 6 daily notification data taken at the end of a blast (phase A), during the middle of a cruise (phase B), and at the end of a cruise period (phase C) for single items measuring craving, doping MD, doping SRE, and Affect.....	p105
Figure 4.5 Participant 6 daily notification data taken at the end of a blast (phase A), during the middle of a cruise (phase B), and at the end of a cruise period (phase C) for single items measuring craving, doping MD, doping SRE, and Affect.....	p107
Figure 4.6 Participant 6 daily notification data taken at the end of a blast (phase A), during the middle of a cruise (phase B), and at the end of a cruise period (phase C) for single items measuring craving, doping MD, doping SRE, and Affect.....	p110

Glossary of Abbreviated Terms

AAS – Anabolic-Androgenic Steroids

AASCS – Anabolic-Androgenic Steroid Craving Scale

AASDS – Anabolic-Androgenic Dependence Scale

AAS-WSWS – Anabolic-Androgenic Steroid Specific Wisconsin Smoking Withdrawal Scale

AIC – Akaike Information Criterion

ASIH – Anabolic Steroid Induced Hypogonadism

CFA – Confirmatory Factor Analysis

CFI – Comparative Fit Index

DMDS – Doping Moral Disengagement Scale

DSMV – Diagnostic and Statistical Manual of Mental Health Disorders 5th Edition

DSRES – Doping Self-Regulatory Efficacy Scale

IPEDs – Image and Performance Enhancing Drugs

MD – Moral Disengagement

ML – Maximum Likelihood Estimation

RMSEA – Root Mean Square Error Approximation

SDS – Severity of Dependence Scale

SRE – Self-Regulatory Efficacy

SRMR – Standardised Root Mean Residual

χ^2 – Chi Square

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Chapter 1: General Introduction

Use of anabolic-androgenic steroids (AAS) is becoming increasingly commonplace within recreational gym going populations (Basaria, 2018; Jacka et al., 2017; McVeigh & Begley, 2017). Misuse¹ of AAS including administration of supraphysiological doses can produce a myriad of undesired physical and psychological effects (Goldman & Basaria, 2018), and the presence of AAS dependence (Brower et al., 1989; Kanayama, et al., 2009a, 2009b 2009c, 2010b) is thought to affect around 30% of those using AAS (Pope et al., 2014a). Individuals with AAS dependence exhibit more undesired effects associated with their use of AAS (Ip et al., 2012), including irreversible neurological effects than non-dependent individuals (Bjørnebekk et al., 2021; Hauger et al., 2019). Despite research identifying characteristics of those displaying AAS dependence (Brower et al., 1991; Ip et al., 2012; Kanayama et al., 2009a, 2010b), there remains a deficit of studies identifying the psychosocial factors which may facilitate the development of AAS dependence. Therefore, the overarching aim of this thesis was to further our understanding of factors that may facilitate the development of AAS dependence, and to develop an assessment of AAS dependence to provide a greater understanding of its dimensionality.

Anabolic-androgenic steroids (AAS) are synthetic derivatives of the male sex hormone testosterone, which exert both anabolic (i.e., muscle building) and androgenic (i.e., masculinising) effects within the user (Pope et al., 2014b). AAS were initially developed for therapeutic use within medical settings (Bain, 2008), however, since the mid-20th century AAS use has migrated towards use within the professional athlete environment (Bhasin et al., 2021). More recently, those who use AAS tend to be recreational gym users who engage in

¹ Misuse is used within this thesis to indicate the use of a substance in a way that was not initially intended for the compound/s in question (i.e., used in a non-clinical setting).

practices of strength training to enhance and develop their physiques (Ip et al., 2012; Kanayama et al., 2020). Global lifetime prevalence of AAS use has been estimated at 3.3%, with the highest prevalence seen within male gym frequenting populations (6.4%; Sagoe et al., 2014). When combined with a regimented diet and training regime, AAS enable those who use them to surpass natural limitations in muscle growth, strength, and aesthetics (Bhasin et al., 1996; Kanayama & Pope, 2018). These ergogenic effects are reportedly dose dependent (Yu et al., 2014), which may cause some individuals to administer larger doses over prolonged periods, increasing the risk of developing undesired effects associated with AAS (Bolding et al., 2002; Harmer, 2010; Pope et al., 2000).

Misuse of these substances is not without consequence, long-term abuse of AAS in supraphysiological doses is associated with a plethora of undesired effects (Baggish et al., 2017; Goldman & Basaria, 2018; Kaufman et al., 2019; Pope et al., 2000, 2014b) ranging from acute to chronic (Pope et al., 2014b). Although there is evidence to suggest the acute effects are reversible upon cessation of use, and/or via self-medication through using ancillary compounds (Kanayama et al., 2010; Pope, Kanayama, et al., 2014), chronic effects can be irreversible (van Amsterdam et al., 2010). The severity and frequency of such effects are thought to be idiosyncratic (Pope et al., 2014b). The undesired consequences of AAS use include cosmetic effects such as acne (Goldman & Basaria, 2018), cardiovascular events (Baggish et al., 2017), impaired hepatic and renal function (Maravelias et al., 2005; Robles-Diaz et al., 2015), compromised neurological function (Bjørnebekk et al., 2017, 2021; Seitz et al., 2017; Westlye et al., 2017), sexual dysfunction (Corona et al., 2022), psychological events (Chegeni et al., 2021; Pagonis et al., 2006), and hormonal imbalance (de Souza & Hallak, 2011).

Despite rationale for breaks in AAS use to provide time for recovery before re-initiating administration (Chester, 2018; Llewellyn et al., 2017), some individuals shorten or avoid these drug free periods due to experiencing withdrawal-like symptoms (Kanayama et al., 2010). Administration of supraphysiological doses of AAS has been demonstrated to suppress the hypothalamic-pituitary gonadal axis leading to anabolic steroid induced hypogonadism (ASIH; see Tan & Scally, 2009). Experience of ASIH in off-cycle² periods is known to elicit symptoms of depression and sexual dysfunction (Brower, 2002; Kanayama et al., 2015). It is believed that experience of these withdrawal-like symptoms may cause some individuals to prematurely reinitiate use of AAS to self-medicate for these effects (Kanayama et al., 2010b). In some cases, suppression of the HPG axis is irreversible (Boregowda et al., 2011), leading to some individuals to permanently administer AAS without breaks³ (Kanayama, et al., 2010b). Using AAS in such a manner dependencies indicative of an underlying syndrome of dependence (Brower, 2009; Hildebrandt et al., 2011; Kanayama et al., 2009b).

Over the last four decades a growing body of literature has demonstrated the presence of AAS dependence (Brower et al., 1989, 1991; Kanayama.,2008, 2009a, 2009b; Pope et al., 2012; Tennant et al., 1988), characterised as sustained use of AAS over prolonged periods of time, despite experiencing undesired effects including symptoms of withdrawal and psychological impairment (Kanayama et al., 2009a, 2009b, 2010). Based on current approaches, AAS dependence is thought to affect around 30% of individuals who administer

² AAS are typically administered in cycles (Kanayama et al., 2003), with the ‘on-cycle’ period spanning 8 to 16 weeks followed up by drug-free periods (i.e., ‘off-cycle’) lasting months at a time (Kanayama et al., 2008), to allow for endogenous testosterone production to recover (Kanayama et al., 2009a).

³ Continuous administration of AAS occurs in blast and cruise protocols (Chandler & McVeigh, 2014), whereby ‘cruising’ is characterised by administering a low to moderate dose of AAS over time (Sagoe et al., 2015) interspersed by periods of ‘blasting’ where doses are increased to encourage increased muscle and strength gains (Underwood, 2016).

AAS (Pope et al., 2014). Despite identification of some characteristics of AAS dependence and associated health risks, to date, there is no single agreed upon theory or model of AAS dependence. As such several multidimensional models have been proposed over the last 30 years in an attempt to explain how AAS use manifests into a syndrome of dependence (Bahrke & Yesalis, 1994; Brower, 1992, 2002; Hildebrandt et al., 2011; Kanayama et al., 2010). Despite models of AAS dependence identifying its multidimensional nature, studies are yet to identify how these factors of dependence interact with other constructs that may facilitate the development of AAS dependence.

Brower (1992) proposes AAS exerts dependence via four mechanisms: 1) primary positive reinforcement through neurological reward pathways, 2) secondary positive reinforcement from increased growth of muscle, 3) primary negative reinforcement via avoidance of biologically mediated withdrawal symptoms, and 4) secondary negative reinforcement through avoidance of psychosocial withdrawal symptoms. Researchers have identified issues with this model, primarily attributed to difficulties in discriminating between each of these reinforcing factors in survey-based research as many of these factors present themselves simultaneously (Yesalis et al., 1990). Disagreement over the psychoactive nature of AAS has caused some researchers to believe that AAS dependence is caused by secondary reinforcing effects due to both personal and socially rewarding properties, rather than being attributed to psychoactive stimulation (Midgley et al., 1999).

Other models have proposed AAS dependence manifests itself through socio-cultural reinforcing effects, motivating individuals to engage in rigorous training behaviours to build highly muscled physiques (Bahrke & Yesalis, 1994). Within this model it is training that is the focus of AAS dependence rather than AAS use, causing associated improvements in mood and self-esteem, associated with AAS dependence. Therefore, reinforcing effects attributed to

AAS dependence are predominantly accredited to the combination of, the muscle building properties of AAS, regimented routines of administration, intense training, and strict dietary protocols (Midgley et al., 1999). As such, this model may be better suited to identify exercise dependence and a desire to increase body capital (Gunnarsson et al., 2022; Kotzé & Antonopoulos, 2021) rather than AAS dependence alone.

A later model by Brower (2002) indicates AAS dependence manifests itself through a two-stage process. The primary stage identifies administration of AAS, when combined with strict dietary protocols, develops positively reinforcing supraphysiological muscle mass. Due to this positive feedback, the behaviour is maintained despite incurring unwanted health effects. The second stage is characterised through administering increased doses which activate neurological reward mediated pathways, inhibiting the cessation of AAS administration (Brower, 2002). The author identifies that there is a lack of evidence for AAS dependence without associated resistance training or an increase in musculature (Brower, 2002), therefore the reinforcing effects of AAS seen here are attributed more towards a dependence on enhancing muscular strength, aesthetics, and physical performance (Mhillaj et al., 2015) rather than a dependency towards AAS.

More recently, research has presented a model of AAS dependence comprising of three distinct mechanisms (Kanayama et al., 2010). First, the anabolic mechanism is moderated by the presence of muscle dysmorphia causing an AAS using individual to continuously administer AAS without breaks due to a 'fear' of losing muscle mass when absconding from AAS use. Second, the androgenic mechanism identifies administration of exogenous testosterone-based compounds causes the suppression of the hypothalamic-pituitary-gonadal axis and the subsequent development of AAS induced hypogonadism and associated symptoms (ASIH; Tan & Scally, 2009). AAS are therefore administered to

alleviate these symptoms in pre-planned drug-free periods (Brower, 2002). Third, the hedonic mechanism identifies similarities of AAS dependence to dependence of other substances of abuse, further demonstrated by animal models (Koob, 2006; Wood, 2002; Wood et al., 2004). The reinforcing effects of AAS in the first two mechanisms are attributed to self-medicating underlying comorbidities (i.e., muscle dysmorphia and ASIH) rather than on the reinforcing behaviours of AAS themselves. Furthermore, the third mechanism is somewhat limited as animal models have contradicted previous findings (Negus et al., 2001; Wood et al., 2011), whilst the hedonic reinforcing effects of AAS are not comparable to substances like heroin or cocaine, but more comparable to less harmful substances like caffeine (Wood, 2008).

A final model proposed by Hildebrandt et al. (2011) indicates AAS dependence may be both a physical and psychological construct. The model portrays AAS dependence as a physical construct attributed to a positive feedback loop of combined exercise and AAS administration leading to pleasurable secondary reinforcing factors (i.e., gratification from others). However, administration of exogenous testosterone inhibits endogenous testosterone, causing ASIH, subjecting the individual to a physical dependence to AAS. Psychological dependence is demonstrated when the AAS using individual experiences positively reinforcing social benefits related to their use of AAS (i.e., improved appearances, gratification from others, or social dominance through aggression). Hildebrandt et al. (2011) identifies that physical dependence is present once the individual begins to use ancillary compounds to combat the undesired effects associated with AAS use (Hildebrandt et al., 2011). However, research demonstrates many who use AAS and experience undesired effects administer ancillary compounds to mitigate them (Kanayama et al., 2010; Pope, Kanayama, et al., 2014), therefore use of ancillary compounds may not be indicative of AAS dependence.

With these models demonstrating reinforcing behaviour and the development of AAS dependence through multiple dimensions, it suggests a need to examine the dimensionality of dependence. To date, scales used to assess AAS dependence capture AAS dependence as a single factor (Gillespie et al., 2007; Gossop et al., 1995; Grant et al., 2007; Lynskey & Agrawal, 2007; Ray et al., 2008; Teesson et al., 2002), and are therefore unable to represent the multidimensional nature of AAS dependence. Based on the current models of AAS dependence, one can identify possible sub-dimensions of AAS dependence. These included unbroken use sustained over time despite incurring undesired (physical, psychological, and social) effects, experience of withdrawal and self-medication with AAS in periods of abstinence (Brower, 2002; Kanayama et al., 2009, 2009b; Pope et al., 2010), and administration of larger doses to improve AAS effectiveness (Brower, 2002; Kutscher et al., 2002; Yu et al., 2014).

Up to now, studies have mainly characterised how AAS dependent users present when compared to non-dependent users and/or non-AAS using controls (Brower et al., 1991; Hauger et al., 2019, 2020; Ip et al., 2012; Kanayama et al., 2009a). Whereby those with AAS dependence self-report use of more AAS compounds, incurring more undesired effects, demonstrate an increased concern over undesired health consequences associated with AAS use, use higher doses and spend less time off-cycle than their non-dependent counterparts (Brower et al., 1991; Hauger et al., 2019, 2020; Ip et al., 2012; Kanayama et al., 2009a).

In an early study, Brower and colleagues (1991) examined a small convenience sample of AAS using males to determine correlates of AAS dependence. The authors identified that increased dosage and body dissatisfaction predicted AAS dependence. However, the authors neglected to identify which model of AAS dependence they were utilising or how they operationalised AAS dependence. Building on the previous findings

Kanayama et al. (2009a) sought to identify associated risk factors of AAS dependence. Despite the authors defining AAS dependence as “continuous use despite adverse medical, psychiatric, social, and occupational effects” (Kanayama et al., 2009a), there was no mention of what model was operationalised. The study demonstrated increased durations of use, larger dosages, polysubstance use, higher prevalence of childhood conduct disorders, and increased body dissatisfaction in AAS dependent users compared to non-AAS dependent users, and a non-AAS using weightlifting control. Ip et al. (2012) demonstrated differences in clinical characteristics between dependent and non-dependent AAS users. Authors recruited a large sample of AAS using males. The study characterised dependence as continued use despite incurring undesired effects, and operationalised Kanayama et al. (2010) model of dependence. Those with AAS dependence reported use of more anabolic compounds, higher doses, shorter off-periods, longer duration of AAS use, and increased concern over AAS related health issues.

Across three studies Hauger and colleagues (2019, 2020, 2021) identified correlates of AAS dependence at both a clinical and neurological level. Characteristics of AAS dependent users were in alignment with previous studies. These studies also demonstrated thinner neurological cortices, compromised executive function, and a positive relationship between violence and aggression with dependence amongst AAS dependent participants. These findings have been supported more recently by Scarth et al. (2022). Findings also identified more negative personality traits (i.e., antagonism, disinhibition, rigid perfectionism, and psychoticism) positively associated with AAS dependence. The study characterised AAS dependence as continued use despite experiencing negative physical and psychological effects including symptoms of withdrawal and impaired psychosocial functioning, and utilised the model for dependence proposed by Kanayama et al. (2010).

A factor often deemed as an antecedent of drug dependence is drug craving (Drummond, 2001). Manifesting in numerous ways, craving presents itself in an intrusive and dominating manner instilling considerable distress in an individual (Beck et al., 1993; Tiffany & Wray, 2012). Craving is considered to be a desire to administer a substance (Tiffany & Wray, 2012), characterised by a want, urge, or a compulsion to engage in drug seeking behaviour, only satiated by using the substance itself (Kozlowski & Wilkinson, 1987).

Research within other substances has demonstrated symptoms of drug craving present during drug-free periods, exacerbated by environmental cues associated with drug administration, drug exposure, and drug expectancy (Donny et al., 2008; Drummond et al., 1990; Franken et al., 2002; Pickens et al., 2011), facilitating drug seeking behaviour (Drummond et al., 1990). Evidence in the extant literature indicates presence of craving within substance use disorders, such as alcohol, smoking, and opioid misuse (Kakko et al., 2019; Serre et al., 2015a; Tiffany, 1990), leading to some researchers suggesting craving contributes towards substance dependence (Tiffany & Wray, 2012). Subsequently, craving has been included in both the International Classification of Disorders 10th edition and within the Diagnostic and Statistical Manual of Mental Disorders 5th edition diagnostic criteria for substance dependence (APA, 2013; WHO, 1992). However, there remains some dispute over the role of craving in drug relapse during periods of non-substance use (Anton, 2000; Perkins, 2009; Wray et al., 2013).

To better understand AAS dependence and targets for potential interventions it is important to explore the relationship between AAS dependence and craving. To date, however, no published research has examined craving with respect to AAS dependence in humans. Outside of AAS use, research on substance dependence has considered dependence as a trait-like construct that is not expected to fluctuate greatly over short time periods (see

Flannery et al., 2019; Geiser et al., 2017). Comparatively, craving is viewed as a state-like construct that fluctuates easily in response to internal and external stimuli during periods of substance withdrawal (Geiser et al., 2017; Huhn et al., 2016; Serre et al., 2015; Tiffany & Wray, 2012). Currently, there is no universal agreement on how craving and dependence affect one another. Theories have postulated craving as the primary motivating factor to sustain substance use, and claimed it is responsible for relapse in times of abstinence (Baker et al., 1987; Ludwig, 1974; Ray & Roche, 2018).

Despite believing craving has a direct role in substance abuse, some researchers believe craving is not responsible for compulsive substance use (Baker et al., 2004; Tiffany, 1990). Even though there is disagreement over the role of craving in drug dependence, it is still believed to be a key feature of substance abuse disorders (Drummond, 2001; Tiffany et al., 2008). As such, many theories in the extant literature operationalise drug craving as an antecedent for substance dependence (Baker et al., 2004; Tiffany, 1990), and have included craving as a criterion in diagnosing dependence (see APA, 2013). In terms of AAS dependence, it is possible that AAS craving motivates individuals to prematurely re-initiate their use of AAS in androgen free periods. It is therefore an important area to explore within AAS research for potential harm reduction intervention.

Researchers have suggested the multidimensionality of drug craving through proposing a series of models (see Skinner & Aubin, 2010). Originally proposed by Wikler (1948) and later built upon by Drummond and colleagues (1990), craving has been thought to exert itself through conditioned response (i.e., environmental cues). In this case an individual will experience unpleasant sensations when exposed to drug related cues (e.g., being within an environment where drug use occurred, or seeing an object related to substance use), in turn, exerting a desire to engage in substance use to alleviate noxious stimuli (Drummond et

al., 1990; Wikler, 1948). It is believed that environmental cues increase the risk for drug relapse in periods of abstinence (Drummond, 2001). However, little evidence within the literature indicates a significant association with this model and drug relapse (Skinner & Aubin, 2010).

Marlatt and Gordon (1985) proposed a model of drug craving, exerted through drug expectancy (Marlatt & Gordon, 1985). In this model, the role of craving in substance relapse is mediated by anticipated effects of administering the drug (i.e., a desire for the positive effects associated with the use of the drug). This model has been paired with aspects of the conditioned response model of Drummond et al. (1990), whereby past gratification associated with drug use in an environment can elicit drug craving (Marlatt & Gordon, 1985). Through the mechanisms proposed within this model, it is possible that AAS craving may present itself in a similar manner. Whereby positive drug expectancy associated with AAS use, and being within the gym environment may facilitate the experience of AAS craving and potentiate subsequent use of AAS in drug free periods.

Building upon the previous models, Niaura et al. (1988) indicate that drug craving may arise from environmental cues, and experience of both positive and negative mood (i.e., affect) states. In this model, initial drug use provides positive reinforcement through a desired mood change (e.g., the improvement of mood through drug administration), and acts directly upon positive feedback mechanisms. This feedback is amplified when the individual is exposed to environmental cues associate with substance use, such as being seeing an abject associated with drug administration (Niaura, 2000; Niaura et al., 1988). This model has demonstrated an inverse association with self-regulatory efficacy, identifying possible action in drug relapse (Niaura, 2000). With the models previously discussed in mind, one would expect to see AAS craving display dimensionality including aspects of, drug expectancy,

environmental cues, and altering both positive and negative mood states (Drummond et al., 1990; Marlatt & Gordon, 1985; Niaura, 2000; Niaura et al., 1988).

With studies demonstrating the presence of drug craving in substance abuse research, it is possible that those who misuse AAS experience symptoms of craving during breaks in their AAS protocols, driving them to seek AAS administration. Existing research has provided evidence for craving-like behaviours and for the relevance of craving for AAS use through testing of animal models. To determine the rewarding properties of AAS, researchers across several studies infused rodents with testosterone (equivalent to 900mg to 3,000mg per week; see Wood, 2008). Rodent models displayed a conditioned place preference associated with AAS associated environmental cues, and voluntarily self-administer AAS (Alexander et al., 1994; Arnedo et al., 2002; de Beun et al., 1992; Schroeder & Packard, 2000; Wood et al., 2004a), even to the point of death (Wood, 2006). Further evidence to suggest AAS display craving-like properties has been provided through a series of experiments where medications used to alleviate the symptoms of craving (e.g., Naltrexone) inhibited behaviours of self-administration of AAS amongst animal models (Peters & Wood, 2005; Pickens et al., 2011; Wood et al., 2004). With the evidence these models provide it is likely that AAS may exert similar rewarding effects upon strength athletes who use AAS. These rewarding effects of AAS may increase craving for AAS in drug-free periods and facilitate AAS dependence.

Evidence for craving within AAS users has been further demonstrated within human studies. Researchers have demonstrated craving-like behaviours in human studies, with some AAS users re-initiating their use of AAS in pre-planned 'off-cycle' periods to self-treat symptoms of withdrawal (Ip et al., 2012; Kanayama et al., 2010). Research has identified that during these 'off-cycle' periods, some individuals experience dysphoric effects associated with both ASI and an impaired mood state (Brower, 1997; Kashkin & Kleber, 1989; Malone

& Dimeff, 1992). It is therefore important to identify key dimensions of AAS craving and develop an instrument to assess them, in order to explore AAS further, and to understand the interrelationships between dimensions of AAS craving and dependence, to better understand how to adequately manage individuals displaying these conditions.

As well as furthering our understanding of AAS craving, it is also important to understand possible antecedents of this construct. A concept previously associated with substance use and craving is moral disengagement (MD; Ahmadi et al., 2019; Kleinjan et al., 2009). Proposed by Bandura (1991) in his social cognitive theory of moral thought and action, MD presents as a collective term for eight psychosocial mechanisms that justify and rationalise engagement in transgressive actions by changing how we think about the behaviour, who is responsible for it, and its repercussions. Research has demonstrated that MD is able to facilitate engagement in harmful behaviours including AAS use (Boardley et al., 2015, 2017, 2018). MD achieves this by allowing individuals to suppress or eradicate personal rebuke of anticipated negative emotions (i.e., guilt and shame) associated with harmful acts (e.g., use of IPEDs; see Bandura, 1991).

Research over the last decade has linked MD with use of IPEDs in sport and exercise contexts. Qualitative research by Boardley and Grix (2014) identified six of the eight mechanisms of MD (e.g., moral disengagement, advantageous comparison, diffusion/displacement of responsibility, distortion of consequences, and euphemistic labelling) originally proposed (Bandura, 1991), via interviews with nine IPED using bodybuilders from England. Building on these findings Boardley and colleagues (2014) interviewed a national sample of 64 IPED using male bodybuilders in England. Analysis identified the utilisation of the same six mechanisms previously identified (Boardley & Grix, 2014). These findings were supported and further developed by Boardley et al. (2015) through

interviews with 12 IPED using athletes from the United States of America and England. Quantitative research has demonstrated support for these qualitative findings. Boardley et al. (2017) assessed the association of doping MD with doping behaviour amongst team- ($n = 195$), individual- ($n = 169$) athletes, and in hardcore- ($n = 125$) and corporate- ($n = 121$) gym goers. Structural equation modelling identified a moderately positive predictive effect between doping MD and IPED use. With this evidence demonstrating association between MD and IPEDs, similar relationships would be expected to be demonstrated between MD and AAS use.

Evidence in the few studies examining the association between MD and substance dependence has demonstrated the association between MD and nicotine dependence (Kleinjan et al., 2009). Using a smoking specific measure of MD, a study by Kleinjan et al. (2009) demonstrated the association between MD, nicotine dependence, and readiness to quit smoking. The study identified a positive association between MD and nicotine dependence, and demonstrated MD negatively predicted readiness to quit smoking through regression analysis (Kleinjan et al., 2009). To date, there is an absence of literature focusing on the association between AAS dependence and MD. However, with the evidence provided from the extant literature similar associations may be demonstrated, whereby elevated levels of AAS dependence are associated with increased scores of MD.

Research on other forms of drug misuse has explored the association between MD and substance craving (Ahmadi et al., 2019). Ahmadi and colleagues (2019) assessed the association between substance craving and MD amongst a population of individuals from substance abuse clinics. This study demonstrated a positive association between MD on substance craving (Ahmadi et al., 2019), whereby elevated levels of MD positively predicted craving through regression analysis. Presently there is a dearth of research looking at the

associations between MD and AAS craving, however from the evidence provided one could postulate that elevated scores of MD may act as a predictor of AAS craving, thereby increasing the risk of engaging in AAS use during periods of abstinence.

Another component of Bandura's social cognitive theory of moral thought and action, which may play a part in facilitating AAS dependence, is self-regulatory efficacy (SRE; (Bandura, 1997; Bandura et al., 2001). SRE represents an individual's self-belief in his or her ability to resist internal and external pressures to partake in harmful and detrimental behaviours (Bandura, 1997). In the AAS context, an individual possessing elevated levels of SRE will display a better capacity to withstand personal and social influences to engage in AAS use. SRE is known to display an antagonistic relationship with MD, whereby high levels of SRE are associated with subdued levels of MD, as there is a reduced need to rationalise and justify engaging in transgressive conduct when one possesses self-belief in the ability to resist engaging in the act (Bandura et al., 2001).

There is evidence within the extant literature to suggest that the process of SRE is utilised within IPED using populations (Boardley et al., 2017). A study by Boardley and colleagues (2017) demonstrated SRE negatively predicted MD when looking at doping behaviours (e.g., use of AAS) within team and individual sport athletes. Based on these findings, in terms of AAS use and dependence, an individual who has high levels of SRE would present low MD and be able to resist engaging in harmful behaviour which in turn may decrease the likelihood of developing AAS dependence. This relationship has been demonstrated within research on alcohol and tobacco whereby individuals displaying high levels of SRE demonstrated more successful attempts to abstain from substances of abuse and avoid relapse (Chavarria et al., 2012; Niaura et al., 1988; Stuart et al., 1994). Presently, there is an absence of research identifying the associations between SRE and AAS dependence. It is

possible therefore, that SRE would display a negative relationship with AAS dependence, and be an important area to therapeutically target for those who wish to abstain from future AAS use.

Through his theory of moral thought and action, Bandura proposes anticipated guilt contributes towards processes self-regulation. Bandura (1991) identifies that anticipated guilt acts as a deterring effect in engaging in transgressive behaviour, via inflicting an unpleasant emotional sensation of tension and regret upon and individual who is considering/partaken in detrimental activities. Bandura states that tension and regret are less likely to be experienced by those displaying high MD (Bandura, 1991). Consistent with Bandura's (1991) theory, IPED research has demonstrated an inverse effect of guilt on MD (Boardley et al., 2017, 2018). With evidence suggesting the mediating effect of guilt on MD, and subsequent substance use, it seems logical to postulate guilt would display a similar mediating effect on MD and AAS dependence.

To date, both qualitative and quantitative literature has identified evidence for the use of constructs from Bandura's (1991) theory of moral thought and action and IPED use (i.e., AAS; see Boardley et al., 2014, 2015, 2017, 2018; Boardley & Grix, 2014). Previous research has established cross-sectional evidence for the association of dependence and craving with MD, SRE, and guilt (Ahmadi et al., 2019; Chavarria et al., 2012; Kleinjan et al., 2009; Niaura et al., 1988; Stuart et al., 1994). However, there is an absence of evidence associating AAS dependence and craving with constructs of MD, SRE, or anticipated guilt within gym using populations. Therefore, further research in these areas would build upon the extant literature and develop our understanding of how dependence and craving manifest within AAS using populations. Offering potential opportunities for harm reduction interventions tailored to suit those who use AAS.

Summary and Aims of the Thesis

In summary, despite research exploring AAS dependence over the last three decades, there remains a dearth of studies identifying the psychosocial factors that may facilitate the development of AAS dependence. Furthermore, with an absence of longitudinal research in this area, research has not been able to explore temporal associations between AAS dependence and constructs aiding in its development. Present measures used to assess AAS dependence are not only unidimensional but are adapted from pre-existing scales to assess intoxicating substances of abuse. Severely limiting our understanding of the multidimensional nature of AAS dependence proposed by hypothetical models for AAS dependence. There also remains an absence of literature exploring craving within the context of AAS use, limiting our understanding of a potentially key driver in sustained AAS administration. Research has postulated craving displaying multidimensionality through multiple models, however with the preference for single item measures and no measure developed specifically for AAS craving, our understanding of how AAS craving manifests within those who use is somewhat limited.

Taking into consideration the evidence presented up to this point, the primary aim of this thesis was to further our understanding of the nature of AAS dependence, as well as its antecedents and outcomes. Based upon the reviewed literature, three empirical studies were designed to address some of the key limitations in knowledge identified to this point.

Specifically, the aims for each of these studies were:

- a) To longitudinally examine whether MD and AAS dependence mediated the relationship between AAS use and AAS harms. These aims were addressed in Study 1 (Chapter 2).

- b) To further our understanding of the dimensionality of AAS dependence and AAS craving, by developing multidimensional measures of each construct. These aims were addressed in Study 2 (Chapter 3).
- c) To explore the longitudinal and real-time co-occurrence of AAS craving and associated psychosocial constructs (i.e., anticipated guilt, SRE, and affect) across different periods of the AAS use cycle within natural environments. These aims were addressed in Study 3 (Chapter 4).

Chapter 2: The mediating role of moral disengagement in the longitudinal relationship between anabolic-androgenic steroid dependence and undesired health effects

Introduction

Anabolic-androgenic steroids (AAS), initially utilised by professional athletes such as bodybuilders and weightlifters (Kanayama et al., 2008), are increasingly being used by recreational strength athletes (Zahnow et al., 2017). Prolonged supraphysiological administration of AAS is associated with an array of undesired acute and chronic physical and psychological health effects (Goldman & Basaria, 2018), as well as the development of a dependency syndrome (Kanayama et al., 2009a, 2009b, 2010). Currently estimated to affect up to 30% of AAS users, AAS dependence is associated with prolonged use of AAS, thus increasing risk of experiencing adverse effects (Ip et al., 2012; Pope et al., 2014a). Research has identified use of image and performance enhancing drugs (IPEDs) may be facilitated by psychosocial mechanisms of moral disengagement (MD; Boardley et al., 2014, 2015a, 2017; Boardley & Grix, 2014). It is possible that over time those with AAS dependence display higher MD, thereby facilitating prolonged use of AAS and increasing the risk of incurring associated undesired effects. Therefore, the overarching aim of this research was to investigate whether the longitudinal relationship between AAS dependence and associated undesired physical and psychological health effects amongst strength athletes was mediated by MD.

AAS are a family of synthetic compounds derived from the male hormone testosterone, that produce anabolic (i.e., muscle building) and androgenic (i.e., masculinising) effects for those that use them (Pope et al., 2014b). When combined with adequate training and diet, AAS aid an individual to surpass their natural potential of enhancing muscle mass, strength and physical aesthetics (Ip et al., 2011; Pope et al., 2012). Evidence suggests that

these ergogenic effects are dose dependent (Yu et al., 2014), therefore increasing the risk that some users of AAS misuse⁴ these compounds (see Christiansen et al., 2017 and Zahnow et al., 2018). This is of some concern as it is believed that those who use higher doses of AAS may be at an increased risk of developing undesired effects (Bolding et al., 2002; Harmer, 2010).

An important motive for developing our understanding of the psychosocial processes that facilitate AAS dependence, are the adverse health consequences associated with their use. Misuse of AAS is associated with a myriad of undesired affects (Goldman & Basaria, 2018; Kanayama et al., 2018), the frequency and severity of which are idiosyncratic. These adverse effects can be acute or chronic (Pope et al., 2014b), with acute effects often being reversible upon cessation of use (van Amsterdam et al., 2010). However, long-term use can increase the risk of chronic, and often irreversible health effects such as cardiac dysfunction, neurological abnormalities, and psychological effects including depression and mood disorders (Bjørnebekk et al., 2021; Hauger et al., 2019; Rasmussen et al., 2016; Sculthorpe et al., 2010; Seitz et al., 2017). Most studies identifying physical and psychological harms are case-reports and cross-sectional studies (Chegeni et al., 2021; D'Andrea et al., 2022; Doleeb et al., 2019; Kaufman et al., 2015; Windfeld-Mathiasen et al., 2022), therefore limiting our understanding the temporal associations between prolonged use and harms. Despite the presence of two studies investigating longitudinal effects of AAS administration (Bjørnebekk et al., 2021; Smit et al., 2021), only one demonstrated longitudinal effects (Bjørnebekk et al., 2021).

Administration of AAS often occurs in 'cycles' (Kanayama et al., 2003), whereby compounds are taken over periods of 8 to 16 weeks followed up with drug free intervals lasting months or even years (Kanayama et al., 2008). Drug free periods aim to re-establish

⁴ Misuse is used to indicate the use of a substance in a way that was not initially intended for the compound/s in question (i.e., used in a non-clinical setting).

endogenous testosterone production (Kanayama et al., 2009a) as exposure to exogenous testosterone-based substances (i.e., AAS) suppresses the function of the hypothalamic-pituitary gonadal axis, a syndrome known as anabolic steroid induced hypogonadism (ASIH; Kanayama et al., 2015a; Tan & Scally, 2009). Abstinence from AAS use can produce withdrawal-like symptoms (Kanayama et al., 2010), likened to those experienced when abstaining from psychoactive drugs (e.g., opioids; Brower, 2002, 2009) and in extreme cases, suicide attempts (Amaral et al., 2020; Papazisis et al., 2007). Research indicates that some individuals use AAS in a continuous manner to avoid symptoms of withdrawal (Christou et al., 2017) and to self-medicate for ASIH (Kanayama et al., 2015). Although continuous use of AAS may prevent withdrawal-like symptoms being experienced, it is likely to increase the risk of developing long-term irreversible harms (Baggish et al., 2017; Pope et al., 2014b) and AAS dependence (Kanayama et al., 2015a).

Over the last three decades, research has developed an ever-growing picture of AAS dependence and its correlates (Brower, 1989, 1992; Kanayama et al., 2009a, 2009b). Empirical evidence to date estimates AAS dependence to affect up to 30% of AAS users (Pope et al., 2014a), predominantly manifesting in those using supraphysiological doses (Brower, 2002). Those with dependence are noted to develop a greater number of AAS related health issues than those who are non-dependent (Ip et al., 2012), exaggerated for those with years of uninterrupted administration (Kanayama et al., 2020). Research in this area remains in its infancy, as such researchers still do not fully understand the exact mechanisms that contribute to the development of AAS dependence (Kanayama et al., 2009a).

Despite not completely comprehending how AAS dependence manifests itself, researchers have identified several underlying characteristics of AAS dependence. Brower et al. (1991) identified AAS dependent users displayed more undesired psychological effects,

including body dissatisfaction and aggression, than non-dependent users. Kanayama et al. (2009a) demonstrated individuals with AAS dependence displayed significantly more psychological events than non-dependent individuals, including increased body dissatisfaction and substance dependence disorders. Ip and colleagues (2012) reported AAS dependent individuals displaying an increased concern over health issues, more undesired psychological and physical effects associated with their AAS use than non-dependent users. More recently the literature has displayed evidence to suggest AAS dependence is associated with concerning aberrations in brain structure, compromised executive function, increased violent behaviour, and increased adverse personality traits in AAS dependent individuals (see Hauger et al., 2019, 2020, 2021; Scarth et al., 2022). However, to date, research in this area has been cross-sectional, limiting our understanding of the processes linking AAS dependence and associated harms over time. Therefore, longitudinal research exploring the temporal relationships (Anstey & Hofer, 2004) between AAS dependence and undesired affects associated with AAS use would make an important contribution to our understanding of AAS dependence.

Given the apparent prevalence of AAS dependence amongst recreational strength athletes, it is important to examine psychosocial processes that may facilitate the effects of AAS dependence on detrimental health outcomes. Bandura's (1991) social cognitive theory of moral thought and action describes processes that have been linked with harmful behaviours such as drug use. More specifically, it proposes that individuals can suppress or eradicate anticipated personal rebuke and associated negative emotions (i.e., guilt and shame) that normally result from engagement in harmful acts (e.g., use of IPEDs) through moral disengagement (MD). MD is a collective term for eight psychosocial mechanisms that justify and rationalise detrimental behaviours by reducing changing how we think about the

behaviour, reduce accountability for it and its consequences, distort the consequences, and dehumanise or blame victims. As such, MD may demonstrate a mediating effect, enabling individuals with AAS dependence to rationalise and justify their behaviour of prolonged use of AAS despite incurring undesired physical and psychological effects.

Empirical research has linked MD with use of IPEDs in sport and exercise contexts, including AAS use in gym populations. Utilisation of MD by athletes and bodybuilders who use IPEDs was identified across three qualitative studies (Boardley et al., 2014, 2015; Boardley & Grix, 2014). This research illustrated the use of six out of the eight MD mechanisms (i.e., moral justification, advantageous comparison, diffusion and distortion of responsibility, distortion of consequences and euphemistic labelling) to justify and rationalise use of IPEDs (Boardley et al., 2014, 2015; Boardley & Grix, 2014). Quantitative research assessing the association of doping MD and doping behaviour amongst athletes and gym populations including AAS using bodybuilders identified doping MD had a positive predictive effect on use of IPEDs. Although research has linked MD and IPED use, to date, researchers have not examined the relationships between AAS dependence, MD, and harmful health effects stemming from AAS use.

Support for a link between AAS dependence and MD is offered by the limited research that has explored the association between substance dependence and MD (Ahmadi et al., 2019; Kleinjan et al., 2009). One study assessed the effects MD to rationalise drug use amongst a sample that were exclusively dependent on opioids, identifying the positive predictive ability of MD to engage in drug seeking behaviour in periods of abstinence (Ahmadi et al., 2019). Another study, explored the association between MD and nicotine dependence, indicating a positive relationship between nicotine dependence and MD, and that MD negatively predicted readiness to quit smoking (Kleinjan et al., 2009). With the evidence

from these studies demonstrating the utilisation of MD amongst diagnosed with substance dependence, it is possible that MD plays a role in facilitating AAS dependence. In order to further our understanding of AAS dependence, it is important to explore whether MD facilitates links between AAS dependence and harmful effects of AAS use over time.

Based upon the arguments made to this point, this study sought to understand whether AAS dependence predicted harmful psychological and physical effects of AAS use over time, and whether MD facilitated any effects over time. Based on the reviewed literature we hypothesised: a) *AAS dependence would positively predict the number of undesired physical effects experienced by those using AAS (H1)*, b) *AAS dependence would positively predict the number of undesired psychological effects experienced by those using AAS (H2)*, and c) *MD would mediate – at least in part – the relationships between AAS dependence and the undesired physical and psychological effects of AAS use (H3)*.

2.2 Methods

2.2.1 Participants

Participants ($n = 118$) were strength athletes originating from 26 countries ($n_{USA} = 52$; $n_{UK} = 25$; $n_{other} = 41$). Strength athletes reported being between; 18 to 20 years of age (8.6%), 21 to 25 years of age (27.2%), 26 to 30 years of age (37.3%). 31 to 35 years of age (16.1%), 36 to 40 years of age (4.2%), and over 41 years of age (6.6%). Participants reporting being single (44.9%), heterosexual (94.1 %), and full-time employed (58.5%). On average, participant's age of AAS initiation was 25.03 years ($SD = 6.70$), total number of cycles run was 4.48 ($SD = 4.11$), and number of cycles run in the last 12-months was 1.60 ($SD = 0.74$).

2.2.2 Measures

Self-reported undesired physical and psychological effects associated with AAS use were assessed at each time-point. Participants reported the presence of adverse physical and

psychological effects currently being experienced by responding to the question “Are you currently experiencing any of these effects associated with the use of anabolic steroids?”. Physical effects included effects well established in the current scientific literature (e.g., acne, fluid retention, injection site pain, cholesterol imbalance, elevated red blood cell count; see van Amsterdam et al., 2010). Psychological effects included items associated with AAS withdrawal (e.g., depressive thoughts, decreased libido, excessive body checking, increased anxiety, insomnia, and mood swings; Brower et al., 1991; Ip et al., 2011; Parkinson & Evans, 2006; Westerman et al., 2016). Participants responded dichotomously via ‘Yes’ or ‘No’ responses for both physical and psychological events, which were summed to produce a total for both undesired physical and undesired psychological effects at each time point.

