

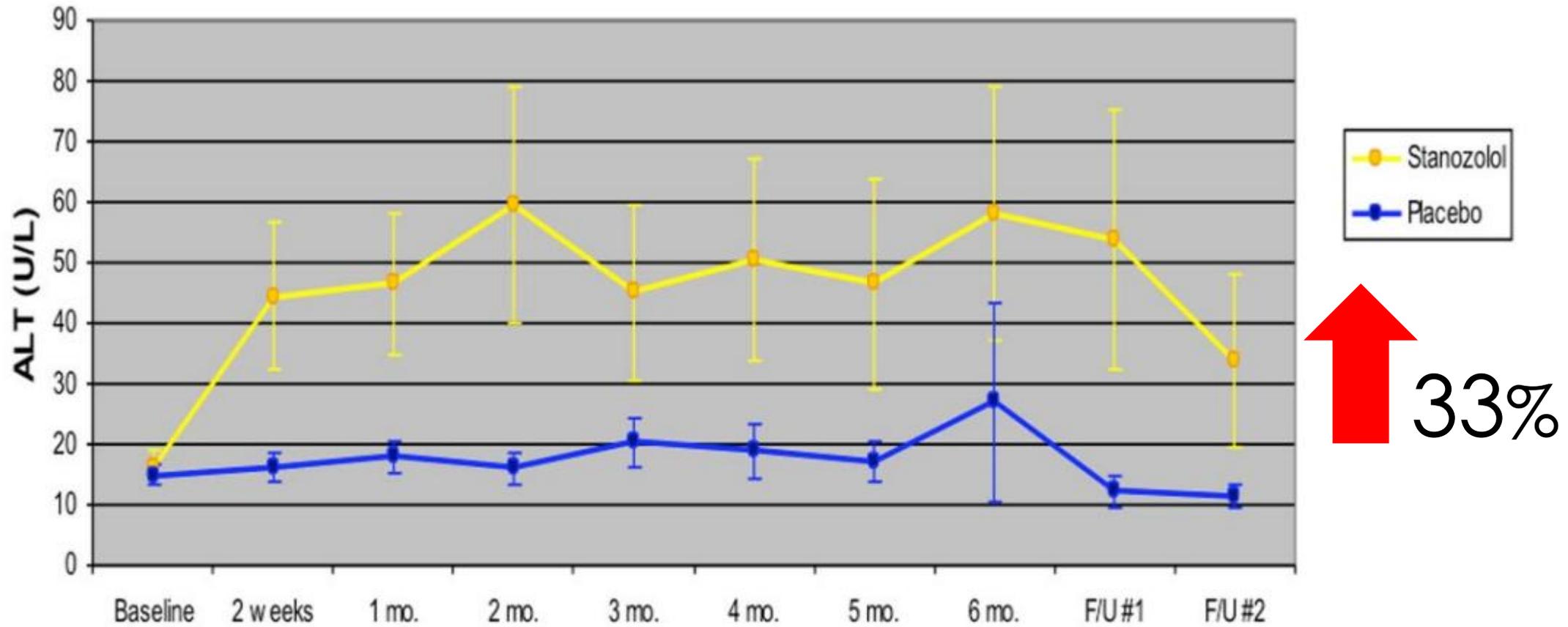


**Uso abusivo de anabolizantes**

**e**

**alterações do sistema hepático e renal**

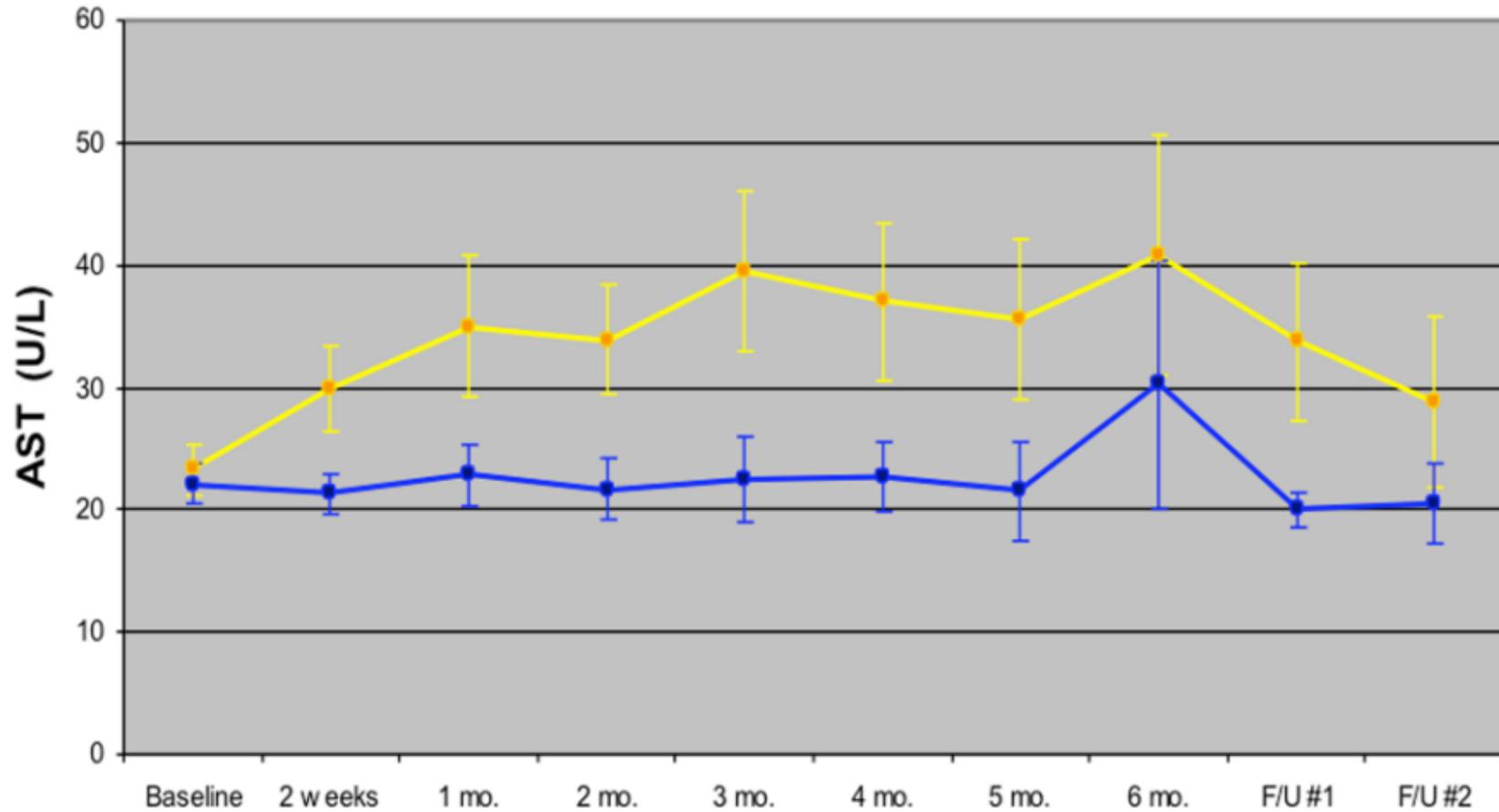
# Enzimas hepáticas: ALT/TGP



## P-values for significance as compared to baseline values

Stanozolol	0.6496	0.0361	0.0182	0.0422	0.0795	0.0876	0.1348	0.0402	0.1792	0.1253
Placebo	>0.05	0.6609	0.2605	0.5011	0.0645	0.4271	0.5078	0.4843	0.4328	0.4070

