Eur J Appl Physiol
DOI 10.1007/s00421-007-0621-6

ORIGINAL ARTICLE

Effects of anabolic androgenic steroids on sleep patterns of individuals practicing resistance exercise

Daniel Paulino Venâncio · Sergio Tufik · Silvério Aparecido Garbuio · Antonio Claudio Lucas da Nóbrega · Marco Túlio de Mello

Accepted: 9 November 2007

Abstract Anabolic androgenic steroid (AAS) abuse has become a public health problem in many countries, and is associated with many psychiatric disorders. Epidemiological studies have also found increasing numbers of sleep disorders reported by individuals using AASs. The purpose of this study was to evaluate sleep patterns and disorders in anabolic androgenic users who practice resistance exercise. The sample comprised 58 males divided into three groups: (1) 20 current AAS users aged 26 ± 1.2, (2) 21 controls with no history of AAS use, aged 26 ± 1 and (3) 17 sedentary men with no sleep disorders aged 27.2 ± 0.34. The volunteers spent a night in the sleep laboratory for polysomnography. Comparing the three groups, the user group showed reduced sleep efficiency and more wakings after sleep onset than the sedentary group ($P = 0.001$). The sedentary group showed a higher percentage of stage 4 than the non-users group. We suggest that using of anabolic steroids reduced sleep efficiency and alters sleep architecture.

D. P. Venâncio · S. Tufik · S. A. Garbuio · M. T. de Mello
Psychobiology Department,
Universidade Federal de São Paulo (UNIFESP),
São Paulo, SP, Brazil

S. Tufik · S. A. Garbuio · M. T. de Mello
Psychopharmacology Support Fund Association (AFIP),
São Paulo, SP, Brazil

A. C. L. da Nóbrega
Universidade Federal Fluminense (UFF), Niterói, Brazil

M. T. de Mello (✉)
Rua Marselhesa, 535,
CEP 04020-060 Vila Clementino, SP, Brazil
e-mail: tmello@psicobio.epm.br

Keywords Anabolic androgenic steroids · Sleep · Resistance training

Introduction

Anabolic androgenic steroids (AAS) abuse has become a public health issue in many countries (Yesalis et al. 1993), and is associated with a large number of side effects, especially in long-term users, including atherogenesis, gynecomastia, liver disorders and suppressed neuroendocrine function (Anthony 1988; Dhar et al. 2005; Lane et al. 2006; O'Connor et al. 1990). In particular, the negative feedback action appear to induce the suppression of the circulation of luteinizing hormone (LH) and follicle stimulating hormone (FSH), by the action of androgen on the hypothalamic-pituitary axis (MacIndoe et al. 1997). Moreover, AAS use may cause psychiatric disorders such as hypomania, aggressiveness and mood disorders, and a reinforcing effect, has also been observed (Brower, 2002). In addition to behavioral effects, epidemiological studies showed increased sleep disorders among AAS users (Eklof et al. 2003; Kindlundh et al. 1999; Korkia and Stimson 1997; Parkinson and Evans 2006). A recent study found that 496 of 500 subjects had side effects related to AAS, and almost half reported insomnia when using anabolic steroids androgenic (Parkinson and Evans 2006). Sexual desire was boosted and there was a feeling of being energized with reduced sleep need after methyltestosterone administration, and use was correlated with elevation of 5-hidroxyindoleacetic acid (5-HIAA; Daly et al. 2001). Leibeluft et al. (1997) described an increase in stage 4 non-REM sleep (NREM) and a tendency for increased latency to onset of REM sleep (Leibenluft et al. 1997). More recently, (Liu et al. 2003) found reduced total sleep time, lower sleep efficiency, and less percentage

NREM sleep as well as increased stage 2 sleep. In relation to sleep respiratory disorders, Sandblom et al. (1983) detected increased apnea rates after high doses of testosterone (T). However, there have been contradictory research findings in relation to sleep architecture and exogenous administration of T (Leibenluft et al. 1997; Liu et al. 2003). It has been suggested that the increase in the plasma levels of T raises nocturnal metabolic expenditure (White et al. 1985), leading to lower sleep quality (Bonnet and Arand 1995).