MD was assessed using the Doping Moral Disengagement Scale (DMDS; see Boardley et al., 2018) and adapting it to suit the use of AAS (i.e., ‘doping’ was replaced with ‘steroid’). This scale consisted of 18-items (e.g., compared to most lifestyles in the general public, steroid use isn’t that bad”), with three items for each of the six mechanisms linked with IPED use (see Boardley et al., 2014, 2015; Boardley & Grix, 2014). Participants were instructed to read statements describing thoughts, feelings and situations associated with the use of AAS and indicate their level of agreement with each statement using a 7-point Likert scale anchored at 1 (*strongly disagree*) and 7 (*strongly agree*). Mean scores for the 18 items were computed for this measure at each time point. The scale demonstrated acceptable to good internal consistency at T1, T2, T3, and T4 ($\alpha = .72, .82, .84, .82$, respectively).

AAS dependence was initially assessed using the severity of dependence scale (SDS; see Gossop et al., 1995) adapted to suit the use of AAS (see Griffiths et al., 2018). This measure consists of five items (e.g., “in the last 4 months did the prospect of missing a dose of steroids make you anxious or worried?”), and participants were instructed to read a number

of statements and respond to the best of their ability to the questions. Items one to four were anchored at 0 (*never/almost never*) and 3 (*always/nearly always*), whereas item five (e.g., in the last 4 months how difficult did you find it to stop, or go without steroids?") was anchored at 0 (*not difficult*) and 3 (*impossible*). The SDS identifies dependence via computing an individual's total score. If it exceeds six the individual is considered dependent. Although previous research indicated an adequate internal consistency ($\alpha = .75$; Griffiths et al., 2018), our data demonstrated an unacceptable internal consistency at T1 ($\alpha = .47$), T2 ($\alpha = .67$), T3 ($\alpha = .37$), and T4 ($\alpha = .59$).

Due to the poor internal consistency of the SDS at T1, we decided to include an alternative measure of AAS dependence from T2. We utilised the AAS specific Diagnostic Statistical Manual of Mental Disorders fourth edition (DSM-IV) criteria adapted to suit AAS by Kanayama and colleagues (2009c). This measure consists of nine items (e.g., "over the last 4 months have you increased the doses of steroids you are using due to being dissatisfied with your previous results?") identifying areas of dependence such as tolerance, withdrawal, increased dosage, unsuccessful attempts to cut down use, time associated with use, cessation of social activities to use, and continued use despite adverse effects. A Likert scale was utilised anchored at 1 (*never*) and 4 (*very often*). Scores were aggregated, higher scores demonstrated an increased risk for the presence of AAS dependence. This scale indicated acceptable levels of internal consistency at T2, T3, and T4 ($\alpha = .76, .71, \text{ and } .78$ respectively).

2.2.3 Procedures

Full ethical approval was obtained from the University of Birmingham Ethics Committee (ERN_19-1955). Data collection occurred across four time points over an 18-

month period, T1 occurred in April – May 2020⁵, followed by T2 in September – October 2020, T3 in February – March 2021, and T4 in July – August 2021)⁶. Inclusion criteria required participants to be male, over the age of 18, and had taken AAS in the last 12 months prior to T1. Participants were excluded from analysis if they had failed to complete two or more time points, 119 participants did not meet this criterion, therefore analysis was conducted with 118 participants who had completed between two-to-four-time points.

Participants were recruited through advertisements on bodybuilding and strength training forums, groups on social media platforms (i.e., Facebook, Twitter, and Reddit) where the use of AAS is regularly discussed, and through existing contacts and gatekeepers. Potential participants were provided with a brief description of the study and a hyperlink to access the online survey. Once accessed, participants were presented with an information sheet, General Data Protection Regulation information and a consent form. Informed consent was obtained from all participants at each time point. Confidentiality was assured to participants of their responses as no personal details (e.g., names, addresses, phone numbers, etc) were gathered from participants. Email addresses were required for further contact for data collection at T2, T3, and T4 to provide participants with Amazon vouchers. T1 took approximately between 10 to 15 minutes to complete, T2, T3, and T4 took approximately 10 minutes to complete. Upon completion of T1 participants were alerted that they would be contacted via the email address they had provided at T1 for the completion of the second survey in four months' time at T2 and subsequent surveys at T3 and T4. Upon completion of

⁵ Data collection within this time period coincided with the national lockdown protocols experienced throughout many nations around the globe due to the COVID pandemic (Lau et al., 2020; Moris & Schizas, 2020).

⁶ The latter three time points occurred during periods of time where the isolation restrictions were relaxed, including the removal of work from home policies that were implemented within the UK (Rathod et al., 2021).

the survey participants were entered into a prize draw to win a £25, £50 or £100 Amazon voucher.

2.2.4 Data Analysis

Preliminary data analysis revealed the presence of missing data between the respective time points. Between time point 1 (T1, $n = 237$), time point 2 (T2, $n = 91$), time point 3 (T3, $n = 81$), and time point 4 (T4, $n = 87$) missing data was reported at each subsequent time point at 59.07%, 65.82%, and 63.29% respectively. Due to the poor performance of the adapted SDS, we used the alternative measure of AAS dependence we introduced from T2 (i.e., the AAS adapted DSM-IV criteria; Kanayama et al., 2009c). Furthermore, we decided to omit data from T1 because of the poor performance of the AAS adapted SDS from any data analysis and the high attrition rate between T1 and T2, using only data from T2, T3, and T4. For simplicity and clarity, from this point forward we will refer to these three time points as T1, T2, and T3. With our longitudinal design and a reduced sample size, we adopted mediated regression models to test two mediational models. The first would look at the effect of AAS dependence on undesired physical effects via doping MD, whilst the second would look at the effect of AAS dependence on undesired psychological effects via doping MD. This would enable us to test the proposed direct and indirect effects, whilst mitigating the issue of our reduced sample size. To examine longitudinal effects, doping AAS dependence from T1, MD from T2, and number of undesired physical and psychological effects from T3 in our analyses.

To address the issue of missing data, we subjected the data to Little's Missing Completely at Random test (Little, 1988), demonstrating data was missing completely at random ($X^2 = 14.17$, $df = 12$, $p > .29$). A multiple imputation model was therefore established to replace missing data; five data sets were generated with the maximum number of

parameters set at 100. The subsequent analyses were undertaken with the average value of the missing data sets. The PROCESS version 4.1 (Hayes, 2017) SPSS macro (model 4) was used to test for direct and indirect effects of AAS dependence on the number of self-reported undesired physical and psychological effects associated with use of AAS, via MD. Direct effects describe the effects of a predictor variable on an outcome variable occurring independently from the effects of the mediator. Indirect effects describe the effects of a predictor variable on an outcome variable via a mediator variable. The total effect represents the sum of the direct and indirect effects. We set bootstrapping to 10,000 samples to account for Type 1 errors (Hayes, 2009; Preacher & Hayes, 2004). Bias-corrected confidence intervals were calculated for each effect. A significant effect is evident when the confidence interval does not contain zero. The Completely Standardised Indirect Effect (CSIE) was utilised to determine small (0.01), medium (0.09), and large (0.25) effect sizes (Preacher & Kelley, 2011). Statistical significance was set as $p < .05$.

2.3 Results

2.3.1 Descriptive statistics and correlations.

Table 2.1 indicates descriptive statistics and correlations for the study variables. Most participants reported being on cycle⁷ at T1 (85.7%), T2 (86.4%), and at T3 (89.6%) whilst the remainder reported being off cycle. Participants demonstrated a moderate doping MD score at T1 ($M = 4.3$, $SD = 0.7$), T2 ($M = 4.2$, $SD = 0.7$), and T3 ($M = 4.2$, $SD = 0.7$). Participants demonstrated a moderate level of AAS dependence at T1 ($M = 16.6$, $SD = 4.1$), T2 ($M = 15.3$, $SD = 3.4$), and T3 ($M = 15.3$, $SD = 4.1$). T1 correlations indicated strong significant positive associations between AAS dependence and doping MD ($r = .47$, $p < .01$), moderate

⁷ 'Use of AAS' refers to participants being either; on-cycle, blasting, cruising, or on testosterone replacement therapy.

significant positive association between AAS dependence and undesired physical effects ($r = .27, p < .01$) and moderate -to-strong positive associations with undesired psychological effects ($r = .32, p < .01$), weak-to-moderate positive associations were also identified between doping MD and undesired psychological effects ($r = .18, p < .05$), and between undesired physical and psychological effects ($r = .25, p < .01$). T2 indicated weak-to-moderate significant positive correlations between AAS dependence and undesired psychological effects ($r = .24, p < .01$), and between undesired physical effects and undesired psychological effects ($r = .23, p < .01$). T3 demonstrated moderate-to-strong significant positive associations between AAS dependence and undesired physical effects ($r = .39, p < .01$) and strong significant positive association between AAS dependence and undesired psychological effects ($r = .45, p < .01$), and a moderate-to-strong significant positive association between undesired physical and psychological effects ($r = .37, p < .01$).

2.3.2 Mediation analysis.

To examine the predictive effect, of AAS dependence on the number of undesired health effects a series of mediated regressions were conducted (see Table 2.2). In the first mediation model, AAS dependence from T1 was entered in as a predictor of undesired physical health effects at T3 via our mediator, doping MD from T2. These analyses showed that AAS dependence from T1 was a significant predictor of doping MD from T2 ($\beta = 0.10$, 95% CI = 0.07 - 0.13), and a significant positive predictor for undesired physical health effects at T3 ($\beta = 0.08$, 95% CI = 0.05 to 0.15). However, there was no significant indirect effect for doping MD at T2 on undesired physical effects at T3 ($\beta = -0.13$, 95% CI = -0.52 to 0.27, CSIE = -0.04, 95% CI = -0.15 to 0.10). These results can be seen in Table 2.2 and Figure 2.1.

The second mediation model, AAS dependence from T1 was entered in as a predictor for number of undesired psychological effects at T3 via our mediator, doping MD from T2. These analyses indicated that AAS dependence from T1 was a significant positive predictor for doping MD at T2 ($\beta = 0.10$, 95% CI = 0.07 to 0.13), and a significant positive predictor for undesired psychological effects experienced at T3 ($\beta = 0.14$, 95% CI = 0.07 to 0.20). However, there was no significant indirect effect for doping MD from T2 on undesired psychological effects experienced at T3 ($\beta = -0.25$, 95% CI = -0.57 to 0.08, CSIE = -0.08, 95% CI = -0.22 to 0.02). These results can be seen in Table 2.2 and Figure 2.2.

Table 2.1. Descriptive statistics and correlations of doping MD, AAS dependence, and number of self-reported undesired effects associated with AAS from T1, T2, and T3 ($n = 118$).

	Variable	Mean	SD	Range	Skewness	Kurtosis	1	2	3	4	5	6	7	8	9	10	11	12
1	AAS Dependence T1	16.55	4.10	9.00-27.00	1.57	4.47	-											
2	Doping MD T1	4.28	0.71	3.00-7.00	0.56	0.95	.47**	-										
3	Undesired Physical Effects T1	1.95	1.57	0.00-8.00	.94	1.02	.27**	.05	-									
4	Undesired Psychological Effects T1	1.14	1.21	0.00-5.00	1.11	0.78	.32**	.18*	.25**	-								
5	AAS Dependence T2	15.28	3.42	9.00-22.00	1.12	3.80	.50**	.32**	.03	.10	-							
6	Doping MD T2	4.24	0.68	1.00-6.00	-0.49	3.95	.18*	.46**	-.11	.05	.11	-						
7	Undesired Physical Effects T2	1.17	1.08	0.00-5.00	0.94	0.80	.08	.07	.13	.17	.09	-.12	-					
8	Undesired Psychological Effects T2	0.95	1.14	0.00-6.00	1.41	2.49	.29**	.10	.33**	.28**	.24**	.15	-.23*	-				
9	AAS Dependence T3	15.30	4.11	9.00-27.00	1.54	2.49	.74**	.48**	.19*	.21*	.55**	.10	.10	.29**	-			
10	Doping MD T3	4.19	0.68	3.00-7.00	0.08	0.26	.12	.55**	-.11	.13	.02	.63**	-.17	.09	.07	-		
11	Undesired Physical Effects T3	1.91	1.44	0.00-7.00	0.95	1.29	.19*	.18*	.25**	.15	.24**	-.06	.09	.20*	-.39**	.03	-	
12	Undesired Psychological Effects T3	1.22	1.27	0.00-5.00	0.69	-0.59	.36**	.16	.23*	.20*	.28**	-.06	.04	.25**	.45**	.03	.37**	-

Note: * $p < .05$, ** $p < .01$.

Table 2.2. Direct and indirect effects for AAS dependence on both undesired physical and undesired psychological effects via doping MD.

Pathways	B	95% CI	CSIE	95% CI
<i>Direct effect of AAS dependence on</i>				
Doping MD	0.10**	0.07 - 0.13		
Undesired physical effects	0.08*	0.05 - 0.15		
Undesired psychological effects	0.14**	0.07 - 0.20		
<i>Indirect effect of</i>				
AAS dependence on undesired physical effects via doping MD	-0.13	-0.52 - 0.27	-0.04	-0.15 - 0.10
AAS dependence on undesired psychological effects via doping MD	-0.25	-0.57 - 0.08	-0.08	-0.22 - 0.02

Note: Unstandardised coefficients are shown. AAS = Anabolic-Androgenic Steroids, CSIE = Completely Standardised Indirect Effect, MD = Moral Disengagement.

* $p < 0.05$; ** $p < 0.01$

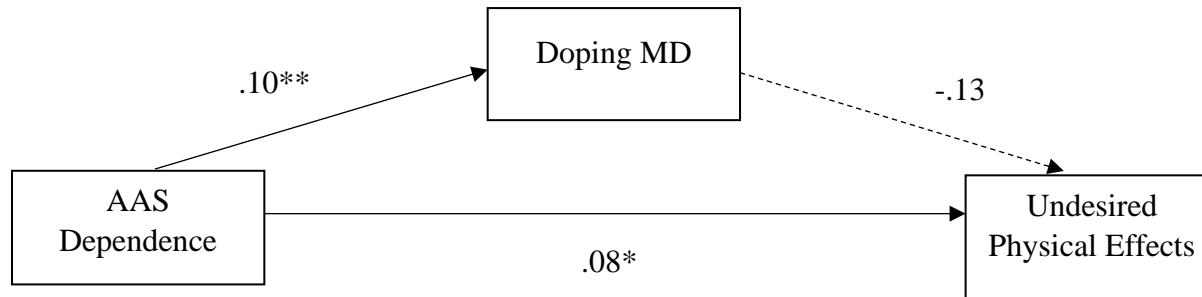


Figure 2.1. The effects of AAS dependence on the undesired physical effects of AAS, and the mediating role of doping MD.

Note. Values are unstandardized regression coefficients. Filled lines indicate significant results * $p < .05$, ** $p < .01$.

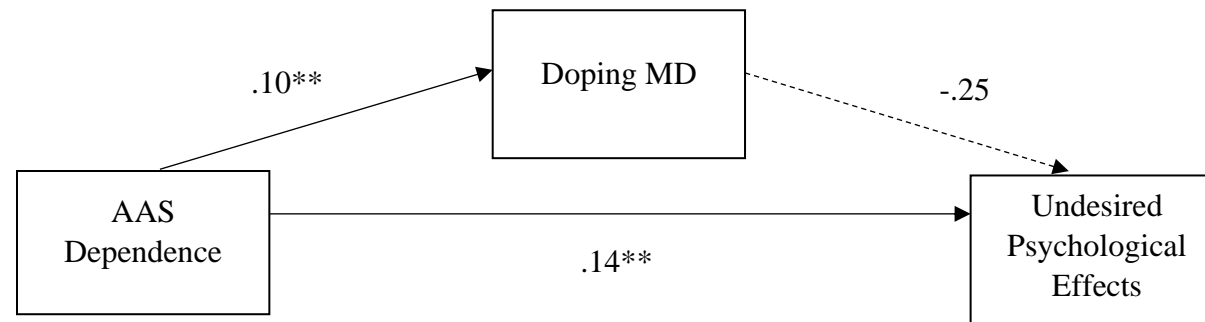


Figure 2.2. The effects of AAS dependence on the undesired psychological effects of AAS, and the mediating role of doping MD.

Note. Values are unstandardized regression coefficients. Filled lines indicate significant results * $p < .05$, ** $p < .01$.

2.4 Discussion

The existence of AAS dependence amongst those who use AAS has become increasingly apparent over the last thirty years (Brower et al., 1991; Hauger et al., 2020; Ip et al., 2012; Kanayama et al., 2009; Pope et al., 2014; Scarth et al., 2022). However, despite identifying underlying physical and psychological comorbidities (Kanayama et al., 2010), little attempt has been made to understand the psychosocial mechanisms through which AAS dependence may influence the number of adverse effects experienced by people who use AAS. Furthermore, there is an absence of longitudinal research on AAS dependence and its relationship with undesired effects associated with AAS. This study sought to address these deficits in knowledge by examining whether AAS dependence predicted undesired physical and psychological effects via MD over time in a sample of strength athletes who use AAS.

The extant literature indicates that AAS dependence is associated with an increased experience of undesired physical effects (Brower et al., 1991; Hauger et al., 2019; Ip et al., 2012; Kanayama et al., 2009a). It was therefore hypothesised that AAS dependence would positively predict the number of undesired physical consequences associated with use of AAS. The direct effects within this study indicated AAS dependence was able to positively predict the increased experience of undesired physical effects across a 12-month period, thereby supporting *H1*. Current literature investigating the harms associated with AAS dependence is cross-sectional (Brower et al., 1991; Hauger et al., 2019; Ip et al., 2012; Kanayama et al., 2009a). Therefore, the findings from this study not only confirm cross-sectional reports to date, but provide stronger support for a causal effect by showing this predictive effect is apparent over time, providing an important novel contribution to the AAS literature.

Our finding that AAS dependence is able to positively predict the increased experience of undesired physical effects across a longitudinal period, provides a novel and significant contribution to the extant understanding of AAS dependence. Over the last 30 years research has indicated AAS dependent users administer elevated dosages and have shorter drug-free periods across AAS administration compared to non-dependent users (Brower et al., 1991; de Zeeuw et al., 2023; Ip et al., 2012). These behaviours are believed to increase the risk of experiencing harms (Baggish et al., 2017; McCullough et al., 2021), including acne, aberrations in the structure and function of the heart and liver, gynecomastia, dyslipidaemia, and hypertension, (Hartgens et al., 2004; Neri et al., 2011; Rothman et al., 2011; Smit et al., 2021). To date researchers have attributed the experience of undesired effects with the larger dosages and duration of use exhibited by AAS dependent users (see Ip et al., 2012), however this research remains cross-sectional. By demonstrating AAS dependence has a direct causal effect on increased experience of undesired physical harms, this study gives us a greater insight of the symptoms associated with AAS dependence.

Presently, literature on AAS dependence indicates the association of dependence with an increased experience of undesired psychological effects attributed to the use of AAS (see Brower et al., 1991; Hauger et al., 2019, 2020; 2021; Ip et al., 2012; Kanayama et al., 2009a; Scarth et al., 2022). We therefore hypothesised that AAS dependence would positively predict the number of undesired psychological effects associated with use of AAS. The direct effects in model testing indicated AAS dependence had a positive longitudinal relationship with undesired psychological harms over a 12-month period, therefore fully supporting *H2*. By providing evidence of a longitudinal effect of AAS dependence on undesired psychological harms stemming from AAS use, this study made a significant novel contribution to the

literature, strengthening evidence of a causal effect of AAS dependence on negative psychological effects of AAS use.

Our finding that AAS dependence is able to positively predict the increased experience of undesired psychological effects across a 12-month period provides a significant contribution to the extant literature of AAS dependence. A growing body of research has categorised those with AAS dependence to use higher dosages and administer more anabolic compounds than non-dependent users (Brower et al., 1991; de Zeeuw et al., 2023; Hauger et al., 2020; Ip et al., 2012; Kanayama et al., 2009a; Scarth et al., 2022). Researchers have identified that administration of supraphysiological dosages of AAS over prolonged periods of time is associated with increased risks of experiencing undesired psychological effects including depression, irritability, mood swings, and hostility (Hauger et al., 2019, 2021; Pagonis et al., 2006). To date cross-sectional research has attributed the experience of undesired effects with the larger dosages and duration of use exhibited by AAS dependent users (see Ip et al., 2012). By demonstrating AAS dependence has a direct causal effect on increased experience of undesired psychological harms, this study gives us a greater insight of the undesired psychological health consequences associated with AAS dependence.

Despite extant literature identifying a positive association between doping MD and use of AAS (see Boardley et al., 2014, 2015, 2017, 2018; Boardley & Grix, 2014), there remains a dearth in research exploring the mediating effect of doping MD on AAS dependence and associated undesired effects of AAS use. We therefore hypothesised that MD would mediate (in part) relationships between AAS dependence and undesired physical and psychological effects. No significant indirect effect of doping MD was identified on AAS dependence and experience of adverse physical or psychological effects, the results in this

study do not support *H3*. Despite this, the study identified a significant direct effect between AAS dependence and doping MD.

One possible explanation for the absence of evidence for MD as a mediator in the relationship between AAS dependence and undesired health effects was potentially attributed to the rewording of the doping MD items to suit AAS. This was achieved by replacing the word ‘doping’ with ‘steroids’. Alteration of the doping MD items may have changed the way participants responded to this measure, exhibited by lower internal consistency values and higher mean scores of the doping MD scale in this study than in previous literature (Boardley et al., 2017, 2018). Furthermore, attrition experienced in the study will have affected the study’s sample size, thereby reducing statistical power. This is evident with in the CSIE values in both models assessed in this study being close to significant (see Table 2.2 and Table 2.3).

By indicating a direct positive direct effect between AAS dependence and MD across a 6-month period, this study presents significant contributions to the understanding of Bandura’s (1991) theory of MD within the AAS literature. Bandura (1991) proposes engagement in transgressive acts are facilitated through mechanisms to rationalise and justify behaviour (i.e., drug use). Despite evidence for MD in substance use behaviours (Boardley et al., 2017; Heyes & Boardley, 2019; Quinn & Bussey, 2015; Sumnall et al., 2022), there are few studies identifying a casual effect between substance dependence and MD. Klienjan et al. (2009) identified a positive association between nicotine dependence and MD, and indicated MD negatively predicted readiness to quit smoking. From the results of Klienjan et al (2009) it is likely that MD is used by those with substance dependence disorders to rationalise and justify their behaviours in order to maintain their prolonged drug use. However, the cross-sectional design of this study limits our understanding on the causal nature between

dependence and MD. Through the findings of our study identifying AAS dependence has a direct causal effect on MD, this study gives us a greater insight of the factors that may facilitate the syndrome of dependence in those who use AAS.

2.4.1 Limitations and Future Directions

As with any study there were a number of limitations that should be considered. A high attrition rate was experienced across the transition between the first and second time points (62.1%), second and third time points (10.9%), and third and fourth time points (4.4%). Attrition played a part in omitting the first time point from analysis in this study. Despite this attrition rate being high, it was not to a level that would cause the results to be non-meaningful (see Angrist et al., 1996). Although statistically significant results were identified within the study, the reduced power attributed to the attrition rate will have reduced the ability to detect statistically significant results. It is possible that attrition was due to a number of reasons including emails being redirected to junk/spam folders, participants forgetting their participation within the study, COVID-19 lockdown restrictions ending causing a reduced motivation to continue with the study, and COVID survey fatigue (see Zoob Carter et al., 2021). Future longitudinal studies may benefit from providing participants with questions at the end of surveys enquiring if their email addresses have change, this will ensure that the most up-to-date email addresses are retained and may aid in the prevention in attrition.

Generalisability could have also been affected as it is possible that we did not capture all typologies of AAS users (Christiansen et al., 2017; Zahnnow et al., 2018). It is possible that we captured data from an array of typologies, including the YOLO type (Christiansen et al., 2017), during the first time point of the study. With the relaxation of COVID lockdown, and subsequent reduced motivations to continue with the study, it is likely that these YOLO participants removed themselves from further participation in the time points used in analysis.

With YOLO users obtaining their knowledge of AAS from ‘steroid gurus’ (see Christiansen et al., 2017), it is possible that a distrust for researchers and medical professionals is passed on to them, causing them to limit interactions with researchers and medical professionals (Bonnacaze et al., 2020). Potentially causing a response bias as some AAS users may not fully disclosing their use and/or give the full picture of undesired effects they experience (Bonnacaze et al., 2020). Advertisement of academic studies via gatekeepers and moderators on forums may be a benefit to future research aiming to recruit AAS users. As approval from respected members of the AAS community may encourage engagement from harder to reach AAS users.

Findings from this study identified limitations with the measures of AAS dependence. Analysis of internal consistency at T1 identified that the SDS was not internally consistent (i.e., unacceptable), leading to the addition the AAS adapted DSM-IV (see Kanayama et al., 2009c) from T2. Our findings contradicted the existing literature, which indicated the SDS as an internally consistent measure for assessing AAS dependence (Griffiths et al., 2018). The DSM-IV was also met with limitation as it is unable to capture the multidimensional nature of AAS dependence exhibited in extant models (see Bahrke & Yesalis, 1994; Brower, 2002; Brower et al., 1991; Hildebrandt et al., 2011; Kanayama et al., 2010), as existing measures of AAS dependence unidimensional (Gillespie et al., 2007; Gossop et al., 1995; Ray et al., 2008). Therefore, we recommend researchers develop a bespoke multidimensional scale specific to identify if specific aspects of AAS dependence are more strongly linked with undesired health effects.

Another common limitation in this thesis was found with the internal consistency of the MD measure used. Currently, MD has been explored within the IPED using community with the doping MD scale (see Boardley et al., 2018). This scale was adapted within this

study to suit AAS, by rewording and replacing the term ‘doping’ with ‘steroid use’ (i.e., “The risks associated with steroid use are exaggerated”). Despite demonstrating good internal consistency scores, the alpha values were smaller than reported in previous literature (see Boardley et al., 2017). Hardcore gym users made up less than half the sample within the study by Boardley et al (2017), with over half the participants belonging to individual and team sports. This was a very different sample to the ones identified within this thesis, which contained participants from hardcore gyms. It is possible that those who use AAS interpret the MD items differently from other athletes, whereby AAS users may dissociate their use of AAS from items concerning ‘doping’ behaviour. The alteration in item wording within this study may have caused participants to answer more defensively as items may have caused participants to reflect more on their own behaviour. Development of a bespoke AAS suited measure of MD would be key in amending this limitation.

2.4.2 Applied Implications

The novel findings identifying the relationship between AAS dependence and MD is important, as it furthers our understanding of the factors which may facilitate AAS dependence. The current literature on AAS dependence has postulated several models in order to explain how AAS using individuals manifest this syndrome (Bahrke & Yesalis, 1994; Brower, 2002; Brower et al., 1991; Hildebrandt et al., 2011; Kanayama et al., 2010) . However, none of these models have explored how psychosocial factors such as MD may attribute to the development of AAS dependence (de Zeeuw et al., 2023; Ip et al., 2012; Kanayama, Hudson, et al., 2009; Scarth et al., 2022a). Despite researchers identifying a number of characteristics of those with AAS dependence, there is little research exploring the mindset of those with AAS dependence and how they rationalise and justify their behaviours associated with their use of AAS. Therefore, the information from this study may offer a

greater insight to the thoughts and characteristics of those with AAS dependence. By identifying the role of MD in AAS dependence these findings may offer novel targets for harm reduction interventions; such as cognitive behavioural therapy (see Quaglio et al., 2009; Smit et al., 2019).

To date research on undesired physical effects associated with AAS use have been reported as being reversible (Goldman & Basaria, 2018); consequently, the impact upon overall health may be understated by some sub-populations of AAS users (Pope et al., 2014b; Christiansen et al., 2017). It is therefore of high importance to present the current findings to both the AAS community, healthcare practitioners, and harm reduction services. Atkinson and colleagues (2021) have indicated the importance of providing credible research to healthcare practitioners and harm reduction workers, in order to establishing effective engagement strategies with AAS users. To date much of the harm reduction surrounding AAS use is focused on blood-borne viruses causing substantial frustration amongst the AAS using community as many other physical harms are more frequently experienced (see Underwood, 2019), and to date there is no evidence indicating transmission of BBVs occurs via AAS use (see McVeigh, 2019). Havnes et al (2019) suggests that providing practitioners with improved knowledge about the adverse effects of AAS use may facilitate engagement and management of AAS users. By understanding the needs and experiences of AAS users, the findings collated from this study will aid in the provision of information and understanding to these harm reduction groups, aiding in the creation of bespoke management plans (see Bates et al., 2021) for AAS dependent users. Furthermore, by circulating the findings from studies, such this, will aid in raising an understanding and awareness within the AAS community about the impact sustained use of AAS has on their physical health.

Presently there is some disagreement within the AAS community over the presence of undesired psychological effects (Kimergård, 2015; Monaghan, 2002); however, it is important to ensure findings from this study are not overlooked by the AAS community and healthcare providers. A recent review has suggested that AAS using individuals experiencing undesired psychological symptoms may benefit from interventions through behavioural health therapists (Bonnecaze et al., 2021). However, researchers like Van de Ven et al (2022) indicate that there is a dearth of evidence from research informing effective harm reduction provision. Therefore, findings from this study demonstrating a causal effect of AAS dependence on psychological harms may offer such evidence and knowledge to practitioners. Furthermore, by identifying the myriad of undesired psychological events experienced by those who use AAS (Goldman & Basaria, 2018), harm reduction services and healthcare professionals may be made aware that a 'one size fits all' approach to management of AAS users is not appropriate. Therefore, novel findings from this study may aid in providing harm reduction of healthcare services with the appropriate information to develop bespoke management and support to AAS dependent users. Furthermore, circulating the findings from this study within the AAS community may aid in raising an understanding and awareness about the impact sustained use of AAS has on their psychological health.

2.5 Conclusion

Through this longitudinal study we demonstrated AAS dependence has a significantly positive direct effect with experience of undesired physical and psychological effects associated with AAS use, and a significant positive direct effect with MD. This study makes a particular contribution to the literature as this is the first study to identify a longitudinal relationship between AAS dependence and MD, furthering our knowledge of how the AAS community utilise MD (Boardley et al., 2017). The study also contributes to the current

literature, as it is the first study to identify a longitudinal relationship between AAS dependence and undesired health effects associated with AAS use, supporting previous cross-sectional associations (see Ip et al., 2012; Kanayama et al., 2009b). We look forward to studies expanding on these findings in future research.

Chapter 3 - The Development and Validation of Dependence and Craving Measures Specific to Athletes Who Use Anabolic-Androgenic Steroids

3.1 Introduction

Anabolic-androgenic steroids (AAS), primarily utilised by weightlifters and bodybuilders, are also used by recreational gym goers (Kanayama et al., 2009b; Zahnow et al., 2018). Misuse⁸ of supraphysiological doses⁹ of AAS has been linked with a myriad of undesired health effects (Pope et al., 2014; Ip et al., 2011; Parkinson et al., 2006; Westerman et al., 2016; Brower et al., 1991), including a dependency syndrome which is estimated to affect up to 30% of people who use AAS (Pope et al., 2014). Although development of substance dependency is thought to be facilitated by drug craving (Donny et al., 2008), craving remains poorly understood (Flannery et al., 2001) and AAS craving has not yet been explored. To explore AAS dependence and craving, valid and reliable assessment instruments are required. The overarching aim of the current research was to develop two such instruments and to validate their scores.

AAS research has demonstrated evidence of a dependency syndrome through case reports since late 1980 (Brower et al., 1989; Tennant et al., 1988). Subsequently, research into this area attempted to diagnose AAS dependence with the utilisation of diagnostic criteria used to identify substance misuse and dependence. The two most accredited criteria for assessing the presence of dependence to substances of misuse are the Diagnostic and Statistical Manual of Mental Disorders criteria (DSM; APA, 2013), and the International Classification of Diseases (ICD; WHO, 1992). However, the DSM and ICD disagree on how AAS are viewed. Whilst the ICD volume 10 (ICD-10) does not consider AAS as substances

⁸ Misuse is used here to refer to the use of a substance in a manner that is not medically recommended (WHO, 1992).

⁹ There have been no reported cases of AAS dependence whilst using therapeutic doses of AAS (Brower, 2002).

that can cause dependence or symptoms of withdrawal (Midgley et al., 1999), the DSM criteria classifies AAS in the ‘other’ category of substance use disorders, which includes substance dependence (APA, 2013). For this reason, research on AAS dependence has primarily utilised the DSM criteria (Kanayama et al., 2009a; Ip et al., 2012; Scarth et al., 2022).

Despite the DSM criteria being primarily used to diagnose dependence towards intoxicating substances of misuse, research identified the presence and prevalence of AAS dependence amongst populations of male weightlifters using the DSM criteria (Brower et al., 1989, 1991; Copeland et al., 2000; Gridley & Hanrahan, 1994; Midgley et al., 1999; Perry et al., 2005). Unlike commonly misused substances, AAS are not immediately intoxicating (Grönbladh et al., 2016; Kanayama et al., 2009). Instead, AAS are consumed over prolonged periods of time to obtain a delayed reward of increased musculature and strength, rather than administered to obtain the sensation of an instantaneous “high” (Kanayama et al., 2009b). To account for this, researchers have provided recommendations to make the DSM criteria more specific to the use of AAS (Kanayama et al., 2009b). Amendments included items identifying unsuccessful attempts to stop AAS use due to anxiety over loss of muscle size in drug-free periods, avoidance of important activities in favour of maintaining supraphysiological muscle mass, and excessive time spent training, attending to diet, associating with other AAS users (see Kanayama et al., 2009b). However, these amendments may actually represent risk factors for AAS use, such as muscle dysmorphia, exercise addiction and eating disorders (Cole et al., 2003; Copeland et al., 2000; Greenway and Price, 2018; Griffiths et al., 2018; Gunnarsson et al., 2022; Hurst et al., 2000), or even different typologies of AAS users (i.e., ‘Expert’; see Christiansen et al., 2017; Zahnow et al., 2018) rather than dependence alone.

Another method of assessing AAS dependence is the Severity of Dependence Scale (SDS; see Gossop et al., 1995). Initially developed to assess dependence on drugs of abuse (e.g., alcohol, heroin, cocaine and amphetamine), the SDS is a concise and internally consistent dependence measure for such substances (Gossop et al., 1995; Lawrinson et al., 2007; Martin et al., 2006). More recently, two studies have reported using an adapted version of the SDS to measure AAS dependence (Cole et al., 2003; Griffiths et al., 2018). Despite this, there are concerns of the use of the adapted SDS to measure AAS dependence. First, like the DSM criteria, the SDS was originally established to identify dependence on intoxicating drugs of abuse (e.g., alcohol, heroin, cocaine and amphetamine; see Gossop et al., 1995). Second, there is very limited evidence for the psychometric properties of the adapted version of the SDS, as the validity and reliability of scores obtained through its use are largely unknown (Cole et al., 2003; Griffiths et al., 2018).

The limitations of the SDS and DSM approaches are further highlighted when one considers current models of AAS dependence. Several models have been presented over the last three decades to try to explain AAS dependence (Bahrke & Yesalis, 1994; Brower, 1992; Brower, 2002; Hildebrandt et al., 2011; Kanayama et al., 2010). In an early model Brower (1992) indicates AAS may cause dependence via four possible mechanisms: 1) primary reinforcement through neurological reward pathways (e.g., opioid pathways), 2) secondary reinforcement from increased musculature (including increased self-esteem, outside admiration and winning competitions), 3) avoidance of biologically mediated withdrawal symptoms, or 4) avoidance of psychosocial withdrawal symptoms (e.g., depression due to decreased athletic performance). Limitations with this proposed model include difficulties in differentiating between each of the reinforcing factors within survey and case report research (Kean, 2003), as many of these factors often present themselves simultaneously (Yesalis et

al., 1990). There also remains a disagreement over the psychoactive nature of AAS. Midgely et al. (1999) indicated AAS dependence is likely to be caused by secondary reinforcing effects due to the personal and socially rewarding nature they exert, rather than via psychoactive stimulation. With the model demonstrating difficulties in practical use within research and the absence of agreement over the psychoactive and reinforcing nature of AAS, use of this model in the development of measures to assess and understand AAS dependence could be problematic.

Bahrke and Yesalis (1994) presented a model of AAS dependence whereby development of dependence originates from socio-cultural contexts, subsequently motivating individuals, primarily males, to engage in an intense and frequent rigmarole of training sessions to build highly muscular physiques. Within this model, it is the training sessions that produce improvements in mood, self-esteem and are associated with controlled dietary programmes. Therefore, the reinforcing effects of these anabolic compounds can be attributed to their muscle-building properties (Midgely et al., 1999), and the regimented routines of AAS administration facilitate compulsive training and dietary protocols. Thus, the positively reinforcing effects of this model may relate more to exercise dependence and a desire to boost body capital (Gunnarsson et al., 2022; Koté & Antonopolous, 2021), rather than AAS dependence alone, potentially causing issues for measures of AAS dependence created from this model.

In a later model, Brower (2002) proposed a two-stage process of AAS dependence. The first stage sees AAS being used in high doses to build supraphysiological muscle mass when combined with a strict diet and training regime, reinforced by the development of increased muscle mass this behaviour is maintained despite encountering any adverse effects. The second stage is characterised by individuals administering high dosages of AAS

activating neurological mediated reward pathways preventing the individual from halting use of AAS (Brower, 2002). The latter stage is characterised by the experience of psychoactive effects (i.e., mood changes, increased aggressive behaviours), and associated with polysubstance use of compounds such as opioids, and can be a target for addiction treatments (Arvary & Pope, 2000; Mhillaj et al., 2015). However, Brower (2002) stipulates there is a dearth of evidence of AAS dependence without associated weight training or ergogenic effects on musculature, therefore the positively reinforcing effects of AAS could be attributed more towards an underlying dependence on improving muscular strength, aesthetics, and physical performance (Mhillaj et al., 2015).

Kanayama et al. (2010) indicated that AAS dependence may present via three distinct mechanisms: anabolic effect, androgenic effects, and hedonic effects. The anabolic mechanism is modulated by the presence of muscle dysmorphic disorder, whereby an individual will maintain the use of AAS due to a 'fear' of losing their musculature when abstaining from AAS administration. The androgenic mechanism points to the effects of hypothalamic pituitary gonadal axis suppression, facilitating the development of anabolic steroid induced hypogonadism (ASIH) and associated symptoms (see Tan & Scally, 2009). Therefore, AAS are administered to alleviate the symptoms of ASIH experienced during substance free periods (Brower, 2002). Lastly, the hedonic mechanism demonstrates AAS dependence sharing similarities with dependence to other substances of abuse (e.g., opioids), further demonstrated via animal models (Koob, 2006; Wood, 2002; Wood et al., 2004a).

More traditional models based upon the allostatic framework of addiction (Hildebrandt et al., 2011) have been presented, whereby the development of AAS dependence is believed to be both a psychological and a physical construct. Hildebrandt et al. (2011) describes how an individual can improve their hedonic state by implementing protocols of

exercise and AAS use simultaneously, thereby improving their hedonic tone and bringing about positive reinforcement through social benefit. Combined with chronic AAS use, psychological dependence is established. Physical dependence is achieved once the individual administers ancillary compounds to negate undesired effects (Hildebrandt et al., 2011). However, evidence in the current literature suggests many individuals who use AAS combine their anabolic compounds with ancillary substances to counteract undesired effects (Kanayama et al., 2010; Pope et al., 2014), therefore this may not be indicative of AAS dependence.

The presence of multiple pathways within models of AAS dependence (Brower, 1992; Bahrke & Yesalis, 1994; Brower, 2002; Kanayama et al., 2010; Hildebrandt et al., 2011), demonstrates that AAS dependence is not unidimensional but contains many underlying dimensions, as such it is important that measures are able to identify this by containing multiple factors. Presently, measures used to assess AAS dependence capture AAS dependence as a single factor (Gillespie et al., 2007; Gossop et al., 1995; Grant et al., 2007; Lynskey & Agrawal, 2007; Ray et al., 2008; Teesson et al., 2002) and therefore are limited in representing the likely multidimensional nature of AAS dependence. Research in this area could benefit from a multidimensional measure to discriminate between the underlying dimensions of AAS dependence allowing researchers to identify if specific dimensions are of a greater importance to AAS dependence than others.

To accurately identify, diagnose and further understand AAS dependence, the multidimensional nature identified within existing theories needs to be addressed. Sub-dimensions have been identified within the extant literature, with researchers categorizing AAS dependence as almost unbroken use sustained over time, despite incurring undesired (physical, psychological, and social) effects, and experience of withdrawal symptoms in

periods of abstinence (Brower, 2002; Kanayama et al., 2009, 2009b; Pope et al, 2010). Another sub-dimension identified within models of AAS dependence is the belief that administering chronic supraphysiological doses of AAS improves effectiveness of their regime in enhancing muscular and strength gains (Brower, 2002; Kutscher et al., 2002). For the purpose of this study, we will be adopting and adapting the multidimensional model of AAS dependence proposed by Kanayama et al. (2010) to produce a multidimensional measure of AAS dependence.

A contributing factor in the development of drug dependency is the presence of drug craving (APA, 1994; Drummond, 2001). Believed to manifest itself in a myriad of ways (see Beck et al., 1993), craving presents as an intrusive and dominating sensation causing an individual substantial distress (Tiffany & Wray, 2012). Craving is recognised as a conscious desire for substance use (Sayette et al., 2000), characterised by a want, urge or compulsion to engage in satiating behaviour (Kozlowski and Wilkinson, 1987). With little consensus over the definition of craving, it remains poorly understood (Flannery et al., 2001; Franken, 2003). Craving is believed to present itself during periods of drug abstinence elicited when experiencing drug-related cues (e.g., environmental cues and drug exposure; see Drummond et al., 1990), and due to drug expectancy (Donny et al., 2008; Franken, 2003; Pickens et al., 2011). This should thereby increase the propensity for drug seeking behaviour in individuals with compromised self-efficacy (Marlatt & Gordon, 1985), only satiated by drug use (Drummond et al., 1990).