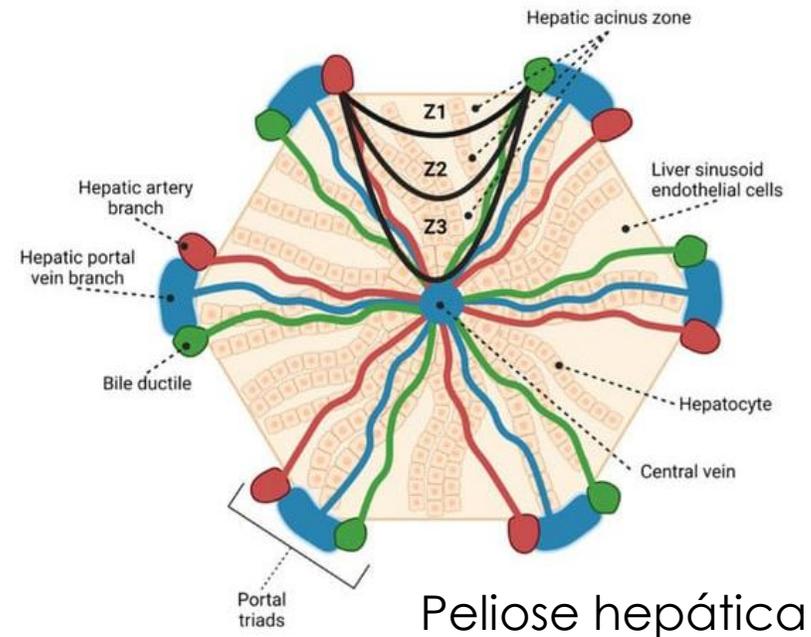
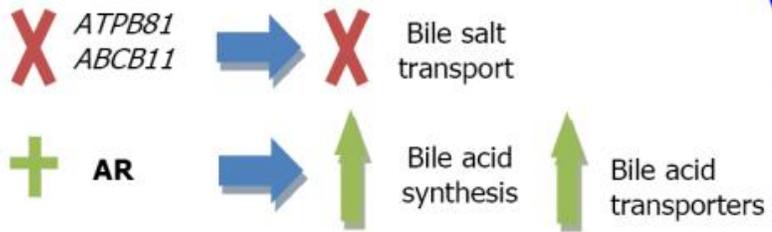
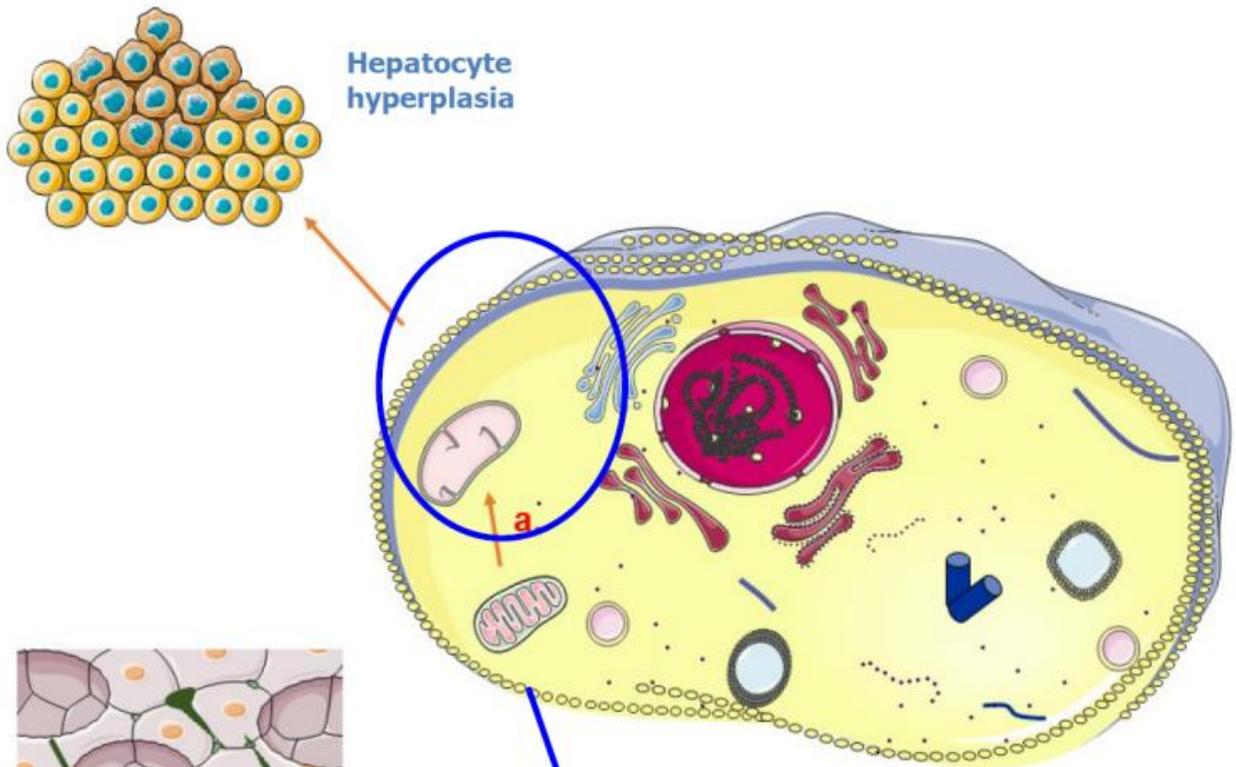
# Enzimas hepáticas: AST/TGO



 29%

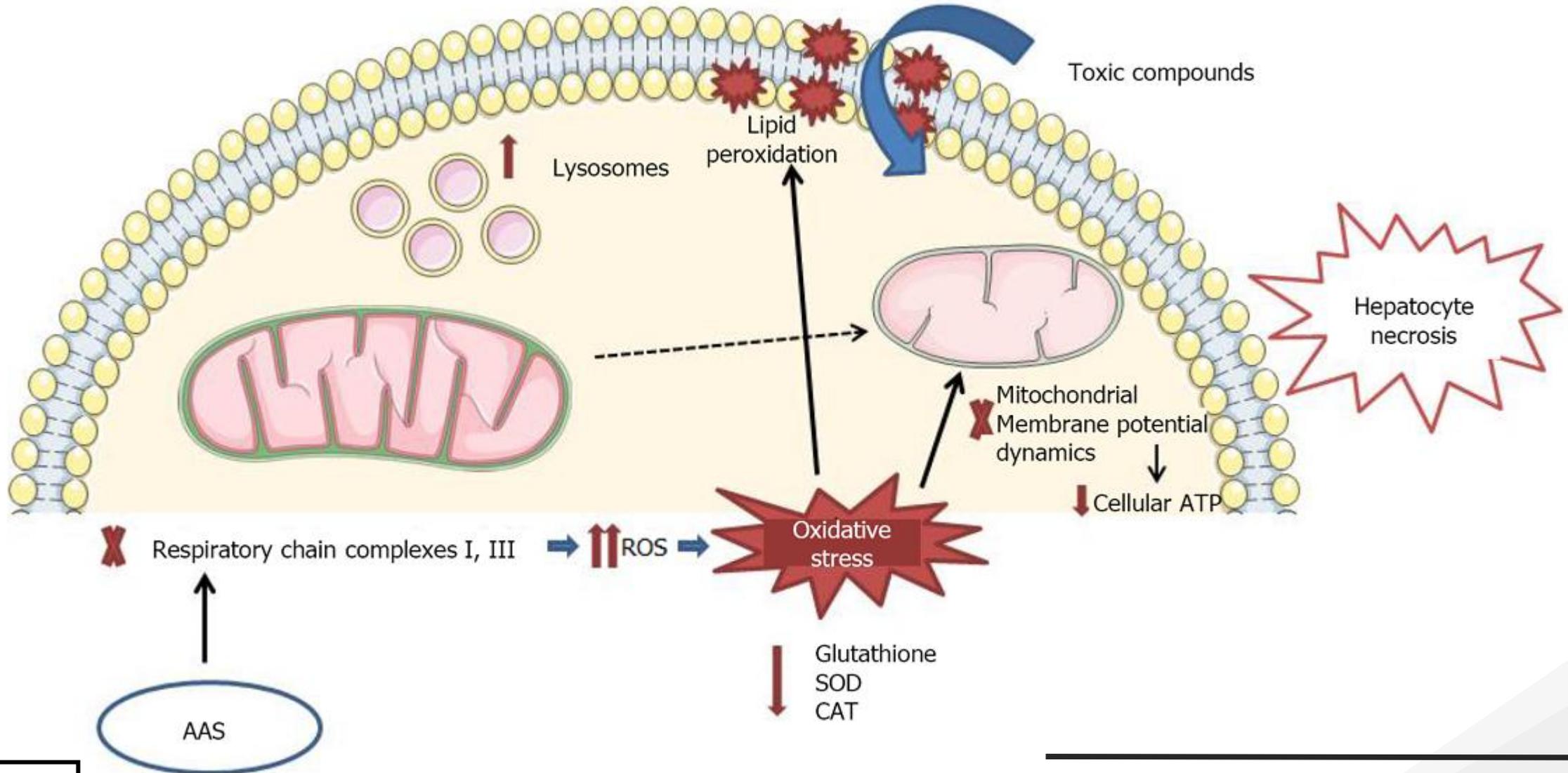
**P-values for significance as compared to baseline values**

Stanozolol	0.6376	0.1180	0.0663	0.0145	0.0365	0.0809	0.1200	0.0500	0.0692	0.4664
Placebo	>0.05	0.9207	0.9541	0.4431	0.9694	0.9175	0.5431	0.4558	0.1067	0.7084

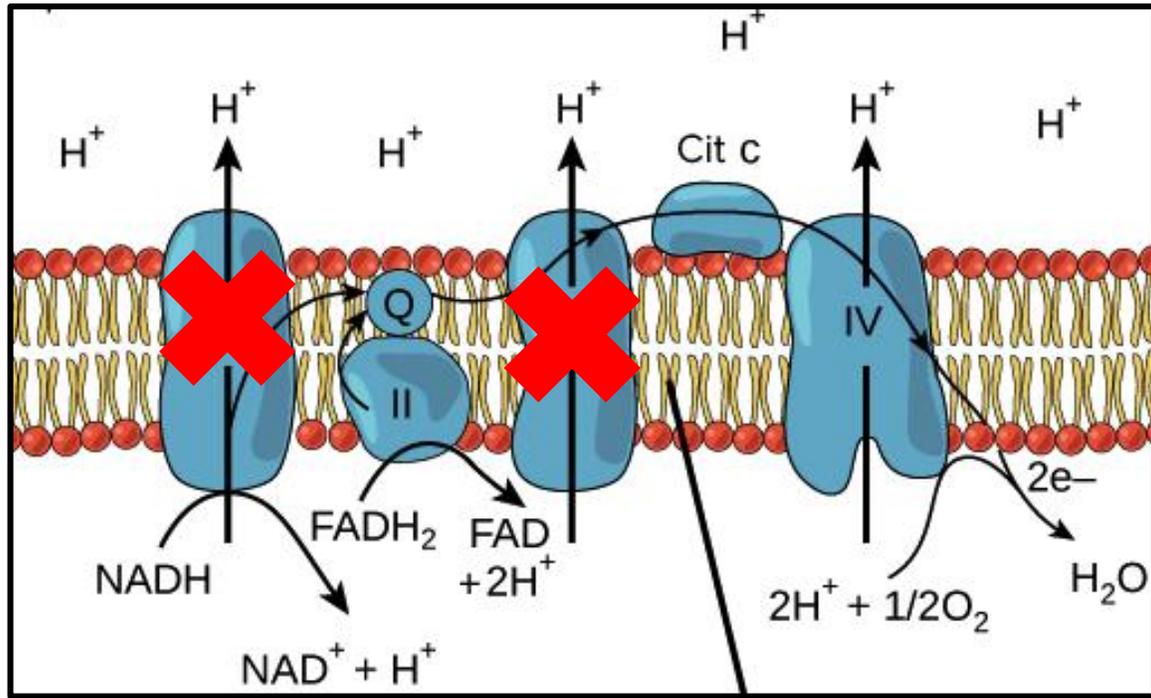


Redução do fluxo de bile nos canais biliares do fígado para o intestino delgado (doenças hepáticas e obstruções biliares)