The individuals who intake AAS are engaged in a typical resistance exercise to improve their muscle mass by hypertrophy (Hartgens and Kuipers 2004). Resistance exercise is known as a method of improving the functional capacity of the neuromuscular system. Depending on program design, resistance exercise is capable of enhancing each of the functional constituents of the neuromuscular system, that is, strength, power, and local muscular endurance (Deschenes and Kraemer 2002).

American Sleep Disorders Association considers physical exercise to be a modality of non-pharmacological treatment for sleep disorders (Driver and Taylor 2000). Acute physical exercise results in a transient reduction of sleepiness that depends on the intensity and the time of day at which the exercise is performed. This reduced sleepiness might be due to the fact that physical exercise increases total sleep time (Youngstedt et al. 1997), delays REM sleep, increases stage 4 and reduces REM sleep time when we compare sedentary and physically active individuals.

We can find these answers in the studies which used aerobic exercise and it is the most studied kind also found in the literature, instead of anaerobic or resistance training that show inconclusive data (Driver and Taylor 2000). Accordingly, Montgomery et al. (1988) found that individuals who have practiced strength training show no alteration in slow wave sleep (SWS) or another stage, but the tendency was that exercise negatively affects sleep. Yet, with respect to the type of training, another study compared aerobically trained endurance runners, power trained weightlifters and bodybuilders, kind of athletes with mixed anaerobic and aerobic, the power training, with unfit, non-athletic sedentary group where only aerobic group showed more SWS and NREM sleep, slept longer, and had shorter sleep onset latencies (Trinder et al. 1985).

Therefore, the objective of the present study was to evaluate sleep pattern, efficiency and potential disorders in AAS users practicing resistance exercise. To the best of our knowledge, this study is the first to use polysomnography to evaluate sleep patterns in AAS users based on compositions and dosages commonly associated with abuse.

Methods

Subjects

The Research Ethics Committee at Universidade Federal de São Paulo authorized all experimental procedures (# 1309/02) and three groups of males were recruited. Two who regularly practiced resistance training and one sedentary. They were classified as follows: (1) 20 current AAS users, via oral or injection routes (user group); (2) 21 non-users doing the same type of training and schedule (hours/week); and (3) 17 sedentary males with no sleep disorders and non-users of AAS. This group was used to exclude the interference of exercise in sleep patterns.

Recruitment process

Volunteers were selected from the responses to the advertisements in gyms in São Paulo and from anabolic androgenic steroids discussion forums on the internet. The exclusion criteria were: users of psychotropic drugs; individuals taking more than 15 doses of alcohol a week ($\cong$30 g/day); those who had made transmeridian journeys (altering sleep/wake cycles) within the past 12 months; and those scoring (>5) on the Pittsburgh scale (Buysse et al. 1989). Individuals voluntarily reporting AAS use and those who said they had never used the drug were submitted to blood level exams for total testosterone, luteinizing- and follicle- stimulant hormones and estradiol to indirectly assess anabolic steroid use. After initial evaluation, we randomly selected user and non-user group members among individuals who agreed to participate in the study.

Those recruited for the research protocol presented no electrocardiogram or echocardiogram alterations during repose, or metabolic alterations in biochemical exams (complete hemogram and fasting glycemia).

The drugs used by members of the user group had been purchased from non-medical sources, and the mean duration of use was 9 ± 2.56 weeks. They were self-administering a mean weekly AAS dose of 653.4 ± 99.47 mg using two or more kinds of AAS simultaneously (stack), and alternating amounts (pyramid). Stack refers to using two or more drugs in the same cycle of drug administration, while pyramid involves moving from lower to higher dose followed by gradual returning to initial dose. AAS varieties administered by intramuscular injections were nandrolone, stanazolol and different esters of testosterone. Substances taken orally included oxandrolone, oxymetholone and stanazolol. Since the time expended and the amounts of drugs were similar in both the ways of administration, we do not consider this as a variable.

