Impact of supraphysiological doses of anabolic steroids on glucose and insulin levels in male bodybuilders: A Systematic Review

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Introduction

Testosterone (TE) is an anabolic androgenic steroid (AAS) that promotes muscle growth and the maintenance of secondary sexual characteristics (Mottram & George, 2000; Tavares et al., 2024. Due to its aesthetic benefits, AASs have become popular in sports, typically administered orally or intramuscularly, with doses up to 100 times higher than the therapeutic dose. A therapeutic dose is safe and sufficient for the desired effect, while a supraphysiological dose is significantly greater than what is needed for normal body functions. Insulin regulates blood glucose levels and metabolic processes (Azevedo, 1993). Insulin resistance occurs when cells do not respond well to this hormone, leading to elevated blood sugar levels (Azevedo, 1993). It is believed that TE disrupts the secretion of adipocytokines, increases the amount of circulating fatty acids, and may negatively affect insulin signaling in muscle and fat cells (Rasmussen et al., 2017). This systematic review aims to understand the influence of administering supraphysiological doses of AASs on blood glucose and insulin levels in male bodybuilding athletes.

Method

According to the PRISMA methodology (Page et al., 2021), articles addressing the use of supraphysiological doses of AASs and their effects on glucose and insulin levels published between 1987 and 2024 and with free access were selected. The study population should include only recreational male bodybuilders, with articles excluded if the population included individuals with diabetes or any condition that limited their ability to participate in the study. Studies that did not present laboratory results for the biochemical parameters of glucose and insulin or did not include laboratory results compared to control groups were also excluded. Additionally, articles that did not address the use of AASs in the abstract, as well as those in ebook and review formats, were removed. The quality of the articles was assessed using the Critical Appraisal Checklist for Cohort Studies by JBI (JBI Critical Appraisal Tools | JBI, no date). The Mendeley platform was used as a reference manager.

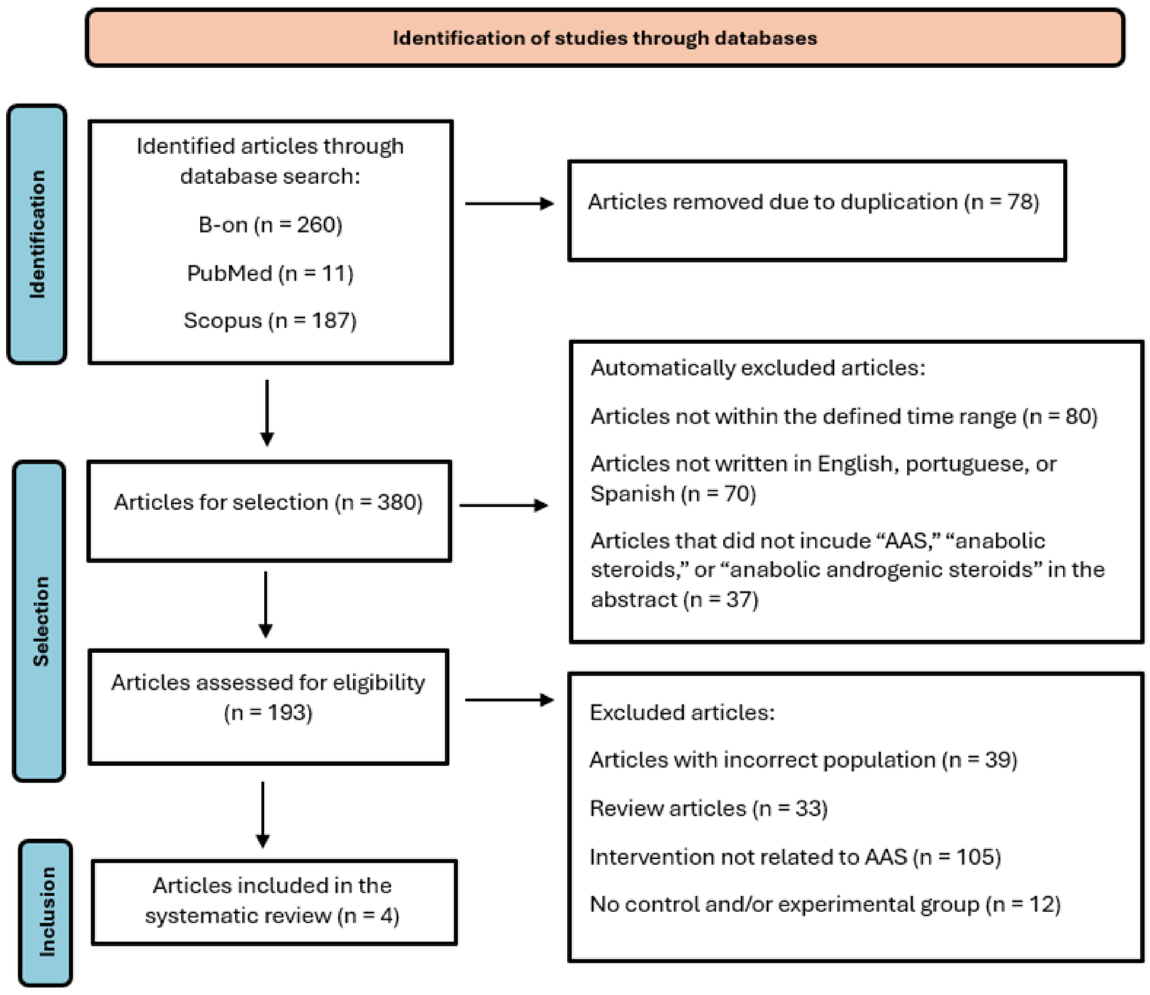

Figure 1 - PRISMA flowchart of the article selection process for the present systematic review.