# Alterações hepáticas

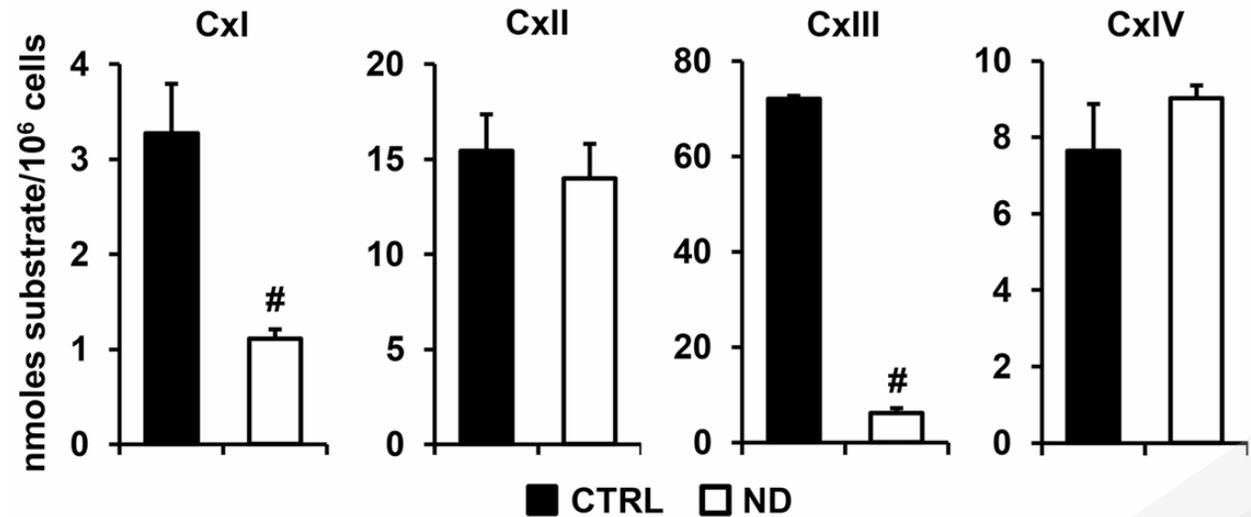
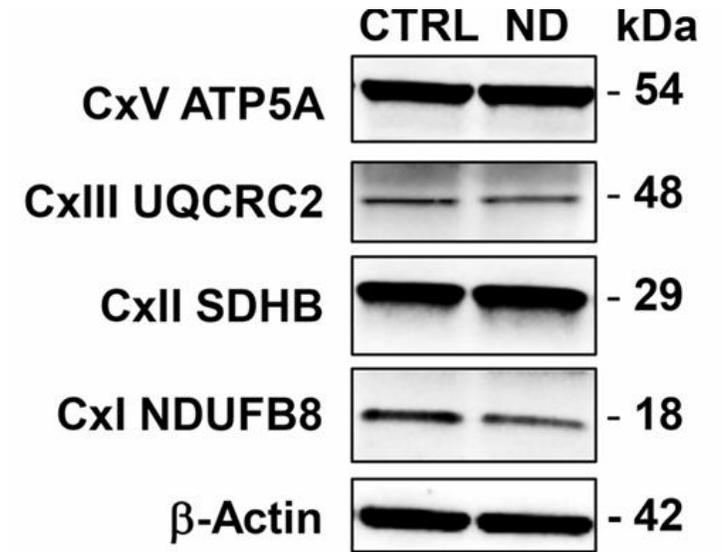


# Alterações na respiração celular

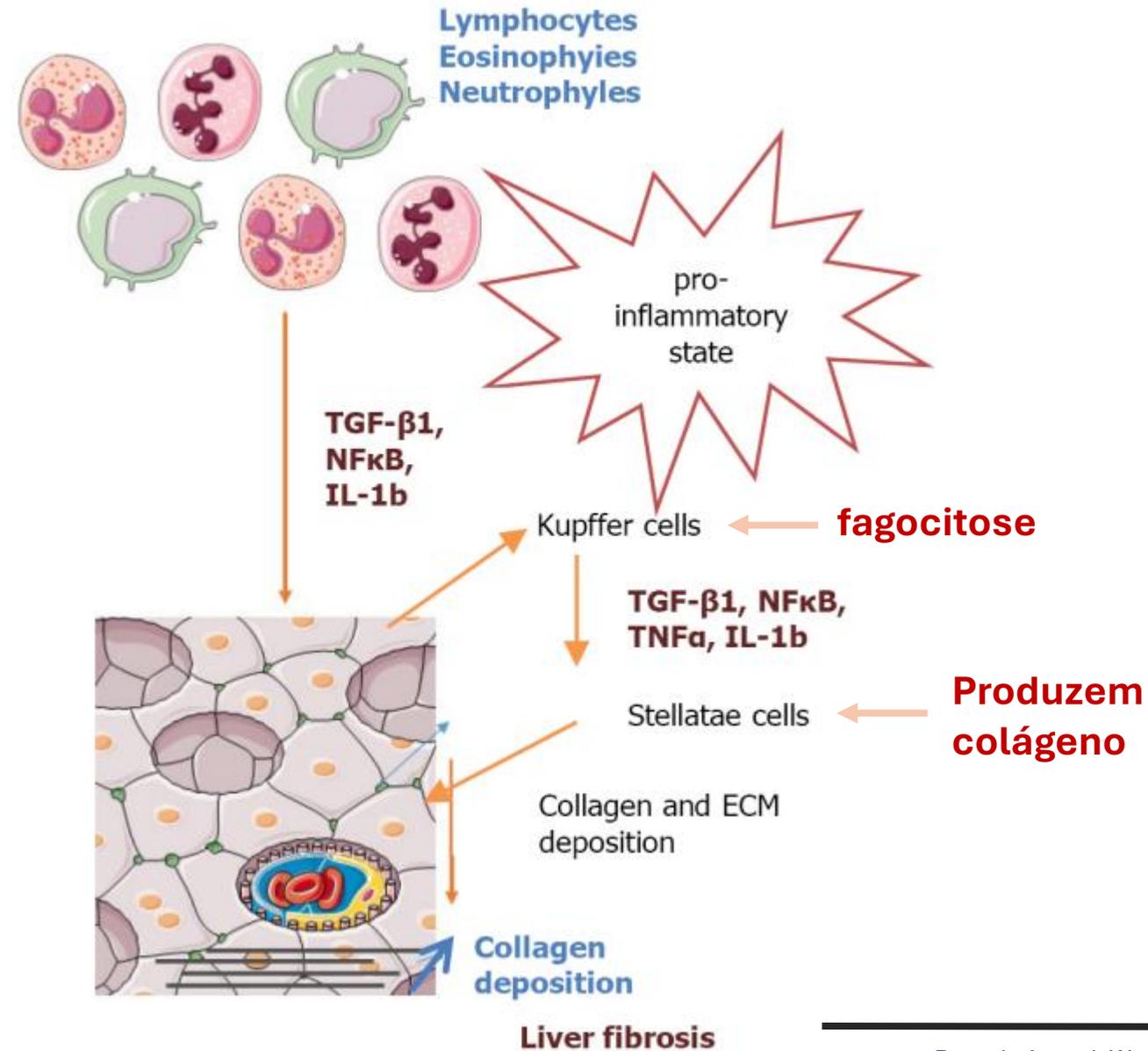


**Estresse oxidativo**

**ROS**



# Estado pró-inflamatório



**Table 1 Different Patterns of Clinical Presentation and Outcomes Associated With AAS Intake.**

Patient details	Case 1	Case 2	Case 3	
Age and gender	31 years, male	24 years, male	36 years, male	
Comorbidities	Diabetes mellitus	None	None	
Type and dosage of AAS intake	Oxymetholone, 50 mg twice a day	Intramuscular testosterone decanoate, fortnightly Oral stanozolol, once daily	Intramuscular nandrolone decanoate, once a week	
Duration of consumption	2 months	3 years	2 months	
Clinical presentation	Progressive jaundice with pruritus, pale stool	Right hypochondrial pain, nausea	Jaundice, right hypochondrial pain	
Blood tests at presentation				
Total bilirubin (mg/dL)	35.6	<b>0.2-1.0 mg/dL</b>	0.6	7.4
Direct bilirubin (mg/dL)	24.7	0.3	4.8	
AST (U/L)	34	30	387	
ALT (U/L)	40	28	498	
ALP (U/L)	294	110	170	
GGT (U/L)	64	28	30	
Albumin (g/dL)	3.7	4.5	3.8	
Platelets (lakh/cumm)	4.2	3.3	4.8	
INR	0.99	1.0	1.8	<b>0.8-1.0</b>
Other investigations	HAV, HBV, HCV and HEV: negative. ANA, ASMA, AMA, anti-LKM antibodies: negative. Serum ceruloplasmin levels and 24-h urinary copper excretion within the normal range.	HBV and HCV: negative. Alpha-fetoprotein level: 4.5 ng/mL (normal).	HAV, HBV, HCV and HEV: negative. ANA, ASMA, AMA, anti-LKM antibodies: negative. Serum ceruloplasmin levels and 24-h urinary copper excretion within the normal range.	
Type of hepatotoxicity	Cholestatic	Hepatocellular carcinoma with multiple adenomas	Steatohepatitis	

# AAS DILI



**Maior chance de lesão hepático com administração oral**

## Anabolic androgenic steroid-induced liver injury

	Hepatocellular (n = 15)	Cholestatic (n = 10)	P value
Age, mean years (range)	32 (20–42)	32 (20–49)	0.80
Compound, n (%)			0.48
Stanozolol	12 (80)	5 (50)	
Methylepitiostanol	3 (20)	4 (40)	
Methasterone	–	1 (10)	
Administration route, n (%)			0.88
Oral	11 (73)	8 (80)	
Intramuscular	4 (27)	2 (20)	
Clinical information			
Daily dose, mean mg (range)	71 (32–150)	38 (1–54)	0.53
Duration of therapy, mean days (range)	81 (23–338)	53 (16–111)	0.65
Time to onset, mean days (range)	84 (13–338)	54 (7–123)	0.92
Jaundice, n (%)	13 (87)	10 (100)	0.42
Hospital admission, n (%)	9 (60)	9 (90)	0.12
Severity			0.017
Mild	2 (13%)	–	
Moderate	13 (87%)	3 (30%)	
Severe	–	7 (70%)	

> J Clin Exp Hepatol. 2025 May-Jun;15(3):102506. doi: 10.1016/j.jceh.2025.102506. Epub 2025 Jan 21.