The volunteers selected for the user group had already been using AAS prior to recruitment, and were fully

responsible for the whole procedure. At no time the researchers encouraged the use of the drug. Moreover, as part of selection, volunteers signed a declaration in which they stated that they were aware of their drug use and the risks of abuse.

Polysomnographic analysis

Volunteers were evaluated at the Psychobiology Department's Sleep Laboratory (UNIFESP) on two consecutive nights. The purpose of the first night was to adapt to sleep measurement equipment and the experimental procedure in order to reduce the novelty effect. Sensors were positioned but no polysomnographic readings were taken until the second night. Starting time was 20:30–21:30 h and data were recorded for at least 7 h. Sleep variables were collected and recorded using amplifiers and preamplifiers (Meditron™) and a computerized sleep system (Sonolabs®Meditron, São Paulo, Brazil), with sampling frequency 256 Hz per channel. We collected a total of 4 EEG derivations (C3-A2, C4-A1, Fz-A1, O1-A1), 2 electrooculogram channels, 2 electromyogram channels (submentonian and legs) and one ECG derivation (V2 modified). The polysomnographic study followed the Rechtschaffen and Kales (1968) classification of sleep stages.

In order to monitor volunteers' breathing we used: (1) nasal cannula with a pressure transducer; (2) oral air-flow monitoring; and (3) two thoracic and abdominal effort channels. We also used a Nellcor™ oximeter to obtain volunteers' pulse oximetry to verify O_2 saturation. To analyze the breathing events utilizing the criterions of the AASM (AASM 1999).

We analyzed the following sleep parameters: sleep latency (three consecutive epochs of certain sleep stages), total sleep time (TST), sleep efficiency (percentage obtained on dividing total sleep time by total recording time), wakefulness after onset of sleep (WASO), REM sleep, non-REM sleep stages 3 and 4 (as percentages), REM sleep latency (time from sleep onset to first REM sleep epoch), AHI (the AHI was calculated as the sum of apneas and hypopneas by hour of sleep) and O_2 saturation.

Analysis of the hypothalamus–pituitary–gonadal axis (HPG)

In the morning after the adaptation night, samples of blood from the anticubital vein were collected to analyze hormones in the HPG axis. An aliquot of serum was frozen at −20°C until assayed for hormones. Testosterone (T), luteinizing hormone (LH), follicle-stimulating hormone (FSH) and estradiol (E2) were measured by chemiluminescent immunometric assay (Advia Centaur, Bayer Corporation, Tarrytown, NY, USA). Detection thresholds were 10 ng/dl for testosterone (Ortho-Clinical Diagnostics Inc., Amersham, England), 7.0 pg/ml for estradiol (Advia Centaur), 0.3 mU/ml for FSH and 0.07 mU/ml for LH. Since the use of AAS lead to increased levels of E2 and a reduction in LH and FSH levels, these results enabled us to indirectly confirm AAS use (Baume et al. 2006; Hartgens and Kuipers 2004; Karila et al. 2004).

Statistical analysis

Data were expressed as mean (SEM). The Kolmogorov–Smirnov test was used to assess normal-distribution data and one-way ANOVA followed by Tukey test to compare groups. The significance level was at ($P < 0.05$). We used STATISTICA (StatSoft, Inc.) for statistical analyses.

Results

Descriptive analysis

Age and height were consistent among all three groups, but user group members presented higher body mass index than the other groups ($P < 0.001$; Table 1).