Craving research has demonstrated its presence within substance use disorders, including alcohol, tobacco, opioid, cocaine, cannabis and other psychoactive substances (Kakko et al., 2019; Serre et al., 2018; WHO, 1992). As such, craving has been included within the ICD-10 diagnostic criteria for substance dependence (WHO, 1992), and more

recently in DSM fifth edition (DSM-V; APA, 2013). However, despite consensus that drug craving has a role in drug dependency (Tiffany and Wray, 2012), there remains a dispute amongst researchers on the presence of a relationship between craving and relapse, indicative of a substance dependency syndrome (Anton, 2000; Hartman et al., 1998; Paliwal et al., 2008; Perkins, 2009; Weiss et al., 2003; Wray et al., 2013). To identify the presence of craving alongside dependence, it may be more beneficial to retain craving as an independent measure rather than consider it a symptom of dependence and incorporate it into measures of substance dependence.

Research on craving has predominantly focused on alcohol and smoking, subsequently seeing the development of many measures suited to these substances (Anton et al., 1995; Cox et al., 2001; Tiffany & Wray, 2012), and their adaptation to suit research of other substances of misuse (Franken et al., 2002; Mol et al., 2003; Tiffany et al., 1993). With no widely accepted or drug specific standardised instruments available (Rosenberg, 2009), the assessment of drug craving has been diverse (Tiffany & Wray, 2012). This leaves researchers in a predicament where they must pick the most suited measure available, leading to inconsistencies when attempting to identify an appropriate measure (Sayette et al., 2000). Despite the development and validation of multi-item craving measures (May et al., 2014; Tiffany et al., 1993; Rabbe et al., 2005) identifying the varied nature of drug craving, single-item scales remain the most commonly used within craving research (Tiffany & Wray, 2012). This somewhat limits our understanding of drug craving as unidimensional single-item measures are unable to reflect the different multifaceted theories of drug craving (Robinson, 1993; Tiffany, 1990; Tiffany et al., 2000).

Extant research looking at the mechanisms of AAS dependence has identified behaviours associated with craving through animal models. Conditioned place preference and

drug seeking behaviours, synonymous with drug craving, have been identified through environmental cues associated with AAS (Arnedo et al., 2002; Schroeder & Packard, 2000; Wood, 2008). Furthermore, medications to alleviate the symptoms of craving (e.g., Naltrexone) have been seen to inhibit behaviours of self-administration of AAS amongst animal models (Peters & Wood, 2005; Pickens et al., 2011; Wood et al., 2004b), supporting the notion AAS can induce a craving-like response. However, some researchers believe animal models are limited in explaining the nature of craving (Mezinkis et al., 2001; Pickens et al., 2011) due to the inability to communicate sensations and perceptions associated with drug use (Drummond et al., 2000; Drummond, 2001). Thus, with literature suggesting the presence of AAS craving it is important to further explore AAS craving to better understand whether it associates with AAS dependence.

To accurately identify and diagnose AAS craving and any existing sub-dimensions, any new measure should aim to represent the multi-dimensional nature of the construct (Tiffany & Wray, 2012). Researchers have characterised craving as eliciting several experiences upon an individual, these include cue-elicited craving (Drummond, 1995), outcome expectancy (Marlatt and Gordon, 1985), and associated positive and negative mood states (Baker et al., 1986, 2004; Cox et al., 2001; Shiffman & Waters, 2004; Tiffany et al., 2000). Cue-elicited craving has been identified within alcohol research, whereby an individual associates their use of a substance (e.g., alcohol) with an environment (e.g., a bar), and elicits a desire or urge to administer the substance if immediate substance use does not take place (Anton, 1999; Drummond et al., 1995). Outcome expectancy is explained by the motivation and desire to administer a substance due to the positive outcome of its use increasing the likelihood of drug-seeking behaviours (Marlatt & Gordon, 1985). The effects of mood on craving have been identified within smoking research, whereby negative affect will increase

craving and increase the risk of substance use in periods of abstinence (Baker et al., 2004; Cox et al., 2001; Shiffman & Waters, 2004), whilst for positive affect craving is facilitated by pleasurable and positively reinforcing effects increasing the risk for drug seeking behaviour (Baker et al., 1986).

Therefore, the overall aim of this study was twofold. First, we aimed to develop measures of AAS dependence and AAS craving and validate their scores. As part of this, we aimed to determine the number of dimensions within each construct. Based on the theories of dependence and craving previously discussed, we hypothesised: *H1a) AAS dependence would have a five-factor structure covering major aspects of AAS dependence (e.g., Increase use of AAS due to dissatisfaction with the effectiveness of current AAS regime, AAS use to self-medicate withdrawal-like symptoms, and continued use despite the experience of adverse physical, psychological and socio-occupational effects attributed to AAS use) and H2a) AAS craving would present with a 4-factor structure, reflecting the various dimensions of AAS-associated craving (e.g., drug expectancy, environmental cues, and positive and negative mood states)*. Second, we aimed to determine the presence of a higher order factor for both scales, which would support the existence of general dimensions of AAS dependence and AAS craving. Based upon our previous arguments, we hypothesised: *H1b) the AAS dependence measure would demonstrate a higher order factor, supporting the existence of an overarching concept of AAS dependence and H2b) the AAS craving measure would demonstrate a higher order factor, supporting the existence of an overarching concept of AAS craving*. To summarize, this research sought to develop two psychometric instruments: an AAS dependence scale and an AAS craving scale. Throughout the study we followed the guidelines and procedures for instrument development and validation present within the

research literature (i.e., Clark & Watson, 1995, 2019; Fabrigar et al., 1999; Haynes et al., 1995; Messick, 1995).

3.2 Methods

Throughout this study we considered five of the six aspects of construct validity proposed by Messick (1995); content, structural, substantive, generalizability, and external. Expert opinion was used for the content aspect via identifying the representativeness and quality of the items for each of the newly developed measures. The structural aspect identifies if the scoring structure is in alignment with the structure of the domains being assessed, this was achieved through factor analysis. The substantive aspect of construct validity was addressed within the study, by examining the association of the scores from our new measures and those from theoretically-associated variables. To identify the extent of score properties and interpretations generalizing to and across groups and settings, multi-sample analysis was carried out. The presence of convergent and discriminant validity was addressed through the external aspect, this was achieved through association with theoretically relevant instrument scores. The final component of construct validity, consequential, is identified through positive and negative consequences occurring from the use of the new measures. As such, this was beyond the scope of an instrument development and validation study, and more applicable to future applications of the measures created within the study.

3.2.1 Item Development

By reviewing existing measures assessing the constructs of interest and the current literature, we developed two pools of items designed to capture the different aspects of AAS dependence and craving (see Clark & Watson, 1995). Twenty-three items representing AAS dependence and 27 items for AAS craving were generated. Items were either adapted from those used in existing scales to make them relevant to AAS ($n = 12$ for AAS dependence and

$n = 15$ for AAS craving; Anton, 2000; Kanayama et al., 2009; Ooteman et al., 2006; Raabe et al., 2005; Welsch et al., 1999; WHO, 1992) or created based upon relevant theory ($n = 9$ for AAS dependence and $n = 12$ for AAS craving). Both the AAS dependence and craving items were provided with a response format of a 7-point Likert scale anchored by 1 (strongly disagree) and 7 (strongly agree), based upon extant guidelines in the literature (see Clark & Watson, 1995, 2019). This response format was used during expert panel analysis and all subsequent data collections. This format has been noted within the literature to present the best compromise between reliability, validity, discriminatory power, and respondent preference (Preston & Coleman, 2000).

Item pools were subjected to content validity assessment to establish whether they represented the phenomenon they intended to measure (Dunn et al., 1999; Haynes et al., 1995; Kline, 2005). The most effective way to evaluate content validity is via expert opinion. Following the guidelines of Dunn et al. (1999), items were sent to 22¹⁰ academics and healthcare workers with cogent experience who had not been involved in item development. Each expert had a PhD in sport psychology, psychology, neuroscience, a medical degree, or were employed within the healthcare sector or as a harm reduction worker with experience of AAS. To establish evidence for the content validity of the items, we presented the item set to our expert panel in a survey consisting of four sections: i) definition of dependence¹¹ and content validity assessment for the dependence items, ii) assessment of format and response items for the dependence items, iii) definition of craving¹² and content validity assessment for

¹⁰ 38 academics and healthcare workers were contacted to be considered members of the expert panel; 22 replied positively and took part in the study.

¹¹ AAS dependence was defined as; A tendency towards a continuous pattern of anabolic steroid use without drug free intervals to either improve effectiveness or avoid withdrawal symptoms, despite experiencing problematic physical, psychological and/or social effects.

¹² AAS craving was defined as; A state-like conscious obsession, desire or compulsion to take anabolic steroids, influenced by an individual's environment, drug expectancy, or mood.

the craving items, iv) assessment of format and response items for the craving items. Within Sections i and iii, the relevant definition was presented followed by the pertinent items. The experts were then asked to evaluate a) how representative each item was of the definition on a 7-point Likert scale anchored at -3 (*not at all representative*) and 3 (*very representative*), and b) comment on each of the item's relevance to the definition.

Mean expert ratings were computed for each item, following guidelines in the literature surrounding item development (see Hambleton, 1980); any rating that deviated considerably from the other expert scores was removed. Deviant scores were defined as those that equated to or exceeded two response options lower than the next score (e.g., scored at -2 when the next lowest item was 0). Items with a mean expert rating of 1.0 or more were retained, whilst items score at 1.0 or less were revised based upon expert panel comments. Out of the 23 items for the AAS dependence scale 11 underwent minor alterations and the remaining 12 remained unchanged. Out of the 27 items for the AAS craving scale, 11 saw minor amendments and the remaining 16 went unchanged. Content validity of the revised items was examined by 10 members of the original expert panel, alongside colleagues from our research group not involved in item creation. Feedback on amended item wording was positive, with only minor adjustments required. Following these stages of item development, the 23 dependence items and 27 craving items were taken forward to the main construct validity phase of the study.

3.2.2 Participants

2.2.3 Sample 1

Participants ($N = 206$) originated from 31 countries ($n_{USA} = 41.7\%$; $n_{UK} = 26.2\%$; $n_{Canada} = 10.7\%$, $n_{Other} = 21.4\%$), and the majority reported being male (90.3%) and heterosexual (85.0%). Participant average age was 32.04 years ($SD = 9.5$), marital status was

reported as being single (35.9%), in a relationship (34.5%), married (28.2%) or divorced (1.5%). Employment status indicated participants as unemployed (1.5%), on temporary benefits (2.4%), students (13.6%), on a pension (1.0%), dependent on others (1.0%), part-time employed (8.7%), full-time employed (65.5%), self-employed (3.9%), or other (2.4%). Participants self-reported their age of AAS initiation ($M = 25.35$ years, $SD = 6.5$), the total number of cycles they had run up to the data collection ($M = 10.3$, $SD = 19.3$), number of cycles they had run in the past 12-months ($M = 1.8$, $SD = 1.2$), and total number of years they have been using AAS ($M = 5.7$, $SD = 6.7$).

3.2.4 Sample 2

Participants ($N = 224$) originated from 17 countries ($n_{UK} = 78.1\%$; $n_{USA} = 12.9\%$; $n_{Canada} = 1.3\%$. $n_{Other} = 7.7\%$). Participants were male (96.4%), average age was 42.47 years ($SD = 10.76$). Employment status was unemployed (1.8%), on temporary benefits (1.8%), on a pension (0.4%), part-time employed (4.0%), full-time employed (67.4%), or self-employed (15.2%). Participants self-reported their age of AAS initiation ($M = 32.48$ years, $SD = 10.79$), the number of cycles they had run in the last 12-months ($M = 2.14$, $SD = 1.45$), and the number of years they have been using AAS ($M = 15.56$, $SD = 11.02$).

3.2.5 Measures

3.2.5.1 Factorial, convergent, and discriminant validity, and internal consistency

To establish evidence for construct validity, we sought evidence for factorial, convergent and discriminant validity via scores from the new measures. Alongside this, we tested for internal consistency. To establish evidence for factorial validity a two-stage approach proposed by Fabrigar et al. (1999) was implemented, whereby Sample 1 identified the dimensions represented in a measure, whilst Sample 2 confirmed the number and nature of

the identified dimensions. Convergent validity was established with Sample 1¹³, by associating scores of the new measures with pre-existing measures identifying the same constructs (Kline, 2005), in this case the measure for AAS dependence and AAS craving were compared to the AAS amended DSM-V criteria (Kanayama et al., 2009b) and the AAS adapted Wisconsin Smoking Withdrawal Scale (AAS-WSWS; Welsch et al., 1999), respectively. Discriminant validity was established by comparing the intercorrelations amongst the subscales of each respective measure. Effect sizes (i.e., small [.10], medium [.30] and large [.50]) for correlation coefficients were determined in accordance with Cohen (1992). Internal consistency was assessed using Cronbach's alpha utilising cut off values predetermined within the present literature (see Nunnally and Bernstein, 1994) of unacceptable ($\alpha < 0.5$), poor ($\alpha \geq 0.5$ to 0.6), questionable ($\alpha \geq 0.6$ to 0.7), acceptable ($\alpha \geq 0.7$ to 0.8), good ($\alpha \geq 0.8$ to 0.9), and excellent ($\alpha \geq 0.9$).

3.2.5.2 AAS dependence

The nine-item AAS adapted DSM-V criteria (Kanayama et al., 2009b) was used to measure AAS dependence. Participants read statements regarding their use and effects associated with their use of AAS (e.g., "over the last 12 months, have you increased the dose/s of steroid/s you are using due to being dissatisfied with your previous results?") and indicated their level of agreement on a 7-point Likert scale, anchored at 1 (*strongly disagree*) to 7 (*strongly agree*). This scale demonstrated good internal consistency ($\alpha = .82$).

¹³ Data collected from Sample 1 included measures of AAS dependence (DSM-V criteria), Moral Disengagement (DMDS), Self-Regulatory Efficacy (DSRE), the AAS adapted Wisconsin Smoking Withdrawal Scale (AAS-WSWS), items assessing patterns of use of AAS, and self-reported items on the experience of undesired effects associated with the use of AAS. Whilst data collated from Sample 2 only included the new measures as it was intended to test for factorial validity alone.

3.2.5.3 Craving

The four-items of craving from the Wisconsin smoking withdrawal scale (Welsch et al., 1999) were adapted to suit AAS craving in this study (AAS-WSWS). The craving items looked at the impact, frequency and thoughts about the use of AAS on the day to day lives of participants (e.g., “I have trouble getting steroids off my mind”). Participants were instructed to respond to these items using a 7-point Likert scale anchored at 1 (*strongly disagree*) and 7 (*strongly agree*). The scale demonstrated excellent internal consistency ($\alpha = .92$).

3.2.5.4 Moral disengagement

The doping moral disengagement scale (DMDS; Boardley et al., 2018) was used to measure doping moral disengagement. This scale consisted of 18 items examining various mechanisms through which people can justify and rationalise doping (e.g., compared to most lifestyles in the general public, doping isn't that bad”). Participants were instructed to indicate their level of agreement with each statement using a 7-point Likert scale anchored at 1 (*strongly disagree*) and 7 (*strongly agree*). The scale demonstrated acceptable to good internal consistency within this study ($\alpha = .88$).

3.2.5.5 Self-regulatory efficacy

The doping self-regulatory efficacy scale (Boardley et al., 2018) was utilised to assess doping self-regulatory efficacy. This measure comprised of six items examining the strength of peoples' beliefs in their ability to resist internal and external pressures to dope (e.g., “How confident are you in your ability to ignore the temptation to dope when feeling down physically?”). Participants were instructed to read a number of statements and indicate their level of confidence using a Likert scale anchored by 1 (*no confidence*) and 5 (*complete confidence*). The scale demonstrated good internal consistency ($\alpha = .88$).

3.2.5.6 Use of AAS

Patterns of use for AAS were also assessed. Status of use was determined by items enquiring if participants were presently ‘on-cycle’, ‘off-cycle’, ‘blasting’, ‘cruising’, or on ‘testosterone replacement therapy (TRT)’. Weekly dose of AAS were self-reported (i.e., “Please indicate what estimated combined dosage of anabolic steroid/s you are currently using”). Response options ranged from ‘Nothing (i.e., off-cycle)’ to ‘Over 2g per week’. Ranges of AAS doses were based upon literature on therapeutic doses (Quaglio et al., 2009), findings from a recent literature review (Pope et al., 2014), and primary research papers (Evans, 1997; Parkinson & Evans, 2006; Pagonis et al., 2006; Yu et al., 2014), indicating current understanding of low (i.e., clinical doses < 300mg per week), medium, and high doses (> 1,000mg per week) of AAS. Participants were presented with a list of different AAS compounds and other IPEDs and instructed to identify what they were currently using (e.g., ancillary drugs, peptide hormones, selective androgen receptor modulators (SARMs), etc). Items on AAS and IPED compounds were based on findings with the current literature (Brower, 2002; Hall et al., 2005; Llewellyn et al., 2017; Parkinson & Evans, 2006; Westerman et al., 2016).

3.2.5.7 Undesired effects of AAS use

Self-report items of detrimental effects associated with AAS use were collected. Items examined the presence of physical and psychological effects currently being experienced by strength athletes who use AAS (i.e., “Are you currently experiencing any of these effects associated with the use of anabolic steroids?”). Physical effects included items well established in the current scientific literature (e.g., acne, fluid retention, injection site pain, cholesterol imbalance, elevated red blood cell count; see van Amsterdam et al., 2010). Psychological effects included items associated with AAS withdrawal (e.g., depressive

thoughts, decreased libido, excessive body checking, increased anxiety, insomnia and mood swings), well established within seminal scientific literature (Brower et al., 1991; Ip et al., 2011; Parkinson & Evans, 2006; Pope et al., 2014; Westerman et al., 2016). Items were self-reported dichotomously via ‘Yes’ and ‘No’ responses.

3.2.6 Procedure

3.2.6.1 Recruitment and data collection

Approval was granted by the first author’s institutional ethics committee (ERN_19-1955) before the study commenced. Participants were recruited through advertisements on bodybuilding and strength training forums where the use of IPEDs such as AAS is regularly discussed, social media platforms (Facebook, Twitter, Reddit), needle and syringe programmes, and via existing contacts (i.e., gatekeepers). Potential participants were provided with a brief description of the study and a hyperlink to access the survey. Once accessed, participants were presented with an information sheet and consent form. Participants were informed that honesty in responding was essential to the study and that the anonymity of participants was assured as no personal details were gathered from participants. Participants were required to provide their informed consent before completing the survey, which took approximately 10 -15 minutes. Data were collected over two phases, data from phase one (i.e., Sample 1) was analysed before data collection began for the second phase (i.e., Sample 2). This allowed for item adjustment/creation between phases if required. Data for phase 2 was collated by the first author and a colleague from a different university with ethical approval from their institution (21/PHI/019). Upon survey completion participants were entered into a prize draw to win a £25, £50 or £100 Amazon voucher. Email addresses were collated for the purpose of the prize draw, but these were stored separately from the study data to protect anonymity.

3.3 Results

3.3.1 Preliminary Analyses

No missing data were present in either of the datasets. To identify the most appropriate items to measure each construct, a two-stage process recommended by Clark and Watson (1995) was utilised. Inter-item correlations were examined within each of the respective constructs and all item with correlations greater than .15 with all other items were retained for further analysis. Exploratory factor analysis (EFA) was then conducted for each of the nine hypothesised subscales (i.e., five dependence subscales and four craving subscales) using principal axis extraction and direct oblimin rotation, with extraction based on eigenvalues ≥ 1.00 . Subscales were analysed individually to determine and retain the best indicators of each latent variable (Jöreskog & Sörbom, 1993). Before conducting these analyses, the appropriateness of these subscales was determined by following criteria of Dziuban and Shirkey (1974) and Kaiser (1974); significant Bartlett's test of sphericity and a Kaiser Meyer Olkin measure of sampling adequacy $> .50$. All items except one had factor loadings of $\geq .52$. The one exception was an AAS dependence expectation item (i.e., "I have been fearful of regressing in my training if I halted my use of steroids"), with a factor loading of .43, alongside a low correlation value ($r < .40$) with all other items loading on that factor; this item was therefore removed from further analysis. A total of 22 items for AAS dependence and 27 items for AAS craving with minimal factor loadings of .50 remained for subsequent confirmatory factor analysis (CFA).

3.3.2 Factorial Validity

CFA was utilised to establish evidence for factorial validity due to its ability to rigorously test and confirm hypothesised factor structures (Fabrigar et al., 1999). As previously discussed, we defined AAS dependence and craving as multidimensional constructs. For dependence we

expected five dimensions to be present; effectiveness, withdrawal symptoms, physical effects, psychological effects and social effects. In turn, for craving we expected four dimensions: environment, drug expectancy, negative mood, and positive mood.

CFA analysis was conducted using Mplus software (Muthén & Muthén, 2017) using Maximum Likelihood (ML) estimation. Kolmogorov-Smirnov and Shapiro-Wilk tests indicated significant deviation from normality ($p < .001$), thereby requiring robust estimation. This is the default setting with Mplus and ML estimation, producing robust standard errors, model fit indices and chi-square values (Muthén & Muthén, 2017). Multiple complimentary fit indices were used to evaluate model fit (see Hu & Bentler, 1999), specifically; chi-square (X^2), Comparative Fit Index (CFI), Standardised Root Mean Residual (SRMR), and Root Mean Square of Error Approximation (RMSEA). Good model fit is achieved when the CFI, RMSEA and SRMR values are $\geq .95$, $< .06$ and $< .08$ respectively (Hu & Bentler, 1999). To compare nested models for best fit, Akaike Information Criterion (AIC) were used, with the lower value being preferred (Hair et al., 1998).

In the first AAS dependence model all 22 items were utilised and loaded onto a single factor; four items for effectiveness, nine for withdrawal, three for physical effects, three for psychological effects and three for social effects (M1a; see Table 3.1). Results demonstrated an inadequate model fit (Row 1), supporting the multidimensional nature of the scale and indicating a requirement for re-specification. Seven items presenting weak factor loadings and large standardised residuals were removed in a series of CFAs in which the hypothesised 5-factor model was specified. A final model with 15 items produced good model fit (M1b) with three items loading onto each of the five factors (Row 2). Each of the factors were hypothesised to represent a form of AAS dependence, therefore we thought it prudent to examine for presence of a higher order factor representing the five first-order factors. When

the fit of a second-order model approaches that of a first-order model, there is sufficient support for the presence of a second-order structure (Marsh, 1987). As this was the case here (see M1c, Row 3), we accepted the higher-order model, and named the second-order factor AAS dependence.

Although the data supported our hypothesised model of a five-factor structure, it was important to ensure that alternative models could be ruled out (see Table 3.1). We compared the model fit of the five-factor model with that of other possible structures. These were a unidimensional model with all 15 items loaded onto a single factor (M2, Row 4) and a three-factor model with undesired physical, psychological, and social effects loaded onto the same “undesired effects” factor (M3, Row 5). Table 3.1 demonstrates the fit of model M1b was superior to that of both alternative models. Therefore, the five-factor model was accepted as the best model for AAS dependence. Factor correlations and internal consistency scores for this model can be seen in Table 3.2, and items, factor loadings and error variance can be seen in Table 3.3. We named the final scale the Androgenic-Anabolic Steroid Dependence Scale (AASDS).

Table 3.1. Model fit indices for each CFA model run for AAS dependence and craving measures for the first ($N = 206$) and second ($N = 224$) samples.

Model	<i>df</i>	χ^2	CFI	SRMR	RMSEA	AIC
Sample 1						
Dependence						
Models						
1. M1a, 22-Items	199	590.59	0.89	0.06	0.09	15817.64
2. M1b, 15-Items	80	151.10	0.97	0.05	0.06	10570.01
3. M1c, Second Order 15-Items	85	162.92	0.96	0.05	0.06	10571.01

Alternative Dependence Models						
4. M2, 15-Items	90	1247.43	0.54	0.12	0.25	11646.35
5. M3, 15-Items	87	853.73	0.69	0.11	0.20	11258.65
Craving Models						
7. M4a, 27-Items	318	909.49	0.90	0.06	0.09	17627.40
8. M4b, 16-Items	98	227.44	0.96	0.04	0.08	9958.65
9. M4c, Second Order 16-Items	100	234.30	0.96	0.04	0.08	9961.51
Alternative Craving Models						
10. M5, 16-Items	104	1393.37	0.65	0.09	0.24	11262.45
11. M6, 16-Items	101	598.33	0.86	0.10	0.15	10473.41
Sample 2						
Dependence Models						
12. M1a, 15-Items	80	192.16	0.96	0.04	0.07	11287.10
13. M1b, Second Order 15-Items	85	195.01	0.96	0.04	0.07	11279.92
Craving Models						
14. M2a, 16-Items	98	243.07	0.97	0.03	0.08	10080.60
15. M2b, Second Order 16-Items	100	251.07	0.97	0.04	0.08	10084.60

Note: df = Degrees of Freedom, X^2 = Chi-square, CFI = Comparative Fit Index, SRMR = Standardized Root Mean Square Residual, RMSEA = Root Mean Square of Error Approximation, AIC = Akaike Information Criterion. M1 = five-factor model; M2 = one-factor model; M3 = alternate item five-factor model; M4 = three-factor model; M5 = three-factor model; M6 = one-factor model; M7 = three-factor model.

Table 3.2. Internal consistency and correlations between factors from final AAS dependence scale models (M1c and M1b 15-item five factor models), DMDS, DSRES and DSMV from Sample 1 ($N = 206$) and Sample 2 ($N = 224$). Sample 2 data is displayed above the diagonal.

Variable	α	1	2	3	4	5	6
1. Effectiveness	.80/.82	-	.42**	.39**	.42**	.41**	
2. Withdrawal	.92/.91	.43**	-	.47**	.66**	.59**	
3. Physical	.94/.95	.31**	.33*	-	.53**	.49**	
4. Psychological	.91/.90	.47**	.56**	.43*	-	.61**	
5. Social	.87/.90	.47**	.62**	.32**	.64**	-	
6. Doping MD	.88	.23**	.18*	.19**	.29**	.38**	-
7. Doping SRE	.88	-.08	-.12	-.15*	-.23**	-.16*	-.04

Note: * = $p < .05$; ** = $p < .05$; *** = $p < .000$

Correlations are presented below the diagonal for Sample 1, and above the diagonal for Sample 2. Alpha scores are presented on the left-hand side for Sample 1 and on the right-hand side for Sample 2. No alpha scores are presented for Doping MD and SRE for Sample 2 as these scores were only collated with Sample 1 data.

Table 3.3. M1c Items, standardized factor loadings and error variances for the AAS dependence scale (AASDS) from Sample 1 ($N = 206$) and Sample 2 ($N = 224$).

Factor	Factor Loadings	Error Variances
<i>Effectiveness</i>		
1. I increased my use of steroids due to being dissatisfied with the effectiveness of my regime.	0.63/0.68	0.60/0.53
2. I went beyond my pre-planned use of steroids to increase my gains.	0.88/0.85	0.22/0.26
3. I sometimes went beyond my pre-planned regime to increase gains.	0.78/0.81	0.38/0.33
<i>Withdrawal</i>		

4. I used steroids to alleviate withdrawal-like symptoms experienced during an “off-cycle” period.	0.81/0.80	0.34/0.34
5. I had a strong compulsion to use steroids when “off-cycle” due to experiencing withdrawal-like symptoms.	0.93/0.93	0.12/0.12
6. I experienced withdrawal-like symptoms which made it difficult for me to stay “off-cycle”.	0.94/0.92	0.11/0.13

Unwanted Physical Effects

7. I continued using steroids despite experiencing unwanted side effects (e.g., gynecomastia, heart complications, cholesterol imbalance, abscesses, tendon/joint damage, testicular atrophy).	0.93/0.91	0.12/0.17
8. I continued to use steroids despite trying to manage undesired side effects (e.g., gynecomastia, heart complications, cholesterol imbalance, abscesses from injections, tendon/joint damage, testicular atrophy).	0.89/0.96	0.20/0.07
9. I have continued with my steroid regime even though I experienced unwanted effects (e.g., gynecomastia, heart complications, cholesterol imbalance, abscesses from injections, tendon/joint damage, testicular atrophy).	0.93/0.91	0.13/0.16

Unwanted Psychological Effects

10. I continued with my steroid regime despite seeking help for problematic psychological effects (e.g., depressive thoughts, a decreased libido, increased anxiety, insomnia, and mood swings).	0.87/0.81	0.23/0.34
11. I continued using steroids despite having experienced depressive thoughts, a decreased libido, increased anxiety, insomnia, or mood swings.	0.88/0.93	0.21/0.13
12. I experienced concern over unwanted psychological effects (e.g., depressive thoughts, decreased libido, increased anxiety, insomnia, mood swings), but continued to use steroids.	0.89/0.87	0.20/0.23

Unwanted Social Effects

13. I avoided social, occupational and/or recreational activities which may have interfered with my steroid regime.	0.67/0.81	0.55/0.34
14. I avoided social, occupational or recreational activities to prioritise my steroid regime, causing problems in my personal life (e.g., with close family, friends, partner/significant other, boss/manager).	0.94/0.90	0.10/0.18
15. I prioritised my steroid regime over social, occupational and/or recreational activities, even when the outcome was problematic.	0.91/0.91	0.16/0.15

Similar procedures were followed to develop the measure for AAS craving. Initially all 27 of the items were loaded onto a single factor (M4), this model demonstrated an inadequate fit (Row 7), supporting the multidimensional nature of the scale and indicating a requirement for re-specification. Subsequently, 11 items presenting weak factor loadings and large standardised residuals were removed in a series of CFAs in which the hypothesised four-factor structure was specified. The fit of the final 16-item model with four items for each of the four factors can be seen in Table 3.1 (M4b), demonstrating good model fit. Each of the factors were hypothesised to represent a form of AAS craving, therefore we assessed the data for the presence of a higher order factor representing the four first-order factors. The second order model represented fit similar to that of the first order model, thus the higher-order model was accepted, and we named the second order factor AAS craving.

Alternative model structures were assessed to ensure that M4c was the best model for AAS craving. The first was with a unidimensional model with all items 16-items run on one factor (M5), indicating poor model fit. The second was a three-factor model with “positive mood” and “negative mood” combined into a single “mood” factor (M6), indicating poor fit. Table 3.1 contrasts the fits of the various models, demonstrating M4c as the superior model.

This model was therefore accepted as the final model for AAS craving. Table 3.4 identifies the factor correlations and internal consistency scores, and Table 3.5 indicates the items, factor loadings and error variances. We named the scale the Anabolic-Androgenic Steroid Craving Scale (AASCS).

Table 3.4. Internal consistency and correlations between factors in the final craving model (Sample 1 M4c and Sample 2 M2b 4 factor models), Doping MD, Doping SRE and AAS dependence.

Variable	α	1	2	3	4	5	6
1. Expectation	.90/.93	-	.62**	.75**	.67**		
2. Environment	.92/.95	.67**	-	.57**	.64**		
3. Positive Mood	.92/.94	.73**	.66**	-	.68**		
4. Negative Mood	.98/.98	.61**	.58**	.70**	-		
5. Doping MD	0.88	.36**	.46**	.45**	.34**	-	
6. Doping SRE	0.88	-.31**	-.27**	-.26**	-.16*	-0.04	-
7. AAS Dependence	0.82	.55**	.56**	.48**	.58**	.27**	-.13*

Note: * = $p < .05$; ** = $p < .05$; *** = $p < .000$

Correlations are presented below the diagonal for Sample 1, and above the diagonal for Sample 2. Alpha scores are presented on the left-hand side for Sample 1 and on the right-hand side for Sample 2. No alpha scores are presented for Doping MD and SRE for Sample 2 as these scores were only collated with Sample 1 data.

Table 3.5. Items, standardized factor loadings and error variances for the AAS craving scale (AASCS) from Sample 1 ($N = 206$) and Sample 2 ($N = 224$).

Factor	Factor Loadings	Error Variances
Item		
<i>Expectation</i>		

1. I have trouble getting steroids off my mind because of what they can do for me.	0.84/0.91	0.29/0.16
2. I frequently think about my steroid routine because of how it makes me feel.	0.82/0.89	0.31/0.20
3. Much of my time is occupied by ideas, thoughts, impulses, and images relating to what I can achieve whilst using steroids.	0.85/0.87	0.28/0.23
4. It takes a lot of effort to disregard my thoughts and feelings about my use of steroids.	0.80/0.87	0.35/0.24
<i>Environment</i>		
5. Being around my gym friends makes me want to use steroids.	0.91/0.93	0.15/0.12
6. Talking to other gym users about training makes me want to use steroids.	0.90/0.93	0.18/0.13
7. Being around my gym friends makes me desire steroids.	0.74/0.91	0.45/0.16
8. Passing by a gym can make me want to use steroids.	0.94/0.89	0.10/0.19
<i>Positive Mood</i>		
9. The thought of using steroids makes me feel more relaxed.	0.82/0.86	0.32/0.25
10. I think about using steroids as they improve my mood.	0.82/0.89	0.32/0.20
11. Knowing I will be using steroids improves my mood.	0.93/0.92	0.12/0.14
12. I feel more content when I anticipate the use of steroids.	0.90/0.93	0.19/0.12
<i>Negative Mood</i>		
13. I have a desire to use steroids when I feel down.	0.95/0.90	0.08/0.17
14. When I am in a low mood, I want to use steroids.	0.96/0.98	0.06/0.02
15. The feeling of being down makes me desire steroids.	0.97/0.98	0.05/0.02
16. I sometimes have urges to use steroids when I feel low.	0.95/0.98	0.09/0.02

To further examine the construct validity of scores generated using the AASDS and AASCS, we collected evidence relating to their convergent, concurrent and discriminant

validity with Sample 1. To examine convergent validity, correlations between the AAS amended DSM-V criteria (AMA, 2013) with the AASDS and the AAS-WSWS (Welsch et al., 1999) with the AASCS were computed (see Table 3.6). Evidence for convergent validity would be established if the AASDS and AASCS were correlated at moderately high levels with the DSM-V and AAS-WSWS respectively. If the correlation is too high ($r > .90$) then the construct is redundant (Kline, 2005). AASDS was positively associated with the DSM-V ($r = .71, p < .001$). AASDS subscales of ‘Effectiveness’ ($r = .51, p < .001$), ‘Withdrawal’ ($r = .58, p < .001$), ‘Physical Effects’ ($r = .36, p < .001$), ‘Psychological Effects’ ($r = .64, p < .001$), and ‘Social Effects’ ($r = .64, p < .001$) demonstrated moderate to strong significantly positive associations with the DSM-V. AASCS was positively related to the AAS-WSWS ($r = .85, p < .001$). AASCS subscales of ‘Expectancy’ ($r = .87, p < .001$), ‘Environment’ ($r = .74, p < .001$), ‘Negative Mood’ ($r = .64, p < .001$), and ‘Positive Mood’ ($r = .70, p < .001$) demonstrated strong positively significant associations with the AAS-WSWS.

Concurrent validity was assessed by measuring the associations between AASDS and experience of undesired effects (see Table 3.6). Past research has indicated that AAS dependence should correlate positively with experience of undesired effects associated with AAS use (Pope et al., 2010; Ip et al., 2011). Analysis demonstrated that there was a significant positive association between AASDS subscales and number of undesired effects experienced; effectiveness ($r = .18, p < .01$), withdrawal ($r = .12, p < .01$), physical effects ($r = .44, p < .01$), psychological effects ($r = .27, p < .01$), and social effects ($r = .21, p < .05$). Further evidence of concurrent validity was explored with moral disengagement (MD) and self-regulatory efficacy (SRE), due to research identifying presence of high MD and low SRE within the AAS using community (Boardley et al., 2018). Therefore, we would expect a positive association with MD and negative associations with SRE with AAS dependence.

Table 3.6 demonstrates AASDS association with both MD ($r = .33, p < .01$) and SRE ($r = -.18, p < .01$), AASDS subscales also demonstrated significant associations with MD and SRE (see Table 3.2).

Concurrent validity for AASCS scores was assessed by determining the associations of AAS craving with DMDS, DSRE and the DSM-V scores (see Table 3.6). Past research has indicated that craving is associated with self-regulatory efficacy (Shadel & Cervone, 2006), moral disengagement (Ahmadi et al., 2019), and drug dependence syndromes (Donny et al., 2008). Analysis indicated significant positive associations between AASCS subscales (see Table 3.4); expectation ($r = .36, p < .01$), environment ($r = .46, p < .01$), positive mood ($r = .45, p < .01$) and negative mood ($r = .34, p < .01$) with MD. SRE was significantly and negatively associated with expectation ($r = -.31, p < .01$), environment ($r = -.27, p < .01$), positive mood ($r = -.26, p < .01$), and negative mood ($r = -.16, p < .05$). DSM-V scores were significantly associated with expectation ($r = .55, p < .01$), environment ($r = .56, p < .01$), positive mood ($r = .48, p < .01$), and negative mood ($r = .58, p < .01$). AASCS scores demonstrated significant associations with both MD ($r = .47, p < .001$) and SRE ($r = -.29, p < .001$; see Table 3.6).

Discriminant validity of the AASDS and AASCS scores was examined through intercorrelations among subscales scores for the AASDS and AASCS. Correlations for AASDS ranged from .31 to .64 (see Table 3.2) indicating distinct separation between all subscale pairs. AASCS indicated correlations ranging from .58 to .73 (see Table 3.4), indicating distinct separation between all factors within the construct. This analysis provided evidence for sufficient levels of discriminant validity within both the AASDS and AASCS. Reliability of the new measures was assessed with internal consistency (see Table 3.6), presenting with high scores for both AASDS (.91) and its subscales ranging from .80 to .94.

Internal consistency of AASCS (.96) and its subscales (.89 to .98) also indicated excellent levels of internal consistency.

Table 3.6. Descriptive statistics, internal consistencies, and correlations between AASDS, number of self-reported undesired effects, AASCS, DSMV, AAS-WSCS, DMDS, and DSRE from Sample 1 ($N = 206$).

	Variable	α	Mean	SD	Range	1	2	3	4	5	6	7
1	AASDS	0.91	3.09	1.35	1.00-7.00	-						
2	Undesired Effects		1.68	2.06	1.00-9.00	.29**	-					
3	AASCS	0.96	3.00	1.52	1.00-7.00	.72**	.23**	-				
4	DSMV	0.82	1.86	0.58	1.00-4.00	.72**	.23**	.63**	-			
5	AAS-WSCS	0.92	2.93	1.73	1.00-7.00	.67**	.25**	.85**	.57**	-		
6	Doping MD	0.88	4.51	0.99	2.28-7.00	.33**	.19**	.47**	.27**	.34**	-	
7	Doping SRE	0.88	3.55	1.13	1.00-5.00	-.18**	-.26**	-.29**	-.14*	-.30**	-.04	-

Note: * = $p < .05$; ** = $p < .05$; *** = $p < .001$

No internal consistency was computed for Undesired effects as this was an aggregated value from a multiple-choice list of undesired effects experienced associated with AAS use, and not a measure.

3.3.3 Confirmation of Factor Structure

Following on from the CFA analyses on the first sample, CFA was conducted on the two (i.e., first- and higher-order) final AAS dependence models with the data gathered from Sample 2: a) the 15-item, five-factor, first order model confirmed with data from the first sample; b) a hierarchical model with five first-order factors and one second-order factor. CFA was also conducted on the two (i.e., first- and higher-order) final AAS craving models presented with data from the first sample: a) the 16-item, four-factor, first order model confirmed with data from the first sample; b) a hierarchical model with four first-order factors and one second-order factor. The results of these CFA are presented in Table 3.1.

The five-factor first-order AAS dependence model was the first to be tested (M1a). The results demonstrate a good model fit (Table 3.1, row 12). The second AAS dependence model analysed was the hierarchical five-factor second-order model (M1b), presented with good model fit and achieved a similar fit to the first model (Table 3.1, row 13), with a very similar AIC value. Based on this we accepted M1b. Factor loadings, error variances and the final 15-items are shown in Table 3.4. For AAS craving the four-factor first-order model was tested first (M2a). The model showed good model fit (Table 3.1, row 14). The second AAS craving model to be tested was the hierarchical four-factor second-order model (M2b). This model demonstrated good model fit (Table 3.1, row 15), with similar fit indices to the first model. Due to no major differences in the model fit we accepted M2b. Factor loadings, error variances and the final 15-items are shown in Table 3.5. The final versions of the AASDS and AASCS can be found in Appendix A and B respectively.

3.4 Discussion

Research has highlighted the prevalence of AAS dependence amongst those who use AAS (Brower et al., 1991; Hauger et al., 2020; Kanayama et al., 2009; Pope et al., 2014; Scarth et

al., 2022). It is also possible – but to date not examined – that craving plays a role in AAS dependence. However, to examine these constructs fully, it is important to measure them using valid instruments tailored specifically to the characteristics of AAS use. Existing measures of AAS dependence (see Kanayama et al., 2009; Griffiths et al., 2018) have significant limitations (Gossop et al., 1995; Kanayama et al., 2009b; Bahrke & Yesalis, 1994; Brower, 1992; Brower, 2002; Hildebrandt et al., 2011; Kanayama et al., 2010). There also remains no current measure to assess craving in people who use AAS. Therefore, the overarching aim of this study was to develop psychometrically robust scales to assess dependence and craving specifically with populations who use AAS.

Previous research suggested multiple dimensions may underlie and contribute to AAS dependence (Bahrke & Yesalis, 1994; Brower, 1992; Brower, 2002; Hildebrandt et al., 2011; Kanayama et al., 2010). Consequently, we developed a multidimensional scale and hypothesised that we would see evidence of five lower-order factors and one higher-order factor with scores obtained using the final instrument. Factor analysis of the data from both samples supported these hypotheses, providing evidence for the multifaceted nature of AAS dependence and supporting *H1a* and *H1b*. This is important in furthering our understanding of AAS dependence as present measures such as the DSM-V and SDS capture only a single factor (Gillespie et al., 2007; Gossop et al., 1995; Grant et al., 2007; Lynskey & Agrawal, 2007; Ray et al., 2008; Teesson et al., 2002). In contrast, our measure of AAS dependence can capture and aid our understanding of the complex multidimensional nature of AAS dependence.