# Stanozolol-induced Liver Injury: A Distinctive Cholestatic Clinical and Biochemical Phenotype at Presentation

Vinícius Nunes<sup>1 2</sup>, Maria I Schinoni<sup>1</sup>, Fernando Bessone<sup>3</sup>, Maria I Lucena<sup>4</sup>,  
Inmaculada Medina-Cáliz<sup>4</sup>, Nelia Hernandez<sup>5</sup>, Maria C Moura Costa<sup>1</sup>, Isadora Lins<sup>1</sup>,  
Ana J Cardoso<sup>6</sup>, Barbara Freire<sup>7</sup>, Leonardo Schiavon<sup>8</sup>, Marcelo Silva<sup>1</sup>, Eduardo R Cançado<sup>3</sup>,  
Raymundo Paraná<sup>1 2</sup>

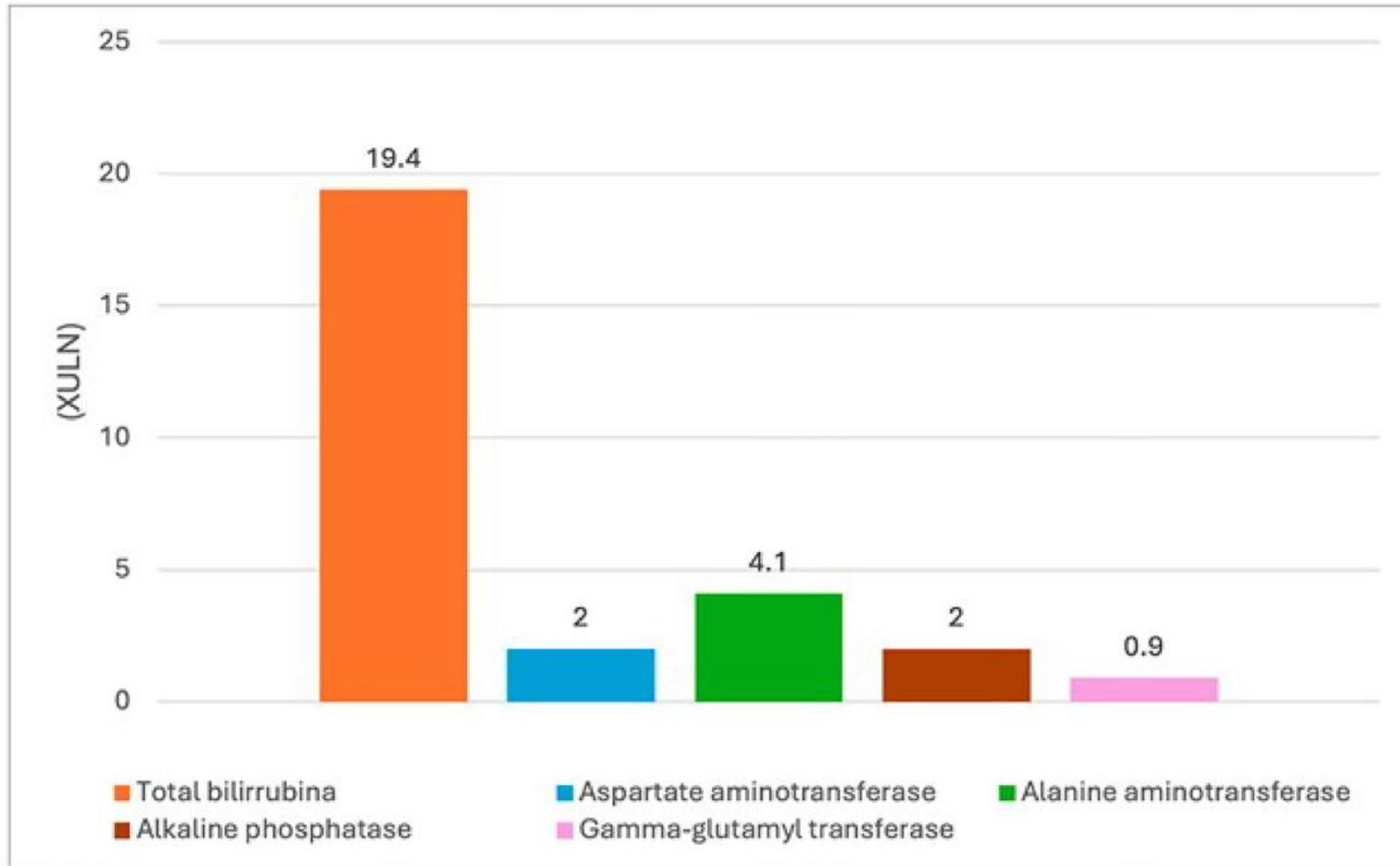
Affiliations + expand

PMID: 40040852 PMCID: PMC11872475 (available on 2026-05-01)

DOI: 10.1016/j.jceh.2025.102506



# Função hepática vs. lesão estrutural (Colestase branda)



Não foi documentado lesões hepáticas estruturais

# AAS DILI

**2017-2023**

**TABLE 1** Patient characteristics and drug/supplement combinations.

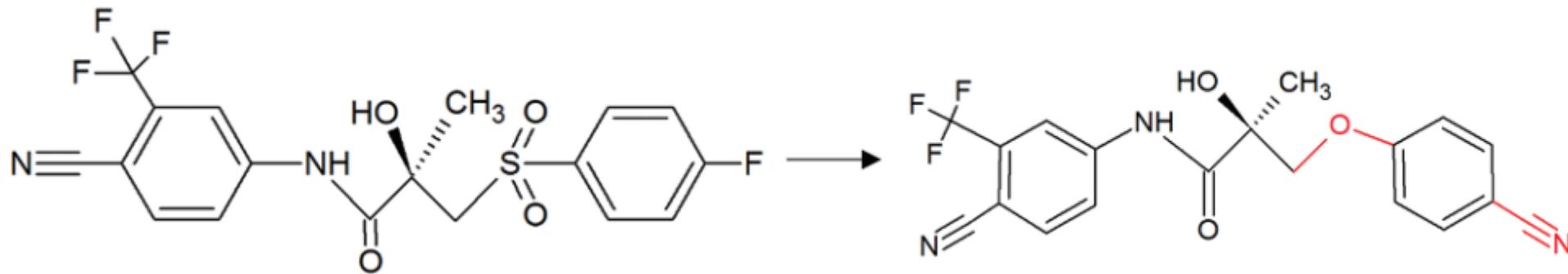
Characteristic	All patients (n = 23)	AAS only (n = 11)	SARMs only (n = 7)	AAS vs SARMs, p value	BBS only (n = 2)	Combination (n = 3)
Age, years (median, IQR)	30 (26–42)	30 (25–38)	30 (24–46)	0.66	41 (40–41)	29 (26–29)
Male, n (%) <sup>a</sup>	22 (96)	11 (100)	7 (100)	1.00	1 (50)	3 (100)
Symptomatic, n (%)	21 (91)	10 (91)	6 (86)	0.86	2 (100)	3 (100)
Latency of onset, days (median, IQR)	58 (28–112)	58 (20–90)	44 (28–60)	0.60	135 (60–135)	112 (21–112)
Time to normalisation of liver biochemistry, days (median, IQR)	175 (70–292)	75 (64–138)	236 (125–308)	0.04	257 (196–257)	216 (39–216)
Hospitalisation for DILI, n (%)	17 (74)	8 (73)	5 (71)	1.00	1 (50)	3 (100)
Length of stay, days, median (IQR)	7 (5–13)	6 (4–21)	11 (6–13)	0.52	11 (11–11)	6 (6–6)
Death due to liver injury, n	0	0	0	1.00	0	0
Liver transplantation, n (%)	1 (4)	0	0	1.00	0	1 (33)

# AAS DILI

TABLE 2 Names of drugs used.