Hormonal parameters

The AAS use causes a significant alteration in HPG axis hormones. The one-way ANOVA test showed differences for LH ($F2.49 = 17.58$; $P < 0.0001$), FSH ($F2.49 = 17.58$; $P < 0.0001$) and E2 ($F2.49 = 12.70$; $P < 0.0001$). The post hoc test showed significantly reduced plasmatic concentrations of LH, FSH and higher levels of the E2 in the user group compared with the non-users and sedentary groups ($P < 0.0001$). The user group showed higher levels of testosterone than the sedentary group ($P < 0.04$; Table 2).

Polysomnographic evaluation

The AAS users showed altered sleep efficiency ($F2.56 = 7.97$; $P < 0.001$). The post hoc test revealed a difference between the users compared with the non-users and sedentary group ($P < 0.001$; Fig. 1a). No differences

Table 1 Sample—descriptive analysis

	Sedentary group	Non-user group	User group
Age	27.2 ± 0.34	26.01 ± 1.0	26 ± 1.2
Body mass index (BMI)	25.35 ± 0.4	23.35 ± 0.42	26.68 ± 0.97*#
Height (m)	1.75 ± 0.01	1.75 ± 0.01	1.79 ± 0.02

Anova test * different from sedentary group and # different from non-user group

Table 2 Hormonal parameters at time of study in sedentary, non-user and user groups

Hormones	Sedentary group	Non-user group	User group
Total testosterone (nmol/L)	554.8± 41.00	697.51 ± 50.39	873.33 ± 131.66*
LH (IU/L)	4.53 ± 0.43	3.99 ± 0.27#	1.29 ± 0.50*
FSH (IU/L)	3.15± 0.411	2.40 ± 0.24#	1.06 ± 0.30*
Estradiol (pg/mL)	32.4 ± 2.9	38.20 ± 4.29#	93.86 ± 14.70*

Values presented as mean ± standard error of mean. * Different from sedentary group and # different from user group. Anova test followed by post hoc Tukey test

were detected between groups in REM sleep onset latency ($P > 0.05$).

There were no statistically significant differences in sleep onset latency, stage 1 sleep, REM sleep or stage 3 sleep ($P > 0.05$). However, the one-way ANOVA test showed a significant difference in WASO (F2.56 = 5.52; $P < 0.05$). Post hoc analyses showed that the user group had higher proportion of WASO than the sedentary group ($P < 0.001$). Analysis showed difference in stage 2 sleep (F2.56 = 8.02; $P < 0.05$) and Tukey's test showed difference between the users and non-user group in relation to the sedentary group ($P < 0.001$). There was a difference in the percentage of stage 4 sleep (F2.56 = 8.94; $P < 0.001$). The post hoc analysis showed that the sedentary group had a higher percentage of stage 4 sleep than the non-user group ($P < 0.0001$). There was no difference regarding basal saturation of O_2, PLM index or AHI ($P > 0.05$; Table 3).

Table 3 Mean (SEM) values for polysomnographic parameters in the sedentary, non-user and user groups

	Sedentary	Non-user group	User group
TST (min)	387. 44 ± 7.99	377.10 ± 7.48	351.92 ± 16.26
Sleep latency (min	6.49 ± 2.02	13.97 ± 3.16	12.32 ± 2.53
WASO (%)	5.32±0.48	10.02 ± 1.42	18.15 ± 4.37*
Stage 1 (%)	3.05±0.35	1.99 ± 0.24	2.34 ± 0.41
Stage 2 (%)	51.86±1.52	60.98 ± 1.71*	59.41 ± 1.76*
Stage 3 (%)	3.97±0.31	4.56 ± 0,44	3.87 ± 0.61
Stage 4 (%)	19.00±1.42	11.29 ± 1.0*	14.79 ± 1.35
REM Sleep (%)	22.04±1.16	21.16 ± 1.39	18.66 ± 1.53
AHI/h	2.85±0.35	3.16 ± 0.35	4.98 ± 1.30
SaO2 (%)	95.2±0.87	95.09 ± 0.38	95.15 ± 0.37
PLM/h	1.56±0.54	1.81± 0.60	4.11 ± 1.58

Values presented as mean ± standard error of mean. * Different from sedentary group. Anova test followed by post hoc Tukey's test

Discussion

The results of the present study show that AAS use associated with resistance exercise may alter user sleep patterns. In this respect, we found reduced sleep efficiency and a higher percentage of WASO. Interestingly, both user and non-user groups showed an elevated percentage of stage 2 sleep compared with the sedentary group. Furthermore, both groups (users and non-users) showed the same percentage of stage 4 SWS, while the sedentary group showed statistically significant differences for this parameter.