This development of a valid and reliable multidimensional scale to assess AAS dependence will further our understanding of this syndrome through the assessment of novel research questions that could not be answered with current assessment instruments. For

example, previous research has identified those dependent on AAS administer higher doses of AAS per week, and higher number of anabolic compounds than non-dependent individuals (see Ip et al., 2012). However, through use of the AASDS, it is now possible to further our understanding on this by answering depth questions such as, ‘which dimension of AAS dependence is linked most strongly with higher dosages of AAS?’, or ‘which dimension of AAS dependence is associated most strongly with use of more anabolic compounds?’.

Furthermore, evidence from the current literature indicates, in some cases, AAS being administered to self-medicate against withdrawal-like symptoms when off-cycle (Christou et al., 2017; Kanayama et al., 2015). Therefore, researchers could use the AASDS to address research questions such as ‘is the subdimension of withdrawal linked with a greater propensity to shorten off-cycle periods, or progress to a blast and cruise protocol?’. Evidence suggests a link between AAS dependence and a greater number of self-reported undesired effects associated with use of AAS (Ip et al., 2012). The AASDS now allows researchers to investigate possible links between the three subdimensions linked to undesired effects (physiological, psychological, and social) and an increased number and severity of undesired effects. Answering questions such as these could help practitioners by identifying primary areas to be targeted by therapeutic interventions and therefore the development of more effective support services.

With no existing measure for AAS craving and evidence in the existing literature identifying the presence of craving-like behaviours in people who use AAS (Arnedo et al., 2002; Peters & Wood, 2005; Schroeder & Packard, 2000; Wood et al., 2004; Wood, 2007) we also sought to develop a measure of AAS craving. As existing literature indicates potential for drug craving to be multifaceted (May et al., 2014; Raabe et al., 2005; Tiffany et al., 1993; Tiffany & Wray, 2012), we aimed to develop a multidimensional scale and hypothesised the

presence of four lower-order and one higher-order factor in the final instrument. Factor analysis with both samples provided support for these suppositions, therefore supporting hypotheses *H2a* and *H2b*. The development of a multidimensional measure of AAS craving is important as it will allow researchers to examine the potential role of the different dimensions of AAS craving in the development of AAS dependency specifically and use of AAS more generally.

The AASCS will allow researchers to address a wide range of important research questions that until now could not be addressed. For instance, researchers could seek to determine which dimension/s of drug craving predict increases in AAS dependence over time, whether environmentally influenced craving is associated with the amount of time athletes spend in the gym environment, or whether manipulation of athletes' mood moderates any relationship between mood-related craving and AAS use. Addressing research questions such as these will not only benefit our current understanding of how AAS craving may contribute to AAS dependence, but it will also enable practitioners to target specific subdimensions that have stronger associations with AAS dependence and can therefore be the target of therapeutic intervention by harm reduction practitioners.

Convergent validity is a key component of scale development, constituting one of the six essential aspects of construct validity that should be addressed when developing and validating new scales (Messick, 1995). Presence of convergent validity requires scores obtained with the newly constructed measure to correspond with those from existing measures of the same construct (Byrne, 2012; Clark & Watson, 1995, 2019). Such evidence provides support for the new measure's ability to capture the latent construct it was designed to assess. In terms of the study, we sought evidence for convergent validity of the AASDS through correlations with the AAS adapted DSM-V criteria (Kanayama et al., 2009b) and of the

AASCS through associations with the AAS adapted WSWS (Welch et al., 1995). Our analyses testing these relationships provided evidence for the convergent validity of scores obtained with both the AASDS and AASCS. Convergent validity of AASDS scores was evidenced by strong positive correlations with AAS adapted DSM-V scores, whereas for the AASCS this was provided by strong positive associations with AAS-WSWS scores. These associations are consistent with recommendations for convergent validity, whereby the magnitude of correlations should be at least moderate (Byrne, 2012; Kline, 2005). This is important as it provides evidence the AASDS and AASCS assess their respective psychological constructs of dependence and craving.

Another important element of establishing construct validity is providing evidence of concurrent validity (Adams et al., 2014; Kline, 2005). Here, evidence should be provided showing the new scale can predict theoretically-related constructs when data on the two constructs are collected at the same time (Kline, 2005). Presently, we provided evidence for the concurrent validity of AASDS scores by correlating them with scores of self-reported undesired effects from AAS use, whereas for the AASCS we associated scores with those of doping MD and doping SRE (Boardley et al., 2018). Support for the concurrent validity of both new measures was provided through strong positive correlations between AASDS scores and self-reported undesired effects from AAS use and for AASCS scores through moderate positive associations with doping MD and moderate negative associations with doping SRE. Support for the concurrent validity of AASDS and AASCS scores is important, as it indicates scores with both measures are capable of predicting theoretically related constructs.

Correlations examining concurrent validity showed differences across the subscales of the AASDS and AASCS. For the AASDS, the *“use of AAS despite experiencing associated undesired physical effects”* subscale had the strongest association with self-reported

experience of undesired effects, and “*use of AAS despite experiencing associated undesired social effects*” the weakest. This is consistent the extant literature on AAS dependence, whereby AAS dependent users reported experiencing more undesired physical effects whilst using AAS (Ip et al., 2012), and reported a higher frequency of undesired physical effects compared to undesired psychological and social effects (Pope et al., 2010; Ip et al., 2012). For the AASCS, the “*environmental cues*” and “*AAS expectancy*” subscales had the strongest associations with doping MD and doping SRE, and “*negative mood*” indicated the weakest association with these constructs. These findings are consistent with non-AAS research on craving, where SRE has shown a negative association with craving (Shadel & Cervone, 2006) and a positive association with MD (Ahmadi et al., 2019). The differential associations with theoretically related constructs for the AASDS and AASCS subscales are important as they provide evidence the subscales are capturing different elements of dependency and craving.

Evidence for discriminant validity is also required when validating new scales. Messick (1995) indicates this can be provided by examining the strength of associations amongst subscale scores. Providing evidence of discriminant validity discounts the possibility that alternative constructs influence the scores of a new measure, and items within the measure are related but distinct to one another (Clark & Watson, 1995). With regards to this study, we determined the degree to which AASDS and AASCS subscales were associated with one another. For the AASDS, subscale scores were moderately to strongly associated with one another, indicating whilst they are all underpinned by a higher-order dependency construct, they are distinct from one another. More specifically, the “*experience of undesired physical effects*” subscale had weaker associations with the other four subscales, suggesting discriminant validity was highest for scores for this subscale. In contrast, scores for the “*experience of undesired psychological effects*” subscale had the strongest associations with

the other subscales, so evidence of discriminant validity was weakest for scores for this subscale. For AASCS scores, there was evidence for similar levels of discriminant validity. For the specific subscales, scores for the “environmental cues” subscale evidenced weaker associations than other subscales, providing the strongest evidence for discriminant validity. In contrast, the “*positive mood*” subscale showed the strongest associations with other subscales, and therefore evidenced lower levels of discriminant validity.

Finally, new measures should also provide evidence supporting the internal consistency of scale and subscale scores as this provides evidence for the homogeneity of items (Clark & Watson, 1995, 2019). Assessment of the internal consistency values for both the AASDS and AASCS and their respective subscales exceeded the minimum criterion levels recommended when developing novel scales (i.e., 0.80; Clark & Watson, 1995, 2019) in both samples. This provides strong and consistent evidence for the internal consistency of scale scores for the AASDS and AASCS as well as their respective subscales.

3.4.1 Limitations and future directions

This project developed and validated the scores of two multidimensional psychological instruments measuring AAS dependence and craving, providing strong evidence for the psychometric properties of both scales. However, this research was not without limitations which should be acknowledged. First, it is possible that individuals recruited within the study may not reflect all typologies of individuals who use AAS, identified in previous research (Christiansen et al., 2017) potentially indicating a degree of sampling bias. Therefore, future studies should look to validate scores of the AASDS and AASCS with other populations of AAS users. Use of different methods of recruitment to the ones used within this study may facilitate this process, therefore we recommend the use of ‘insiders’ to help recruit harder to reach AAS using populations.

A further limitation is that we did not examine all aspects of validity. Validation of measures is an ongoing process (Clark & Watson, 1995), and researchers are encouraged to examine other aspects of validity of AASDS and AASCS scores in future work. For instance, researchers could examine the predictive validity of scores through longitudinal research that examines possible links with theoretically related constructs over time. For instance, associations between AASDS and AASCS scores and emotional states (e.g., positive and negative affect) could be tested. Our data were collected largely from westernised cultures, too. Further validation of the scales with non-westernised cultures is therefore another important avenue for future work.

Finally, it was beyond the scope of this article to measure test-retest reliability to establish the consistency of AASDS and AASCS scores across time, and as such future researchers should aim to address this. For those engaging in such work, we recommend short time intervals between data collection periods for craving, given it is a state-like construct (Drummond et al., 2000; Sayette et al., 2000; Shiffman et al., 1996). In contrast, longer time intervals could be used for dependence given it is a more enduring construct (see APA, 2013). It is important to keep this in mind, as lower stability scores from craving may reflect changes in levels of craving over time rather than inconsistencies in measurement (Sayette et al., 2000).

3.5 Conclusion

Through a rigorous set of procedures, we developed psychometric scales to assess AAS dependence and AAS craving, providing strong evidence for the validity of scores obtained with both measures. Specifically, evidence for several aspects of construct validity (i.e., convergent, concurrent and discriminant validity) and internal consistency was provided for the AASDS and AASCS. During item development we also provided evidence for high levels

of content and face validity of items through feedback from relevant experts. The AASDS makes important contribution to the literature as it is the first AAS dependency measure specifically designed for those who use AAS rather than being adapted from existing measures. In turn, the development of the AASCS is important because it represents the first AAS craving measure. Further, the multidimensional nature of both measures provides exciting possibilities for future research. We look forward to seeing further evaluation and use of these measures in future research.

Chapter 4 - Temporal Changes in AAS Craving, Anticipated Guilt, Self-Regulatory Efficacy and Mood in Anabolic-Androgenic Steroid Use: A Naturalistic Single Case

Experimental Investigation

4.1 Introduction

Anabolic-androgenic steroids (AAS) are a family of synthetic derivatives of testosterone, increasing in prevalence within recreational gym settings (Zahnow et al., 2018). Traditionally administered in cycles, some users move towards a continuous method of use (Christou et al., 2017), increasing the risk of experiencing a plethora of undesired effects (Brower, 1989, 1992; Ip et al., 2012; Kanayama et al., 2009a, 2009b, and the development of AAS dependence (Kanayama et al., 2009a, 2009b, 2010; Pope et al., 2014). A construct thought to facilitate substance dependence is drug craving (Donny et al., 2008). Experienced in periods of drug abstinence, craving elicits drug seeking behaviours satiated only through substance administration (Kozlowski & Wilkinson, 1987). Previous research has demonstrated that craving behaviours are mediated via psychosocial factors (see Sayette, 2004). Although links have been established between dependence and craving for other forms of drug use, to date, researchers have not examined AAS craving as a manifestation of dependence nor the temporal dynamics of it along with theoretically associated psychosocial variables. The overarching aim of this study was to address this gap in the literature.

Administration of AAS traditionally occurs in 'cycles' (Kanayama et al., 2003), with anabolic compounds being used over 8-to-16-weeks whilst on-cycle, proceeded by drug free intervals (off-cycle) lasting months, sometimes years (Kanayama et al., 2008). Off-cycle periods aim to re-establish endogenous testosterone production (Kanayama et al., 2009a), which is often suppressed due to exogenous testosterone administration. Abstaining from AAS has been noted to produce withdrawal-like symptoms (Kanayama et al., 2010), likened to

those experienced when abstinence from psychoactive drugs (e.g., opioids; Brower, 2002, 2009). Research indicates an increasing trend of administering AAS in a continuous manner to avoid symptoms of androgen withdrawal (Bonnecaze et al., 2020; Christou et al., 2017; Cohen et al., 2007) through ‘blast’ and ‘cruise’ protocols (Sagoe et al., 2015). Sustained use of AAS is believed to increase the risk of developing long-term irreversible harms (Baggish et al., 2017; Pope et al., 2014) and increase the risk of developing AAS dependence (Kanayama et al., 2015).

A growing body of literature over the last 30 years has demonstrated the presence of AAS dependence within the AAS using community (Brower, 1989, 1992; Hauger et al., 2019, 2020, 2021; Ip et al., 2012; Kanayama et al., 2009a, 2009b, 2015; Scarth et al., 2022). Based on current approaches to assessment, AAS dependence is believed to affect around 30% of individuals who use AAS compounds (Pope et al., 2014), and is defined as sustained use of androgens over prolonged periods of time, despite experiencing undesired effects including symptoms of withdrawal and psychological impairment (Kanayama et al., 2009a, 2009b, 2010).

A construct thought to manifest from drug dependence is drug craving (Drummond, 2001). Craving is characterised by a want, urge or compulsion to engage in satiating drug behaviour (Kozlowski and Wilkinson, 1987) in periods of drug abstinence (Donny et al., 2008; Drummond et al., 1990; Franken, 2003; Pickens et al., 2011). Craving is believed to manifest as intrusive and dominating sensations causing an individual substantial distress and disruption within day-to-day life (Tiffany & Wray, 2012). Existing research has indicated craving is present within various substance use disorders, including alcohol, tobacco, opioid, cocaine, cannabis, and other psychoactive substance dependence (Kakko et al., 2019; Shiffman et al., 1995; Serre et al., 2018). Moreover, drug craving has been demonstrated to

be higher within drug dependent individuals (Auriacombe et al., 2018). As such, it is reasonable to expect craving to manifest in individuals who experience AAS dependence.

A recent systemic review investigated a series of studies indicating craving is able to predict smoking relapse in nicotine dependent individuals (Serre et al., 2015). With craving being a state-like construct it (Flannery et al., 2019), has been explored using temporal methods of data collection, such as ecological momentary assessment (EMA; see Shiffman et al., 2008). Utilising this method of data collection, the extant literature has indicated temporal self-reported responses of opioid and cocaine craving in dependent individuals is elevated when approaching periods of drug use (Preston et al., 2018). Therefore, within the context of AAS use, individuals may present fluctuating levels of AAS craving across different phases of AAS administration, with highest scores being reported when approaching a new ‘on-cycle’ period. Therefore, it is not only important to examine how AAS craving manifests, but to investigate if this manifestation is affected by recent use of or abstention from AAS. Recent cross-sectional research has demonstrated a strong association between AAS dependence and craving (see Zoob Carter & Boardley, In Review). However, thus far there has been no research examining the temporal relationships between AAS dependence and craving. From the extant literature it would be plausible that craving would present with an increasing trend¹⁴ and level¹⁵ throughout AAS-free periods.

As well as furthering our understanding on AAS craving, it is also important to examine possible correlates of it. A concept previously associated with substance dependence and craving is self-regulatory efficacy (SRE; Bandura, 1999; Chavarria et al., 2012; Niaura et al., 1988; Stuart et al., 1994). SRE represents an individual’s belief in his or her ability to

¹⁴ The term ‘trend’ within this study implicates the direction exhibited by the data from the study (see Gast, 2005).

¹⁵ The term ‘level’ within this study represents the magnitude of the data (see Gast, 2005).

resist internal and external pressures to partake in harmful and detrimental behaviours (Bandura et al., 2001). Research within opioid and nicotine dependent individuals has indicated a reciprocal association between craving and SRE (Shadel & Cervone, 2006; Yuan et al., 2018), more specifically low levels of SRE have been associated with increased craving (Sayette, 2004). This relationship has been further demonstrated within models of drug craving whereby high levels of craving are associated with low SRE in periods of substance abstinence (Marlatt & Gordon, 1985). In the context of AAS use, it is probable that elevated levels of AAS craving are associated with impaired SRE. From the findings within the extant literature, one would expect SRE to display a lower trend and level during 'off-cycle' periods. Presently, there is an absence of literature exploring the interaction between AAS craving and SRE.

Another variable potentially linked with harmful drug use is anticipated guilt. Anticipated guilt represents an unpleasant emotional state whereby an individual experiences tension and regret from the thought of partaking in harmful behaviours (Bandura, 1991). West and Shiffman (2016) considered the self-regulatory role of guilt with respect to nicotine dependence and craving, suggesting an individual with nicotine dependence will have an increased decreased desire to smoke, despite incurring symptoms of craving when experiencing impaired anticipated guilt. Recent research has provided empirical support for this by identifying an inverse predictive effect of anticipated guilt on IPED use including AAS (Boardley et al., 2017). However, there remains an absence of literature exploring how AAS craving interacts with anticipated guilt. From the extant literature, one would expect a lower trend and level of anticipated guilt across 'off-cycle' periods.

Psychological constructs known to influence the experience of drug craving are alterations in positive and negative mood (i.e., affect) systems (Tiffany, 2010). Extant

literature indicates that negative affect (e.g., stress) can induce sensations of drug seeking behaviours to alleviate negative emotions (Tiffany, 1999). Whereas positive effect (e.g., excitement) is associated with positively reinforcing emotions associated with the positive sensations of administering a drug of abuse (Chiang et al., 2023; Tiffany, 1999). Recent studies in the literature have demonstrated positive affect has an inverse relationship with craving response whilst negative affect positively predicts craving responses amongst individuals with opioid, alcohol, nicotine and sedative abuse (Huhn et al., 2016; Lydon-Staley et al., 2017; Schlauch et al., 2013). It is possible that affect plays a similar role in AAS craving, whereby individuals experiencing elevated levels of negative affect self-reported increased AAS craving, during an ‘off-cycle’ period. Whereas, individuals self-reporting increased positive affect associated with their use of AAS will present with lower levels of AAS craving.

Overall, the overarching aim of this study was to investigate trends and levels of craving and associated psychosocial variables, and mood within and between different phases of AAS administration. Specifically, we investigated the direction (e.g., trend) and magnitude (e.g., level) of craving, SRE, anticipated guilt and affect at the end of an ‘on-cycle’/‘blast’ period (Phase A), middle of an ‘off-cycle’/‘cruise’ period (Phase B), and end of an ‘off-cycle’/‘cruise’ period (Phase C), and ii) whether trend and level of craving, SRE, anticipated guilt and affect differ between; phases A, B, and C. Researchers within the craving literature have indicated the importance of investigating craving events and their antecedents during drug use and non-using phases (Paty et al., 1992). Therefore, with the information presented previously indicating experience of drug seeking and associated behaviours during drug-free periods and increasing when approaching drug use (Donnay et al., 2008; Kanayama et al.,

2010; Preston et al., 2018), it was decided to focus phases of data collection at the end of an 'on-cycle' phase, middle of an 'off-cycle', and the end of an 'off-cycle'.

Based on the reviewed literature, we hypothesised within-condition analysis would indicate: *AAS craving would present with a low level with no change in trend in Phase A, a moderate level with an increasing trend at Phase B, and a high level with no change in trend at Phase C (H1). Anticipated guilt would display a high level with no change in trend at Phase A, a moderate level with a decreasing trend at Phase B, and a low level with no change in trend at Phase C (H2). SRE would present a high level with no change in trend at Phase A, a moderate level with a decreasing trend at Phase B, and a low level with no change in trend at Phase C (H3). Affect would display a high level with no change in trend at Phase A, a moderate level with a decreasing trend at Phase B, and a low level with no change in trend at Phase C (H4). It was further hypothesised that between-condition analysis would indicate level of craving would be higher in Phase B than in Phase A, and would be higher in Phase C than in Phase B (H5), levels of anticipated guilt, SRE, and affect would be lower in Phase B than in Phase A, and would be lower in Phase C than in Phase B (H6, H7, and H8 respectively).*

4.2 Methods

Participants ($n = 6$) were strength athletes originating from five countries ($n_{USA} = 2$; $n_{England} = 2$; $n_{Cyprus} = 1$; $n_{Germany} = 1$). They reported a mean age of 33.2 years, were single (50.0%) or married (50.0%), largely heterosexual (83.3 %), and predominantly employed full-time (66.7%). At the time of study participants self-reported their administration as cruising (66.7%) or blasting (33.3%), and administered dosages ranging from less than 300mg per week to 2,000mg per week during the study (see Appendix C).

4.2.1 Measures

AAS-Craving. For daily completions, AAS Craving was assessed using a single item derived from the extant literature and re-worded to make it more applicable to AAS use (“Presently, I feel a strong desire to use steroids”; see American Psychiatric Association, 2013). Participants were asked to respond using a 7-point Likert scale anchored at 1 (*strongly disagree*) to 7 (*strongly agree*). Means were computed across three daily responses, with higher scores indicated higher levels of craving.

Doping SRE. Daily doping SRE was assessed with a single item measure adapted from Boardley and colleagues’ work (Boardley et al., 2018). As no current single item exists to identify doping SRE the item was worded to be more specific to AAS (“How confident are you in your ability to resist personal and external pressures to increase your dose of steroids”). Participants were asked to rate how confident they were using a 5-point Likert scale anchored at 1 (*no confidence*) to 5 (*complete confidence*). Means were taken from the three daily responses, with higher scores indicating the participant displayed elevated SRE.

Anticipated Guilt. A single-item measure of anticipated guilt was developed based on the definition within the existing literature (see Marschall et al., 1994). This was adapted to become more specific to AAS use (“Presently, I feel remorse and/or regret about my use of steroids”) to assess daily anticipated guilt. Participants were asked to respond with their level of agreement using a 7-point Likert scale anchored at 1 (*strongly disagree*) to 7 (*strongly agree*). Means were computed across the three daily responses, with higher scores indicating higher levels of guilt.

Affect. To assess daily affect, we asked participants to select one item that best captured their current mood from a selection of 16 items. Items included eight positive (e.g.,

happy, affectionate, enthusiastic, calm) and eight negative (e.g., ashamed, sad, irritable, angry) mood states (see Russell, 1980; Huhn et al., 2016). Items were ranked in alignment with previous literature and were scored accordingly (e.g., Happy = 1, Joyful = 2, Loving = 3, Sad = 14, Ashamed = 15, and Guilty = 16; see Huhn et al., 2016). Means were computed across the three daily responses with higher scores indicating more negative affect.

4.2.2 Procedures

Recruitment. Participants were recruited through advertisements on bodybuilding and strength training forums, groups on social media platforms (i.e., Facebook, Twitter, and Reddit) where the use of AAS is regularly discussed, through existing contacts and via gatekeepers. Potential participants were provided with a brief description of the study and a hyperlink to access a pre-screening survey. Once accessed, participants were presented with an information sheet, General Data Protection Regulation information and a consent form. Informed consent was obtained from all participants at the beginning of each 12-day phase, via an item worded ‘Do you consent in taking part in this study?’. Confidentiality was assured to participants of their responses as no personal details (e.g., names, addresses, phone numbers, etc) were gathered from participants. Email addresses were provided to enable follow-up contact to arrange data collection at later phases within the study, and to provide participants with Amazon vouchers for compensation.

Pre-Screening Surveys. The purpose of the pre-screening survey was to establish each participant’s current AAS phase (i.e., on-/off-cycle, or blasting/cruising), how long they had been running this phase, the duration of the phase, and the timing and duration of subsequent phases. This was to establish a timeline to set up the three 12-day data collection phases for each participant. Pre-screening also assessed what times of day participants preferred to be notified for completion of daily surveys, with one time selected in the

morning, one in the afternoon, and another in the evening. This was to minimise disruption of each participant's lifestyle and reduce attrition due to non-adherence with the survey protocol. Participants were provided information on the smartphone application being used for data collection, and assured that use of this application would not require information that would be traceable to them, to aid in maintaining their anonymity.

Data collection. This study utilised an intensive longitudinal design (see Bolger et al., 2003). Data collection occurred across three independent 12-day phases spanning a 5-month period (July 2022 – December 2022); each phase was timed to coincide with a specific period within each participant's AAS protocol (i.e., the end of an on-cycle/blast period; the middle of an off-cycle/cruise period; the end of an off-cycle/cruise period). EMA was utilised as the method of data collection within this study, whereby data were collated within the participant's own environment and within their day-to-day lifestyles, therefore increasing the ecological (see Shiffman et al., 2008). Data were collected using a bespoke smartphone application from SEMA3 (Koval et al., 2019) which sent notifications to participants to complete short (two to three minute long) surveys three times per day across each 12-day phase. The timing of the daily notifications and wording of the aforementioned items was to ensure data was collated from that moment in time in order to limit response bias (Shiffman et al., 2008).

Inclusion criteria required participants to be over the age of 18, had taken AAS in the last 12-months, and be currently using AAS (i.e., on/off-cycle, or blasting/cruising). Participants were excluded from analysis if they failed to complete two or more 12-day phases. Six participants (P1 through P6) completed all three of the 12-day phases and were therefore utilised in data analysis. Upon completion of the survey participants who has a completion rate of more than 80% were entered into a prize draw to win a £50 Amazon

voucher. Full ethical approval was obtained from the University of Birmingham Ethics Committee (ERN_19-1955).

4.2.3 Data Analysis

Data were analysed through graphical analysis (see Barker et al., 2011; Lane & Gast, 2014), an appropriate technique for single-case research (Kennedy, 2005). We considered this a single-case naturalistic design as the differing phases (e.g., on/off cycle) of AAS use represent naturally occurring interventions with the potential to influence the study variables. Furthermore, data collection occurred within the natural environment of participants and the researcher acted as an observer (see Dahl, 2017; Eby, 2011; Smith, 1981).

Analysis for the within-condition was established by analysing each individual condition within the study (e.g., end of an on-cycle phase; see Gast & Spriggs, 2014). Following the guidance of Lane and Gast (2013) level and trend were calculated to interpret within-condition analysis. Analysis also focused on the between-condition, whereby analysis focused across adjacent conditions (e.g., from being on-cycle to being off-cycle; see Gast & Spriggs, 2014). Following the steps provided by Lane and Gast (2013) change in trend and level (i.e., relative, absolute, mean, and median level change) were also calculated. Rationale for their inclusion was attributed to work by Lane and Gast (2013), who indicated the importance for reporting each of the respective level change scores together despite the preference for median level change scores in visual analysis (see Lane & Gast, 2013).

Graphical analysis was used to compare changes in trend and level across the three different conditions (i.e., end of a blast [Phase A]; middle of a cruise [Phase B]; end of a cruise [Phase C]) within the study, with each participant acting as their own control (Gast & Hammond, 2012). During each phase there were 36 data collection points for each participant,

with a notification sent via the app for each collection point (e.g., three notifications per day over 12-days). Daily responses were aggregated to provide daily scores for each variable within each respective phase. Descriptive statistics, tables and graphical representation of the data and analyses were produced using IBM SPSS Statistics version 29. Descriptive statistics included means, standard deviations, and median values. To determine the degree of overlap between conditions the percentage of non-overlapping data (PND; see Scruggs et al., 1987)¹⁶ was calculated. Researchers recommend the PND over other overlap metrics as it is less sensitive to outliers (Ledford et al., 2018).

4.3 Results

4.3.1 Participant 1

Within-Condition Analysis. Evaluation of AAS craving indicated data presented a high level ($M = 5.7$, $SD = 0.5$) with a decreasing trend in Phase A, high level ($M = 5.0$, $SD = 0.0$) with no change in trend in Phase B, and a high level ($M = 5.6$, $SD = 0.7$) with an increasing trend in Phase C (Table 4.1). Observations of anticipated guilt indicated data presented a low level ($M = 3.1$, $SD = 1.2$) with an increasing trend in Phase A, a low level ($M = 1.8$, $SD = 0.4$) with a decreasing trend in Phase B, and a low level ($M = 3.4$, $SD = 1.3$) with a decreasing trend in Phase C (Table 4.2). Data for SRE were observed as low level ($M = 3.2$, $SD = 0.4$) with no change in trend in Phase A, low level ($M = 2.9$, $SD = 0.4$) with no change in trend in Phase B, and low level ($M = 3.4$, $SD = 1.3$) with a decreasing trend in Phase C (Table 4.3). Observations of affect indicated a moderate level ($M = 3.1$, $SD = 4.8$) with an increasing trend in Phase A, a moderate level ($M = 2.6$, $SD = 3.9$) with an increasing trend in

¹⁶ These calculations are utilised in SCED to measure effect size (see Tarlow & Penland, 2016) with larger PND scores indicating a larger effect size.

Phase B, and a moderate level ($M = 3.4$, $SD = 1.3$) with a decreasing trend in Phase C (Table 4.4).

Between-Condition Analysis. Level change scores for AAS craving indicated a lower level of craving in Phase B than in A, and a higher level of craving in Phase C than in Phase B. PND scores indicated there was 0.0% non-overlap of AAS craving observed between Phase A and B, and a 52.4% non-overlap between Phase B and C (Table 4.5). Level change scores for anticipated guilt indicated a lower level of guilt in Phase B than in Phase A, and higher level of guilt in Phase C than in Phase B. PND indicated there was 11.1% non-overlap between Phases A and B, and a 0.0% non-overlap and a 97.2% overlap between Phase B and C (Table 4.6). SRE indicated a lower level of SRE in Phase B than in Phase A, and lower levels of SRE in Phase C than in Phase B. PND scores indicated a 11.1% non-overlap between Phase A and B. PND scores indicated a 0.0% non-overlap between Phase B and C (Table 4.7). Affect indicated a lower level of affect in Phase C than in Phase B, and a higher level of affect in Phase C than in Phase B. PND scores between indicated a 0.0% non-overlap between Phase A and B. PND scores indicated a 0.0% non-overlap between Phase B and C (Table 4.8 and Figure 4.1).

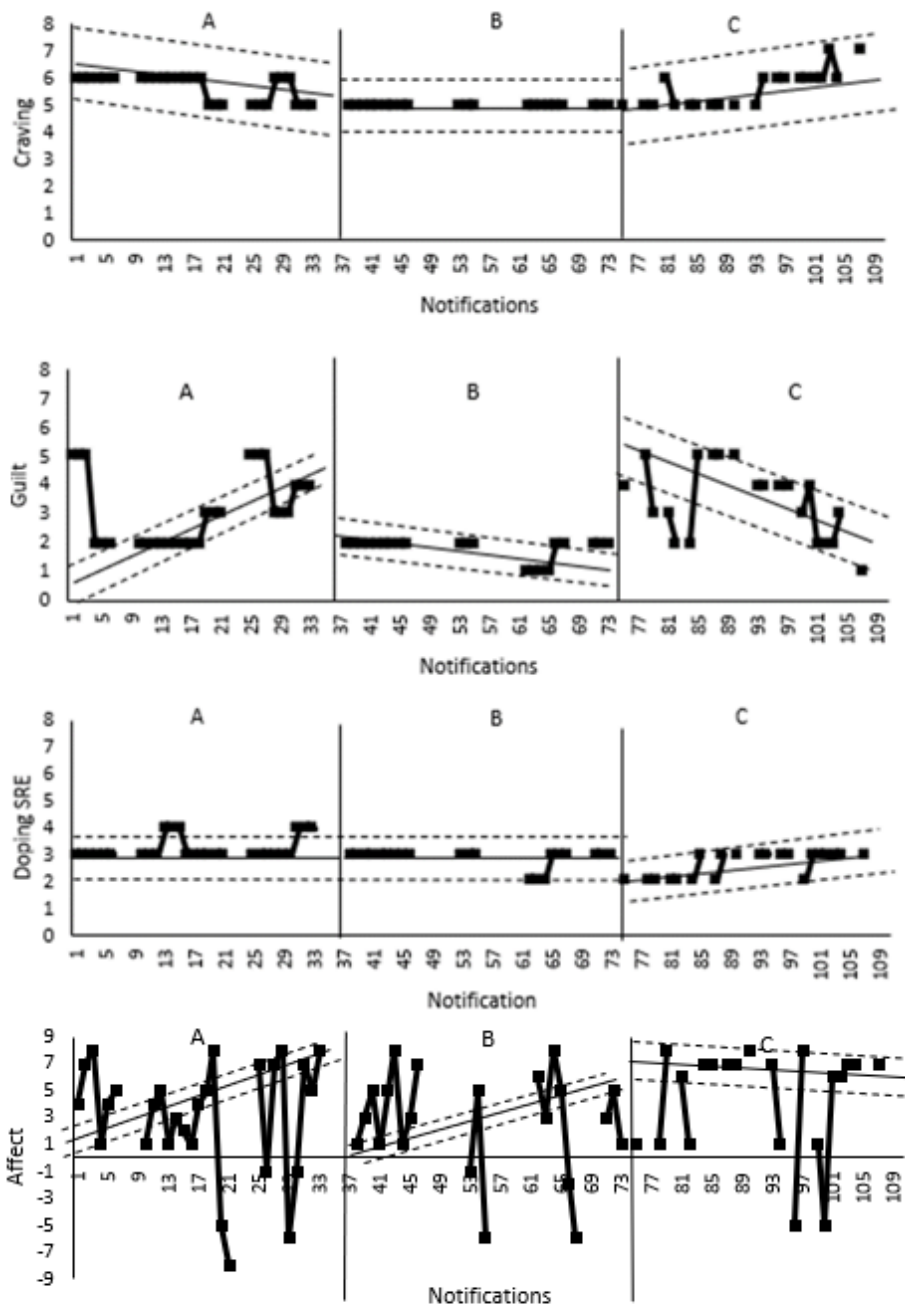


Figure 4.1. Participant 1 daily notification data taken at the end of a blast (Phase A), during the middle of a cruise (Phase B), and at the end of a cruise period (Phase C) for single items measuring craving, anticipated guilt, doping SRE, and Affect. Within-condition analysis, estimation of trend indicated by solid line and trend stability ($\pm 25\%$ of median value) indicated by dotted lines.

4.3.2 Participant 2

Within-Condition Analysis. Evaluation of AAS craving within Phase A indicated a high level ($M = 6.2$, $SD = 0.6$) with an increasing trend, Phase B indicated a high level ($M = 6.0$, $SD = 0.8$) with an increasing trend, and Phase C indicated a high level ($M = 6.1$, $SD = 0.5$) with an increasing trend (Table 4.1). Observations of anticipated guilt indicated a low level ($M = 1.4$, $SD = 0.6$) with no change in trend in Phase A, a low level ($M = 1.2$, $SD = 0.6$) with a decreasing trend in Phase B, and a low level ($M = 1.4$, $SD = 0.7$) with no change in trend in Phase C (Table 4.2). Data for SRE were overserved with a high level ($M = 5.4$, $SD = 0.7$) with a decreasing trend in Phase A, a high level ($M = 5.1$, $SD = 0.7$) with an increasing trend in Phase B, and a low level ($M = 1.4$, $SD = 0.7$) with no change in trend in Phase C (Table 4.3). Observations of affect indicated data had a high level ($M = 7.1$, $SD = 0.8$) with a decreasing trend in Phase A, a high level ($M = 5.6$, $SD = 1.7$) with an increasing trend in Phase B, and a high level ($M = 5.0$, $SD = 2.6$) with an in increasing trend in Phase C (Table 4.4).

Between-Condition Analysis. Level change scores for AAS craving indicated a lower level of craving in Phase B than in A, and lower levels of craving in Phase C than in Phase B. PND scores indicated there was a 0.0% non-overlap across Phase A to B, and 0.0% non-overlap across Phase B and C (Table 4.5). Level change scores for anticipated guilt indicated a lower level of guilt in Phase B than in A, and a higher level of guilt in Phase C than in B. PND scores indicated that there was a 0.0% non-overlap across Phase A to B, and a 0.0% non-overlap across Phase B to C (Table 4.6). Level change scores for SRE indicated a lower level in Phase B than in A, and a higher level in Phase C than in B. PND indicated a 13.9% non-overlap across Phase A to B, and a 0.0% non-overlap across Phase B to C (Table 4.7). Level change scores for affect indicated a lower level in B than in A, and a higher level in C

than in B. PND and a 27.8% non-overlap across phase A to B, and a 2.8% non-overlap across Phase B to C (Table 4.8 and Figure 4.2).

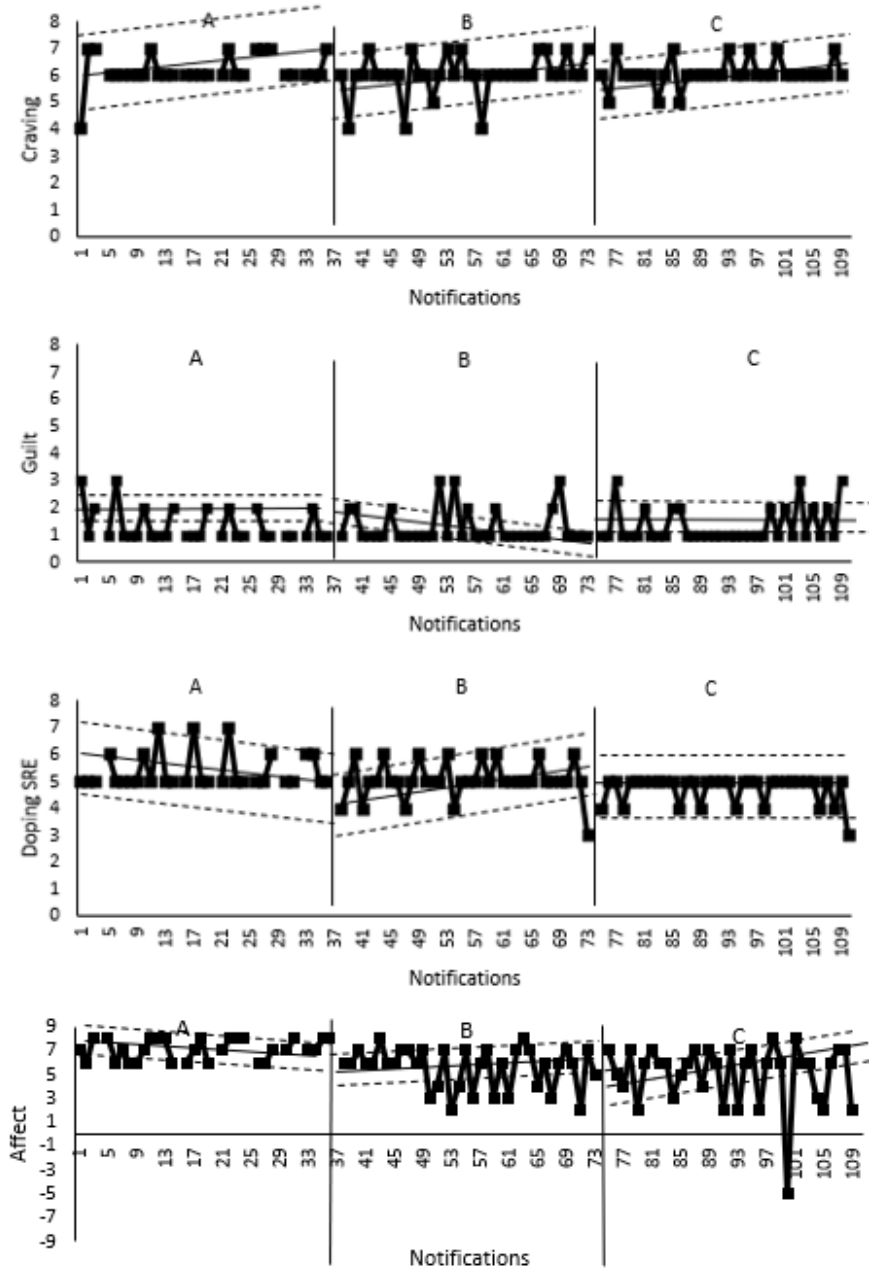


Figure 4.2. Participant 2 daily notification data taken at the end of a blast (Phase A), during the middle of a cruise (Phase B), and at the end of a cruise period (Phase C) for single items measuring craving, doping MD, doping SRE, and Affect. Within-condition analysis, estimation of trend indicated by solid line and trend stability ($\pm 25\%$ of median value) indicated by dotted lines.

4.3.3 Participant 3

Within-Condition Analysis. Evaluation of AAS craving indicated a high level ($M = 6.0$, $SD = 0.0$) with no change in trend in Phase A, a high level ($M = 5.7$, $SD = 0.5$) with a decreasing trend in Phase B, and a high level ($M = 6.3$, $SD = 0.6$) with no change in trend in Phase C (Table 4.1). Observations of anticipated guilt data identified a low level ($M = 2.0$, $SD = 0.0$) with no change in trend in Phase A, a low level ($M = 1.9$, $SD = 0.5$) with no change in trend in Phase B, and a low level ($M = 1.8$, $SD = 0.4$) with decreasing trend in Phase C (Table 4.2). Data for SRE were observed to have a low level ($M = 2.7$, $SD = 0.4$) with no change in trend in Phase A, low level ($M = 1.8$, $SD = 0.4$) with no change in trend in Phase B, and a low level ($M = 1.8$, $SD = 0.4$) with a decreasing trend in Phase C (Table 4.3). Observations of affect identified data having a high level ($M = 5.8$, $SD = 2.5$) with a decreasing trend in Phase A, a moderate level ($M = 0.3$, $SD = 4.8$) with a decreasing trend in Phase B, and a moderate level ($M = 2.75$, $SD = 4.8$) with no change in trend in Phase C (Table 4.4).

Between-Condition Analysis. Level change scores for AAS craving demonstrated a higher level of craving in B than in A, and a higher level of craving in C than in B. PND indicated there was a 22.2% non-overlap across Phase A and B, and a 0.0% non-overlap across Phase B and C (Table 4.5). Level change scores for anticipated guilt demonstrated a lower level of guilt in Phase B than in A, a lower level of guilt in Phase C than in B. PND indicated a 13.9% non-overlap between Phase A and B, and a 0.0% non-overlap between Phase B and C (Table 4.6). Level scores for SRE indicated a lower level of SRE in Phase B than in A, a lower level of SRE in Phase C than in B. PND indicated a 16.7% non-overlap between Phase A and B, and a 0.0% non-overlap between Phase B and C (Table 4.7). Level scores for affect demonstrated a higher level of affect in Phase B than in A, and a lower level of affect in Phase

C than in B C. PND indicated a 16.7% non-overlap between Phase A and B, and a 5.6 non-overlap between Phase B and C (Table 4.8 and Figure 4.3).