AAS (n = 18)	SARMs (n = 14)
Testosterone (6/18)	Ligandrol (5/14)
Trenbolone (3/18)	Testolone (5/14)
Oxymetholone (2/18)	Ostarine (2/14)
Nandrolone (2/18)	Unspecified (2/14)
Oxandrolone (1/18)	
Boldenone (1/18)	
Methandrostenolone (1/18)	
Stanozolol (1/18)	
Unspecified (1/18)	

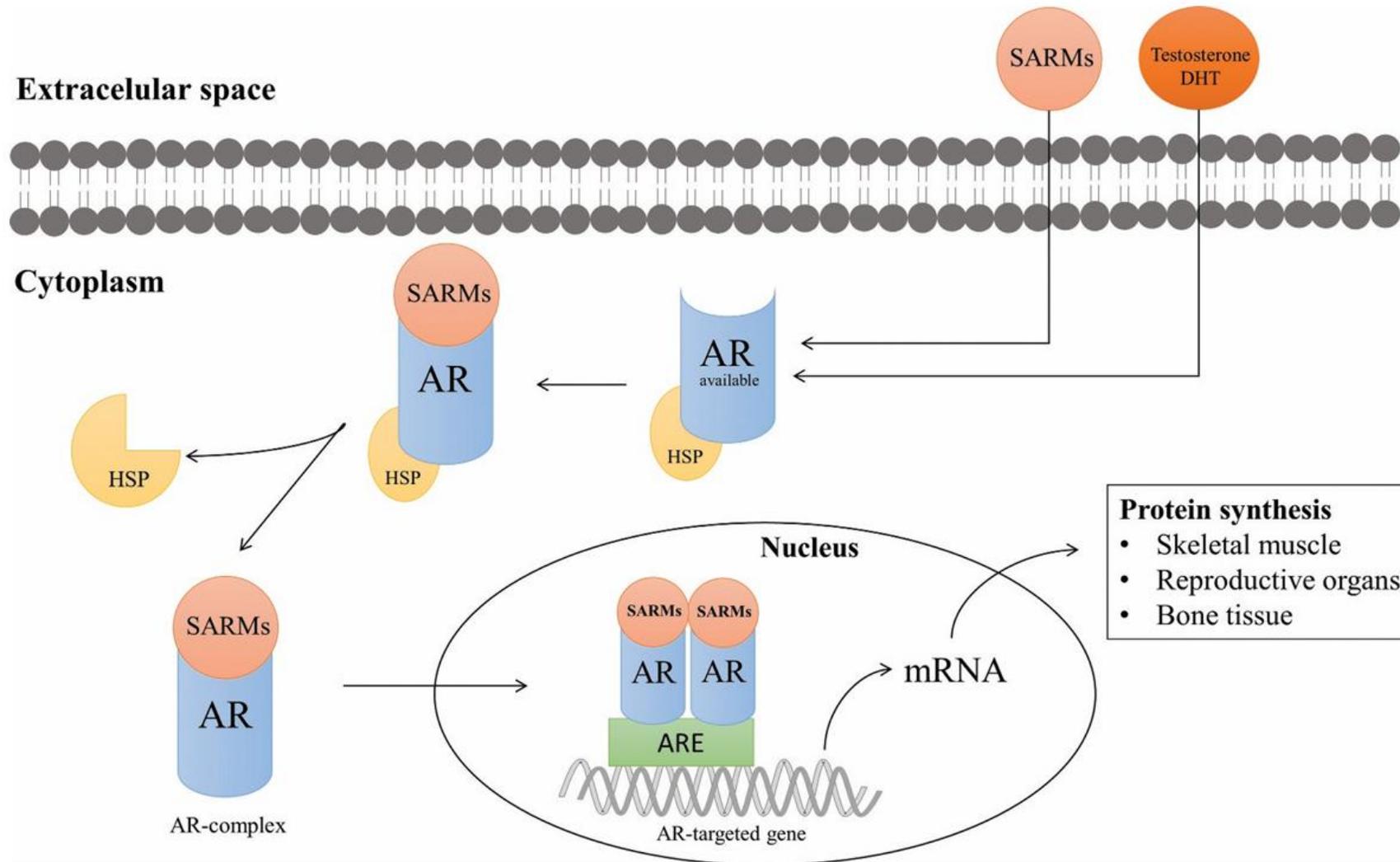
a



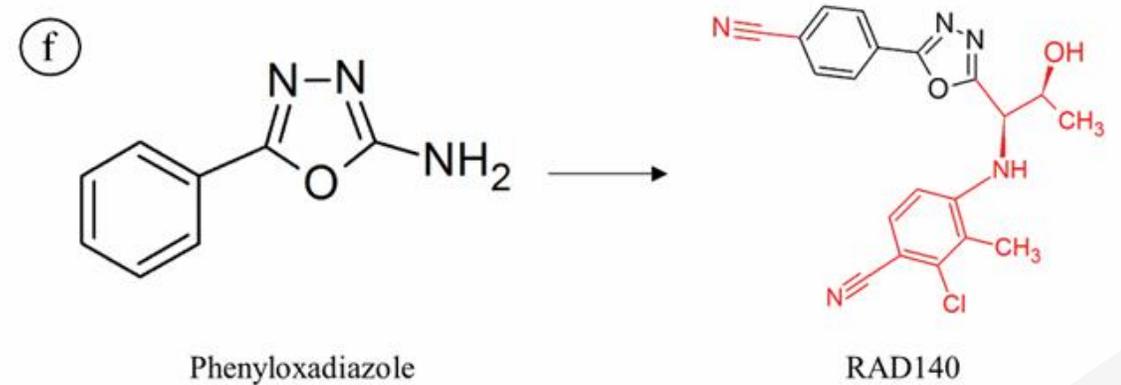
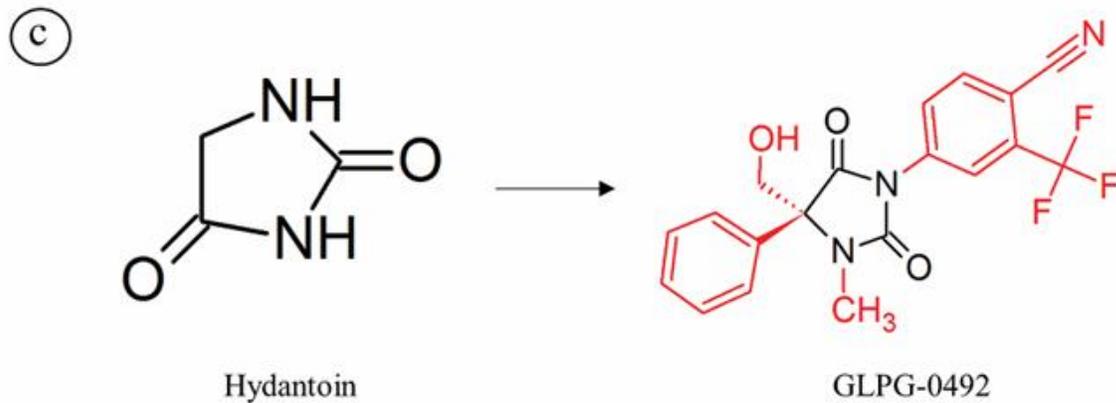
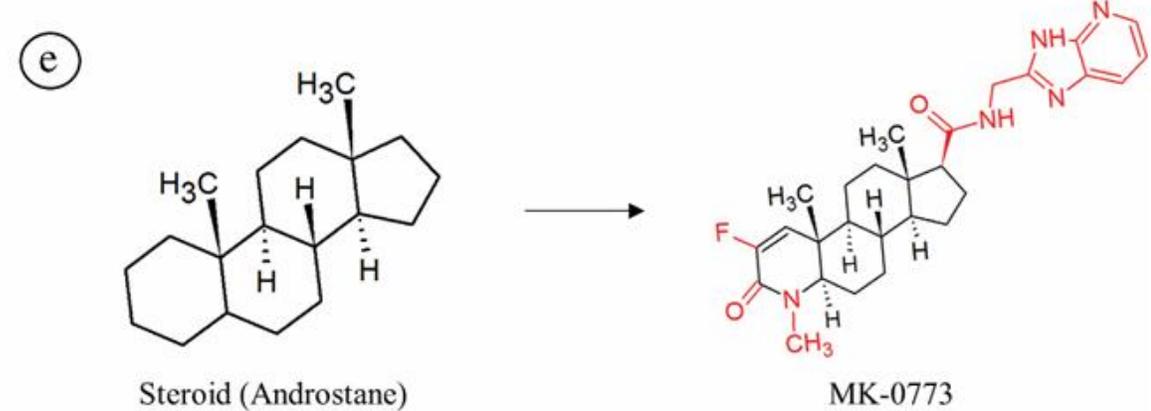
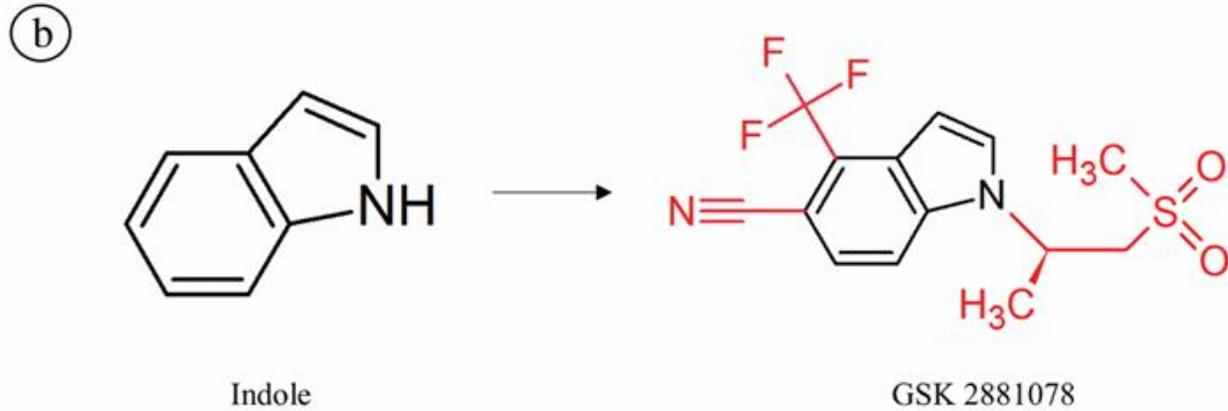
Bicalumatide