In 1997, was observed a tendency for an increase in REM sleep latency and stage 4 SWS in normal sedentary individuals who were using 200-mg testosterone enanthate doses every 2 weeks for 1 month (Leibenluft et al. 1997). On the other hand, they found reduced sleep efficiency, increased stage 2 sleep, a lower proportion of non-REM sleep and less total sleep time in volunteers who received T in 200, 250 and 500 mg doses every 2 weeks (Liu et al. 2003). Other researchers failed to find alterations in parameters related to distribution of sleep stages or any other sleep architecture variables. However, administration of T increased the AHI (Matsumoto et al. 1985; Sandblom et al. 1983; Schneider et al. 1986).

Our results agree with data reported by Liu et al. (2003) in relation to reduced sleep efficiency. We may infer that high doses used by volunteers in the user group affected reduced sleep efficiency, since higher plasmatic and androgen levels raise nocturnal metabolic expenditure (White

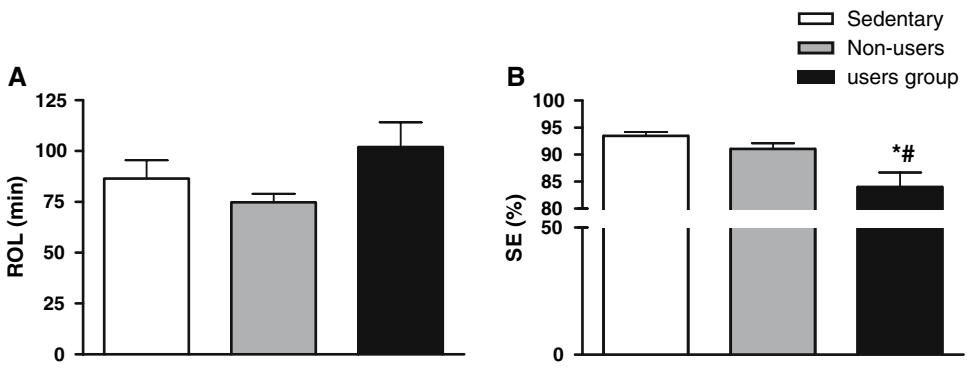

Fig. 1 **a** REM sleep onset latency (ROL) and **b** sleep efficiency (SE) for sedentary, non-user and user groups. * Different from sedentary group and # different from non-user group. Anova test followed by post hoc Tukey test ($P < 0.001$)

et al. 1985), which may cause sleep fragmentation and so lower-quality sleep (Bonnet and Arand 1995). It is also postulated that androgens, which have the capacity to interfere in serotoninergic neurotransmission (Fink et al. 1999), alter the action of the latter and induce longer periods of wakefulness (Daly et al. 2001), which would in turn reduce sleep efficiency.

In addition, steroid hormones act on the central nervous system by means of neurosteroids, compounds derived from sexual and steroid hormones. These substances may or may not be synthesized in the central nervous system, altering neuronal excitability through interaction with membrane receptors of the various neurotransmitters, mainly those of the GABAergic system (Rupprecht 2003; Stoffel-Wagner 2001). The high E2 concentrations observed in the user group might have increased neurosteroid levels and contributed to the down-regulation mechanism of the ligand sites in the GABAA receptors (Beyenburg et al. 2001). This reduction in ligand sites may have boosted neuronal excitability, thus reducing sleep efficiency.