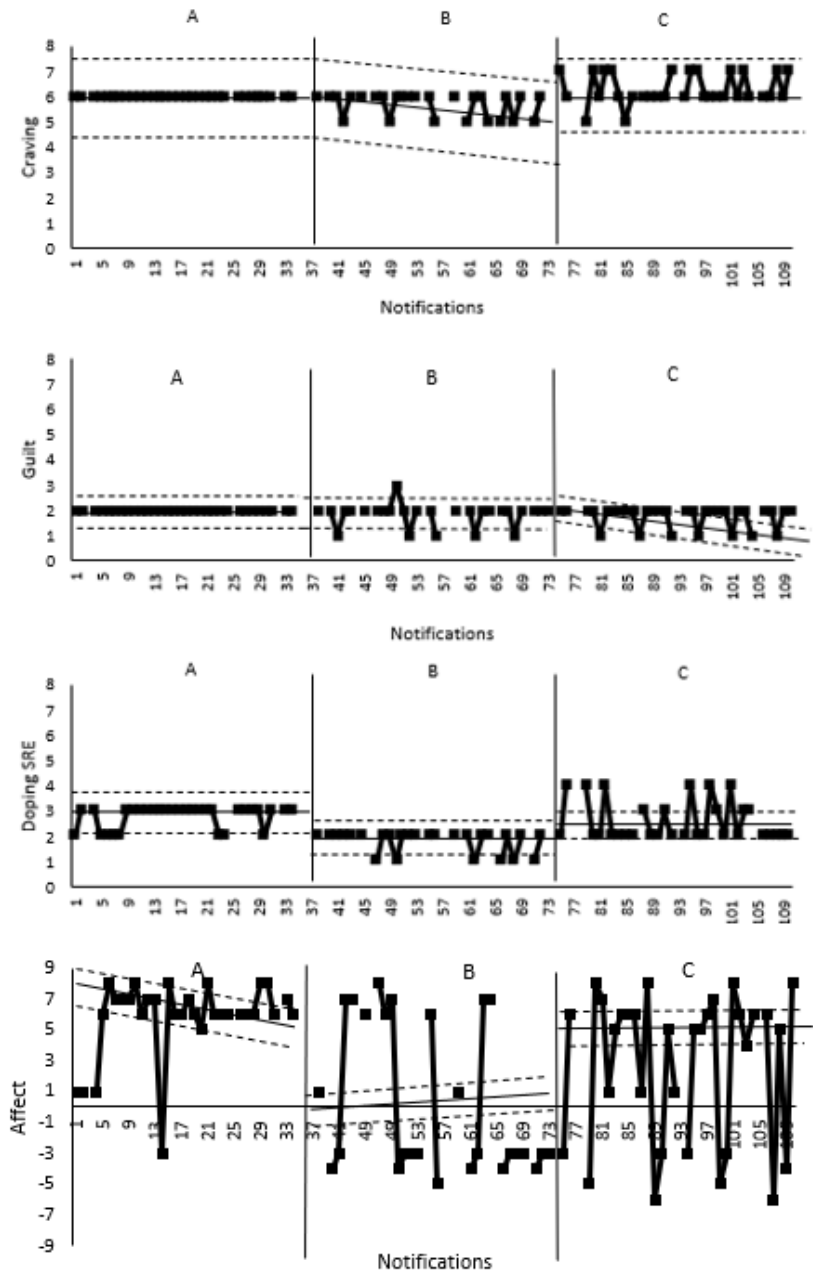


Figure 4.3. Participant 3 daily notification data taken at the end of a blast (Phase A), during the middle of a cruise (Phase B), and at the end of a cruise period (Phase C) for single items measuring craving, doping MD, doping SRE, and Affect. Within-condition analysis, estimation of trend indicated by solid line and trend stability ($\pm 25\%$ of median value) indicated by dotted lines.

4.3.4 Participant 4

Within-Condition Analysis. Evaluation of AAS craving indicated a low level ($M = 2.6$, $SD = 1.3$) with a decreasing trend in Phase A, a low level ($M = 1.7$, $SD = 0.9$) with an increasing trend in Phase B, and a low level ($M = 2.2$, $SD = 1.3$) with an increasing trend in Phase C (Table 4.1). Observations of anticipated guilt identified a low level ($M = 1.0$, $SD = 0.0$) with no change in trend in Phase A, a low level ($M = 1.0$, $SD = 0.0$) with no change in trend in Phase B, and a low level ($M = 1.0$, $SD = 0.0$) with no change in trend in Phase C (Table 4.2). Data for SRE were observed to have a moderate level ($M = 4.6$, $SD = 0.5$) with an increasing trend in Phase A, a moderate level ($M = 4.1$, $SD = 0.5$) with no change in trend in Phase B, and a low level ($M = 1.0$, $SD = 0.0$) with no change in trend in Phase C (Table 4.3). Observations of affect data indicated data had a high level ($M = 5.5$, $SD = 3.6$) with an increasing trend in Phase A, a high level ($M = 5.6$, $SD = 4.3$) with no change in trend in Phase B, and a high level ($M = 6.1$, $SD = 3.7$) with a decreasing trend in Phase C (Table 4.4).

Between-Condition Analysis. Level change scores for AAS craving indicated a lower level of craving in Phase B than in A, a lower level of craving in Phase C than in B. PND there was a 0.0% non-overlap across Phase A to B, and a 0.0% non-overlap across Phase B to C (Table 4.5). Level change scores for anticipated guilt indicated a no level change across each Phase. PND indicated that there was a 0.0% non-overlap between all Phases (Table 4.6). Level change scores for SRE indicated a lower level in Phase B than in A, a lower level of SRE in Phase C than in B. PND indicated that there was a 5.6% non-overlap between Phase A and B, and a 0.0% non-overlap between Phase B and C (Table 4.7). Level change scores for affect indicated a lower level in Phase B than in A, and a higher level in Phase C than in B. PND indicated there was a 5.6% non-overlap between Phase A and B, and a 0.0% non-overlap between Phase B and C (Table 4.8 and Figure 4.4).

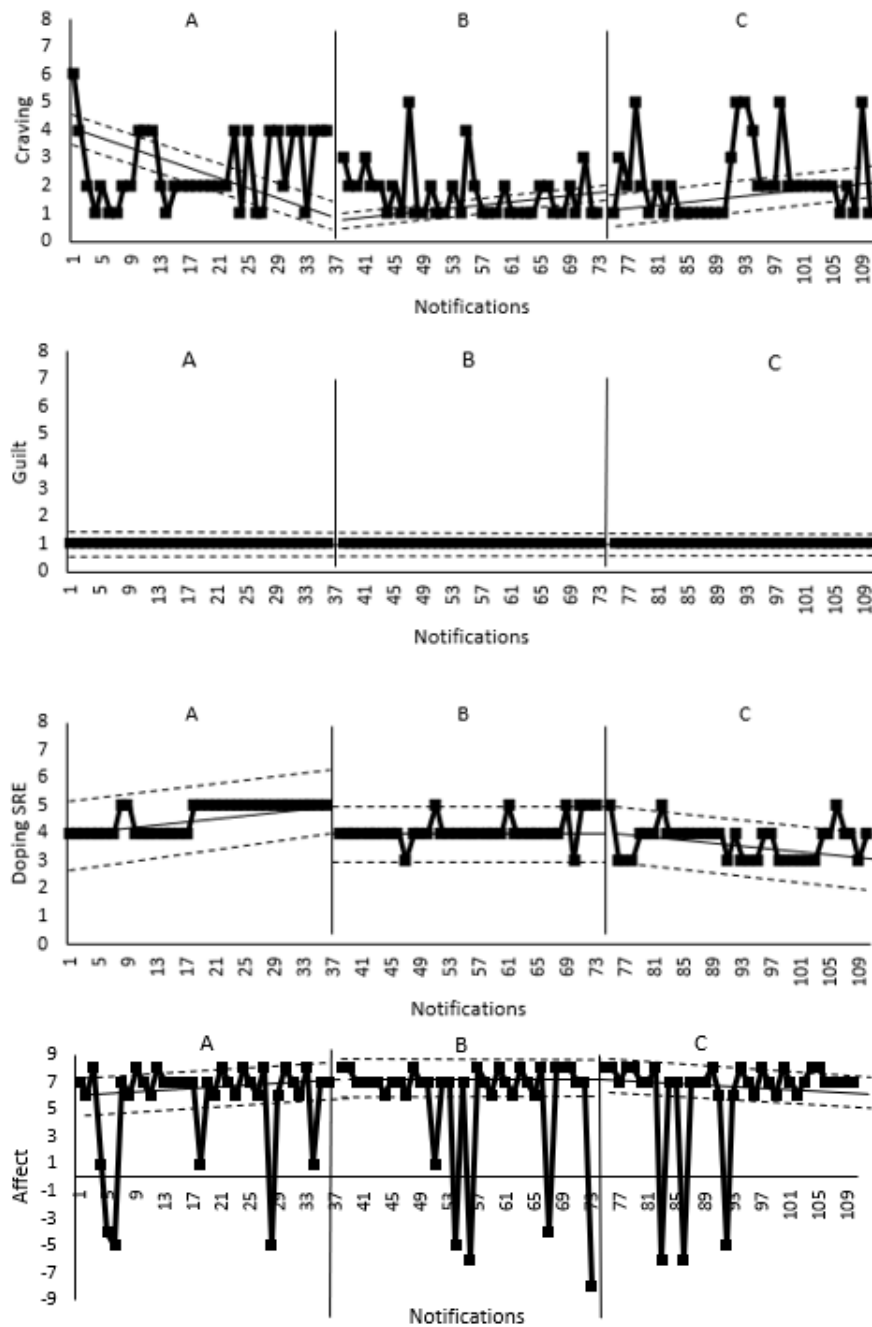


Figure 4.4. Participant 4 daily notification data taken at the end of a blast (Phase A), during the middle of a cruise (Phase B), and at the end of a cruise period (Phase C) for single items measuring craving, doping MD, doping SRE, and Affect. Within-condition analysis, estimation of trend indicated by solid line and trend stability ($\pm 25\%$ of median value) indicated by dotted lines.

4.3.5 Participant 5

Within-Condition Analysis. Evaluation of AAS craving identified a high level ($M = 6.0$, $SD = 0.0$) with no change in trend in Phase A, a high level ($M = 6.0$, $SD = 0.0$) with no change in trend in Phase B, and a high level ($M = 6.0$, $SD = 0.0$) with no change in trend in Phase C (Table 4.1). Observations of anticipated guilt identified a low level ($M = 1.0$, $SD = 0.0$) with no change in trend in Phase A, a low level ($M = 1.0$, $SD = 0.0$) with no change in trend in Phase B, and a low level ($M = 1.0$, $SD = 0.0$) with no change in trend in Phase C (Table 4.2). SRE data were observed to have a moderate level ($M = 4.0$, $SD = 0.0$) with no change in trend in Phase A, a moderate level ($M = 4.4$, $SD = 0.5$) with a decreasing trend in Phase B, and a low level ($M = 1.0$, $SD = 0.0$) with no change in trend in Phase C (Table 4.3). Observations of affect identified data having a high level ($M = 3.7$, $SD = 2.6$) with a decreasing trend in Phase A, a high level ($M = 4.9$, $SD = 3.1$) with no change in trend in Phase B, and a high level ($M = 3.9$, $SD = 2.2$) with a decreasing trend in Phase C (Table 4.4).

Between-Condition Analysis. Level change scores for AAS craving indicated a positive change across Phase A and B, and no change was seen between Phase B and C. PND indicated that there was a 0.0% non-overlap between all three phases (Table 4.5). Level change scores for anticipated guilt indicated a higher level in Phase B than in A, and no change between Phase B and C. PND indicated a 0.0% non-overlap between each of the phases (Table 4.6). Level change scores for SRE indicated a higher level in Phase B than in A, and a lower level in Phase C than in B. PND indicated that there was a 0.0% non-overlap between Phase A and B, and a 0.0% non-overlap between Phase B and C (Table 4.7). Level change scores for affect indicated a lower level of affect in Phase B than in A, and a higher level in Phase C than in B. PND indicated that there was a 5.6% non-overlap between Phase A and B, and at 0.0% non-overlap between Phase B and C (Table 4.8 and Figure 4.5).

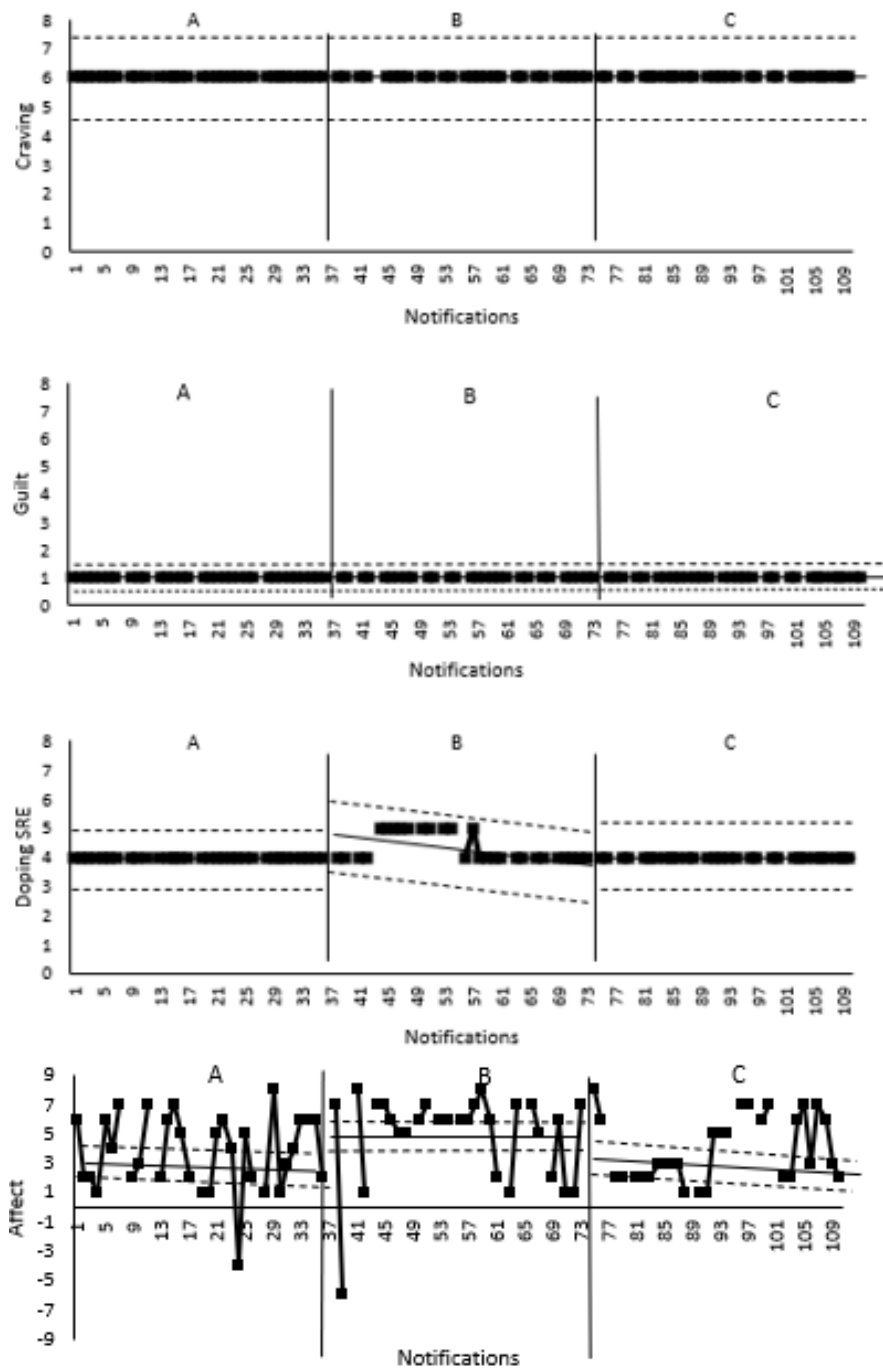


Figure 4.5. Participant 5 daily notification data taken at the end of a blast (Phase A), during the middle of a cruise (Phase B), and at the end of a cruise period (Phase C) for single items measuring craving, doping MD, doping SRE, and Affect. Within-condition analysis, estimation of trend indicated by solid line and trend stability ($\pm 25\%$ of median value) indicated by dotted lines.

4.3.6 Participant 6

Within-Condition Analysis. Evaluation of AAS craving identified a low level ($M = 1.1$, $SD = 0.3$) with no change in trend in Phase A, a low level with no change in trend in Phase B ($M = 1.2$, $SD = 0.6$), and a moderate level ($M = 3.0$, $SD = 0.0$) with no change in trend in Phase C (Table 4.1). Observations of anticipated guilt identified a low level ($M = 1.9$, $SD = 0.2$) with no change in trend in Phase A, a low level ($M = 1.9$, $SD = 0.2$) with no change in trend in Phase B, and a low level ($M = 2.0$, $SD = 0.0$) with no change in trend in Phase C (Table 4.2). SRE data were observed to have a moderate level ($M = 4.9$, $SD = 0.3$) with no change in trend in Phase A, a moderate level ($M = 4.6$, $SD = 0.7$) with no change in trend for Phase B, and a low level ($M = 2.0$, $SD = 0.0$) with no change in trend in Phase C (Table 4.3). Observations of affect indicated data had a high level ($M = 7.7$, $SD = 1.8$) with no change in trend in Phase A, a high level ($M = 5.7$, $SD = 4.0$) with an increasing trend in Phase B, and a high level ($M = 5.8$, $SD = 4.1$) with no change in trend in Phase C (Table 4.4).

Between-Condition Analysis. Level change scores for AAS craving indicated a higher level of craving in Phase B than in A, and a higher level in Phase C than in B. PND indicated that there was a 2.8% non-overlap across Phase A and B, and a 0.0% non-overlap across Phase B to C (Table 4.5). Change scores for anticipant guilt indicated a lower level in Phase B than in A, and a higher level in Phase C than in B. PND indicated that there was a 0.0% non-overlap between Phase A to B and from 0.0% non-overlap between Phase B to C (Table 4.6). Level change scores for SRE indicated a lower level in SRE in Phase B than in A, and higher level in Phase C than B. PND indicated that there was a 27.8% non-overlap across Phase A to B, and a 0.0% non-overlap across Phase B to C (Table 4.7). Level change scores for affect indicated a lower level in Phase B than in A, and a higher level in Phase C than in

B. PND indicated that there was an 8.3% non-overlap between Phase A and B, and a 0.0% non-overlap between Phase B to C (Table 4.8 and Figure 4.6).

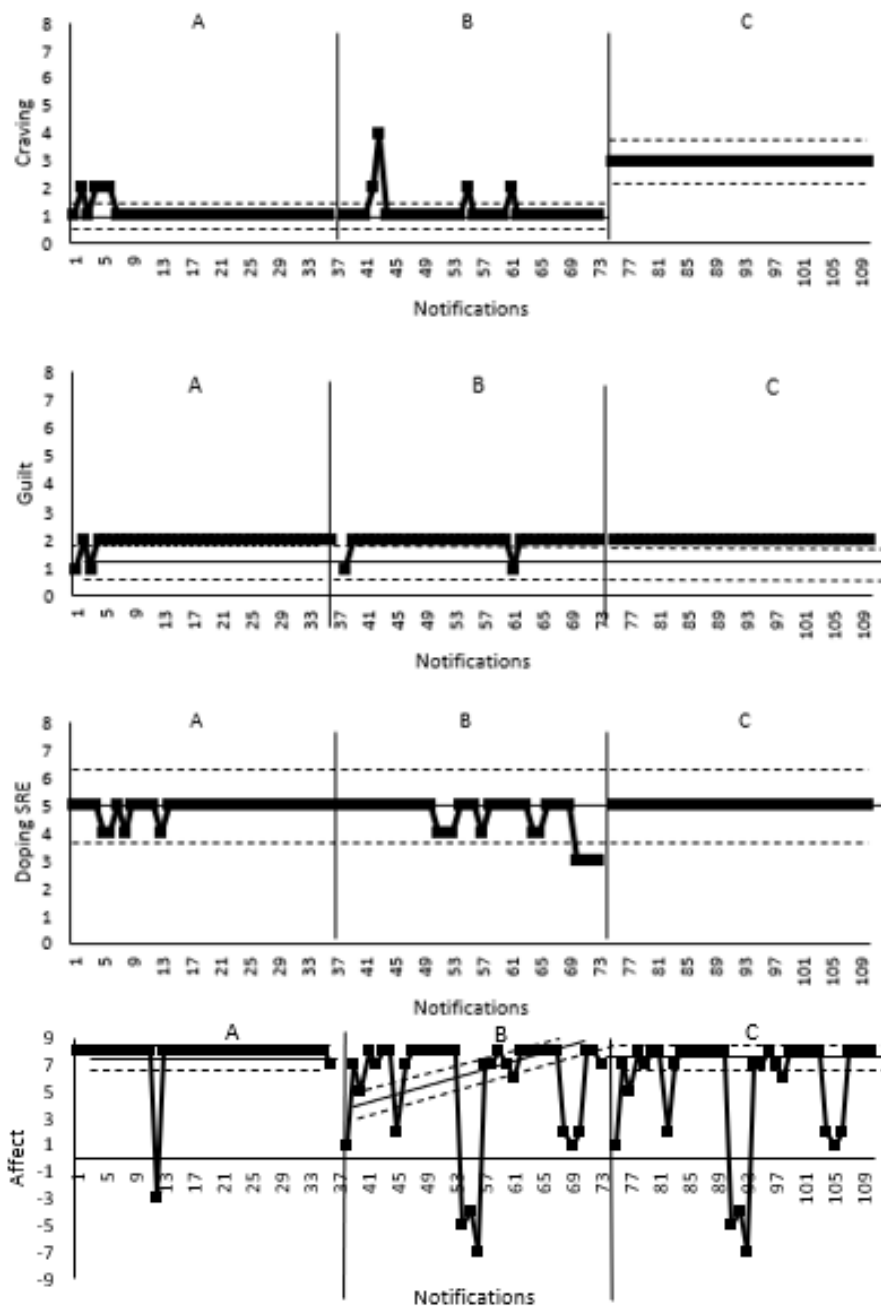


Figure 4.6. Participant 6 daily notification data taken at the end of a blast (Phase A), during the middle of a cruise (Phase B), and at the end of a cruise period (Phase C) for single items measuring craving, doping MD, doping SRE, and Affect. Within-condition analysis, estimation of trend indicated by solid line and trend stability ($\pm 25\%$ of median value) indicated by dotted lines.

Table 4.1. Within-condition analysis for AAS craving during Phase A (end of blast), B (middle of a cruise), and C (end of a cruise) identifying descriptive statistics, level and trend of responses.

Participant	Mean	SD	Median	Range	Trend Stability %	Level Change	Trend
Phase A							
1	5.67	0.48	6.00	5-6	100.00	-1.00	Decreasing
2	6.20	0.61	6.00	4-7	96.67	+3.00	Increasing
3	6.00	0.00	6.00	6-6	100.00	0.00	No Change
4	2.61	1.34	2.00	1-6	11.11	-2.00	Decreasing
5	6.00	0.00	6.00	6-6	100.00	0.00	No Change
6	1.11	0.32	1.00	1-2	88.89	0.00	No Change
Phase B							
1	5.00	0.00	5.00	5-5	100.00	0.00	No Change
2	6.03	0.77	6.00	4-7	83.33	+1.00	Increasing
3	5.69	0.47	6.00	5-6	100.00	0.00	Decreasing
4	1.69	0.95	1.00	1-5	5.56	-2.00	Increasing
5	6.00	0.00	6.00	6-6	100.00	0.00	No Change
6	1.17	0.56	1.00	1-4	88.89	0.00	No Change
Phase C							
1	5.62	0.67	6.00	5-6	100.00	+2.00	Increasing
2	6.09	0.51	6.00	5-7	88.57	0.00	Increasing
3	6.29	0.59	6.00	5-7	100.00	0.00	No Change
4	2.17	1.34	2.00	1-5	38.89	0.00	Increasing
5	6.00	0.00	6.00	6-6	100.00	0.00	No Change
6	3.00	0.00	3.00	3-3	100.00	0.00	No Change

Note: Increase in level change is represented by +, decrease in level change is represented by -.

Table 4.2. Within-condition analysis for guilt during Phase A (end of blast), B (middle of a cruise), and C (end of a cruise) identifying descriptive statistics, level and trend of responses.

Participant	Mean	SD	Median	Range	Trend Stability %	Level Change	Trend
Phase A							
1	3.11	1.22	3.00	2-5	58.33	-1.00	Increasing
2	1.37	0.61	1.00	1-2	19.44	-2.00	No Change
3	2.00	0.00	2.00	2-2	100.00	0.00	No Change
4	1.00	0.00	1.00	1-1	100.00	0.00	No Change
5	1.00	0.00	1.00	1-1	100.00	0.00	No Change
6	1.94	0.23	2.00	1-2	94.00	+1.00	No Change
Phase B							
1	1.81	0.40	2.00	1-2	44.44	0.00	Decreasing
2	1.22	0.63	1.00	1-3	52.78	0.00	Decreasing
3	1.85	0.46	2.00	1-3	16.67	0.00	No Change
4	1.00	0.00	1.00	1-1	100.00	0.00	No Change
5	1.00	0.00	1.00	1-1	100.00	0.00	No Change
6	1.94	0.23	2.00	1-2	94.00	+1.00	No Change
Phase C							
1	3.42	1.25	4.00	1-5	30.56	-3.00	Decreasing
2	1.37	0.65	1.00	1-3	0.00	+2.00	No Change
3	1.78	0.42	2.00	1-2	38.89	-2.00	Decreasing
4	1.00	0.00	1.00	1-1	100.00	-1.00	No Change
5	1.00	0.00	1.00	1-1	100.00	0.00	No Change
6	2.00	0.00	2.00	2-2	100.00	0.00	No Change

Note: Increase in level change is represented by +, decrease in level change is represented by -.

Table 4.3. Within-condition analysis for doping SRE during Phase A (end of blast), B (middle of a cruise), and C (end of a cruise) identifying descriptive statistics, level and trend of responses.

Participant	Mean	SD	Median	Range	Trend Stability %	Level Change	Trend
Phase A							
1	3.22	0.42	3.00	3-4	77.78	+1.00	No Change
2	5.37	0.67	5.00	5-7	90.00	0.00	Decreasing
3	2.74	0.44	3.00	2-3	74.19	+1.00	No Change
4	4.58	0.50	5.00	4-5	100.00	+1.00	Increasing
5	4.00	0.00	4.00	4-4	100.00	0.00	No Change
6	4.89	0.32	5.00	4-5	100.00	0.00	No Change
Phase B							
1	2.86	0.36	3.00	2-3	90.48	0.00	No Change
2	5.06	0.67	5.00	4-6	86.11	-1.00	Increasing
3	1.77	0.43	2.00	1-2	77.78	0.00	No Change
4	4.11	0.46	4.00	4-5	77.78	+1.00	No Change
5	4.36	0.49	4.00	4-5	100.00	0.00	Decreasing
6	4.61	0.69	5.00	3-5	88.89	-2.00	No Change
Phase C							
1	3.42	1.25	4.00	1-5	30.56	+1.00	Decreasing
2	1.37	0.65	1.00	1-3	0.00	-1.00	No Change
3	1.78	0.42	2.00	1-2	38.89	0.00	Decreasing
4	1.00	0.00	1.00	1-1	100.00	-2.00	No Change
5	1.00	0.00	1.00	1-1	100.00	0.00	No Change
6	2.00	0.00	2.00	2-2	100.00	0.00	No Change

Note: Positive change is represented by +, negative change is represented by -.

Table 4.4. Within-condition analysis for Affect during Phase A (end of blast), B (middle of a cruise), and C (end of a cruise) identifying descriptive statistics, level and trend of responses.

Participant	Mean	SD	Median	Range	Trend Stability %	Level Change	Trend
Phase A							
1	3.11	4.38	4.00	-5-8	22.22	+4.00	Increasing
2	7.10	0.84	7.00	6-8	100.00	+1.00	Decreasing
3	5.81	2.49	6.00	-3-8	63.89	+5.00	Decreasing
4	5.52	3.60	7.00	-5-8	83.33	0.00	Increasing
5	3.71	2.60	4.00	-4-8	22.22	-4.00	Decreasing
6	7.67	1.83	8.00	-3-8	97.22	-1.00	No Change
Phase B							
1	2.62	3.94	3.00	-6-8	11.11	0.00	Increasing
2	5.58	1.71	6.00	2-7	63.69	-1.00	Increasing
3	0.29	4.85	-3.00	-5-8	36.11	+10.00	Increasing
4	5.56	4.25	7.00	-8-8	86.11	+8.00	No Change
5	4.89	3.10	6.00	-6-8	52.82	0.00	No Change
6	5.67	4.02	8.00	-7-8	75.00	+6.00	Increasing
Phase C							
1	4.42	4.09	7.00	-5-8	38.89	+6.00	Decreasing
2	5.02	2.59	6.00	-5-8	63.89	-3.00	Increasing
3	2.75	4.81	5.00	-6-8	41.67	+1.00	No Change
4	6.11	3.66	7.00	-6-8	91.67	-1.00	Decreasing
5	3.97	2.23	3.00	1-8	41.67	-6.00	Decreasing
6	5.81	4.07	8.00	-7-8	75.00	+7.00	No Change

Note: Increase in level change is represented by +, decrease in level change is represented by -.

Table 4.5. Between-condition analysis for each participant’s response to single -item AAS craving.

Participant	Relative Level Change (A-B)	Relative Level Change (B-C)	Absolute Level Change (A-B)	Absolute Level Change (B-C)	Median Level Change (A-B)	Median Level Change (B-C)	Mean Level Change (A-B)	Mean Level Change (B-C)	PND (A-B)	PND (B-C)
1	-1.00	+1.00	-1.00	0.00	-1.00	+1.00	-1.00	+1.00	0.00	52.77
2	0.00	+1.00	-1.00	+1.00	0.00	0.00	-1.00	+1.00	0.00	0.00
3	+3.00	+1.00	0.00	+1.00	0.00	0.00	0.00	+1.00	22.22	0.00
4	-4.00	0.00	-1.00	0.00	-1.00	-1.00	-1.00	0.00	0.00	0.00
5	+5.00	0.00	+1.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
6	+1.00	+2.00	0.00	+2.00	0.00	+2.00	0.00	+2.00	2.77	0.00

Note: Percentage of non-overlapping data – PND. Improved change is represented by +, deteriorating change is represented by -.

Table 4.6. Between-condition analysis for each participant’s response to single-item anticipated guilt.

Participant	Relative Level Change (A-B)	Relative Level Change (B-C)	Absolute Level Change (A-B)	Absolute Level Change (B-C)	Median Level Change (A-B)	Median Level Change (B-C)	Mean Level Change (A-B)	Mean Level Change (B-C)	PND (A-B)	PND (B-C)
1	-2.00	+1.67	-3.00	+2.00	-1.00	+2.00	-1.30	+1.61	11.11	0.00
2	-0.18	+0.05	-2.00	+1.00	0.00	0.00	-0.15	+0.15	0.00	0.00
3	-0.17	-0.22	0.00	0.00	0.00	0.00	-0.15	-0.07	13.89	0.00
4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	+1.00	0.00	0.00	0.00	+1.00	0.00	0.00	0.00	0.00	0.00
6	-0.13	+0.08	0.00	+1.00	0.00	0.00	0.00	+0.06	0.00	0.00

Note: Percentage of non-overlapping data – PND. Improved change is represented by +, deteriorating change is represented by -.

Table 4.7. Between-condition analysis for each participant's response to single-item of SRE.

Participant	Relative Level Change (A-B)	Relative Level Change (B-C)	Absolute Level Change (A-B)	Absolute Level Change (B-C)	Median Level Change (A-B)	Median Level Change (B-C)	Mean Level Change (A-B)	Mean Level Change (B-C)	PND (A-B)	PND (B-C)
1	0.00	-1.00	-1.00	-1.00	0.00	0.00	-0.37	-0.24	11.11	0.00
2	0.00	0.00	-1.00	+1.00	0.00	0.00	-0.03	+0.33	13.89	0.00
3	-1.00	0.00	-1.00	0.00	-1.00	0.00	-0.97	-0.78	16.67	0.00
4	-1.00	0.00	-1.00	0.00	-1.00	0.00	-0.47	-0.42	5.56	0.00
5	+1.00	0.00	0.00	0.00	+1.00	0.00	+0.36	-0.36	0.00	0.00
6	0.00	0.00	0.00	+2.00	0.00	0.00	-0.28	+0.39	27.77	0.00

Note: Percentage of non-overlapping data – PND. Improved change is represented by +, deteriorating change is represented by -.

Table 4.8. Between-condition analysis for each participant’s response to single-item of affect.

Participant	Relative Level Change (A-B)	Relative Level Change (B-C)	Absolute Level Change (A-B)	Absolute Level Change (B-C)	Median Level Change (A-B)	Median Level Change (B-C)	Mean Level Change (A-B)	Mean Level Change (B-C)	PND (A-B)	PND (B-C)
1	-3.00	+7.00	-7.00	0.00	0.00	+2.00	-0.58	+0.57	0.00	0.00
2	-1.00	+1.00	-2.00	+2.00	-1.00	0.00	-1.52	+0.04	27.78	2.78
3	+1.50	0.00	+5.00	0.00	+3.00	+5.00	+2.85	+1.74	16.67	5.56
4	0.00	-3.50	+1.00	-8.00	0.00	0.00	+0.72	-0.03	5.56	0.00
5	+2.00	-6.00	+5.00	-1.00	+2.00	-3.00	+1.39	-1.64	5.56	0.00
6	0.00	+2.00	-6.00	+6.00	0.00	0.00	-0.83	+0.03	8.33	0.00

Note: Percentage of non-overlapping data – PND. Improved change is represented by +, deteriorating change is represented by -.

4.4 Discussion

A growing body of evidence has contributed to our knowledge and understanding of AAS dependence (Brower et al., 1991; Ip et al., 2012; Kanayama et al., 2009; Pope et al., 2014) and recently its association with craving (see Zoob Carter & Boardley, in review). To date there is a dearth of research identifying the temporal trends between manifestations of AAS dependence (i.e., craving), and the associated psychosocial constructs of anticipated guilt, SRE, and affect across different periods of AAS use. Therefore, the overarching aim of this study was to explore the trends and levels of craving, anticipated guilt, SRE and mood within- and between- different phases of AAS administration.

One of the major contributions of this study was the observation of AAS craving across different patterns of AAS use. Despite models of AAS dependence indicating the presence of craving-like behaviours (see Wood, 2008), there remains limited research in this area; with just one study exploring the concept of craving within the AAS using community (see Zoob Carter & Boardley, In Review). With the inclusion of craving in the latest edition of the Diagnostic and Statistical Manual of Mental Disorders (APA, 2013) and researchers providing the provisions to adapt it for AAS users (see Kanayama et al., 2009c), it is important to further explore AAS craving to gain a greater insight into factors that may facilitate prolonged misuse of AAS.

Data analyses showed patterns of craving partly consistent with *HI* for three participants (P3, P5, and P6). Visual analysis of P6 observed consistency with *HI* through observing a low level of AAS craving with no change in trend in Phase A. However, with Phase B and C indicating P6 having a low level of craving with no change in trend, the consistencies with *HI* ended. Observations in P3 and P5 of high levels of AAS craving with no change in trend within Phase C were also consistent with *HI*. However, there were no

further consistencies with as P3 demonstrated a high level of craving with no change in trend in Phase A, and a high level of craving with a decreasing trend in phase B. P5 was observed to have a high level of craving with no change in trend in Phase A, and a high level of craving with no change in trend was observed in Phase B. The low level of AAS craving in phase A (P6) may be explained by findings from Shiffman et al (1995), whereby levels of craving have been found to be lowest whilst engaging in drug use. This may also provide further explanation for the observations in P6 that were not consistent with *HI* (i.e., low levels of craving in Phase B and C). The absence of trend observed in P6 within phase A can be explained by research from Drummond (2000), who postulates that drug use satiates craving by alleviating the desire to seek and take a drug. As such, craving would be at its lowest point and have no room to decrease any further. Again, Drummond's (2000) observation may provide an explanation for P6 demonstrating no change in trend in Phase B and C.

High levels of craving like those identified in P3 and P5 may be explained through the craving model of outcome expectancy (Marlatt, 1985), whereby positive expectations of how a drug will take effect elicits drug seeking behaviour. It is therefore possible that the types of anabolic compounds being consumed are not meeting the expectancies of some participants in this study. This may be attributed to AAS procurement from illicit drug markets (Evans-Brown et al., 2009) containing poor quality and/or the wrong type of active substances (Frude et al., 2020), different patterns of use (i.e., inclusion of other muscle building and ergogenic compounds in AAS protocols; see Kanayama et al., 2010b), and past drug use histories across participants (Skårberg et al., 2008). The observations of no change in trend in those experiencing high levels of craving (i.e., P3 and P5 in Phase C) may be explained with research from Schlauch et al (2013), who indicates craving for nicotine and alcohol is highest when approaching periods of substance use; therefore, indicating experience of craving

cannot increase anymore. The decreasing trend of craving observed in participants with high levels of craving (P3 in Phase B) may be explained by patterns of AAS use. Long-chain AAS have a slow method of action (de Souza & Hallak, 2011; Graham et al., 2008) lasting up to 4-weeks within the body (Llwelllyn et al., 2017). With dosages as high as 1,000mg per week combined with use of long-chain AAS as seen in P3, it is possible that the decreasing trend in craving during Phase B was due to the combined effect of the AAS use during that phase along with the remaining long-chain AAS still present from Phase A.

Between-condition analysis showed two participants demonstrating consistency with some, but not all, patterns of *H5*. Analysis identified higher craving levels in Phase B than in Phase A, and higher levels of craving in Phase C than Phase in B for P2, P3 and P6; demonstrating consistency with *H5*. Differences in dosages may explain the high levels of craving in P3. Yu et al (2014) indicate the dose dependent nature of AAS. It is possible that high levels of craving observed in P2, P3 and P6 are attributed to the lower dosages administered within Phase B and C establishing high levels of AAS expectancy for the next blast period. In contrast, no consistencies with *H5* were in seen in P1, P4, or P5. Between-condition analysis demonstrated P1 had a lower level of craving in Phase B than Phase A, and a higher level of craving in Phase C than Phase B. P4 were observed to have a lower level of craving in phase B than phase A, and a lower level in Phase C than Phase B. Whilst P5 was observed to display higher level of craving in Phase B than Phase A, with no change in level between Phase B and C. The low levels of craving observed in P1 and P4 may be attributed to the number and types of anabolic compounds being administered as they were observed to administer more anabolic agents, including fast acting oral compounds, than other participants. It is possible that the ergogenic effects from these different compounds used and the short time in which they were noticed may have met AAS expectancy of P1 and P4,

causing a reduced experience of craving (see Marlatt, 1985; Zoob Carter & Boardley in Review).

Within-condition observations demonstrated some but not all patterns of anticipated guilt responses proposed by *H2*. A low level of anticipated guilt with no change in trend was observed in four participants (P2, P4, P5, and P6) within Phase C. However, no consistency with *H2* was observed in any other phase of the study. In phase A the same four participants demonstrated a low level of anticipated guilt with no change in trend. Phase B indicated P2 presenting with a low level of anticipated guilt with a decreasing trend, whilst P4, P5, and P6 were observed to have a low level of anticipated guilt with no change in trend. Furthermore, two participants demonstrated no consistency with any aspects of *H2*. P1 was observed with low guilt with an increasing trend in Phase A, low guilt with decreasing trend in Phase B, and low guilt with a decreasing trend in Phase C. P3 was observed to have low guilt with no change in trend in Phase A and in Phase B; whilst in Phase C, P3 was observed with low guilt and a decreasing trend. An explanation for the low levels of anticipated guilt within this study can be found within the current literature, whereby Bandura (1996) proposes that the effects of anticipated guilt may be diminished by the use of moral disengagement (MD). It is possible the each of the participants were utilising MD within Phase A, B, and C, causing a low level of guilt across all phases of the study and not demonstrating patterns in anticipated guilt consistent with *H2* (i.e., changes in trend).

Between-condition observations from this study showed one participant demonstrating some, but not all, consistencies with *H6*. Lower levels of anticipated guilt were observed in Phase B than Phase A, and lower levels of guilt were identified in Phase C than Phase B in P3. The remaining five participants indicate no consistency with *H6*. Observations from P1, P2, and P6 were observed to demonstrate lower levels of anticipated guilt in Phase B than in

Phase A, and higher levels in Phase C than in Phase B. P5 was observed to show higher levels of anticipated guilt in Phase B than in Phase A, with no change in level identified in Phase B or C. P4 demonstrated no change in craving level in any phase. Within the current literature, anticipated guilt is proposed to deter individuals from engaging in acts that oppose societal norms (see Bandura, 1991). It is common practice within the steroid using community to pre-plan AAS use (Kanayama, et al., 2009; Llewellyn et al., 2017). Therefore, the higher levels of craving identified in Phase B within P5 and in Phase C within P1, P2, and P6 may be indicative of a desire to deviate from the pre-planned cruise phases. For example, the higher levels of guilt in Phase B for P5 may have been in response to a desire to increase the duration of Phase A, whereas the higher levels of guilt in Phase C may have been in response of a desire to prematurely initiate a blast phase in P1, P2, and P6; and thereby engaging self-regulatory processes to continue with the current cruise protocols. Boardley et al (2017) indicated an inverse relationship between anticipated guilt and AAS use; therefore, continuous administration of AAS across the study period may have suppressed anticipated guilt causing no level change to occur between phases.