Enobosarm

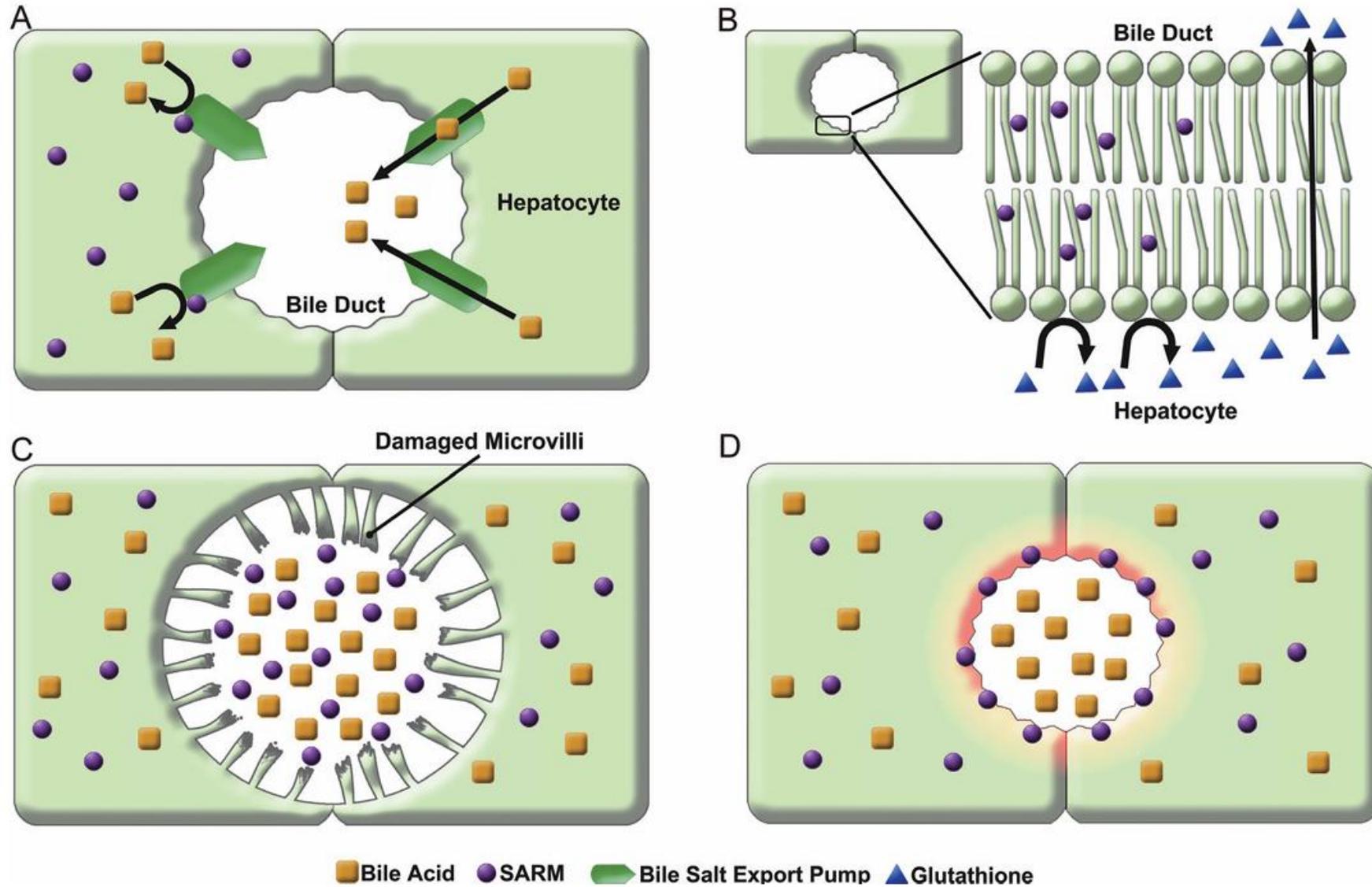
# SARMs e sarcopenia/caquexia



# Selective androgen receptor modulators (SARMs)



# Selective androgen receptor modulators (SARMs)



# AAS DILI

TABLE 4 Histopathological patterns of drug-induced liver injury, by drugs taken per patient.

AAS (n=4)	SARMs (n=5)	BBS (n=1)	Combinations (n=3)
<ul style="list-style-type: none"> <li>• Cholestatic hepatitis (n=1)</li> <li>• Acute cholestasis (n=1)</li> <li>• Hepatocellular carcinoma (well-differentiated) and focal nodular hyperplasia (n=1)</li> <li>• Hepatocellular adenoma with atypical features and focal nodular hyperplasia (n=1)</li> </ul>	<ul style="list-style-type: none"> <li>• Chronic cholestasis (n=2)</li> <li>• Cholestatic hepatitis (n=2)</li> <li>• Mixed acute hepatitis and chronic cholestasis (n=1)</li> </ul>	<p>Acute hepatitis (n=1)</p>	<ul style="list-style-type: none"> <li>• Acute cholestasis (AAS &amp; SARM &amp; BBS) (n=1)</li> <li>• Cholestatic hepatitis (AAS &amp; BBS) (n=1)</li> <li>• Cholestatic hepatitis and focal nodular hyperplasia (SARM &amp; BBS) (n=1)</li> </ul>

Abbreviations: AAS, anabolic-androgenic steroids; BBS, bodybuilding supplements; SARMs, selective androgen receptor modulators.

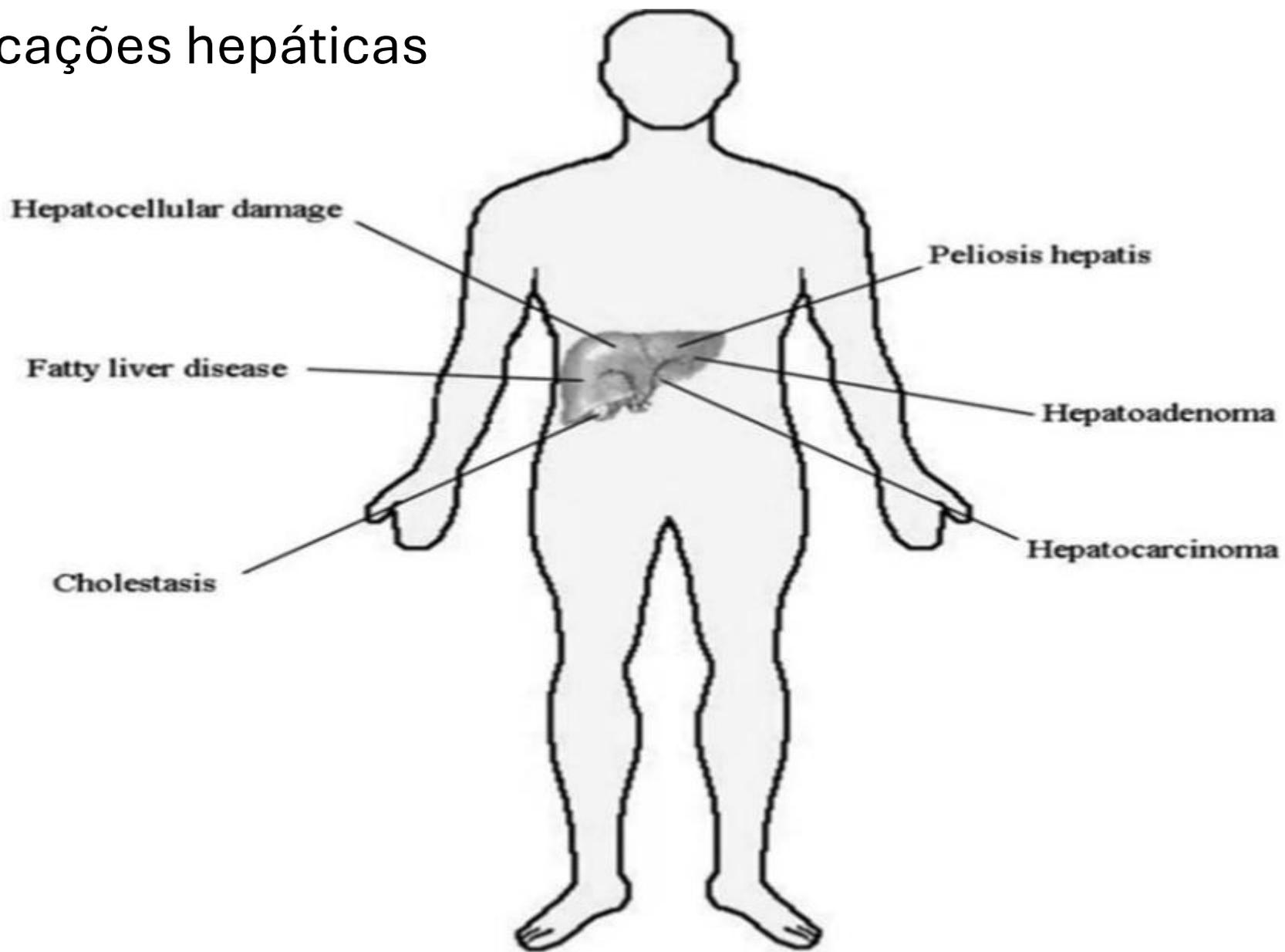
# AAS DILI

**Assintomáticos = 21/23**

**TABLE 3** Laboratory values on presentation of drug-induced liver injury and at peak.

Parameter	All patients (n = 23)		Patients using AAS only (n = 11)		Patients using SARMs only (n = 7)	
	Presentation median (IQR, maximum)	Peak (nadir <sup>a</sup> ) median (IQR, maximum)	Presentation median (IQR)	Peak (nadir <sup>a</sup> ) median (IQR)	Presentation median (IQR)	Peak (nadir <sup>a</sup> ) Median (IQR)
ALP (U/L)	150 (108–190, 570)	190 (155–289, 818)	130 (97–218)	181 (112–419)	150 (108–186)	252 (185–289)
GGT (U/L)	103 (50–171, 792)	96 (61–224, 1061)	116 (46–330)	116 (60–330)	89 (48–157)	63 (48–157)
ALT (U/L)	169 (125–589, 5530)	281 (161–2731, 6954)	160 (82–412)	252 (75–2731)	229 (158–589)	229 (161–589)
AST (U/L)	91 (59–298, 3203)	150 (91–860, 5571)	66 (54–377)	133 (60–934)	111 (90–175)	141 (96–175)
Bilirubin (μmol/L)	126 (68–268, 831)	258 (77–541, 831)	83 (54–223)	175 (54–469)	95 (68–268)	268 (116–583)
Albumin (g/L) <sup>a</sup>	40 (35–45, 48)	33 (26–34, 46)	38 (35–40)	32 (27–34)	46 (41–48)	34 (26–38)
Creatinine (μmol/L)	106 (85–121, 133)	119 (94–134, 927)	97 (82–124)	119 (91–136)	115 (93–120)	115 (106–134)
INR	1.0 (0.9–1.1, 1.5)	1.1 (1.0–1.3, 2.4)	1.0 (0.9–1.1)	1.0 (0.9–1.3)	1.0 (0.8–1.0)	1.2 (1.1–1.3)
Platelets (×10 <sup>9</sup> /L) <sup>a</sup>	279 (225–416, 586)	244 (179–304, 840)	351 (197–423)	232 (168–344)	257 (229–302)	245 (196–270)