Results

Table 1 - Summary table of the articles that make up this systematic review.

Authors	Year	Country	*N*	Age,mean(SD)	Steroid administered to AAS users	Route of administration	Effects obtained on glucose and insulin levels
Cohen *et al* (Cohenf & Hickman, 1987)	1987	South Africa	ACE (n=8); ANCE (n=7)	ACE (27 ± 2); ANCE (24 ± 2)	- Dianabol -Depo-Testosterone - Primobolan - Anapolon	Oral and intramuscular	ACE have impaired glucose tolerance compared to ANCE
Lane *et al* (Lane et al., 2006)	2006	United Kingdom	ACE (n=10); ACEP (n=8); ANCE (n=10)	ACE (26 ± 7,2) ACEP (32 ± 7,1) ANCE (24 ± 7,1)	- Testosterone - Nandrolone - Stanozolol	Parenteral	Glucose and Insulin levels were similar between the three groups
Rasmussen *et al* (Rasmussen et al., 2017)	2017	Denmark	ACE (n=37); ACEP (n=33); ANCE (n=30)	ACE (31,4) ACEP (34,8) ANCE (31,5)	- Testosterone - Androstendione Dehydroepiandrosterone Sulfate (DHEAS) -17OH-progesterone	----	Both ACE and ACEP showed lower insulin sensitivity compared to ANCE
Girolamo, *et al* (Di Girolamo et al., 2024)	2024	Italy	ACE (n=13); ANCE (n=52)	ACE (28 ± 1) ANCE (32 ± 1)	----	----	Both ACE and ANCE showed normal insulin sensitivity values. However, despite being normal, this value is lower in ACE

Caption:
ACE – Athletes who consume AASs
ANCE – Athletes who have never consumed AASs
ACEP – Athletes who have consumed AASs in the past

Table 2 - Results of selected studies.

Authors	Parameters	Resuls ACE	Results ANCE	Results ACEP	*p* value
Cohen *et al (Cohenf & Hickman, 1987)*	Fasting Glucose (mmol/L)	4,8±0,3	5,0±0,3	ND	----
	Glycemia 120 min (mmol/L)	14±5	7±3		*p* <0.05
	Fasting insulin (mU/L)	14±10	4±1		*p* <0.05
	Insulin 120 min (mU/L)	683±281	176±86		*p* <0.005
	Insulin spike (mU/L)	144±46	42±15		*p* <0.005
Lane *et al* (Lane et al., 2006)	Fasting Glucose (mmol/L)	4,3±0,4	4,5±0,3	4,6±0,3	ND
	Insulin (mU/L)	4,2±0,7	3,2±0,5	3,8±1,0	
Rasmussen *et al* (Rasmussen et al., 2017)	Glycemia 120 min (mmol/L)*	830	743	800	0,029
	Insulin 120min (nmol/L)	35,1	29,5	41,8	p < 0,001
	Adiponectin (mg/L)	6,4	8,6	7,7	0,009
	Leptin (µg/L)	1,2	4,4	6,1	p < 0,001
	PCR-as (mg/L)	1,2	0,4	0,6	p < 0,001
Girolamo *et al (Di Girolamo et al., 2024)*	Fasting Glucose (mg/dL)	89±2	93±1	ND	0,15
	Fasting insulin (µU/mol)	7,1±1,0	6,3±0,4		0,22
	Leptin (ng/mL)	0,63±0,18	0,86±0,12		p < 0,001
	PCR-as (mg/dL)	0,14±0,05	0,07±0,02		0,01

Caption:
ACE – Athletes who consume AASs
ANCE – Athletes who have never consumed AASs
ACEP – Athletes who have consumed AASs in the past
hs-CRP – High sensitivity C-reactive protein
*Value obtained from the area under the curve

Discussion and conclusion

Observational studies cannot fully control for factors such as diet, family history, and health habits, which may influence the relationship between AAS use and the risk of type 2 diabetes, and they do not establish cause-and-effect relationships. Future studies should investigate the effects of different AAS dosages to identify a potential risk threshold, as well as expand the diversity of participants. Additionally, it is necessary to explore the biological mechanisms of AAS on glucose and insulin, especially in individuals with pre-existing conditions, and to use methods like HOMA-IR and the Matsuda Index to quantify insulin resistance.

Based on the analysis of the studies included in this systematic review, it was possible to identify a relationship between the use of anabolic-androgenic steroids (AAS) and blood glucose and insulin levels in male bodybuilding athletes. It was found that prolonged use of AAS may lead to an increase in glucose levels and a reduction in insulin sensitivity, suggesting a potential risk for the development of insulin resistance and possibly type 2 diabetes. However, the heterogeneity of the studies in terms of dosages and types of AAS used limits the generalization of the findings.

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