Within-condition observations from this study demonstrated some but not all patterns of SRE responses in five participants were consistent with *H3*. P5 was observed to have a moderate level of SRE with a decreasing trend in Phase B, however results from Phase A (moderate level of SRE with no change in trend) and C (low level of SRE with no change in trend) were not consistent with *H3*. P2, P4, and P6 were observed to have a low level of SRE with no change in trend in Phase C; indicating their only consistency with patterns of *H3*. Observations of P2 indicated a high level of SRE with a decreasing trend in Phase A, and a high level of SRE with an increasing trend in Phase B. P4 was observed to have a moderate level of SRE with an increasing trend in Phase A, and a moderate level of SRE with no

change in trend in Phase B. P6 was observed to have a moderate level with no change in trend in Phase A and a moderate level of SRE with no change in Phase B. P1 was observed to have a low level of SRE with no change in trend in Phase A and Phase B, and have a low level with a decreasing trend in Phase C. P3 was observed to have a low level of SRE with no change in trend in Phase A and Phase B, and a low level of SRE with a decreasing trend in Phase C.

Explanations for the predominantly medium and low within-condition levels of SRE observed are as follows. Sayette (2004) proposes effective SRE overrides sensations of craving to avoid drug seeking behaviours. Therefore, as AAS were continually being used, the effectiveness of SRE may have been compromised and caused a moderate level to be observed within P4, P5 and P6 in Phase A and Phase B. Bandura (1991) proposing self-regulatory behaviour prevents the engagement in transgressive acts, therefore the low levels of SRE observed may be attributed to participants by complying with pre-planned AAS protocols. With AAS protocols being pre-planned (see Kanayama et al., 2009), high levels of SRE may be preventing P2 from administering larger doses of AAS. One explanation for decreasing trends of SRE may be attributed to an increasing trend of craving occurring during the same phase. A recent study has identified an inverse association between SRE and AAS craving (Zoob Carter & Boardley, In Review), therefore by experiencing high levels of AAS craving in Phase B SRE may be becoming impaired and present with a decreasing trend. Research has identified an inverse association between SRE and AAS use (see Boardley et al., 2017), therefore, by using AAS continuously SRE may have been compromised. With SRE being impaired in this way, it is also possible that this construct could not decrease any further, providing an explanation for the absence of a change in trend observed within participants.

Between-condition analysis identified three participants who demonstrated some consistency with *H7*, however four participants demonstrating no consistency with *H7*. Levels of SRE were reported to be lower in Phase B than in A, and lower in Phase C than in B within P1, P3, and P4. However, not all participants showed consistency with *H7*. P2 and P6 demonstrated lower levels of SRE in Phase B than in Phase A, and higher levels of SRE in C than in B. Whilst P5 indicated higher levels of SRE in Phase B than in Phase A, and lower levels of SRE in Phase C than in Phase B. One explanation for the predominantly low levels of SRE observed within these participants may be due to the continuous method of AAS administration. This has been identified in the current literature as Boardley et al (2017) indicated a negative link between SRE and AAS use. Therefore, with no time without AAS the effects of SRE may not be experienced (see Gwaltney et al., 2001) and therefore remain permanently impaired (i.e., in P1, P3, and P4).

Within-condition observations demonstrated one participant showed some consistency with *H4*. P6 was observed to indicate a high level of affect with no change in trend within Phase A, a high level of affect with an increasing trend in Phase B, and a high level with no change in trend in Phase C. No observed general pattern was identified in the responses from the other five participants. P1 indicated a moderate level with an increasing trend in Phase A and B, and a high level with a decreasing trend in Phase C. P2 indicated a high level and decreasing trend in Phase A, and a high level with a decreasing trend in Phase B and C. P3 was observed to have a high level with an increasing trend in Phase A, a moderate level with an increasing trend in Phase B, and a moderate level with no change in trend in Phase C. P4 indicated a high level with an increasing trend in Phase A, a high level with no change in trend in Phase B, and a high level with a decreasing trend in Phase C. P5 indicated a moderate level with a decreasing trend in Phase A, a high level with an increasing trend in Phase B, and

a high level with no change in trend in Phase C. Administration of AAS has been noted to elicit positive emotional effects onto users (i.e., increased self-confidence, heightened energy levels, improved mood; Bonnecaze et al., 2020; de Zeeuw et al., 2023; Yates, 2000). With affect demonstrated an increased variability, compared to the other variables of interest within each participant across each phase of the study, it is therefore possible that high levels and increasing trends experienced were attributed to AAS administration leading to the observed fluctuations in affect. Furthermore, by experiencing such positive effects on mood it is possible that participants with high levels of affect could not increase any further therefore demonstrating no change in trend. Moderate levels of affect, and decreasing trends in Phase A may be attributed to participants not getting the ergogenic effects they expected from their AAS administration, causing the experience of salient stimuli that may contribute to drug seeking behaviours (see Marlatt, 1985).

Between-condition analysis identified no participants who demonstrated consistency with *H8*. Between-condition analysis for P1, P2, and P6 displayed lower levels of affect in Phase B than in A, and higher levels of affect in Phase C than in B. P3 demonstrated higher levels of affect in Phase B than in A, and higher levels of affect in Phase C than in B. P4 and P5 indicate higher levels of affect in Phase B than in A, and lower levels of affect in Phase C than in B. With the ergogenic effects of AAS being dependent on dosages use (see Yu et al., 2014) lower dosages used in cruise phases may have caused some participants not to see the same results in musculature gains they would with high doses and thereby experience more incidences of negative affect; explaining the lower levels of affect in Phase B than in A found in P1, P2, and P6. The higher levels of affect observed in P1, P2, and P6 in Phase C may be attributed to positive expectation for the approaching blast phase and higher doses that accompany it.

4.4.1 Limitations and Future Directions

One of the major limitations of this research was the absence of participants on a traditional ‘on-cycle/off-cycle’ protocol. Within this study all participants were on ‘blast’ and ‘cruise’ protocols, therefore, no participants were in stages of AAS abstinence at any point. This may have reduced the magnitude of the expected differing patterns between the three phases (see Chavarria et al., 2012b; Sayette et al., 2004; Shadel & Cervone, 2006; West & Shiffman, 2016). Evidence of this limitation can be seen by the small effect sizes identified by the PND calculations. It is therefore recommended to future researchers who wish to conduct a similar study design to focus their data collection on ‘on-/off-cycle’ protocols, and exclude AAS users running ‘blast’ and ‘cruise’ administration. Furthermore, it is important to note that the language surrounding AAS protocols may be changing. It was apparent within this study that the phrase ‘on-cycle’ was also attributed to ‘blasting’ administration. Understanding the true nature of participants patterns of use is vitally important for researchers wishing to undertake similar studies. It is recommended that researchers explore patterns of use in their participants in a more detailed manner. This may be obtained by allowing participants to explain their AAS administration via text box responses, rather than getting participants to fill in a tick box.

Further limitations include the small sample size of this study. This limited the researcher in the types of analysis available for analysis single-case design research (see Barker et al., 2011). A larger sample of 100 participants (e.g., 50 participants for on-cycle period and 50 in an off-cycle period) following a similar study protocol would allow for a statistical method of analysis such as multilevel modelling (see Maas & Hox, 2005).

Analysis with methods such as multi-level modelling may aid in distinguishing differences within- and between-phases of AAS use (see Snijders & Bosker, 2012). As AAS users are a

notoriously hard to reach populace, we would recommend future studies embed researchers within multiple online forums and social media pages specific to AAS using communities to make amicable relationships with potential participants. This may facilitate recruitment of a larger sample of AAS users.

4.5 Conclusion

Through this naturalistic single case experimental investigation, we observed patterns in temporal levels and trends of AAS craving, anticipated guilt, and affect across three phases of AAS administration. This study provided novel contributions to the extant literature as it is the first study to observe AAS craving longitudinally within a single case design, furthering our knowledge of how those within the AAS community may be experiencing craving (see Zoob Carter & Boardley, In Review). This study provides further novel contributions to the current literature by demonstrating the use of EMA methods of data collection can be used within the AAS community. To date the EMA literature has focused mainly on alcohol, nicotine and opioids, as such this study has filled a gap in the extant literature. We look forward to studies building upon this method of data collection and utilising this research design in future endeavours.

Chapter 5 – General Discussion

5.1 Thesis Summary

Based on current conceptualisations, research has estimated that anabolic-androgenic steroid (AAS) dependence is experienced by up to 30% of those who use steroids (Pope et al., 2014). Existing literature indicates dependence is associated with increased experience of undesired physical and psychological effects (Brower et al., 1991; Hauger et al., 2019, 2020, 2021; Ip et al., 2012; Kanayama et al., 2009a; Scarth et al., 2022). To date, there is a dearth of research identifying the longitudinal relationship between AAS dependence and associated adverse effects. Presently, there is no universally agreed upon model of AAS dependence (Brower, 1992; Bahrke & Yesalis, 1994; Brower, 2002; Kanayama et al., 2010a; Hildebrandt et al., 2011), limiting our understanding of the construct. This is particularly problematic when most of the proposed models are multifactorial, which contrasts with measurement approaches to date that have adopted unidimensional approaches (Gillespie et al., 2007; Gossop et al., 1995; Grant et al., 2007; Kanayama et al., 2009c; Lynskey & Agrawal, 2007; Ray et al., 2008; Teesson et al., 2002). Also, researchers have not thus far examined craving with respect to AAS use, even though it represents a construct thought to aid in the development of dependence (Tiffany & Wray, 2012). A first step in examining craving in the context of AAS use would be to develop a measure that captures possible dimensions of AAS craving. Finally, due to the absence of literature examining AAS craving current knowledge and understanding of how this is experienced within AAS users maybe sparse. Therefore, observations of the temporal patterns in craving across periods of use may provide an insight as to how craving presents within individuals who use AAS.

To address these limitations in current knowledge, the line of research described in this thesis aimed to: a) longitudinally examine whether MD mediated the relationship between

AAS dependence and undesired physical and psychological effects (Study 1), b) develop and validate multidimensional measures of AAS dependence and AAS craving (Study 2), and c) examine the temporal patterns in AAS craving, anticipated guilt, SRE, and affect different stages of AAS administration (Study 3).

Chapter 1 provided an introduction to AAS, AAS dependence, and highlighted potential factors facilitating the development of AAS dependence, including AAS craving and moral disengagement. This introduction also provided a detailed background and theory regarding AAS dependence, and AAS craving, as well as other key variables from the subsequent empirical chapters.

Chapter 2 presented a longitudinal study building upon previous research on AAS dependence, associated undesired effects (Bower et al., 1991; Ip et al., 2012; Kanayama et al., 2009a, 2009b, 2010a) and doping moral disengagement (MD; Boardley et al., 2014, 2015, 2017, 2018; Boardley & Grix, 2014). Using mediated regression analysis, the study explored whether MD mediated longitudinal relationships between AAS dependence and negative psychological and physical health consequences of AAS use. Despite not identifying an indirect effect, this study was the first to demonstrate significant positive longitudinal effects of AAS dependence on undesired health effects, and AAS dependence on MD.

Chapter 3 developed and validated psychometric measures of AAS dependence and AAS craving. Current measures of AAS dependence are unidimensional, and therefore incapable of representing the multidimensional nature of AAS dependence depicted in current multi-component models of AAS dependence (Bahrke & Yesalis, 1994; Brower, 1992; Brower, 2002; Hildebrandt et al., 2011; Kanayama et al., 2010a). This study addressed this limitation in current assessment approaches, and by developing a multidimensional measure

of AAS craving, it also addressed this gap in measurement capability. Through a rigorous set of analyses this study developed and validated two novel and reliable measures, to assess AAS dependence and AAS craving.

Chapter 4 sought to evaluate daily patterns in AAS craving, anticipated guilt, and SRE across different stages of AAS administration via ecological momentary assessment (EMA). To date research has explored temporal trends in craving within opioid, and dependent users (Huhn et al., 2016; Shiffman & Waters, 2004). However due to a dearth in research on AAS craving, there has been no attempt to identify temporal trends in craving or associated constructs across AAS administration. Using visual analysis (see Barker et al., 2011; Lane & Gast, 2014), some expected patterns were identified across different periods of AAS administration, however, this was not consistent across the whole sample. These results were potentially due to the ‘blast’ and ‘cruise’ nature of AAS administration across the six participants.

5.2 Discussion and Findings

This thesis provides significant novel contributions to the AAS dependence literature. To date, researchers have demonstrated a positive association of AAS dependence and increased experience of undesired physical and psychological effects linked to AAS (see Brower et al., 1991; Hauger et al., 2019, 2020, 2021; Kanayama et al., 2009a; Kanayama et al., 2010a; Scarth et al., 2022). Findings from Chapter 2 indicate the causal relationship between AAS dependence and increased experience of undesired physical and psychological effects associated with AAS use. Despite researchers postulating multidimensional models for dependence (Brower, 1992, 2002; Hildebrandt et al., 2011; Knayama et al., 2010), measures to assess this syndrome are adapted unidimensional measures suit AAS (see Griffiths et al., 2018; Kanayama et al., 2009c). Chapter 3 produced a novel measure to assess multiple

dimensions of AAS dependence within the AAS community. To date, research in AAS dependence has identified demonstration of craving-like behaviours in animal models (see Arnedo et al., 2002; Schroeder & Packard, 2000; Wood, 2008), and included items for craving in diagnostic criteria for dependence (APA, 2013; de Zeeuw et al., 2023). Despite dependence and craving being considered distinct constructs (see Koob & Le Moal, 2008), no endeavours have sought to identify and explore craving within the AAS using community. Chapter 3 produced a novel multi-dimensional measure to explore craving within the AAS community. With this dearth of research on AAS craving in the steroid community, Chapter 4 observed AAS craving and associated psychosocial constructs across different period of AAS administration, demonstrating some – not but not all – evidence of patterns in level and trend consistent with current theory of craving across AAS administration.

Several models have been proposed to explain the manifestation of AAS dependence (Brower, 1992, 2002; Hildebrandt et al., 2011; Kanayama et al., 2010); the findings from this thesis support elements from each of these models. A common element within two of the proposed models of AAS dependence is use of AAS to self-medicate for symptoms of withdrawal (Brower, 1992; Kanayama et al., 2010a). Chapter 2 demonstrated evidence in support of this mechanism, as AAS dependence displayed a positive direct causal relationship with experience of undesired health consequences of AAS. The adverse health events identified within Chapter 2 contained withdrawal-like symptoms, attributed to AAS induced hypogonadism (ASIH; see Tan & Scally, 2009). Chapter 3 indicated support for the withdrawal mechanism of dependence (Brower, 2002; Kanayama et al., 2010) by identifying the sub-dimension of ‘withdrawal’. Three items within the ‘withdrawal’ sub-dimension (see Appendix A, items 4 – 6) were established to identify the use of AAS to self-medicate withdrawal-like symptoms; demonstrating significant positive association with experience of

undesired effects in Chapter 3. Throughout the thesis we saw a high prevalence of blast and cruise regimes, highlighted by the participants from Chapter 4 exclusively running this method of AAS administration. Increasing in prevalence (Bonnecaze et al., 2020; Cohen et al., 2007), Blast and cruise regimes are recognised by researchers as an approach used to mitigate and/or self-medicate AAS withdrawal symptoms, including undesired health effects and loss of musculature (Kanayama et al., 2010a; Underwood et al., 2021). From evidence within the current literature, it is possible that the blast and cruise protocols identified across this study may have been implemented to self-treat symptoms of withdrawal. Overall, evidence from this thesis supports use of AAS to self-medicate symptoms of withdrawal proposed by Brower (1992) and Kanayama et al (2010a).

Kanayama and colleagues (2010a) postulate a further mechanism for AAS dependence, that development of dependence is attributed to the positively reinforcing effects of AAS administration. Evidence for the positive reinforcing effects of AAS dependence has been identified in rodent models through environmental cues via conditioned place preference (see Alexander et al., 1994; Arnedo et al., 2002; de Beun et al., 1992; Schroeder & Packard, 2000; Wood et al., 2004a). Environmental cues were identified within Chapter 3, within the ‘environment’ sub-dimension. The four items within this sub-dimension (see Appendix B, items 5 – 8) were established to identify how environmental cues may influence desire to use AAS. It is important to note that this sub-dimension was identified within the higher order dimension of AAS craving, not AAS dependence. As craving has been identified as a key antecedent of dependence (Drummond, 2001; Ray & Roche, 2018; Tiffany et al., 2008), current models, especially Kanayama’s (2010a), should incorporate the facilitating role of AAS craving. It is possible that in doing so, AAS craving be a subsidiary element to this mechanism in the model proposed by Kanayama et al (2010a).

A shared mechanism between two of the existing models of AAS dependence is continued use of AAS despite experiencing undesired effects (see Brower, 2002; Hildebrandt et al., 2011). The findings from Chapter 2 build upon the mechanism, of continued use of AAS despite incurring harms, by providing a novel understanding about the undesired effects associated with AAS dependence. This was achieved by demonstrating a direct causal relationship between AAS dependence and increased experience of harms, and differentiating harms into undesired physical and psychological effects. Chapter 3 provides further evidence in support of this mechanism by identifying three sub-dimensions of undesired effects within the higher order construct of AAS dependence. These sub-dimensions include ‘unwanted physical effects’, ‘unwanted psychological effects’, and ‘unwanted socio-occupational effects’, allowing for clear identification of the more prevalent adverse effects experienced by AAS users. Hildebrandt et al (2011) proposing a further consideration, that physical dependence occurs when ancillary substances are administered in an attempt to self-medicate for undesired physical effects associated with AAS use. Evidence for this hypothesis was identified within Chapter 3 via the sub-dimension of ‘unwanted physical effects’. An item within this sub-dimension explores continued use of AAS despite attempts to self-medicate for undesired physical harms (see Appendix A, item 8). Ancillary substances are widely used by the AAS community to self-medicate for undesired effects (Kanayama et al., 2010a, 2010b; Pope et al., 2014), and was seen within each of the studies conducted within this thesis. Overall, the findings from this thesis demonstrate agreement with the mechanism put forward by both Brower (2002) and Hildebrandt et al (2011).

The identification of the higher order factor of AAS dependence within Chapter 3 makes an important contribution to the literature as it is the first AAS dependency measure specifically designed for those who use AAS, rather than being adapted from

existing measures (see Kanayama et al., 2009c; Cole et al., 2003). The findings from Chapter 3 may aid in the development of our current understanding of AAS dependence in a number of ways, including identification of which sub-dimensions of AAS dependence are most strongly linked with harmful use of AAS. With Chapter 2 demonstrating direct causal effects between AAS dependence and experience of physical and psychological harms, it would be of interest to further explore different aspects of behaviours attributed to AAS dependence. Increased dosages and prolonged administration of AAS has been identified as a characteristic for AAS dependence (see de Zeeuw et al., 2023; Ip et al., 2012; Scarth et al., 2022), and is associated with increased risk of experiencing harms (Bolding et al., 2002; Harmer, 2010). Prolonged use of high doses (of up to 2g per week) of AAS were seen in use of AAS in Chapter 4, with large doses being reported across blast and cruise protocols. Therefore, it would be of interest to explore how each sub-dimension of AAS dependence is associated with undesired effects across different stages of AAS use, including blast and cruise administration.

Through identifying ‘AAS effectiveness’ as an important aspect in AAS dependence, findings from Chapter 3 present an opportunity to explore why some individuals progress from traditional cyclic use of AAS to continuous use. Sustained use of AAS was reported across all three studies in this thesis, but were made most apparent in Chapter 4 with participants exclusively running blast and cruise protocols. Researchers have proposed that this behaviour may be deemed necessary by AAS users to not only maintain their musculature, but to further develop increased muscle mass (Smit et al., 2020). With researchers hypothesising that prolonged use of AAS increases the risk of incurring medical harms (see Kanayama et al., 2009), and findings from Chapter 2 corroborating this; it is important to explore effectiveness as a means for the transition from cyclic use to permanent

use of AAS. Therefore, exploring how ‘AAS effectiveness’ is associated with different methods of AAS administration (i.e., on-off-cycle compared to blast/cruise), may give greater insight on why this transition takes place.

By showing experience of ‘withdrawal’ is a key aspect of AAS dependence, findings from Chapter 3 allow for researchers to assess levels of withdrawal across AAS use, and determine if this is associated with self-medication of AAS. Exploring the effects of withdrawal in this way, gives this sub-dimension the propensity to be an indicator for self-medication of harms experienced in drug-free periods (see Kanayama et al., 2020; Tan & Scally, 2009). Chapter 2 has demonstrated the importance in further exploring this topic as a direct causal relationship was identified between AAS dependence and harms associated with symptoms of withdrawal. Koob and La Moal (2008) highlight experience of withdrawal is impacted by multiple stimuli including, effects of cessation of drug intake, internal/external pressure to obtain the drug (i.e., SRE; see Bandura, 2001), and a desire to administer the substance (i.e., craving). Furthermore, experience of withdrawal is currently believed to be a motivating factor for the progression from intermittent use (i.e., on-/off-cycle) to prolonged use (i.e., blast and cruise), and increasing the risk of incurring undesired health effects (Chandler & McVeigh, 2017). An example of this can be seen in Chapter 4 which observed patterns in craving and SRE across blast and cruise administration. In some participants, observations indicated patterns in levels and trends associated with drug seeking behaviour postulated in craving literature (see Drummond, 2001). Therefore, examining which of the four sub-dimensions of AAS craving are most associated with withdrawal and psychosocial factors like SRE across different stages of AAS administration may give a greater insight to how withdrawal aids establishing in drug seeking behaviour and AAS dependence in periods of abstinence.

Studies within this thesis provide a significant contribution to our understanding of the undesired effects associated with AAS dependence. Chapter 2 not only confirms previous research by identifying the association of undesired physical and psychological effects with AAS dependence (see Hauger et al., 2019), but demonstrates a direct causal effect between AAS dependence and experience of physical and psychological harms. Chapter 3 provides additional evidence by presenting three distinct sub-dimensions to identify undesired consequences of AAS use within ‘physical effects’, ‘psychological effects’, and ‘socio-occupational’ effects. These findings support previous work (see de Zeeuw et al., 2023; Ip et al., 2012). However, measures used in current research are unable to distinguish between each category of undesired effects experienced by AAS users. To date, research has identified a link between AAS dependence and undesired socio-occupational events (see Hauger et al., 2021). However, there remains an absence of studies identifying how socio-occupational events contribute to the development of AAS dependence. Therefore, the identification and discrimination between each of the three sub-dimensions of undesired effects provides substantial findings and tools to further our knowledge on the unpleasant consequences of AAS use.

By identifying a higher order factor of AAS craving within Chapter 3, this thesis presents a novel and important contribution to the extant craving literature as it is the first measure to assess AAS craving, providing an insight to how craving affects those who misuse AAS. Furthermore, through single case naturalistic observations in AAS users across three independent 12-day periods, Chapter 4 provides an innovative insight into how AAS craving is experienced across periods blast and cruise protocols. To date, craving is identified as a poorly understood topic (Flannery et al., 2001), surmised by the absence of a universally accepted model of craving. With current models predominantly focused on psychoactive

substances (i.e., alcohol, cocaine, nicotine; Kakko et al., 2019; Serre et al., 2018), there is an absence of research on AAS craving. Therefore, the findings from this thesis offer a greater insight of how craving is experienced within the AAS community.

Several models of craving have been proposed to explain the action of craving within drug users, evidence from this thesis demonstrates support with some mechanisms of these models in the experience of AAS craving. The conditioned response model (see Drummond, 1990) identifies the importance of environmental cues in causing drug seeking behaviour. Findings from Chapter 3 provide evidence in support of this model conditioned response (Drummond, 1990) by identifying the sub-dimension of ‘environment’ in Chapter 3, as a key sub-dimension of AAS craving (Appendix B, items 5 – 8). Observations in Chapter 4 identified temporal patterns in AAS craving within some of the participants. With notifications for this study being sent out at three different intervals during the day, it is likely that some participants answered the surveys within the gym environment or in contexts associated with muscle building activities (e.g., socialising with gym partners outside of the training environment); potentially contributing to the patterns in craving observed. Overall, findings from this thesis provide support for the effect of environment to elicit craving amongst those who use AAS users. To further knowledge of AAS craving and the effect of ‘environment’ on eliciting drug seeking behaviour, it may be of interest to explore links between negative and/or positive experiences within AAS associated environments to determine if these have a strong association with this sub-dimension of AAS craving.

Evidence in support of the outcome expectancy model of drug craving (Marlatt, 1985) has been identified across the findings of this thesis. Marlatt (1985) proposes experience of strong expectations about the effect administering the substance drive drug seeking behaviour. Outcome expectancy demonstrates some cross-over with substance withdrawal in syndromes

of drug dependence, whereby during periods of abstinence salient stimuli may be experienced, driving drug seeking behaviours and establishing relapse behaviours (Koob & La Moal, 1997). By showing experience of ‘expectancy’ is a key aspect of AAS craving, Chapter 3 identifies offers support for Marlatt’s (1985) model. The sub-dimension of ‘expectancy’ (see Appendix 2, items 1 – 4) may offer a greater understanding of an individual’s experience during AAS withdrawal, whilst accounting for some of the undesired effects noted to be experienced by AAS dependent users (e.g., mood disturbance, insomnia, etc; see Chapter 2). Furthermore, it may offer some insight how individuals view the use of their AAS across different phases of AAS administration (e.g., on-/off-cycle or blasting/cruising; see Chapter 4). By identifying ‘expectancy’ is an intrinsic element of AAS craving, researchers may identify how this relates to AAS dependence. More specifically, which of the five sub-dimensions of AAS dependence identified within Chapter 3 are most strongly associated with ‘expectancy’. This will offer a greater insight on the relationship between AAS dependence and craving.

Experience of different mood (affect) states has been identified across this thesis in regard to AAS craving. Chapter 3 identified the presence of both positive and negative mood subdimensions within the higher order factor of AAS craving. Chapter 4 was able to observe some patterns in affect across AAS administration periods on a day-to-day basis, highlighting a real-world application of affect within those who use AAS. The dual effect model of craving indicates changes in mood states (i.e., positive/negative affect) have the ability to elicit craving (Baker et al., 1987; Niaura et al., 1988). Negative affect has been demonstrated to have a positive association with drug seeking behaviour (Tiffany, 1999, 2010), whilst positive affect negatively predicts drug seeking behaviour (Huhn et al., 2016; Lydon-Staley et al., 2017; Schlauch et al., 2013). The dual effect model demonstrates some overlap with

substance withdrawal in drug dependence, whereby negative emotional states drive drug seeking behaviours (Koob & Volkow, 2016). Chapter 2 indicates support for the impact of negative affect influencing drug seeking behaviour, whereby AAS dependence indicated a direct causal effect on undesired psychological effects; associated with symptoms of AAS withdrawal. Therefore, evidence from this thesis offers support for the role of affect on AAS craving behaviour.

These findings from Chapter 3 provide an important aspect for research in AAS misuse. By providing evidence for two distinct higher order factors: one for AAS dependence and the other for AAS craving, the measures developed in Chapter 3 will aid in distinguishing between dependence and craving within the AAS community. Despite the extant literature providing distinct definitions for drug dependence¹⁷ and craving¹⁸. Craving has been added to the diagnostic statistical manual of mental health disorders 5th edition (DSM-V; APA, 2013), and is present within the international classification of diseases (WHO, 1992) muddling the distinction between these two constructs in research. With the adaptations proposed for the DSM-V for use in AAS research (see Kanayama et al., 2009c), its use in recent studies (see de Zeeuw et al., 2023). It is important for AAS researchers to have a means to explore dependence and craving of AAS in a more succinct and bespoke manner in order to distinguish between the effects of dependence and craving and to explore interactions between each construct. Therefore, the measures from Chapter 3 offer a unique way of exploring dependence and craving within the AAS community.

¹⁷ Dependence is defined as; 'a chronically relapsing disorder characterised by compulsions to find and use a drug, loss of control over intake, and the presence of a negative emotional state when drug use is inhibited' (Koob & La Moal, 2005).

¹⁸ Craving is defined as; 'a memory of rewarding aspects of drug use whilst in a negative mood state' (see Koob & La Moal, 2008).

Findings from Chapter 2 and Chapter 3 provided novel findings to further our understanding of the association of AAS use with the psychosocial construct of moral disengagement (MD) from Bandura's (1991) theory of moral thought and action. Evidence for the relationship between AAS dependence and MD was supported in Chapter 2, which showed significant direct causal effect between AAS dependence and MD. Further support of the link between AAS dependence and MD was shown in Chapter 3, which identified a positive association between each sub-dimension of AAS dependence with MD. Presently, research has explored the use of MD in those who administer AAS, and has demonstrated a positive association between MD and AAS use (Boardley et al., 2014, 2015, 2017, 2018; Boardley & Grix, 2014). The findings from this thesis go beyond the current research by demonstrating AAS dependence causes rationalisation and justification of AAS misuse through MD. These findings provide evidence for the relevance Bandura's (1991) theory in the AAS using community, demonstrating how MD may aid in the development of AAS dependence.

Other elements of Bandura's (1991) theory were explored within this thesis. Chapter 3 provided evidence for the negative association between AAS dependence and self-regulatory efficacy (SRE), whilst Chapter 4 observed temporal patterns of SRE and anticipated guilt, in some participants, across three different phases of AAS administration. Up to now researchers have explored the links between AAS use and SRE, whereby Boardley et al (2017) established a negative association between use of AAS and SRE. With this thesis indicating the presence of SRE within AAS dependence, it is possible that prolonged impairment of SRE may facilitate AAS dependence. It would be of interest to determine how each of the sub-dimensions of AAS dependence and AAS craving are associated with SRE across AAS administration. The findings from this thesis provide evidence for the relevance SRE theory in

the AAS using community, demonstrating how SRE may aid in the development of AAS dependence and craving.

5.3 Limitations and Future Directions

A shared limitation across all studies within this thesis concerned generalisability, with the possibility that we did not recruit all typologies of AAS users (Christiansen et al., 2017; Zahnw et al., 2018) across the three studies in this thesis. It is possible that we captured data from an array of typologies, including the YOLO type (Christiansen et al., 2017) within the first time point of Study 1 due to the implementation of lockdown procedures during the COVID pandemic. With the absence of activities that would normally fill the time of these participant no longer available, more time may have been spent on online forums, and subsequently taking part within the study. However, with the relaxation of COVID lockdown during subsequent time points of Study 1, and experienced throughout Study 2 and 3, it is likely that these YOLO participants did not engage with the other studies within this thesis. As YOLO users obtain their knowledge of AAS from ‘steroid gurus’ (see Christiansen et al., 2017), it is possible that a distrust for researchers and medical professionals is passed down, limit interactions academics (Bonnacaze et al., 2020). We suggest advertisement of academic studies via gatekeepers and moderators on forums may benefit future research, as approval from respected members of the AAS community may encourage engagement from harder to reach AAS users. Furthermore, embedding researchers in gyms to recruit AAS users may be of some use. Evidence in qualitative literature has indicated those who use AAS are more trusting of researchers who demonstrate knowledge of training and AAS (Boardley & Grix, 2014; Monaghan, 2002; Underwood, 2017), and understand the culture of the ‘brotherhood of iron’ (see Smith & Stewart, 2012). Therefore, snowball sampling in this way may provide access to participants researchers would not

normally have access to. By conducting this method of data collection with large research groups and in collaboration with other research groups (nationally and internationally), larger and more generalisable samples of AAS users may be collated.

Another common limitation in this thesis was found with the internal consistency of the MD measure used. Currently, MD has been explored within the IPED using community with the doping MD scale (see Boardley et al., 2018). This scale was adapted within Chapter 2 to suit AAS, by rewording and replacing the term ‘doping’ with ‘steroid use’. Despite demonstrating good internal consistency scores, the alpha values were smaller than reported in previous literature (see Boardley et al., 2017). This was also found to be the case in Study 2 (Chapter 3) when reverting item wording back to ‘doping’ (see Boardley et al., 2018). MD internal consistency was unacceptable in Chapter 4, and was therefore omitted from the study. The participants from this thesis were predominantly hardcore gym users; this is different from the study by Boardley and colleagues (2017), as their sample was mainly made up of individual and team sports athletes. It is possible that gym users respond to MD items differently from sporting athletes, whereby gym users dissociate their use of AAS from the term ‘doping’. Therefore, not viewing MD items as critiquing their personal beliefs of their own drug use. By changing the items in Chapter 2 to suit AAS, the apparent change in alpha levels may have been attributed to some AAS using individuals interpreting the items in terms of their own use, subsequently answering in a defensive manner attributed to existing beliefs surrounding stigmatisation by academics and medical professionals (van de Ven et al., 2022a; Yu et al., 2015). Development of a bespoke AAS suited measure of MD would be key in amending this limitation.

Study 1 was subject to attrition, experienced across the transition between the first and section time points (62.1%), second a third time points (10.9%), and third and fourth time

points (4.4%).¹⁹ Attrition played a part in omitting the first time point from analysis in this study. Despite this, attrition was not to a level that would cause the results to be non-meaningful (see Angrist et al., 1996). Although statistically significant results were identified within the study, we believe that the reduced power attributed to the attrition rate will have reduced the ability to detect statistically significant results. We believe that attrition was due to a number of reasons including; follow up emails being redirected to junk/spam folders, COVID survey fatigue, participants forgetting their involvement in the study, reduced motivation to continue attributed to the fluctuations of lockdown protocols (see Zoob Carter et al., 2021). We therefore recommend to future longitudinal studies to include questions at the end of each survey enquiring about current email addresses for participant. This will ensure that the most up-to-date email addresses are retained and may aid in the prevention in attrition.

Further limitations in Study 1 were identified within the measures of AAS dependence used. Analysis of internal consistency at T1 identified that the SDS was not internally consistent (i.e., unacceptable), leading to the addition the AAS adapted DSM-IV criteria (see Kanayama et al., 2009c) from T2. The DSM-IV was also met with limitation as it is unable to capture the multidimensional nature of AAS dependence exhibited in extant models (see Bahrke & Yesalis, 1994; Brower, 2002; Brower et al., 1991; Hildebrandt et al., 2011; Kanayama et al., 2010), as existing measures of AAS dependence unidimensional (Gillespie et al., 2007; Gossop, Darke, Griffiths, Hando, et al., 1995; Ray et al., 2008). Therefore, we recommend researchers utilise the multidimensional measure curated in Study 3 to identify if specific aspects of AAS dependence are more strongly linked with undesired health effects.

¹⁹ Time points 2, 3 and 4 were retained, whilst time point 1 was omitted from analysis. The time points were renamed time point 1, 2, and 3 in the writing up of Study 1.

Study 2 was met with specific limitations, specifically by not examine all aspects of validity. Clark and Watson (2019) identify that validation of measures is an ongoing process. As such we recommend future work to examine other aspects of validity such as predictive validity. This would be achievable via examining scores from the new measures with theoretically related constructs over time. As data for this study was primarily collected from westernised cultures, further validation of the scales with non-westernised cultures would present an important avenue to explore in future work. Furthermore, it was beyond the scope of this study to measure test-retest reliability to establish the consistency of AASDS and AASCS scores across time. Therefore, researchers should aim to address this in future endeavours.

Limitations specific to Study 3 included its small sample size. This limited the researcher in the types of analysis available for analysis single-case design research (see Barker et al., 2011). A larger sample would allow for more statistical methods of analysis such as multi-level modelling may aid in distinguishing differences within phases of AAS administration, and between different phases of AAS use (see Snijders & Bosker, 2012). As AAS users are a notoriously hard to reach populace, we would recommend future studies embed researchers within the AAS community and utilise snowball sampling to facilitate the data collection process (see Miller, 2003). Recruitment in this manner will provide researchers with a sample suitable for EMA based studies (see Shiffman, 2009). Finally, by utilising the help of other researchers and/or larger research groups embedded within other gyms and training environments, the study has a propensity to reach a larger sample size, more suited for longitudinal research (see Ployhart & Ward, 2011).

5.4 Applied Implications

The novel findings identifying the relationships between AAS dependence and psychosocial mechanisms of MD and SRE in Chapter 2 and Chapter 3, and the patterns between AAS craving guilt and SRE in Chapter 4 furthering our understanding of the factors that may facilitate AAS dependence. The current literature on AAS dependence has postulated several models in order to explain how AAS using individuals manifest this syndrome (Bahrke & Yesalis, 1994; Brower, 2002; Brower et al., 1991; Hildebrandt et al., 2011; Kanayama et al., 2010) . However, none of these models have explored how psychosocial factors such as MD, SRE or anticipated guilt may attribute to the development of AAS dependence (de Zeeuw et al., 2023; Ip et al., 2012; Kanayama et al., 2009; Scarth et al., 2022). Despite researchers identifying a number of characteristics of those with AAS dependence, there is little research exploring the mindset of those with AAS dependence and how they rationalise and justify their behaviours to mitigate self-rebuke associated with their use of AAS. Therefore, the information from this thesis may offer a greater insight to the thoughts and characteristics of those with AAS dependence. By identifying the role of MD, SRE and anticipated guilt in AAS dependence and AAS craving they may offer novel targets for harm reduction interventions; such as cognitive behavioural therapy (see Quaglio et al., 2009; Smit et al., 2019).

To date research on undesired physical effects associated with AAS use have been reported as being reversable (Goldman & Basaria, 2018); consequently, the impact upon overall health may be understated by some sub-populations of AAS users (Pope et al., 2014b; Christiansen et al., 2017). It is therefore of high importance to present the current findings from Chapter 2 and Chapter 3 to both the AAS community, healthcare practitioners, and harm reduction services. Atkinson and colleagues (2021) have indicated the importance of

providing credible research to healthcare practitioners and harm reduction workers, in order to establishing effective engagement strategies with AAS users. To date much of the harm reduction surrounding AAS use is focused on blood-borne viruses causing substantial frustration amongst the AAS using community as many other physical harms are more frequently experienced (see Underwood, 2019), and to date there is no evidence indicating transmission of BBVs occurs via AAS use (see McVeigh, 2019). Havnes et al (2019) suggests that providing practitioners with improved knowledge about the adverse effects of AAS use may facilitate engagement and management of AAS users. By understanding the needs and experiences of AAS users, the findings collated in this thesis will aid in the provision of information and understanding to these harm reduction groups, aiding in the creation of bespoke management plans (see Bates et al., 2021) for AAS dependent users. Furthermore, by circulating the findings from studies, such as the ones in this thesis, will aid in raising an understanding and awareness within the AAS community about the impact sustained use of AAS has on their physical health.

Presently there is some disagreement within the AAS community over the presence of undesired psychological effects (Kimergård, 2015; Monaghan, 2002); however, it is important to ensure findings from Chapter 2 and Chapter 3 are not overlooked by the AAS community and healthcare providers. A recent review has suggested that AAS using individuals experiencing undesired psychological symptoms may benefit from interventions through behavioural health therapists (Bonnecaze et al., 2021). However, researchers like Van de Ven et al (2022) indicate that there is a dearth of evidence from research informing effective harm reduction provision. Therefore, findings from this thesis demonstrating a causal effect of AAS dependence on psychological harms, and the presence of psychological harms as a sub-dimension for AAS dependence may offer such evidence and knowledge to practitioners.

Furthermore, by identifying the myriad of undesired psychological events experienced by those who use AAS (Goldman & Basaria, 2018), harm reduction services and healthcare professionals may be made aware AAS users are often met with multiple comorbidities. Therefore, novel findings from this thesis may aid in providing harm reduction of healthcare services with the appropriate information to develop bespoke management and support to AAS dependent users. Furthermore, circulating the findings from studies, such as Chapter 2 and Chapter 3, may aid in raising an understanding and awareness within the AAS community about the impact sustained use of AAS has on their psychological health.

By developing and validating the multidimensional measures of AAS dependence (Chapter 3), this measure may aid in harm reduction and healthcare workers further understand how dependence affects those who use AAS. By getting individuals who use AAS to complete this measure, services may identify the primary areas to be targeted by therapeutic interventions and therefore develop more effective support services. By identifying the presence of AAS craving in AAS users (Chapter 4) and with the development and validation of the multi-dimensional measure of AAS craving (Chapter 3), findings from this thesis will provide practitioners with a greater understanding of factors that may facilitate the development of AAS dependence. The AAS craving measure will enable practitioners to target specific subdimensions that have stronger associations with AAS dependence, and therefore be the target for therapeutic intervention by harm reduction practitioners.

5.5 Conclusion

The aim of this thesis was to investigate the psychosocial factors that facilitate AAS dependence, and in doing so identified several novel contributions to the current literature. Findings presented here not only support current understanding within the AAS literature but further current knowledge of AAS dependence and the psychosocial factors that facilitate its

development. This thesis has contributed to the current literature by demonstrating the casual relationship between AAS dependence and psychosocial mechanisms of MD, whilst demonstrating a causal relationship between AAS dependence and undesired effects of AAS. Secondly, this thesis has produced bespoke, validated, and reliable multidimensional measures to assess AAS dependence and AAS craving. These measures will aid in identifying which dimensions of dependence and craving are experienced by individuals who use AAS, whilst identifying areas within each construct that may provide therapeutic target for harm reduction interventions. Finally, this thesis has observed patterns in AAS craving that was consistent, in part, with existing theories of drug craving and associated psychosocial constructs. This was the first study of its kind within the AAS community and revealed interesting observations of blast and cruise protocols across AAS administration. The findings identified within this thesis provide exciting and novel avenues which we hope researchers will use in order to explore and expand our current understanding of AAS dependence and its correlates.