# Complicações hepáticas



**Fig. (6).** Potential harmful hepatic effects of AAS [33]. Modified from Vanberg and Atar 2010.

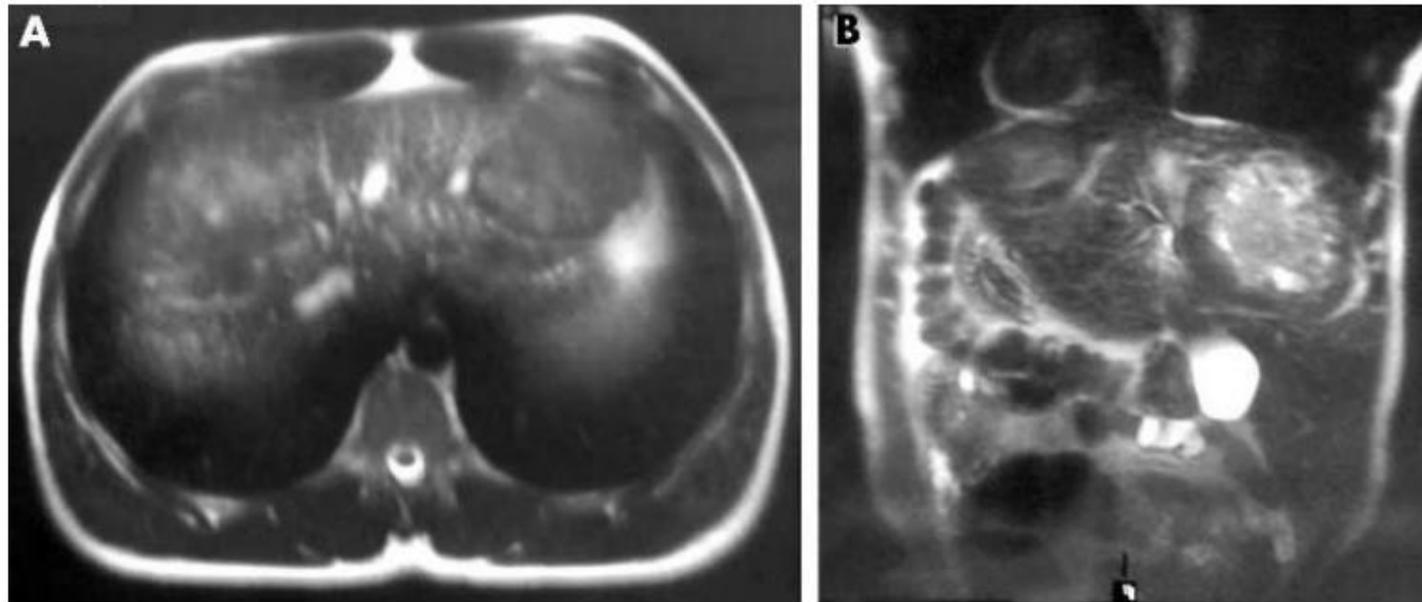
## CASE REPORT

# Hepatocellular adenomas associated with anabolic androgenic steroid abuse in bodybuilders: a report of two cases and a review of the literature

L Socas, M Zumbado, O Pérez-Luzardo, A Ramos, C Pérez, J R Hernández, L D Boada

---

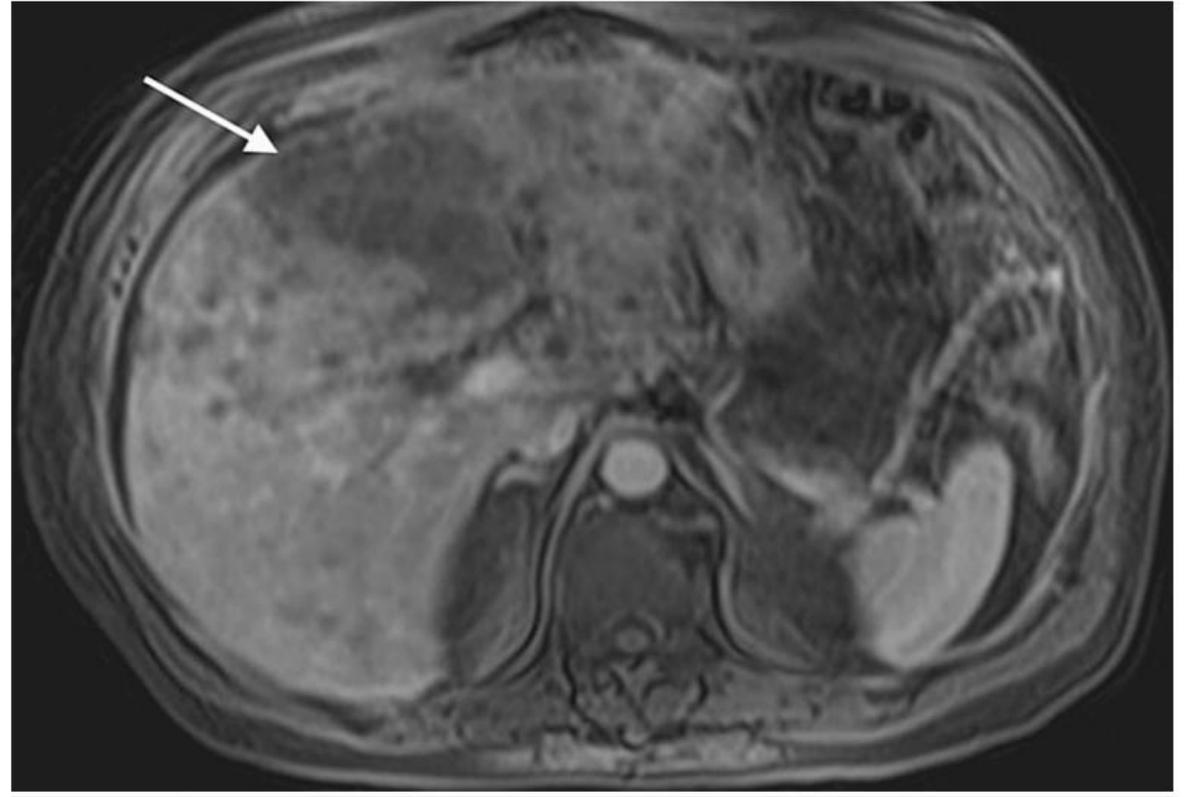
*Br J Sports Med* 2005;**39**:e27 (<http://www.bjsportmed.com/cgi/content/full/39/5/e27>). doi: 10.1136/bjism.2004.013599



**Figure 2** Magnetic resonance imaging showing heterogeneous signal in both liver tumours of the first patient.

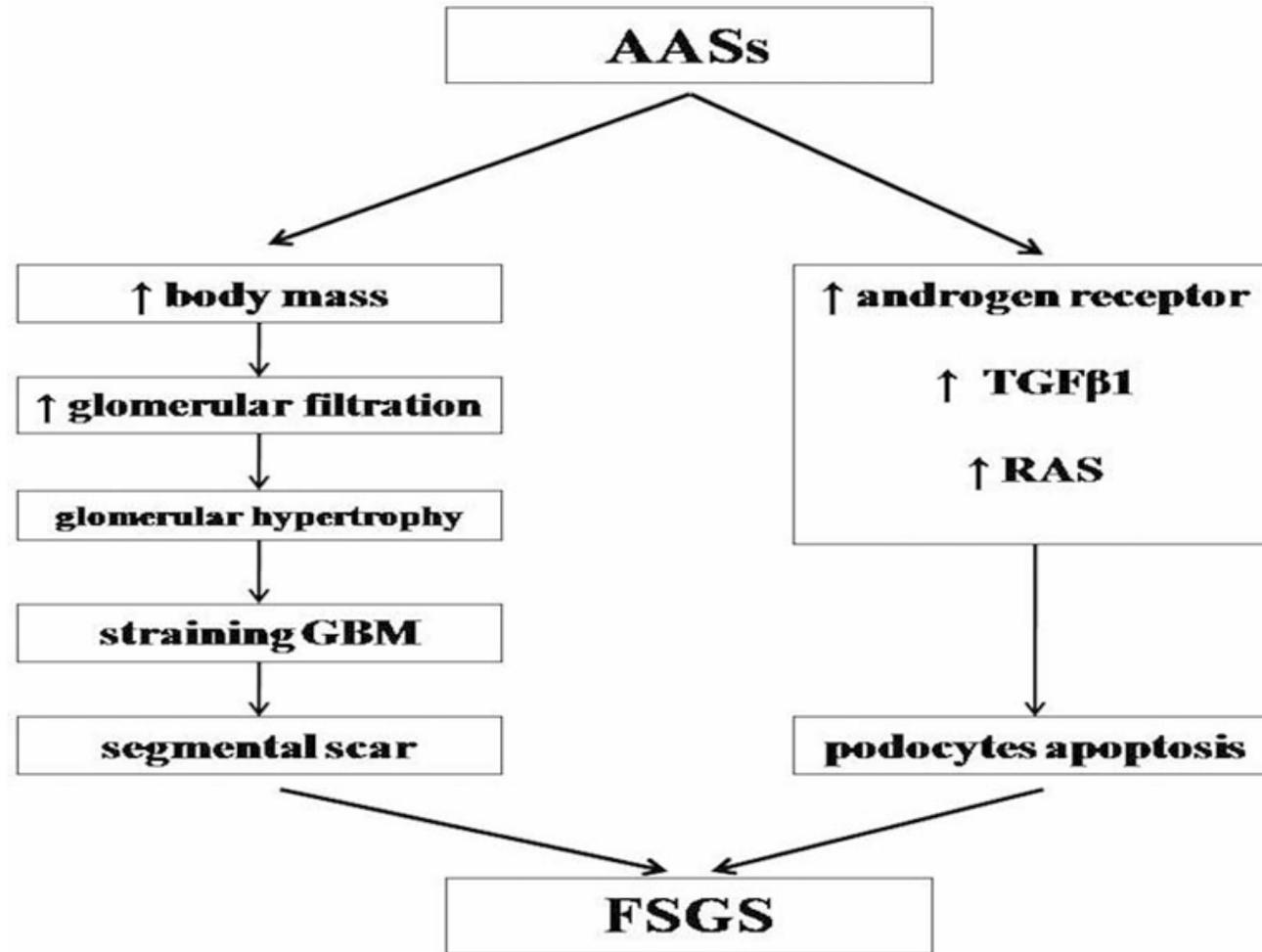
# Development of Liver Cancers as an Unexpected Consequence of Anabolic Androgenic Steroid Use