Another hypothesis is that highly intensive training without sufficient recovery time between sessions may lead to wakefulness periods after sleep onset by increasing sleep fragmentation and changing sleep duration stage 2 in both user and non-user groups (Driver et al. 1994; Shapiro 1981).

Certain limitations in our research should be considered when incorporating findings to the literature. Firstly, we obtained our results from a single polysomnographic analysis while volunteers were using AAS. Therefore, there may be changes in relation to the variables measured over the drug administration cycle. Secondly, users were using many compounds simultaneously so we were unable to determine which if any caused alterations observed in the study.

In conclusion, AAS use reduced sleep efficiency and have relatively discrete effects on sleep architecture. Moreover, strength exercise may cause the slight change in sleep stage 2 and WASO found in both user and non-user groups.

Acknowledgments We would like to thank the Psychopharmacology Support Fund Association (AFIP), FAPESP (CEPID/SLEEP #98/14303-3 S.T), CENESP/UNIFESP and CEPE/UNIFESP, and CNPq. We thank Drs. Beatriz Helena Pizarro De Lorenzo, Monica Levy Andersen and Ben Edwards.

References

AASM (1999) Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. Sleep 22:667–689

Anthony PP (1988) Liver tumours. Baillieres Clin Gastroenterol 2:501–522

Baume N, Schumacher YO, Sottas PE, Bagutti C, Cauderay M, Mangin P, Saugy M (2006) Effect of multiple oral doses of androgenic anabolic steroids on endurance performance and serum indices of physical stress in healthy male subjects. Eur J Appl Physiol 98:329–340

Beyenburg S, Stoffel-Wagner B, Bauer J, Watzka M, Blumcke I, Bidlingmaier F, Elger CE (2001) Neuroactive steroids and seizure susceptibility. Epilepsy Res 44:141–153

Bonnet MH, Arand DL (1995) 24-Hour metabolic rate in insomniacs and matched normal sleepers. Sleep 18:581–588

Brower KJ (2002) Anabolic steroid abuse and dependence. Curr Psychiatry Rep 4:377–387

Buysse DJ, Reynolds CF III, Monk TH, Berman SR, Kupfer DJ (1989) The Pittsburgh sleep quality index: a new instrument for psychiatric practice and research. Psychiatry Res 28:193–213

Daly RC, Su TP, Schmidt PJ, Pickar D, Murphy DL, Rubinow DR (2001) Cerebrospinal fluid and behavioral changes after methyltestosterone administration: preliminary findings. Arch Gen Psychiatry 58:172–177

Deschenes MR, Kraemer WJ (2002) Performance and physiologic adaptations to resistance training. Am J Phys Med Rehabil 81:S3–S16

Dhar R, Stout CW, Link MS, Homoud MK, Weinstock J, Estes NA III (2005) Cardiovascular toxicities of performance-enhancing substances in sports. Mayo Clin Proc 80:1307–1315

Driver HS, Taylor SR (2000) Exercise and sleep. Sleep Med Rev 4:387–402

Driver HS, Rogers GG, Mitchell D, Borrow SJ, Allen M, Luus HG, Shapiro CM (1994) Prolonged endurance exercise and sleep disruption. Med Sci Sports Exerc 26:903–907

Eklof AC, Thurelius AM, Garle M, Rane A, Sjoqvist F (2003) The anti-doping hot-line, a means to capture the abuse of doping agents in the Swedish society and a new service function in clinical pharmacology. Eur J Clin Pharmacol 59:571–577

Fink G, Sumner B, Rosie R, Wilson H, McQueen J (1999) Androgen actions on central serotonin neurotransmission: relevance for mood, mental state and memory. Behav Brain Res 105:53–68

Hartgens F, Kuipers H (2004) Effects of androgenic-anabolic steroids in athletes. Sports Med 34:513–554

Karila T, Hovatta O, Seppala T (2004) Concomitant abuse of anabolic androgenic steroids and human chorionic gonadotrophin impairs spermatogenesis in power athletes. Int J Sports Med 25:257–263