Chapter 6 – References

- Adams, H., Cervantes, P., Jang, J., & Dixon, D. (2014). Standardized Assessment. In D. Granpeesheh, J. Tarbox, A. C. Najdowski, & J. Kornack (Eds.), *Evidence-Based Treatment for Children with Autism* (pp. 501–516). Elsevier.
<https://doi.org/10.1016/B978-0-12-411603-0.00025-2>
- Ahmadi, S., Basharpour, S., & Narimani, M. (2019). The role of sensitivity to reward and punishment and moral disengagement in the prediction of craving among people with substance dependency. *International Journal of Psychology (IPA)*, *13*(1), 40–62.
<https://doi.org/10.24200/ijpb.2018.125014.1012>
- Alexander, G. M., Packard, M. G., & Hines, M. (1994). Testosterone has rewarding affective properties in male rats: Implications for the biological basis of sexual motivation. *Behavioral Neuroscience*, *108*(2), 424–428. <https://doi.org/10.1037/0735-7044.108.2.424>
- Amaral, J. M. X., Padilha, M. C., Chagas, S. v, Baker, J. S., Mullen, C., Neto, L. v, Neto, F. R. A., & Cruz, M. S. (2020). Treatment and prevention of attempted suicide, anxiety, and aggressiveness with fluoxetine, despite proven use of androgenic anabolic steroids. *Drug Testing and Analysis*, 1–6. <https://doi.org/10.1002/dta.2912>
- American Psychiatric Association DSM-5, T. F. (2013). Diagnostic and statistical manual of mental disorders : DSM-5. In *American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 5th edition* (5th ed.). Arlington, Va: American Psychiatric Association.

- Angrist, J. D., Imbens, G. W., & Rubin, D. B. (1996). Identification of causal effects using instrumental variables. *Journal of the American Statistical Association*, *91*(434), 444–455. <https://doi.org/10.1080/01621459.1996.10476902>
- Anstey, K. J., & Hofer, S. M. (2004). Longitudinal Designs, Methods and Analysis in Psychiatric Research. *Australian & New Zealand Journal of Psychiatry*, *38*(3), 93–104. <https://doi.org/10.1080/j.1440-1614.2004.01343.x>
- Anton, R. F. (1999). What is craving? Models and implications for treatment. *Alcohol research & health: the journal of the National Institute on Alcohol Abuse and Alcoholism*, *23*(3), 165–173.
- Anton, R. F. (2000). Obsessive-compulsive aspects of craving: development of the Obsessive Compulsive Drinking Scale. *Addiction*, *95*(2), 211–217. <https://doi.org/10.1046/j.1360-0443.95.8s2.9.x>
- Anton, R. F., Moak, D. H., & Latham, P. (1995). The obsessive-compulsive drinking scale: A self-rated instrument for the quantification of thoughts about alcohol and drinking behavior. *Alcoholism, Clinical and Experimental Research*, *19*(1), 92-99. [doi:10.1111/j.1530-0277.1995.tb01475.x](https://doi.org/10.1111/j.1530-0277.1995.tb01475.x)
- Arnedo, M. T., Salvador, A., Martínez-Sanchís, S., & Pellicer, O. (2002). Similar rewarding effects of testosterone in mice rated as short and long attack latency individuals. *Addiction Biology*, *7*(4), 373–379. <https://doi.org/10.1080/1355621021000005955>
- Arvary, D., & Pope, H. G. (2000). Anabolic-Androgenic Steroids as a Gateway to Opioid Dependence. *New England Journal of Medicine*, *342*(20), 1532–1532. <https://doi.org/10.1056/NEJM200005183422018>

- Atkinson, A. M., van de Ven, K., Cunningham, M., de Zeeuw, T., Hibbert, E., Forlini, C., Barkoukis, V., & Sumnall, H. R. (2021). Performance and image enhancing drug interventions aimed at increasing knowledge among healthcare professionals (HCP): reflections on the implementation of the Dopinglinkki e-module in Europe and Australia in the HCP workforce. *International Journal of Drug Policy*, *95*, 103141. <https://doi.org/10.1016/j.drugpo.2021.103141>
- Auriacombe, M., Serre, F., Denis, C., & Fatséas, M. (2018). Diagnosis of Addictions. In H. Pickard & S. Ahmed (Eds.), *The Routledge Handbook of Philosophy and Science of Addiction* (1st ed., pp. 1–13). Routledge. <https://doi.org/10.4324/9781315689197>
- Baggish, A. L., Weiner, R. B., Kanayama, G., Hudson, J. I., Lu, M. T., Hoffmann, U., & Pope, H. G. (2017). Cardiovascular Toxicity of Illicit Anabolic-Androgenic Steroid Use. *Circulation*, *135*(21), 1991–2002. <https://doi.org/10.1161/CIRCULATIONAHA.116.026945>
- Bahrke, M. S., & Yesalis, C. E. (1994). Weight training: a potential confounding factor in examining the psychological and behavioral effects of anabolic-androgenic steroids. *Sports Medicine*, *18*, 309–318. <https://doi.org/10.2165/00007256-199418050-00003>
- Bain, J. (2008). The many faces of testosterone. *Clinical Interventions in Aging*, *Volume 2*, 567–576. <https://doi.org/10.2147/CIA.S1417>
- Baker, T. B., Morse, E., & Sherman, J. E. (1986). The motivation to use drugs: a psychobiological analysis of urges. *Nebraska Symposium on Motivation*. *Nebraska Symposium on Motivation*, *34*, 257–323.
- Baker, T. B., Morse, E., & Sherman, J. E. (1987). The motivation to use drugs: a psychobiological analysis of urges. In P. C. Rivers (Ed.), *The Nebraska Symposium on*

Motivation: Alcohol and Addictive Behavior (pp. 257–323). University of Nebraska Press.

Baker, T. B., Piper, M. E., McCarthy, D. E., Majeskie, M. R., & Fiore, M. C. (2004).

Addiction Motivation Reformulated: An Affective Processing Model of Negative Reinforcement. *Psychological Review*, *111*(1), 33–51. <https://doi.org/10.1037/0033-295X.111.1.33>

Bandura, A. (1991). *Social cognitive theory of moral thought and action* (W. M. Kurtines, J. Gewirtz, & J. L. Lamb, Eds.; 1st ed.). Psychology Press.

<https://doi.org/10.4324/9781315807294>

Bandura, A. (1997). *Self-efficacy : the exercise of control*. New York, N.Y: W.H. Freeman.

Bandura, A., Barbaranelli, C., Caprara, G. V., & Pastorelli, C. (1996). Mechanisms of Moral Disengagement in the Exercise of Moral Agency. *Journal of Personality and Social Psychology*. <https://doi.org/10.1037/0022-3514.71.2.364>

Bandura, A., Caprara, G. v, Barbaranelli, C., Pastorelli, C., & Regalia, C. (2001).

Sociocognitive self-regulatory mechanisms governing transgressive behavior. *Journal of Personality and Social Psychology*, *80*(1), 125–135. <https://doi.org/10.1037//0022-3514.80.1.125>

Barker, J., McCarthy, P., Jones, M., & Moran, A. (2011). Analysing data in single-case

research. In *Single-Case Research Methods in Sport and Exercise Psychology* (1st ed., pp. 145–166). Routledge.

- Basaria, S. (2018). Use of performance-enhancing (and image-enhancing) drugs: A growing problem in need of a solution. *Molecular and Cellular Endocrinology*, *464*, 1–3.
<https://doi.org/10.1016/j.mce.2018.02.004>
- Bates, G., Shepherd, S., & McVeigh, J. (2021). Harm reduction strategies for androgen users: providing appropriate support and improving engagement with healthcare. *Current Opinion in Endocrinology, Diabetes & Obesity*, *28*(6), 630–635.
<https://doi.org/10.1097/MED.0000000000000676>
- Bates, M., Bates, G., Begley, E., Tod, D., Jones, L., Leavey, C., & Mcveigh, J. (2017). A systematic review investigating the behaviour change strategies in interventions to prevent misuse of anabolic steroids Title: A systematic review investigating the behaviour change strategies in interventions to prevent misuse of anabolic steroids. *Journal of Health Psychology*. <http://researchonline.ljmu.ac.uk/>
- Beck, A. T., Wright, F. D., Newman, C. F., & Liese, B. (1993). *Cognitive therapy of substance abuse*. New York: Guilford Press.
- Bhasin, S., Hatfield, D. L., Hoffman, J. R., Kraemer, W. J., Labotz, M., Phillips, S. M., & Ratamess, N. A. (2021). Anabolic-Androgenic Steroid Use in Sports, Health, and Society. *Medicine & Science in Sports & Exercise*, *53*(8), 1778–1794.
<https://doi.org/10.1249/MSS.00000000000002670>
- Bjørnebekk, A., Kaufmann, T., Hauger, L. E., Klonteig, S., Hullstein, I. R., & Westlye, L. T. (2021). Long-term Anabolic–Androgenic Steroid Use Is Associated With Deviant Brain Aging. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *6*(5), 579–589. <https://doi.org/10.1016/j.bpsc.2021.01.001>

- Bjørnebekk, A., Walhovd, K. B., Jørstad, M. L., Due-Tønnessen, P., Hullstein, I. R., & Fjell, A. M. (2017). Structural Brain Imaging of Long-Term Anabolic-Androgenic Steroid Users and Nonusing Weightlifters. *Biological Psychiatry*, 82(4), 294–302.
<https://doi.org/10.1016/j.biopsych.2016.06.017>
- Boardley, I. D., & Grix, J. (2014). Doping in bodybuilders: a qualitative investigation of facilitative psychosocial processes. *Qualitative Research in Sport, Exercise and Health*, 6(3), 422–439. <https://doi.org/10.1080/2159676X.2013.766809>
- Boardley, I. D., Grix, J., & Dewar, A. J. (2014). Moral disengagement and associated processes in performance-enhancing drug use: a national qualitative investigation. *Journal of Sports Sciences*, 32(9), 836–844.
<https://doi.org/10.1080/02640414.2013.862842>
- Boardley, I. D., Grix, J., & Harkin, J. (2015). Doping in team and individual sports: a qualitative investigation of moral disengagement and associated processes. *Qualitative Research in Sport, Exercise and Health*, 7(5), 698–717.
<https://doi.org/10.1080/2159676X.2014.992039>
- Boardley, I. D., Smith, A. L., Mills, J. P., Grix, J., & Wynne, C. (2017). Empathic and Self-Regulatory Processes Governing Doping Behavior. *Frontiers in Psychology*, 8.
<https://doi.org/10.3389/fpsyg.2017.01495>
- Boardley, I. D., Smith, A. L., Mills, J., Grix, J., Wynne, C., & Wilkins, L. (2018). Development of moral disengagement and self-regulatory efficacy assessments relevant to doping in sport and exercise. *Psychology of Sport and Exercise*, 36, 57–70.
<https://doi.org/10.1016/j.psychsport.2018.01.007>

- Bolding, G., Sherr, L., & Elford, J. (2002). Use of anabolic steroids and associated health risks among gay men attending London gyms. *Addiction*, *97*(2), 195–203.
<https://doi.org/10.1046/j.1360-0443.2002.00031.x>
- Bonnecaze, A. K., O'Connor, T., & Aloï, J. A. (2020). Characteristics and Attitudes of Men Using Anabolic Androgenic Steroids (AAS): A Survey of 2385 Men. *American Journal of Men's Health*, *14*(6), 155798832096653. <https://doi.org/10.1177/1557988320966536>
- Bonnecaze, A. K., O'Connor, T., & Burns, C. A. (2021). Harm Reduction in Male Patients Actively Using Anabolic Androgenic Steroids (AAS) and Performance-Enhancing Drugs (PEDs): a Review. *Journal of General Internal Medicine*, *36*(7), 2055–2064.
<https://doi.org/10.1007/s11606-021-06751-3>
- Boregowda, K., Joels, L., Stephens, J. W., & Price, D. E. (2011). Persistent primary hypogonadism associated with anabolic steroid abuse. *Fertility and Sterility*, *96*(1), e7–e8. <https://doi.org/10.1016/j.fertnstert.2011.04.029>
- Brower, K. (1992). Addictive potential of anabolic steroids. *Psychiatric Annals*, *22*, 30–34.
- Brower, K. (2002). Anabolic steroid abuse and dependence. *Current Psychiatry Reports*, *4*(5), 377–387. <https://doi.org/10.1007/s11920-002-0086-6>
- Brower, K. J. (1992). Anabolic Steroids: Addictive, Psychiatric, and Medical Consequences. *American Journal on Addictions*, *1*(2), 100–114.
<https://doi.org/10.3109/10550499209004011>
- Brower, K. J. (1997). Withdrawal from anabolic steroids. *Current Therapy in Endocrinology and Metabolism*, *6*, 338–343.

- Brower, K. J. (2009). Anabolic Steroid Abuse and Dependence in Clinical Practice. *The Physician and Sportsmedicine*, 37(4), 131–140.
<https://doi.org/10.3810/psm.2009.12.1751>
- Brower, K. J., Blow, F. C., Beresford, T. P., & Fuelling, C. (1989). Anabolic-androgenic steroid dependence. *The Journal of Clinical Psychiatry*, 50, 31–33.
- Brower, K. J., Blow, F. C., Young, J. P., & Hill, E. M. (1991). Symptoms and correlates of anabolic-androgenic steroid dependence. *Addiction*, 86(6), 759–768.
<https://doi.org/10.1111/j.1360-0443.1991.tb03101.x>
- Byrne, B. M. (2012). *Structural Equation Modeling with Mplus: Basic Concepts, Applications, and Programming*. Routledge.
- Chandler, M., & McVeigh, J. (2014). Steroids and Image Enhancing Drugs 2013 Survey Results. In *Liverpool John Moores University*. Centre for Public Health.
<https://www.ljmu.ac.uk/~media/phi-reports/pdf/2014-06-steroids-and-image-enhancing-drugs-2013-survey-results.pdf>
- Chavarria, J., Stevens, E. B., Jason, L. A., & Ferrari, J. R. (2012). The Effects of Self-Regulation and Self-Efficacy on Substance Use Abstinence. *Alcoholism Treatment Quarterly*, 30(4), 422–432. <https://doi.org/10.1080/07347324.2012.718960>
- Chegeni, R., Notelaers, G., Pallesen, S., & Sagoe, D. (2021). Aggression and Psychological Distress in Male and Female Anabolic-Androgenic Steroid Users: A Multigroup Latent Class Analysis. *Frontiers in Psychiatry*, 12. <https://doi.org/10.3389/fpsy.2021.629428>
- Chester, N. (2018). Anabolic agents. In D. R. Mottram & N. Chester (Eds.), *Drugs in Sport* (7th ed., pp. 129–150). Routledge.

- Chiang, S.-C., Knapp, K. S., Bai, S., Cleveland, H. H., & Harris, K. S. (2023). Examining within- and between-person facets of negative affect and associations with daily craving among young adults in substance use disorder recovery. *Addiction Research & Theory*, 31(1), 52–59. <https://doi.org/10.1080/16066359.2022.2102611>
- Christiansen, A. V., Vinther, A. S., & Liokaftos, D. (2017). Outline of a typology of men's use of anabolic androgenic steroids in fitness and strength training environments*. *Drugs: Education, Prevention and Policy*, 24(3), 295–305. <https://doi.org/10.1080/09687637.2016.1231173>
- Christou, M. A., Christou, P. A., Markozannes, G., Tsatsoulis, A., Mastorakos, G., & Tigas, S. (2017). Effects of anabolic androgenic steroids on the reproductive system of athletes and recreational users: A systematic review and meta-analysis. *Sports Medicine (Auckland)*, 47(9), 1869–1883. <https://doi.org/10.1007/s40279-017-0709-z>
- Clark, L. A., & Watson, D. (1995). Constructing validity: Basic issues in objective scale development. *Psychological Assessment*, 7(3), 309-319. doi:10.1037/1040-3590.7.3.309
- Clark, L. A., & Watson, D. (2019). Constructing validity: New developments in creating objective measuring instruments. *Psychological Assessment*, 31(12), 1412–1427. <https://doi.org/10.1037/pas0000626>
- Cohen, J. (1992). Statistical Power Analysis. *Current Directions in Psychological Science*, 1(3), 98–101. <https://doi.org/10.1111/1467-8721.ep10768783>
- Cohen, J., Collins, R., Darkes, J., & Gwartney, D. (2007). A league of their own: demographics, motivations and patterns of use of 1,955 male adult non-medical anabolic steroid users in the United States. *Journal of the International Society of Sports Nutrition*, 4(1). <https://doi.org/10.1186/1550-2783-4-12>

- Cole, J. C., Smith, R., Halford, J. C. G., & Wagstaff, G. F. (2003). A preliminary investigation into the relationship between anabolic-androgenic steroid use and the symptoms of reverse anorexia in both current and ex-users. *Psychopharmacology*, *166*(4), 424–429. <https://doi.org/10.1007/s00213-002-1352-3>
- Copeland, J., Peters, R., & Dillon, P. (2000). Anabolic-androgenic steroid use disorders among a sample of Australian competitive and recreational users. *Drug and Alcohol Dependence*, *60*, 91–96. [https://doi.org/10.1016/S0376-8716\(00\)80011-3](https://doi.org/10.1016/S0376-8716(00)80011-3)
- Corona, G., Rastrelli, G., Marchiani, S., Filippi, S., Morelli, A., Sarchielli, E., Sforza, A., Vignozzi, L., & Maggi, M. (2022). Consequences of Anabolic-Androgenic Steroid Abuse in Males; Sexual and Reproductive Perspective. *The World Journal of Men's Health*, *40*(2), 165. <https://doi.org/10.5534/wjmh.210021>
- Cox, L. S., Tiffany, S. T., & Christen, A. G. (2001). Evaluation of the brief questionnaire of smoking urges (QSU-brief) in laboratory and clinical settings. *Nicotine & Tobacco Research*, *3*(1), 7–16. <https://doi.org/10.1080/14622200020032051>
- D'Andrea, A., Radmilovic, J., Russo, V., Sperlongano, S., Carbone, A., di Maio, M., Ilardi, F., Riegler, L., D'Alto, M., Giallauria, F., Bossone, E., & Picano, E. (2022). Biventricular dysfunction and lung congestion in athletes on anabolic androgenic steroids: a speckle tracking and stress lung echocardiography analysis. *European Journal of Preventive Cardiology*, *28*(17), 1928–1938. <https://doi.org/10.1093/eurjpc/zwab086>
- Dahl, A. (2017). Ecological Commitments: Why Developmental Science Needs Naturalistic Methods. *Child Development Perspectives*, *11*(2), 79–84. <https://doi.org/10.1111/cdep.12217>

- de Beun, R., Jansen, E., Slangen, J. L., & van de Poll, N. E. (1992). Testosterone as appetitive and discriminative stimulus in rats: Sex- and dose-dependent effects. *Physiology & Behavior*, *52*(4), 629–634. [https://doi.org/10.1016/0031-9384\(92\)90389-J](https://doi.org/10.1016/0031-9384(92)90389-J)
- de Souza, G. L., & Hallak, J. (2011). Anabolic steroids and male infertility: a comprehensive review. *BJU International*, *108*(11), 1860–1865. <https://doi.org/10.1111/j.1464-410X.2011.10131.x>
- de Zeeuw, T. I., Brunt, T. M., van Amsterdam, J., van de Ven, K., & van den Brink, W. (2023). Anabolic Androgenic Steroid Use Patterns and Steroid Use Disorders in a Sample of Male Gym Visitors. *European Addiction Research*, 1–10. <https://doi.org/10.1159/000528256>
- Doleeb, S., Kratz, A., Salter, M., & Thohan, V. (2019). Strong muscles, weak heart: testosterone-induced cardiomyopathy. *ESC Heart Failure*, *6*(5), 1000–1004. <https://doi.org/10.1002/ehf2.12494>
- Donny, E., Griffin, K., Shiffman, S., & Sayette, M. (2008). The relationship between cigarette use, nicotine dependence, and craving in laboratory volunteers. *Nicotine & Tobacco Research*, *10*(3), 447–455. <https://doi.org/10.1080/14622200801901906>
- Drummond, D. C. (2001). Theories of drug craving, ancient and modern. *Addiction*, *96*, 33–46. <https://doi.org/10.1080/09652140020016941>
- Drummond, D. C., & Glautier, S. (1995). A controlled trial of cue exposure treatment in alcohol dependence. *Journal of Consulting and Clinical Psychology*, *62*, 800–817. [doi:10.1037//0022-006x.62.4.809](https://doi.org/10.1037//0022-006x.62.4.809)

- Drummond, D. C., Cooper, T., & Glautier, S. P. (1990). Conditioned learning in alcohol dependence: implications for cue exposure treatment. *British Journal of Addiction*, 85. <https://doi.org/10.1111/j.1360-0443.1990.tb01685.x>.
- Drummond, D. C., Litten, R. Z., Lowman, C., & Hunt, W. A. (2000). Craving research: future directions. *Addiction*, 95(8s2), 247–255. <https://doi.org/10.1046/j.1360-0443.95.8s2.13.x>
- Drummond, D. C., Tiffany, S. T., Glautier, S., & Remington, B. (1995). Cue exposure in understanding and treating addictive behaviours. In D. C. Drummond, S. T. Tiffany, S. Glautier, & B. Remington (Eds.), *Addictive behaviour: Cue exposure theory and practice* (pp. 1–17). John Wiley & Sons.
- Dunn, J. G. H., Bouffard, M., & Rogers, W. T. (1999). Assessing item content-relevance in sport psychology scale-construction research: Issues and recommendations. *Measurement in Physical Education and Exercise Science*, 3(1), 15–36. https://doi.org/10.1207/s15327841mpee0301_2
- Dziuban, C. D., & Shirkey, E. C. (1974). When is a correlation matrix appropriate for factor analysis? Some decision rules. *Psychological Bulletin*, 81(6), 358-361. [doi:10.1037/h0036316](https://doi.org/10.1037/h0036316)
- Eby, D. W. (2011). Naturalistic Observational Field Techniques for Traffic Psychology Research. In *Handbook of Traffic Psychology* (pp. 61–72). Elsevier. <https://doi.org/10.1016/B978-0-12-381984-0.10005-0>
- Evans, N. A. (1997). Gym and tonic: A profile of 100 male steroid users. *British Journal of Sports Medicine*, 31(1), 54-58. [doi:10.1136/bjism.31.1.54](https://doi.org/10.1136/bjism.31.1.54)

- Evans-Brown, M., Kimergård, A., & McVeigh, J. (2009). Elephant in the room? The methodological implications for public health research of performance-enhancing drugs derived from the illicit market. *Drug Testing and Analysis*, *1*(7), 323–326.
<https://doi.org/10.1002/dta.74>
- Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological Methods*, *4*, 272–299. doi:10.1037//1082-989X.4.3.272
- Flannery, B. A., Roberts, A. J., Cooney, N., Swift, R. M., Anton, R. F., & Rohsenow, D. J. (2001). The Role of Craving in Alcohol Use, Dependence, and Treatment. *Alcoholism: Clinical and Experimental Research*, *25*(2), 299–308. [https://doi.org/0145-6008/01/2502-0299\\$03.00/0](https://doi.org/0145-6008/01/2502-0299$03.00/0)
- Flannery, J. S., Riedel, M. C., Poudel, R., Laird, A. R., Ross, T. J., Salmeron, B. J., Stein, E. A., & Sutherland, M. T. (2019). Habenular and striatal activity during performance feedback are differentially linked with state-like and trait-like aspects of tobacco use disorder. *Science Advances*, *5*(10). <https://doi.org/10.1126/sciadv.aax2084>
- Franken, I. H. A. (2003). Drug craving and addiction: Integrating psychological and neuropsychopharmacological approaches. In *Progress in Neuro-Psychopharmacology and Biological Psychiatry* (Vol. 27, Issue 4, pp. 563–579). Elsevier Inc.
[https://doi.org/10.1016/S0278-5846\(03\)00081-2](https://doi.org/10.1016/S0278-5846(03)00081-2)
- Franken, I. H. A., Hendriks, V. M., & van den Brink, W. (2002). Initial validation of two opiate craving questionnaires. *Addictive Behaviors*, *27*(5), 675–685.
[https://doi.org/10.1016/S0306-4603\(01\)00201-5](https://doi.org/10.1016/S0306-4603(01)00201-5)

- Frude, E., McKay, F. H., & Dunn, M. (2020). A focused netnographic study exploring experiences associated with counterfeit and contaminated anabolic-androgenic steroids. *Harm Reduction Journal*, *17*(1), 42. <https://doi.org/10.1186/s12954-020-00387-y>
- Gast, D. L., & Hammond, D. (2012). Single subject research methodology in behavioral sciences. In D. L. Gast (Ed.), *Withdrawal and reversal designs* (pp. 234–275). Routledge.
- Gast, D. L., & Spriggs, A. D. (2014). Visual Analysis of Graphic Data. In D. L. Gast & J. R. Ledford (Eds.), *Single Case Research Methodology* (2nd ed., pp. 176–210). Routledge.
- Geiser, C., Götz, T., Preckel, F., & Freund, P. A. (2017). States and Traits. *European Journal of Psychological Assessment*, *33*(4), 219–223. <https://doi.org/10.1027/1015-5759/a000413>
- Gillespie, N. A., Neale, M. C., Prescott, C. A., Aggen, S. H., & Kendler, K. S. (2007). Factor and item-response analysis DSM-IV criteria for abuse of and dependence on cannabis, cocaine, hallucinogens, sedatives, stimulants and opioids. *Addiction*, *102*(6), 920–930. <https://doi.org/10.1111/j.1360-0443.2007.01804.x>
- Goldman, A., & Basaria, S. (2018). Adverse health effects of androgen use. *Molecular and Cellular Endocrinology*, *464*, 46–55. <https://doi.org/10.1016/j.mce.2017.06.009>
- Gossop, M., Darke, S., Griffiths, P., Hando, J., Powis, B., Hall, W., & Strang, J. (1995). The Severity of Dependence Scale (SDS): psychometric properties of the SDS in English and Australian samples of heroin, cocaine and amphetamine users. *Addiction (Abingdon, England)*, *90*(5), 607–614. <https://doi.org/10.1046/j.1360-0443.1995.9056072.x>

- Graham, M. R., Davies, B., Grace, F. M., Kicman, A., & Baker, J. S. (2008). Anabolic Steroid Use. *Sports Medicine*, 38(6), 505–525. <https://doi.org/10.2165/00007256-200838060-00005>
- Grant, B. F., Harford, T. C., Muthén, B. O., Yi, H., Hasin, D. S., & Stinson, F. S. (2007). DSM-IV alcohol dependence and abuse: Further evidence of validity in the general population. *Drug and Alcohol Dependence*, 86(2–3), 154–166. <https://doi.org/10.1016/j.drugalcdep.2006.05.019>
- Greenway, C. W., & Price, C. (2018). A qualitative study of the motivations for anabolic-androgenic steroid use: The role of muscle dysmorphia and self-esteem in long-term users. *Performance Enhancement and Health*, 6(1), 12–20. <https://doi.org/10.1016/j.peh.2018.02.002>
- Gridley DW, & Hanrahan SJ. (1994). Anabolic-androgenic steroid use among male gymnasium participants: knowledge and motives. *Sports Health*, 12, 11–14. <https://doi.org/10.1016/j.jsps.2018.11.007>
- Griffiths, S., Jacka, B., Degenhardt, L., Murray, S. B., & Larance, B. (2018). Physical appearance concerns are uniquely associated with the severity of steroid dependence and depression in anabolic–androgenic steroid users. *Drug and Alcohol Review*, 37(5), 664–670. <https://doi.org/10.1111/dar.12688>
- Grönbladh, A., Nylander, E., & Hallberg, M. (2016). The neurobiology and addiction potential of anabolic androgenic steroids and the effects of growth hormone. In *Brain Research Bulletin* (Vol. 126, pp. 127–137). Elsevier Inc. <https://doi.org/10.1016/j.brainresbull.2016.05.003>

- Gunnarsson, B., Entezarjou, A., Fernández-Aranda, F., Jiménez-Murcia, S., Kenttä, G., & Håkansson, A. (2022). Gunnerson er al., 2022 - Understanding exercise addiction characteristics as AAS use. *Frontiers in Sports and Active Living*, 1–9. <https://doi.org/10.3389/fspor.2022.903777>
- Gwaltney, C. J., Shiffman, S., Normal, G. J., Paty, J. A., Kassel, J. D., Gnys, M., Hickcox, M., Waters, A., & Balabanis, M. (2001). Does smoking abstinence self-efficacy vary across situations? Identifying context-specificity within the Relapse Situation Efficacy Questionnaire. *Journal of Consulting and Clinical Psychology*, 69(3), 516–527. <https://doi.org/10.1037/0022-006X.69.3.516>
- Hair, J. F., Anderson, R. E., Tatham, R. L., & Black, W. C. (1998). *Multivariate data analysis* (Fifth ed.). Upper Saddle River, NJ: Prentice Hall.
- Hall, R. C. W., Hall, R. C. W., & Chapman, M. J. (2005). Psychiatric complications of anabolic steroid abuse. *Psychosomatics*, 46(4), 285-290. doi:10.1176/appi.psy.46.4.285
- Hambleton, R. K. (1980). Test score validity and standard-setting method. In R. A. Berk (Ed.), *Criterion-referenced measurement: State of the art* (pp. 80–123). Johns Hopkins University Press.
- Harmer, P. A. (2010). Anabolic-androgenic steroid use among young male and female athletes: is the game to blame? *British Journal of Sports Medicine*, 44(1), 26–31. <https://doi.org/10.1136/bjism.2009.068924>
- Hartgens, F., Rietjens, G., Keizer, H. A., Kuipers, H., & Wolffenbuttel, B. H. R. (2004). Effects of androgenic-anabolic steroids on apolipoproteins and lipoprotein (a). *British Journal of Sports Medicine*, 38(3), 253–259. <https://doi.org/10.1136/bjism.2003.000199>

Hartman, N., Caskey, N. H., Olmstead, R. E., & Jarvik, M. E. (1998). Nicotine Craving and Psychiatric Diagnosis: Past, Present, and Future. *Psychiatric Annals*, *28*(10), 547–551. <https://doi.org/10.3928/0048-5713-19981001-05>

Hauger, L. E., Havnes, I. A., Jørstad, M. L., & Bjørnebekk, A. (2021). Anabolic androgenic steroids, antisocial personality traits, aggression and violence. *Drug and Alcohol Dependence*, *221*, 108604. <https://doi.org/10.1016/j.drugalcdep.2021.108604>

Hauger, L. E., Westlye, L. T., & Bjørnebekk, A. (2020). Anabolic androgenic steroid dependence is associated with executive dysfunction. *Drug and Alcohol Dependence*, *208*, 107874. <https://doi.org/10.1016/j.drugalcdep.2020.107874>

Hauger, L. E., Westlye, L. T., Fjell, A. M., Walhovd, K. B., & Bjørnebekk, A. (2019). Structural brain characteristics of anabolic–androgenic steroid dependence in men. *Addiction*, *114*(8), 1405–1415. <https://doi.org/10.1111/add.14629>

Havnes, I. A., Jørstad, M. L., & Wisløff, C. (2019). Anabolic-androgenic steroid users receiving health-related information; health problems, motivations to quit and treatment desires. *Substance Abuse Treatment, Prevention, and Policy*, *14*(1), 20. <https://doi.org/10.1186/s13011-019-0206-5>

Hayes, A. F. (2009). Beyond Baron and Kenny: Statistical Mediation Analysis in the New Millennium. *Communication Monographs*, *76*(4), 408–420. <https://doi.org/10.1080/03637750903310360>

Hayes, A. F. (2017). *Introduction to Mediation, Moderation, and Conditional Process Analysis, Second Edition: A Regression-Based Approach* (3rd ed.). Guilford Publications.

- Haynes, S. N., Richard, D. C., & Kubany, E. S. (1995). Content validity in psychological assessment: A functional approach to concepts and methods. *Psychological Assessment, 7*, 238-247. doi:10.1037/1040-3590.7.3.238.
- Heyes, A. R., & Boardley, I. D. (2019). Psychosocial factors facilitating use of cognitive enhancing drugs in education: a qualitative investigation of moral disengagement and associated processes. *Drugs: Education, Prevention and Policy, 26*(4), 329–338.
<https://doi.org/10.1080/09687637.2019.1586831>
- Hildebrandt, T., Yehuda, R., & Alfano, L. (2011). What can allostasis tell us about anabolic–androgenic steroid addiction? *Development and Psychopathology, 23*(1), 907–919.
<https://doi.org/10.1017/S0954579411000393>
- Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modelling, 9*, 1-55.
doi:10.1080/10705519909540118
- Huhn, A. S., Harris, J., Cleveland, H. H., Lydon, D. M., Stankoski, D., Cleveland, M. J., Deneke, E., & Bunce, S. C. (2016). Ecological momentary assessment of affect and craving in patients in treatment for prescription opioid dependence. *Brain Research Bulletin, 123*, 94–101. <https://doi.org/10.1016/j.brainresbull.2016.01.012>
- Hurst, R., Hale, B., Smith, D., & Collins, D. (2000). Exercise dependence, social physique anxiety, and social support in experienced and inexperienced bodybuilders and weightlifters. *British Journal of Sports Medicine, 34*, 431–435.
<https://doi.org/10.1136/bjism.34.6.431>

- Ip, E. J., Barnett, M. J., Tenerowicz, M. J., & Perry, P. J. (2011). The anabolic 500 survey: Characteristics of male users versus nonusers of anabolic-androgenic steroids for strength training. *Pharmacotherapy*, *31*(8), 757-766. doi:10.1592/phco.31.8.757
- Ip, E. J., Lu, D. H., Barnett, M. J., Tenerowicz, M. J., Vo, J. C., & Perry, P. J. (2012). Psychological and Physical Impact of Anabolic-Androgenic Steroid Dependence. *Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy*, *32*(10), 910–919. <https://doi.org/10.1002/j.1875-9114.2012.01123>
- Jacka, B., Peacock, A., Degenhardt, L., Bruno, R., Clare, P., Kemp, R., Dev, A., & Larance, B. (2017). Trends in PIEDs use among male clients of needle–syringe programs in Queensland, Australia; 2007–2015. *International Journal of Drug Policy*, *46*, 74–78. <https://doi.org/10.1016/j.drugpo.2017.05.048>
- Jöreskog, K. G., & Sörbom, D. (1993). *LISREL8: User's Reference Guide*. Scientific Software International
- Kaiser, H. F. (1974). An index of factorial simplicity. *Psychometrika*, *39*, 31-36. doi:10.1007/BF02291575
- Kakko, J., Alho, H., Baldacchino, A., Molina, R., Nava, F. A., & Shaya, G. (2019). Craving in opioid use disorder: From neurobiology to clinical practice. In *Frontiers in Psychiatry* (Vol. 10, Issue AUG). Frontiers Media S.A. <https://doi.org/10.3389/fpsyt.2019.00592>
- Kanayama, G., & Pope, H. G. (2018). History and epidemiology of anabolic androgens in athletes and non-athletes. *Molecular and Cellular Endocrinology*, *464*, 4–13. <https://doi.org/10.1016/j.mce.2017.02.039>

- Kanayama, G., Brower, K. J., Wood, R. I., Hudson, J. I., & Pope, H. G. (2009c). Issues for DSM-V: Clarifying the diagnostic criteria for anabolic-androgenic steroid dependence. In *American Journal of Psychiatry* (Vol. 166, Issue 6, pp. 642–644).
<https://doi.org/10.1176/appi.ajp.2009.08111699>
- Kanayama, G., Brower, K. J., Wood, R. I., Hudson, J. I., & Pope, H. G. (2010a). Treatment of anabolic-androgenic steroid dependence: Emerging evidence and its implications. In *Drug and Alcohol Dependence* (Vol. 109, Issues 1–3, pp. 6–13).
<https://doi.org/10.1016/j.drugalcdep.2010.01.011>
- Kanayama, G., Brower, K. J., Wood, R. I., Hudson, J. I., & Pope, H. G. (2009a). Anabolic-Androgenic Steroid Dependence: An Emerging Disorder. *Addiction*, *104*(12), 1966–1978. <https://doi.org/10.1111/j.1360-0443.2009.02734.x>.
- Kanayama, G., Hudson, J. I., & Pope, H. G. (2008). Long-term psychiatric and medical consequences of anabolic-androgenic steroid abuse: A looming public health concern? In *Drug and Alcohol Dependence*. <https://doi.org/10.1016/j.drugalcdep.2008.05.004>
- Kanayama, G., Hudson, J. I., & Pope, H. G. (2009b). Features of men with anabolic-androgenic steroid dependence: A comparison with nondependent AAS users and with AAS nonusers. *Drug and Alcohol Dependence*, *102*(1), 130–137.
<https://doi.org/10.1016/j.drugalcdep.2009.02.008>
- Kanayama, G., Hudson, J. I., & Pope, H. G. (2010b). Illicit anabolic–androgenic steroid use. *Hormones and Behavior*, *58*(1), 111–121. <https://doi.org/10.1016/j.yhbeh.2009.09.006>
- Kanayama, G., Hudson, J. I., & Pope, Jr., H. G. (2020). Anabolic-Androgenic Steroid Use and Body Image in Men: A Growing Concern for Clinicians. *Psychotherapy and Psychosomatics*, *89*(2), 65–73. <https://doi.org/10.1159/000505978>

- Kanayama, G., Hudson, J. I., DeLuca, J., Isaacs, S., Baggish, A., Weiner, R., Bhasin, S., & Pope, H. G. (2015). Prolonged hypogonadism in males following withdrawal from anabolic-androgenic steroids: an under-recognized problem. *Addiction, 110*(5), 823–831. <https://doi.org/10.1111/add.12850>
- Kanayama, G., Kaufman, M. J., & Pope Harrison G, J. (2018). Public health impact of androgens. *Current Opinion in Endocrinology, Diabetes, and Obesity, 25*(3), 218–223. <https://doi.org/10.1097/MED.0000000000000404>
- Kanayama, G., Pope, H. G., Cohane, G., & Hudson, J. I. (2003). Risk factors for anabolic-androgenic steroid use among weightlifters: a case–control study. *Drug and Alcohol Dependence, 71*(1), 77–86. [https://doi.org/10.1016/S0376-8716\(03\)00069-3](https://doi.org/10.1016/S0376-8716(03)00069-3)
- Kashkin, K. B., & Kleber, H. D. (1989). Hooked on Hormones? An Anabolic Steroid Addiction Hypothesis. *Journal of the American Medical Association, 262*(22), 3166–3170. <https://doi.org/10.1001/jama.262.22.3166>.
- Kaufman, M. J., Janes, A. C., Hudson, J. I., Brennan, B. P., Kanayama, G., Kerrigan, A. R., Jensen, J. E., & Pope, H. G. (2015). Brain and cognition abnormalities in long-term anabolic-androgenic steroid users. *Drug and Alcohol Dependence, 152*, 47–56. <https://doi.org/10.1016/j.drugalcdep.2015.04.023>
- Kaufman, M. J., Kanayama, G., Hudson, J. I., & Pope, H. G. (2019). Supraphysiologic-dose anabolic–androgenic steroid use: A risk factor for dementia? *Neuroscience & Biobehavioral Reviews, 100*, 180–207. <https://doi.org/10.1016/j.neubiorev.2019.02.014>
- Kean, H. (2003). Anabolic steroids and dependence. *Contemporary Drug Problems, 30*(3), 541–562.

- Kennedy, C. H. (2005). *Single-case designs for educational research*. Pearson.
- Kimergård, A. (2015). A qualitative study of anabolic steroid use amongst gym users in the United Kingdom: motives, beliefs and experiences. *Journal of Substance Use*, 20(4), 288–294. <https://doi.org/10.3109/14659891.2014.911977>
- Kleinjan, M., van den Eijnden, R. J. J. M., & Engels, R. C. M. E. (2009). Adolescents' rationalizations to continue smoking: The role of disengagement beliefs and nicotine dependence in smoking cessation. *Addictive Behaviors*, 34(5), 440–445. <https://doi.org/10.1016/j.addbeh.2008.12.010>
- Kline, R. (2005). *Principles and practice of structural equation modelling* (Second ed.). New York: The Guilford Press.
- Koob, G. F. (2006). The neurobiology of addiction: a neuroadaptational view relevant for diagnosis. *Addiction*, 101, 23–30. <https://doi.org/10.1111/j.1360-0443.2006.01586.x>
- Koob, G. F., & le Moal, M. (2005). Plasticity of reward neurocircuitry and the “dark side” of drug addiction. *Nature Neuroscience*, 8(11), 1442–1444. <https://doi.org/10.1038/nn1105-1442>
- Koob, G. F., & le Moal, M. (2008). Addiction and the brain antireward system. *Annual Review of Psychology*, 59(1), 29–53. <https://doi.org/10.1146/annurev.psych.59.103006.093548>
- Koob, G. F., & Moal, M. le. (1997). Drug Abuse: Hedonic Homeostatic Dysregulation. *Science*, 278(5335), 52–58. <https://doi.org/10.1126/science.278.5335.52>
- Koob, G. F., & Volkow, N. D. (2016). Neurobiology of addiction: a neurocircuitry analysis. *The Lancet Psychiatry*, 3(8), 760–773. [https://doi.org/10.1016/S2215-0366\(16\)00104-8](https://doi.org/10.1016/S2215-0366(16)00104-8)

- Kotzé, J., & Antonopoulos, G. A. (2021). Boosting bodily capital: Maintaining masculinity, aesthetic pleasure and instrumental utility through the consumption of steroids. *Journal of Consumer Culture*, 21(3), 683–700. <https://doi.org/10.1177/1469540519846196>
- Koval, P., Hinton, J., Dozo, N., Gleeson, J., Alvarez, M., Harrison, A., Vu, D., Susanto, R., Jayaputera, G., & Sinnott, R. (2019). *SEMA3: Smartphone Ecological Momentary Assessment, Version 3*. [Computer Software]. <http://www.sema3.com>
- Kozlowski, L. T., & Wilkinson, D. A. (1987). Use and Misuse of the Concept of Craving by Alcohol, Tobacco, and Drug Researchers. *Addiction*, 82(1), 31–36. <https://doi.org/10.1111/j.1360-0443.1987.tb01430.x>
- Kozlowski, L. T., & Wilkinson, D. A. (1987). Use and misuse of the concept of craving by alcohol, tobacco, and drug researchers. *British Journal of Addiction*, 82, 31-36. [doi:10.1111/j.1360-0443.1987.tb01430.x](https://doi.org/10.1111/j.1360-0443.1987.tb01430.x)
- Kutscher, E. C., Lund, B. C., & Perry, P. J. (2002). Anabolic Steroids: A Review for the Clinician. *Sports Medicine*, 32, 285–296. <https://doi.org/10.2165/00007256-200232050-00001>
- Lane, J. D., & Gast, D. L. (2014). Visual analysis in single case experimental design studies: Brief review and guidelines. *Neuropsychological Rehabilitation*, 24(3–4), 445–463. <https://doi.org/10.1080/09602011.2013.815636>
- Lau, H., Khosrawipour, V., Kocbach, P., Mikolajczyk, A., Schubert, J., Bania, J., & Khosrawipour, T. (2020). The positive impact of lockdown in Wuhan on containing the COVID-19 outbreak in China. *Journal of Travel Medicine*, 27(3). <https://doi.org/10.1093/jtm/taaa037>

- Lawrinson, P., Copeland, J., Gerber, S., & Gilmour, S. (2007). Determining a cut-off on the Severity of Dependence Scale (SDS) for alcohol dependence. *Addictive Behaviours*, *32*, 1474–1479. <https://doi.org/10.1016/j.addbeh.2006.09.005>
- Ledford, J. R., Lane, J. D., & Severini, K. E. (2018). Systematic Use of Visual Analysis for Assessing Outcomes in Single Case Design Studies. *Brain Impairment*, *19*(1), 4–17. <https://doi.org/10.1017/BrImp.2017.16>
- Lim, M. A. (2021). Exercise addiction and COVID-19-associated restrictions. *Journal of Mental Health*, *30*(2), 135–137. <https://doi.org/10.1080/09638237.2020.1803234>
- Little, R. J. A. (1988). A Test of Missing Completely at Random for Multivariate Data with Missing Values. *Journal of the American Statistical Association*, *83*(404), 1198–1202. <https://doi.org/10.1080/01621459.1988.10478722>
- Llewellyn, W., O'Connor, T., Touliatos, G., Koert, W., & Shelley, J. (2017). *William Llewellyn's Anabolics* (11th ed.). Molecular Nutrition.
- Ludwig, A. M. (1974). The First Drink. *Archives of General Psychiatry*, *30*(4), 539. <https://doi.org/10.1001/archpsyc.1974.01760100093015>
- Lydon-Staley, D. M., Cleveland, H. H., Huhn, A. S., Cleveland, M. J., Harris, J., Stankoski, D., Deneke, E., Meyer, R. E., & Bunce, S. C. (2017). Daily sleep quality affects drug craving, partially through indirect associations with positive affect, in patients in treatment for nonmedical use of prescription drugs. *Addictive Behaviors*, *65*(1), 275–282. <https://doi.org/10.1016/j.addbeh.2016.08.026>
- Lynskey, M. T., & Agrawal, A. (2007). Psychometric properties of DSM assessments of illicit drug abuse and dependence: results from the National Epidemiologic Survey on Alcohol

and Related Conditions (NESARC). *Psychological Medicine*, 37(9), 1345–1355.