Kindlundh AM, Isacson DG, Berglund L, Nyberg F (1999) Factors associated with adolescent use of doping agents: anabolic-androgenic steroids. Addiction 94:543–553

Korkia P, Stimson GV (1997) Indications of prevalence, practice and effects of anabolic steroid use in Great Britain. Int J Sports Med 18:557–562

Lane HA, Grace F, Smith JC, Morris K, Cockcroft J, Scanlon MF, Davies JS (2006) Impaired vasoreactivity in bodybuilders using androgenic anabolic steroids. Eur J Clin Invest 36:483–488

Leibenluft E, Schmidt PJ, Turner EH, Danaceau MA, Ashman SB, Wehr TA, Rubinow DR (1997) Effects of leuprolide-induced hypogonadism and testosterone replacement on sleep, melatonin, and prolactin secretion in men. J Clin Endocrinol Metab 82:3203–3207

Liu PY, Yee B, Wishart SM, Jimenez M, Jung DG, Grunstein RR, Handelsman DJ (2003) The short-term effects of high-dose testosterone on sleep, breathing, and function in older men. J Clin Endocrinol Metab 88:3605–3613

MacIndoe JH, Perry PJ, Yates WR, Holman TL, Ellingrod VL, Scott SD (1997) Testosterone suppression of the HPT axis. J Investig Med 45:441–447

Matsumoto AM, Sandblom RE, Schoene RB, Lee KA, Giblin EC, Pierson DJ, Bremner WJ (1985) Testosterone replacement in hypogonadal men: effects on obstructive sleep apnoea, respiratorydrives, and sleep. Clin Endocrinol (Oxf) 22:713–721

Montgomery I, Trinder J, Paxton S, Harris D, Fraser G, Colrain I (1988) Physical exercise and sleep: the effect of the age and sex of the subjects and type of exercise. Acta Physiol Scand Suppl 574:36–40

O'Connor JS, Baldini FD, Skinner JS, Einstein M (1990) Blood chemistry of current and previous anabolic steroid users. Mil Med 155:72–75

Parkinson AB, Evans NA (2006) Anabolic androgenic steroids: a survey of 500 users. Med Sci Sports Exerc 38:644–651

Rechtschaffen A, Kales A (1968) A manual of standardized terminology, techniques and scoring systemfor sleep stages of human subjects. Ref Type: Serial (Book, Monograph)

Rupprecht R (2003) Neuroactive steroids: mechanisms of action and neuropsychopharmacological properties. Psychoneuroendocrinology 28:139–168

Sandblom RE, Matsumoto AM, Schoene RB, Lee KA, Giblin EC, Bremner WJ, Pierson DJ (1983) Obstructive sleep apnea syndrome induced by testosterone administration. N Engl J Med 308:508–510

Schneider BK, Pickett CK, Zwillich CW, Weil JV, McDermott MT, Santen RJ, Varano LA, White DP (1986) Influence of testosterone on breathing during sleep. J Appl Physiol 61:618–623

Shapiro CM (1981) Sleep and the athlete. Br J Sports Med 15:51–55

Stoffel-Wagner B (2001) Neurosteroid metabolism in the human brain. Eur J Endocrinol 145:669–679

Trinder J, Paxton SJ, Montgomery I, Fraser G (1985) Endurance as opposed to power training: their effect on sleep. Psychophysiology 22:668–673

White DP, Schneider BK, Santen RJ, McDermott M, Pickett CK, Zwillich CW, Weil JV (1985) Influence of testosterone on ventilation and chemosensitivity in male subjects. J Appl Physiol 59:1452–1457

Yesalis CE, Kennedy NJ, Kopstein AN, Bahrke MS (1993) Anabolic-androgenic steroid use in the United States. JAMA 270:1217–1221

Youngstedt SD, O'Connor PJ, Dishman RK (1997) The effects of acute exercise on sleep: a quantitative synthesis. Sleep 20:203–214