<https://doi.org/10.1017/S0033291707000396>

Maas, C. J. M., & Hox, J. J. (2005). Sufficient Sample Sizes for Multilevel Modeling.

Methodology, 1(3), 86–92. <https://doi.org/10.1027/1614-2241.1.3.86>

Malone, D. A., & Dimeff, R. J. (1992). The use of fluoxetine in depression associated with anabolic steroid withdrawal: a case series. *The Journal of Clinical Psychiatry*, 53(4), 130–132.

Maravelias, C., Dona, A., Stefanidou, M., & Spiliopoulou, C. (2005). Adverse effects of anabolic steroids in athletes. *Toxicology Letters*, 158(3), 167–175.

<https://doi.org/10.1016/j.toxlet.2005.06.005>

Marlatt, G. A. (1985). Cognitive factors in the relapse process. In G. A. Marlatt & J. R.

Gordon (Eds.), *Relapse Prevention: maintenance strategies in the treatment of addictive behaviors* (pp. 128–200). Guilford Press.

Marlatt, G. A., & Gordon, J. R. (1985). *Relapse Prevention: Maintenance Strategies in the Treatment of Addictive Behaviors*. Guilford.

Marsh, H. W. (1993). The multidimensional structure of physical fitness: Invariance over gender and age. *Research Quarterly for Exercise and Sport*, 64, 256–273.

doi:10.1080/02701367.1993.10608810

Martin, G., Copeland, J., Gates, P., & Gilmour, S. (2006). The Severity of Dependence Scale (SDS) in an adolescent population of cannabis users: Reliability, validity and diagnostic cut-off. *Drug and Alcohol Dependence*, 83, 90–93.

<https://doi.org/10.1016/j.drugalcdep.2005.10.014>

- May, J., Andrade, J., Kavanagh, D. J., Feeney, G. F. X., Gullo, M. J., Statham, D. J., Skorka-Brown, J., Connolly, J. M., Cassimatis, M., Young, R. M., & Connor, J. P. (2014). The Craving Experience Questionnaire: A brief, theory-based measure of consummatory desire and craving. *Addiction, 109*(5), 728–735. <https://doi.org/10.1111/add.12472>
- McCullough, D., Webb, R., Enright, K. J., Lane, K. E., McVeigh, J., Stewart, C. E., & Davies, I. G. (2021). How the love of muscle can break a heart: Impact of anabolic androgenic steroids on skeletal muscle hypertrophy, metabolic and cardiovascular health. *Reviews in Endocrine and Metabolic Disorders, 22*(2), 389–405. <https://doi.org/10.1007/s11154-020-09616-y>
- McVeigh, J., & Begley, E. (2017). Anabolic steroids in the UK: an increasing issue for public health. *Drugs: Education, Prevention and Policy, 24*(3), 278–285. <https://doi.org/10.1080/09687637.2016.1245713>
- Messick, S. (1995). Validity of psychological assessment: Validation of inferences from persons' responses and performances as scientific inquiry into score meaning. *American Psychologist, 50*(9), 741–749. <https://doi.org/10.1037/0003-066X.50.9.741>
- Mezinkis, J. P., Honos-Webb, L., Kropp, F., & Somoza, E. (2001). The measurement of craving. *Journal of Addictive Diseases, 20*(3), 67-85. doi:10.1300/J069v20n03_07
- Mhillaj, E., Morgese, M. G., Tucci, P., Bove, M., Schiavone, S., & Trabace, L. (2015). Effects of anabolic-androgens on brain reward function. *Frontiers in Neuroscience, 9*. <https://doi.org/10.3389/fnins.2015.00295>
- Midgley, S. J., Heather, N., & Davies, J. B. (1999). Dependence-producing potential of anabolic-androgenic steroids. *Addiction Research and Theory, 7*(6), 539–550. <https://doi.org/10.3109/16066359909004404>

- Miller, R. L. (2003). Sampling, snowball: accessing hidden and hard-to-reach populations. In R. L. Miller & J. D. Brewer (Eds.), *The A - Z of Social Research A Dictionary of Key Social Science Research Concepts* (pp. 274–280). SAGE Publications.
- Mol, A. J. J., Voshaar, R. C. O., Gorgels, W. J. M. J., Breteler, M. H. M., van Balkom, A. J. L. M., van de Lisdonk, E. H., van der Ven, A. H. G. S., & Zitman, F. G. (2003). Development and psychometric evaluation of the Benzodiazepine Craving Questionnaire. *Addiction*, 98(8), 1143–1152. <https://doi.org/10.1046/j.1360-0443.2003.00428.x>
- Monaghan, L. (2002). *Bodybuilding, Drugs and Risk* (G. Hart, Ed.). Routledge. <https://doi.org/10.4324/9780203136522>
- Moris, D., & Schizas, D. (2020). Lockdown during COVID-19: The Greek success. *In Vivo (Athens)*, 34(3 suppl), 1695–1699. <https://doi.org/10.21873/invivo.11963>
- Muthén, L. K., & Muthén, B. O. (2017). *Mplus user's guide* (Eighth ed.). Los Angeles, CA: Muthén & Muthén.
- Negus, S. S., Pope, Jr. , H. G., Kanayama, G., Wines, Jr. , J. D., & Fischer, B. D. (2001). Lack of evidence for opioid tolerance or dependence in rhesus monkeys following high-dose anabolic–androgenic steroid administration. *Psychoneuroendocrinology*, 26(8), 789–796. [https://doi.org/10.1016/S0306-4530\(01\)00028-2](https://doi.org/10.1016/S0306-4530(01)00028-2)
- Neri, M., Bello, S., Bonsignore, A., Cantatore, S., Riezzo, I., Turillazzi, E., & Fineschi, V. (2011). Anabolic Androgenic Steroids Abuse and Liver Toxicity. *Mini-Reviews in Medicinal Chemistry*, 11(5), 430–437. <https://doi.org/10.2174/138955711795445916>
- Niaura, R. (2000). Cognitive social learning and related perspectives on drug craving. *Addiction*, 95(8s2), 155–163. <https://doi.org/10.1046/j.1360-0443.95.8s2.4.x>

- Niaura, R. S., Rohsenow, D. J., Binkoff, J. A., Monti, P. M., Pedraza, M., & Abrams, D. B. (1988). Relevance of cue reactivity to understanding alcohol and smoking relapse. *Journal of Abnormal Psychology, 97*(2), 133–152. <https://doi.org/10.1037/0021-843X.97.2.133>
- Nunnally, J. C., & Bernstein, I. H. (1994). *Psychometric theory* (3rd ed.). McGraw-Hill.
- Ooteman, W., Koeter, M. W. J., Vserheul, R., Schippers, G. M., & Brink, W. (2006). Measuring craving: An attempt to connect subjective craving with cue reactivity. *Alcoholism, Clinical and Experimental Research, 30*(1), 57-69. [doi:10.1111/j.1530-0277.2006.00019.x](https://doi.org/10.1111/j.1530-0277.2006.00019.x)
- Pagonis, T. A., Angelopoulos, N. v., Koukoulis, G. N., & Hadjichristodoulou, C. S. (2006). Psychiatric side effects induced by supraphysiological doses of combinations of anabolic steroids correlate to the severity of abuse. *European Psychiatry, 21*(8), 551–562. <https://doi.org/10.1016/j.eurpsy.2005.09.001>
- Paliwal, P., Hyman, S. M., & Sinha, R. (2008). Craving predicts time to cocaine relapse: Further validation of the Now and Brief versions of the cocaine craving questionnaire. *Drug and Alcohol Dependence, 93*(3), 252–259. <https://doi.org/10.1016/j.drugalcdep.2007.10.002>
- Papazisis, G., Kouvelas, D., Mastrogianni, A., & Karastergiou, A. (2007). Anabolic androgenic steroid abuse and mood disorder: a case report. *The International Journal of Neuropsychopharmacology, 10*(2), 291–293. <https://doi.org/10.1017/S1461145706007243>

- Parkinson, A. B., & Evans, N. A. (2006). Anabolic androgenic steroids: A survey of 500 users. *Medicine & Science in Sports & Exercise*, 38(4), 644–651.
<https://doi.org/10.1249/01.mss.0000210194.56834.5d>
- Paty, J. A., Kassel, J. D., & Shiffman, S. (1992). Assessing stimulus control of smoking: The importance of base rates. *The Experience of Psychopathology*, 347–353.
- Perkins, K. A. (2009). Does smoking cue-induced craving tell us anything important about nicotine dependence? *Addiction*, 104(10), 1610–1616. <https://doi.org/10.1111/j.1360-0443.2009.02550.x>
- Perry, P. J., Lund, B. C., Deninger, M. J., Kutscher, E. C., & Schneider, J. (2005). Anabolic Steroid Use in Weightlifters and Bodybuilders. *Clinical Journal of Sport Medicine*, 15(5), 326–330. <https://doi.org/10.1097/01.jsm.0000180872.22426.bb>
- Peters, K. D., & Wood, R. I. (2005). Androgen dependence in hamsters: Overdose, tolerance, and potential opioidergic mechanisms. *Neuroscience*, 130(4), 971–981.
<https://doi.org/10.1016/j.neuroscience.2004.09.063>
- Pickens, C. L., Airavaara, M., Theberge, F., Fanous, S., Hope, B. T., & Shaham, Y. (2011). Neurobiology of the incubation of drug craving. *Trends in Neurosciences*, 34(8), 411–420. <https://doi.org/10.1016/j.tins.2011.06.001>
- Ployhart, R. E., & Ward, A. K. (2011). The “Quick Start Guide” for conducting and publishing longitudinal research. In *Journal of Business and Psychology*, 26(4), pp. 413–422. <https://doi.org/10.1007/s10869-011-9209-6>

- Pope, H. G., Kanayama, G., & Hudson, J. I. (2012). Risk factors for illicit anabolic-androgenic steroid use in male weightlifters: A cross-sectional cohort study. *Biological Psychiatry, 71*(3), 254–261. <https://doi.org/10.1016/j.biopsych.2011.06.024>
- Pope, H. G., Kanayama, G., Athey, A., Ryan, E., Hudson, J. I., & Baggish, A. (2014a). The lifetime prevalence of anabolic-androgenic steroid use and dependence in Americans: Current best estimates. *The American Journal on Addictions, 23*, 371–377. <https://doi.org/10.1111/j.1521-0391.2013.12118.x>.
- Pope, H. G., Kean, J., Nash, A., Kanayama, G., Samuel, D. B., Bickel, W. K., & Hudson, J. I. (2010). A diagnostic interview module for anabolic-androgenic steroid dependence. *Experimental and Clinical Psychopharmacology, 18*(3), 203-213. doi:10.1037/a0019370
- Pope, H. G., Kouri, E. M., & Hudson, J. I. (2000). Effects of Supraphysiologic Doses of Testosterone on Mood and Aggression in Normal Men. *Archives of General Psychiatry, 57*(2), 133. <https://doi.org/10.1001/archpsyc.57.2.133>
- Pope, H. G., Wood, R. I., Rogol, A., Nyberg, F., Bowers, L., & Bhasin, S. (2014b). Adverse Health Consequences of Performance-Enhancing Drugs: An Endocrine Society Scientific Statement. *Endocrine Reviews, 35*(3), 341–375. <https://doi.org/10.1210/er.2013-1058>
- Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, & Computers, 36*(4), 717–731. <https://doi.org/10.3758/BF03206553>

- Preacher, K. J., & Kelley, K. (2011). Effect size measures for mediation models: Quantitative strategies for communicating indirect effects. *Psychological Methods, 16*(2), 93–115.
<https://doi.org/10.1037/a0022658>
- Preston, C. C., & Colman, A. M. (2000). Optimal number of response categories in rating scales: Reliability, validity, discriminating power, and respondent preferences. *Acta Psychologica, 104*, 1-15. doi:10.1016/S0001-6918(99)00050-5.
- Preston, K. L., Kowalczyk, W. J., Phillips, K. A., Jobes, M. L., Vahabzadeh, M., Lin, J.-L., Mezghanni, M., & Epstein, D. H. (2018). Before and after: craving, mood, and background stress in the hours surrounding drug use and stressful events in patients with opioid-use disorder. *Psychopharmacology, 235*(9), 2713–2723.
<https://doi.org/10.1007/s00213-018-4966-9>
- Quaglio, G., Fornasiero, A., Mezzelani, P., Moreschini, S., Lugoboni, F., & Lechi, A. (2009). Anabolic steroids: Dependence and complications of chronic use. *Internal and Emergency Medicine, 4*, 289-296. doi:10.1007/s11739-009-0260-5
- Quinn, C. A., & Bussey, K. (2015). Moral Disengagement, Anticipated Social Outcomes and Adolescents' Alcohol Use: Parallel Latent Growth Curve Analyses. *Journal of Youth and Adolescence, 44*(10), 1854–1870. <https://doi.org/10.1007/s10964-015-0345-6>
- Raabe, A., Grusser, S., M, Wessa, M., Podschus, J., Flor, H. (2005). The assessment of craving: psychometric properties, factor structure and a revised version of the Alcohol Craving Questionnaire (ACQ). *Addiction 100*, 227-234. doi: 10.1111/j.1360-0443.2004.00960.x.
- Rasmussen, J. J., Selmer, C., Østergren, P. B., Pedersen, K. B., Schou, M., Gustafsson, F., Faber, J., Juul, A., & Kistorp, C. (2016). Former Abusers of Anabolic Androgenic

Steroids Exhibit Decreased Testosterone Levels and Hypogonadal Symptoms Years after Cessation: A Case-Control Study. *PLOS ONE*, *11*(8), 1–16.

<https://doi.org/10.1371/journal.pone.0161208>

- Rathod, S., Pallikadavath, S., Graves, E., Rahman, M. M., Brooks, A., Soomro, M. G., Rathod, P., & Phiri, P. (2021). Impact of lockdown relaxation and implementation of the face-covering policy on mental health: A United Kingdom COVID-19 study. *World Journal of Psychiatry*, *11*(12), 1346–1365. <https://doi.org/10.5498/wjp.v11.i12.1346>
- Ray, L. A., & Roche, D. J. O. (2018). Neurobiology of Craving: Current Findings and New Directions. *Current Addiction Reports*, *5*(2), 102–109. <https://doi.org/10.1007/s40429-018-0202-2>
- Ray, L. A., Kahler, C. W., Young, D., Chelminski, I., & Zimmerman, M. (2008). The Factor Structure and Severity of DSM-IV Alcohol Abuse and Dependence Symptoms in Psychiatric Outpatients. *Journal of Studies on Alcohol and Drugs*, *69*(4), 496–499. <https://doi.org/10.15288/jsad.2008.69.496>
- Robinson, T. (1993). The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Research Reviews*, *18*(3), 247–291. [https://doi.org/10.1016/0165-0173\(93\)90013-P](https://doi.org/10.1016/0165-0173(93)90013-P)
- Robles-Diaz, M., Gonzalez-Jimenez, A., Medina-Caliz, I., Stephens, C., García-Cortes, M., García-Muñoz, B., Ortega-Alonso, A., Blanco-Reina, E., Gonzalez-Grande, R., Jimenez-Perez, M., Rendón, P., Navarro, J. M., Gines, P., Prieto, M., Garcia-Eliz, M., Bessone, F., Brahm, J. R., Paraná, R., Lucena, M. I., & Andrade, R. J. (2015). Distinct phenotype of hepatotoxicity associated with illicit use of anabolic androgenic steroids. *Alimentary Pharmacology & Therapeutics*, *41*(1), 116–125. <https://doi.org/10.1111/apt.13023>

- Rosenberg, H. (2009). Clinical and laboratory assessment of the subjective experience of drug craving. In *Clinical Psychology Review* (Vol. 29, Issue 6, pp. 519–534).
<https://doi.org/10.1016/j.cpr.2009.06.002>
- Rothman, R. D., Weiner, R. B., Pope, H., Kanayama, G., Hutter, A. M., Fifer, M. A., Dec, G. W., & Baggish, A. L. (2011). Anabolic androgenic steroid induced myocardial toxicity: an evolving problem in an ageing population. *Case Reports*, 2011(aug17 1), bcr0520114280–bcr0520114280. <https://doi.org/10.1136/bcr.05.2011.4280>
- Russell, J. A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology*, 39(6), 1161–1178. <https://doi.org/10.1037/h0077714>
- Sagoë, D., McVeigh, J., Bjørnebekk, A., Essilfie, M. S., Andreassen, C. S., & Pallesen, S. (2015). Polypharmacy among anabolic-androgenic steroid users: A descriptive metasynthesis. *Substance Abuse: Treatment, Prevention, and Policy*, 10(1).
<https://doi.org/10.1186/s13011-015-0006-5>
- Sagoë, D., Molde, H., Andreassen, C. S., Torsheim, T., & Pallesen, S. (2014). The global epidemiology of anabolic-androgenic steroid use: a meta-analysis and meta-regression analysis. *Annals of Epidemiology*, 24(5), 383–398.
<https://doi.org/10.1016/j.annepidem.2014.01.009>
- Sayette, M. A. (2004). Self-Regulatory Failure and Addiction. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of Self-Regulation - Research, Theory, and Applications* (pp. 448–466). The Guilford Press.
- Sayette, M. A., Shiffman, S., Tiffany, S. T., Niaura, R. S., Martin, C. S., & Schadel, W. G. (2000). The measurement of drug craving. *Addiction (Abingdon, England)*, 95(8), 189-210. doi:10.1046/j.1360-0443.95.8s2.8.x

- Scarth, M., Havnes, I. A., Jørstad, M. L., McVeigh, J., van Hout, M. C., Westlye, L. T., Torgersen, S., & Bjørnebekk, A. (2022). Severity of anabolic steroid dependence, executive function, and personality traits in substance use disorder patients in Norway. *Drug and Alcohol Dependence*, 231, 109275.
<https://doi.org/10.1016/j.drugalcdep.2022.109275>
- Schlauch, R. C., Gwynn-Shapiro, D., Stasiewicz, P. R., Molnar, D. S., & Lang, A. R. (2013). Affect and craving: Positive and negative affect are differentially associated with approach and avoidance inclinations. *Addictive Behaviors*, 38(4), 1970–1979.
<https://doi.org/10.1016/j.addbeh.2012.12.003>
- Schroeder, J. P., & Packard, M. G. (2000). Differential effects of intra-amygdala lidocaine infusion on memory consolidation and expression of a food conditioned place preference. *Psychobiology*, 28(4), 486–491. <https://doi.org/10.3758/BF03332006>
- Scruggs, T. E., Mastropieri, M. A., & Casto, G. (1987). The Quantitative Synthesis of Single-Subject Research. *Remedial and Special Education*, 8(2), 24–33.
<https://doi.org/10.1177/074193258700800206>
- Sculthorpe, N., Grace, F., Jones, P., & Davies, B. (2010). Evidence of Altered Cardiac Electrophysiology Following Prolonged Androgenic Anabolic Steroid Use. *Cardiovascular Toxicology*, 10(4), 239–243. <https://doi.org/10.1007/s12012-010-9090-y>
- Seitz, J., Lyall, A. E., Kanayama, G., Makris, N., Hudson, J. I., Kubicki, M., Pope, H. G., & Kaufman, M. J. (2017). White matter abnormalities in long-term anabolic-androgenic steroid users: A pilot study. *Psychiatry Research: Neuroimaging*, 260, 1–5.
<https://doi.org/10.1016/j.psychresns.2016.12.003>

- Serre, F., Fatseas, M., Denis, C., Swendsen, J., & Auriacombe, M. (2018). Predictors of craving and substance use among patients with alcohol, tobacco, cannabis or opiate addictions: Commonalities and specificities across substances. *Addictive Behaviors*, *83*, 123–129. <https://doi.org/10.1016/j.addbeh.2018.01.041>
- Serre, F., Fatseas, M., Swendsen, J., & Auriacombe, M. (2015). Ecological momentary assessment in the investigation of craving and substance use in daily life: A systematic review. *Drug and Alcohol Dependence*, *148*, 1–20. <https://doi.org/10.1016/j.drugalcdep.2014.12.024>
- Shadel, W. G., & Cervone, D. (2006). Evaluating social-cognitive mechanisms that regulate self-efficacy in response to provocative smoking cues: An experimental investigation. *Psychology of Addictive Behaviors*, *20*(1), 91–96. <https://doi.org/10.1037/0893-164X.20.1.91>
- Shiffman, S. (2009). Ecological momentary assessment (EMA) in studies of substance use. *Psychological Assessment*, *21*(4), 486–497. <https://doi.org/10.1037/a0017074>
- Shiffman, S., & Waters, A. J. (2004). Negative Affect and Smoking Lapses: A Prospective Analysis. *Journal of Consulting and Clinical Psychology*, *72*(2), 192–201. <https://doi.org/10.1037/0022-006X.72.2.192>
- Shiffman, S., Paty, J. A., Gnys, M., Kassel, J. A., & Hickcox, M. (1996). First lapses to smoking: Within-subjects analysis of real-time reports. *Journal of Consulting and Clinical Psychology*, *64*(2), 366–379. <https://doi.org/10.1037/0022-006X.64.2.366>
- Shiffman, S., Stone, A. A., & Hufford, M. R. (2008). Ecological Momentary Assessment. *Annual Review of Clinical Psychology*, *4*(1), 1–32. <https://doi.org/10.1146/annurev.clinpsy.3.022806.091415>

- Skårberg, K., Nyberg, F., & Engström, I. (2008). The development of multiple drug use among anabolic-androgenic steroid users: six subjective case reports. *Substance Abuse Treatment, Prevention, and Policy*, 3(1), 24. <https://doi.org/10.1186/1747-597X-3-24>
- Skinner, M. D., & Aubin, H.-J. (2010). Craving's place in addiction theory: Contributions of the major models. *Neuroscience & Biobehavioral Reviews*, 34(4), 606–623. <https://doi.org/10.1016/j.neubiorev.2009.11.024>
- Smit, D. L., Buijs, M. M., Hon, O., Heijer, M., & Ronde, W. (2021). Positive and negative side effects of androgen abuse. The HAARLEM study: A one-year prospective cohort study in 100 men. *Scandinavian Journal of Medicine & Science in Sports*, 31(2), 427–438. <https://doi.org/10.1111/sms.13843>
- Smith, M. L. (1981). Naturalistic Research. *The Personnel and Guidance Journal*, 59(9), 585–589. <https://doi.org/10.1002/j.2164-4918.1981.tb00623.x>
- Snijders, T. A. B., & Bosker, R. J. (2012). *Multilevel Analysis: An Introduction to Basic and Advanced Multilevel Modeling* (T. A. B. Snijders & R. J. Bosker, Eds.; 2nd ed.). SAGE Publications.
- Stuart, K., Borland, R., & McMurray, N. (1994). Self-efficacy, health locus of control, and smoking cessation. *Addictive Behaviors*, 19(1), 1–12. [https://doi.org/10.1016/0306-4603\(94\)90046-9](https://doi.org/10.1016/0306-4603(94)90046-9)
- Sumnall, H. R., Montgomery, C., Atkinson, A. M., Gage, S. H., & Boardley, I. D. (2022). Moral disengagement and the harms of cocaine use. *Drugs: Education, Prevention and Policy*, 29(5), 490–500. <https://doi.org/10.1080/09687637.2021.1950126>

- Tan, R. S., & Scally, M. C. (2009). Anabolic steroid-induced hypogonadism – Towards a unified hypothesis of anabolic steroid action. *Medical Hypotheses*, 72(6), 723–728.
<https://doi.org/10.1016/j.mehy.2008.12.042>
- Tarlow, K. R., & Penland, A. (2016). Outcome assessment and inference with the percentage of nonoverlapping data (PND) single-case statistic. *Practice Innovations*, 1(4), 221–233.
<https://doi.org/10.1037/pri0000029>
- Teesson, M., Lynskey, M., Manor, B., & Baillie, A. (2002). The structure of cannabis dependence in the community. *Drug and Alcohol Dependence*, 68(3), 255–262.
[https://doi.org/10.1016/S0376-8716\(02\)00223-5](https://doi.org/10.1016/S0376-8716(02)00223-5)
- Tennant, F., Black, D. L., & Voy, R. O. (1988). Anabolic steroid dependence with opioid-type features. *The New England Journal of Medicine*, 319(9), 578.
- Tiffany, S. T. (1990). A cognitive model of drug urges and drug-use behavior: Role of automatic and nonautomatic processes. *Psychological Review*, 97(2), 147–168.
<https://doi.org/10.1037/0033-295X.97.2.147>
- Tiffany, S. T. (1999). Cognitive concepts of craving. *Alcohol Research & Health*, 23(3), 215–224.
- Tiffany, S. T. (2010). Drug craving and affect. In *Substance abuse and emotion*. (pp. 83–108). American Psychological Association. <https://doi.org/10.1037/12067-004>
- Tiffany, S. T., & Wray, J. M. (2012). The clinical significance of drug craving. *Annals of the New York Academy of Sciences*, 1248(1), 1–17. <https://doi.org/10.1111/j.1749-6632.2011.06298.x>

- Tiffany, S. T., Carter, B. L., & Singleton, E. G. (2000). Challenges in the manipulation, assessment and interpretation of craving relevant variables. *Addiction*, *95*(8), 177–187. <https://doi.org/10.1080/09652140050111753>
- Tiffany, S. T., Singleton, E., Haertzen, C. A., & Henningfield, J. E. (1993). The development of a cocaine craving questionnaire. *Drug and Alcohol Dependence*, *34*(1), 19–28. [https://doi.org/10.1016/0376-8716\(93\)90042-O](https://doi.org/10.1016/0376-8716(93)90042-O)
- Tiffany, S. T., Warthen, M. W., & Goedeker, K. C. (2008). The Functional Significance of Craving in Nicotine Dependence. In A. Caggiula & R. Bevins (Eds.), *The Motivational Impact of Nicotine and its Role in Tobacco Use* (pp. 171–197). Springer. https://doi.org/10.1007/978-0-387-78748-0_10
- Underwood, M. (2017). Exploring the social lives of image and performance enhancing drugs: An online ethnography of the Zyzx fandom of recreational bodybuilders. *International Journal of Drug Policy*, *39*(1), 78–85. <https://doi.org/10.1016/j.drugpo.2016.08.012>
- Underwood, M., van de Ven, K., & Dunn, M. (2021). Testing the boundaries: Self-medicated testosterone replacement and why it is practised. *International Journal of Drug Policy*, *95*, 103087. <https://doi.org/10.1016/j.drugpo.2020.103087>
- van Amsterdam, J., Opperhuizen, A., & Hartgens, F. (2010). Adverse health effects of anabolic–androgenic steroids. *Regulatory Toxicology and Pharmacology*, *57*(1), 117–123. <https://doi.org/10.1016/j.yrtph.2010.02.001>
- van de Ven, K., Boardley, I., & Chandler, M. (2022). Identifying best-practice amongst health professionals who work with people using image and performance enhancing drugs

- (IPEDs) through participatory action research. *Qualitative Research in Sport, Exercise and Health*, 14(2), 199–215. <https://doi.org/10.1080/2159676X.2021.1898457>
- Wang, C. L., & Ahmed, P. K. (2004). The development and validation of the organisational innovativeness construct using confirmatory factor analysis. *European Journal of Innovation Management*, 7(4), 303-313. doi:10.1108/14601060410565056
- Weiss, R. D., Griffin, M. L., Mazurick, C., Berkman, B., Gastfriend, D. R., Frank, A., Barber, J. P., Blaine, J., Salloum, I., & Moras, K. (2003). The Relationship Between Cocaine Craving, Psychosocial Treatment, and Subsequent Cocaine Use. *American Journal of Psychiatry*, 160(7), 1320–1325. <https://doi.org/10.1176/appi.ajp.160.7.1320>
- Welsch, S. K., Smith, S. S., Wetter, D. W., Jorenby, D. E., Fiore, M. C., & Baker, T. B. (1999b). Development and validation of the Wisconsin smoking withdrawal scale. *Experimental and Clinical Psychopharmacology*, 7(4), 354-361. doi:10.1037//1064-1297.7.4.354
- West, R., & Shiffman, S. (2016). *Fast Facts: Smoking Ceassation*. Health Press Limited.
- Westerman, M. E., Charchenko, C. M., Ziegelmann, M. J., Bailey, G. C., Nippoldt, T. B., & Trost, L. (2016). Heavy testosterone use among bodybuilders: An uncommon cohort of illicit substance users. *Mayo Clinic Proceedings*, 91(2), 175–182. <https://doi.org/10.1016/j.mayocp.2015.10.027>
- Westlye, L. T., Kaufmann, T., Alnæs, D., Hullstein, I. R., & Bjørnebekk, A. (2017). Brain connectivity aberrations in anabolic-androgenic steroid users. *NeuroImage: Clinical*, 13, 62–69. <https://doi.org/10.1016/j.nicl.2016.11.014>

- Wikler, A. (1948). Recent progress in research on the neurophysiologic basis of morphine addiction. *American Journal of Psychiatry*, *105*(5), 329–338.
<https://doi.org/10.1176/ajp.105.5.329>
- Windfeld-Mathiasen, J., Christoffersen, T., Strand, N. A. W., Dalhoff, K., Andersen, J. T., & Horwitz, H. (2022). Psychiatric morbidity among men using anabolic steroids. *Depression and Anxiety*. <https://doi.org/10.1002/da.23287>
- Wood, R. I. (2002). Oral testosterone self-administration in male hamsters: dose-response, voluntary exercise, and individual differences. *Hormones and Behavior*, *41*, 247–258.
- Wood, R. I. (2006). Anabolic Steroids: A Fatal Attraction? *Journal of Neuroendocrinology*, *18*(3), 227–228. <https://doi.org/10.1111/j.1365-2826.2006.01407.x>
- Wood, R. I. (2008). Anabolic–androgenic steroid dependence? Insights from animals and humans. *Frontiers in Neuroendocrinology*, *29*(4), 490–506.
<https://doi.org/10.1016/j.yfrne.2007.12.002>
- Wood, R. I., Johnson, L. R., Chu, L., Schad, C., & Self, D. W. (2004). Testosterone reinforcement: intravenous and intracerebroventricular self-administration in male rats and hamsters. *Psychopharmacology*, *171*(3), 298–305. <https://doi.org/10.1007/s00213-003-1587-7>
- Wood, R. I., Vertelkina, N. v., & Antzoulatos, E. (2011). Testosterone as a discriminative stimulus in male rats. *Pharmacology Biochemistry and Behavior*, *100*(1), 185–190.
<https://doi.org/10.1016/j.pbb.2011.08.022>
- World Health Organisation. (1992). *The ICD-10 Classification of Mental and Behavioural Disorders Clinical descriptions and diagnostic guidelines*. World Health Organization.

- Wray, J. M., Gass, J. C., & Tiffany, S. T. (2013). A Systematic Review of the Relationships Between Craving and Smoking Cessation. *Nicotine & Tobacco Research, 15*(7), 1167–1182. <https://doi.org/10.1093/ntr/nts268>
- Yates, W. R. (2000). Testosterone in Psychiatry: Risks and Benefits. *Archives of General Psychiatry, 57*(2), 155. <https://doi.org/10.1001/archpsyc.57.2.155>
- Yesalis, C. E., Vicary, J. R., Buckley, W. E., Streit, A. L., Katz, D. L., & Wright, J. E. (1990). Indications of psychological dependence among anabolic-androgenic steroid abusers. *NIDA Research Monograph, 102*, 196–214. doi:10.1037/e473002004-001
- Yu, J. G., Bonnerud, P., Eriksson, A., Stal, P. S., Tegner, Y., & Malm, C. (2014). Effects of long term supplementation of anabolic androgen steroids on human skeletal muscle. *PLoS ONE, 9*(9). <https://doi.org/10.1371/journal.pone.0105330>
- Yu, J., Hildebrandt, T., & Lanzieri, N. (2015). Healthcare professionals' stigmatization of men with anabolic androgenic steroid use and eating disorders. *Body Image, 15*, 49–53. <https://doi.org/10.1016/j.bodyim.2015.06.001>
- Yuan, M., Guo, X., Li, X., Chen, X., Wang, C., & Li, Y. (2018). The moderating role of regulatory emotional self-efficacy on smoking craving: An ecological momentary assessment study. *PsyCh Journal, 7*(1), 5–12. <https://doi.org/10.1002/pchj.188>
- Zahnow, R., McVeigh, J., Bates, G., Hope, V., Kean, J., Campbell, J., & Smith, J. (2018). Identifying a typology of men who use anabolic androgenic steroids (AAS). *International Journal of Drug Policy, 55*, 105–112. <https://doi.org/10.1016/j.drugpo.2018.02.022>

Zahnow, R., McVeigh, J., Ferris, J., & Winstock, A. (2017). Adverse Effects, Health Service Engagement, and Service Satisfaction Among Anabolic Androgenic Steroid Users.

Contemporary Drug Problems, 44(1), 69–83.

<https://doi.org/10.1177/0091450917694268>

Zoob Carter, B. N., Boardley, I. D., & van de Ven, K. (2021). The Impact of the COVID-19

Pandemic on Male Strength Athletes Who Use Non-prescribed Anabolic-Androgenic

Steroids. *Frontiers in Psychiatry*, 12. <https://doi.org/10.3389/fpsy.2021.636706>

Chapter 7 – Appendices

7.1 Appendix A

The Anabolic-Androgenic Steroid Dependence Scale

A number of statements describing experiences and scenarios you may have had whilst using anabolic steroids are presented below, please rate your level of agreement with the following items.

Over the last 12-months,	Strongly Disagree	Disagree	Slightly Disagree	Neutral	Slightly Agree	Agree	Strongly Agree
1. I have increased my use of steroids due to dissatisfaction with the effectiveness of my regime.	1	2	3	4	5	6	7
2. I have gone beyond my pre-planned use of steroids to increase my gains.	1	2	3	4	5	6	7
3. I have increased my use of steroids to increase gains.	1	2	3	4	5	6	7
4. I have used steroids to alleviate effects induced by stopping my use	1	2	3	4	5	6	7
5. I have used steroids to alleviate withdrawal symptoms experienced during an “off-cycle” period.	1	2	3	4	5	6	7
6. Experiencing withdrawal symptoms has made it difficult to stop using steroids during “off-cycle” periods.	1	2	3	4	5	6	7
7. I have continued using steroids despite experiencing unwanted side effects (e.g., gynecomastia, heart complications, cholesterol imbalance, abscesses, tendon/joint damage, testicular atrophy).	1	2	3	4	5	6	7

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|--|---|---|---|---|---|---|---|
| 8. I have continued to use steroids despite trying to manage undesired side effects (e.g., such as; gynecomastia, heart complications, cholesterol imbalance, abscesses from injections, tendon/joint damage, testicular atrophy). | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| 9. I have continued with my steroid regime since experiencing unwanted effects (e.g., such as; gynecomastia, heart complications, cholesterol imbalance, abscesses from injections, tendon/joint damage, testicular atrophy). | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| 10. I have continued with my steroid regime despite seeking help for problematic psychological effects (e.g., depressive thoughts, a decreased libido, increased anxiety, insomnia, and mood swings). | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| 11. I have experienced depressive thoughts, a decreased libido, increased anxiety, insomnia and mood swings, and continued using steroids. | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| 12. Experiencing unwanted side effects (e.g., depressive thoughts, decreased libido, increased anxiety, insomnia, mood swings) has concern me, but I continue to use steroids. | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| 13. I have avoided social, occupational and/or recreational activities as they would have interfered with my steroid regime. | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| 14. Avoiding social, occupational and/or recreational activities to prioritise my steroid regime has caused me problems within my personal life (i.e., with close family, friends, partner/significant other, boss/manager). | 1 | 2 | 3 | 4 | 5 | 6 | 7 |

15. I always prioritise my steroid regime over social, occupational and/or recreational activities, even if the outcome may be problematic.	1	2	3	4	5	6	7
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7.2 Appendix B

The Anabolic-Androgenic Steroid Craving Scale

A number of statements describing thought and experiences you may have had whilst using anabolic steroids are presented below, please rate your level of agreement with the following items.

Presently,	Strongly Disagree	Disagree	Slightly Disagree	Neutral	Slightly Agree	Agree	Strongly Agree
1. I have trouble getting steroids off my mind because of what they can do for me.	1	2	3	4	5	6	7
2. I frequently think about my steroid routine because of how it makes me feel.	1	2	3	4	5	6	7
3. Much of my time is occupied by ideas, thoughts, impulses, and images relating to what I can achieve whilst using steroids.	1	2	3	4	5	6	7
4. It takes a lot of effort to disregard my thoughts and feelings about my use of steroids.	1	2	3	4	5	6	7
5. Being around my gym friends makes me want to use steroids.	1	2	3	4	5	6	7
6. Being around my gym friends makes me desire steroids.	1	2	3	4	5	6	7
7. Talking to other gym users about training makes me want to use steroids.	1	2	3	4	5	6	7
8. Just passing by a gym makes me want to use steroids.	1	2	3	4	5	6	7

9. The thought of using steroids makes me feel more relaxed.	1	2	3	4	5	6	7
10. Knowing I will be using steroids improves my mood.	1	2	3	4	5	6	7
11. I feel content when anticipating using steroids.	1	2	3	4	5	6	7
12. The thought of using steroids improves my mood.	1	2	3	4	5	6	7
13. I have a desire to use steroids when I am feeling down.	1	2	3	4	5	6	7
14. I desire to use steroids when I feel irritable.	1	2	3	4	5	6	7
15. I have an urge to use steroids when I feel anxious.	1	2	3	4	5	6	7
16. I have a compulsion to use steroids when feeling tense.	1	2	3	4	5	6	7

7.3 Appendix C

Appendix 1. Participant self-reported patterns of AAS use, compounds used and dosages for each phase of the study.

Participant	Phase	Weekly Dose	Anavar	Halotestin	Turinabol	Masteron	Fast Testosterones	Slow Testosterones	Testosterone Blends	Deca-Durabolin	Equipoise	Trenbolone
P1	A	1,000mg to 2,000mg			Yes			Yes		Yes		Yes
	B	501mg to 1,000mg						Yes		Yes		Yes
	C	501mg to 1,000mg						Yes		Yes		Yes
P2	A	1,000mg to 2,000mg	Yes	Yes				Yes		Yes	Yes	Yes
	B	501mg to 1,000mg	Yes		Yes		Yes	Yes	Yes			Yes
	C	501mg to 1,000mg	Yes		Yes		Yes	Yes	Yes			Yes
P3	A	501mg to 1,000mg					Yes	Yes	Yes			
	B	< 300mg						Yes				
	C	< 300mg						Yes				

P4	A	1,000mg to 2,000mg	Yes		Yes	Yes		Yes		Yes
	B	< 300mg	Yes		Yes	Yes	Yes	Yes		Yes
	C	< 300mg	Yes		Yes	Yes	Yes	Yes		Yes
P5	A	300mg to 500mg						Yes		
	B	< 300mg		Yes				Yes		
	C	< 300mg		Yes				Yes		
P6	A	501mg to 1,000mg						Yes	Yes	Yes
	B	< 300mg						Yes		Yes
	C	< 300mg						Yes		Yes