Sameeha Khalid<sup>1</sup>, Gieric Laput<sup>2</sup>, Kamal Khorfan<sup>2</sup>, Marina Roytman<sup>2</sup>



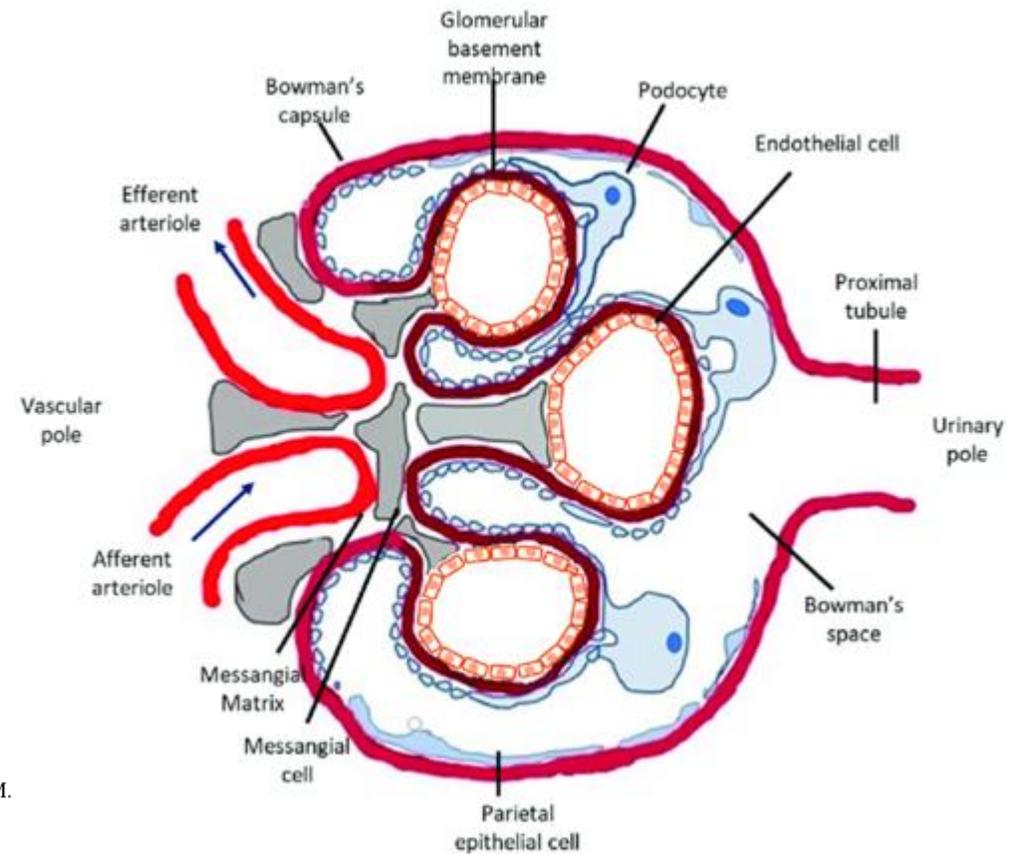
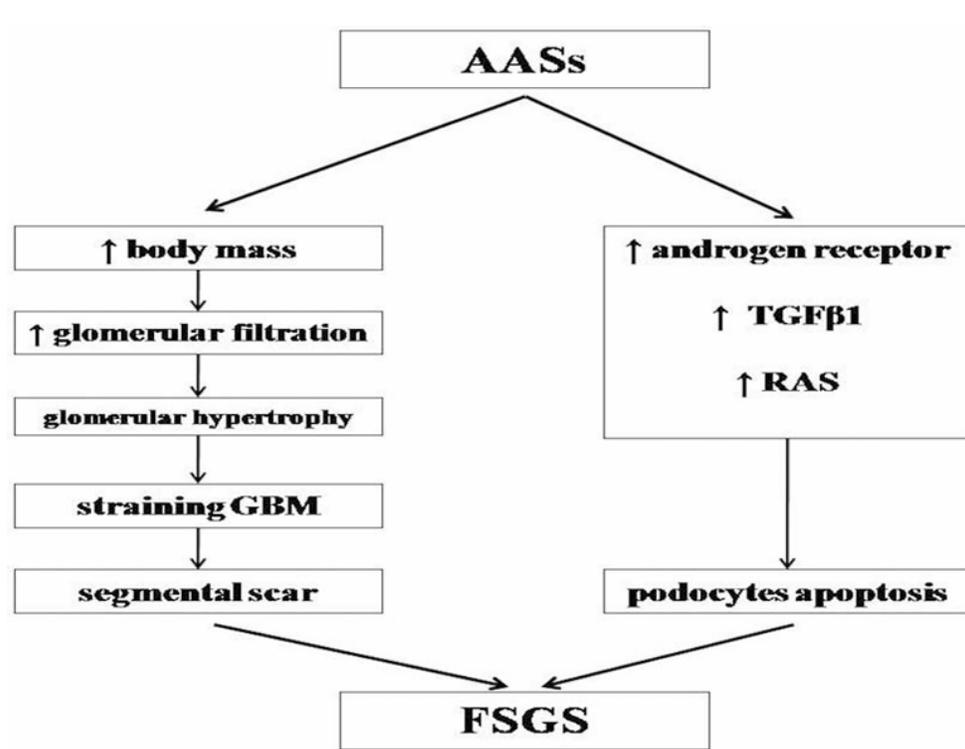
Studies to date indicate that most of AAS-induced tumors occur with use of agents containing a **17-alpha-alkyl group**

# EAA e função renal



**Fig. (1).** AAS-related nephrotoxicity (FSGS). Postadaptive and direct renal damage. [FSGS. Focal segmental glomerulosclerosis. GBM. Glomerular basement membrane. RAS. Rennin-angiotensin system].

# Apoptose de podócitos



**Fig. (1).** AAS-related nephrotoxicity (FSGS). Postadaptive and direct renal damage. [FSGS. Focal segmental glomerulosclerosis. GBM. Glomerular basement membrane. RAS. Rennin-angiotensin system].

Quando os podócitos sofrem apoptose em excesso, isso compromete a integridade da barreira de filtração glomerular, levando a **proteinúria e, eventualmente, à esclerose glomerular (nefropatia diabética, hipertensão glomerular, EAA)**

# EAA e função renal

	AAS+DS	DS	P-value
Urine protein/creatinine ratio(<150mg/day)	69.79±33.44	57.98±15.31	0.006 **
Urine albumin/creatinine ratio (<30 mg/day)	7.47±12.4	4.73±5.64	<0.001
Serum human cystatin C (mg/L) (0.28-2.2)	0.91±0.13	0.88±0.16	0.263 *
eGFR <sub>Cr</sub>	119.67±24.12	122.08±18.03	0.426 *
eGFR <sub>cys</sub> /eGFR <sub>Cr</sub>	121.83±20.62	126.33±21.163	0.036 *
eGFR <sub>cys</sub>	120.67±19.48	127.33±27.526	0.039 **

# Glomeruloesclerose segmentar e focal (FSGS)

- **Glomérulos com esclerose segmentar** (uma parte do glomérulo está colapsada ou fibrosada).
- Está relacionada à **disfunção dos podócitos**;
- Tem maior risco de progressão para **insuficiência renal**.

# Alterações renais (experimental)

Article	Treatment	Rodents	Results
Hoseini et al., 2009	DECA (3 mg/kg)	Female bulb-c mice	↑ Kidney Weight and Volume ↑ The volume of the distal and proximal convoluted tubules (DCT e PCT)
Frankenfeld et al., 2014b	DECA (1 mg.100 g <sup>-1</sup> /Kg)	Adult male Wistar	↓ CAT ↑↑ Carbonyls ↓ Thiol residues ↑ TBARS
Riezzo et al., 2014	DECA (3,75 mg/Kg/week) (10 mg/Kg/week)	Male CD1 mice	↓GR e GPx ↑ IL-1β, TNF-α, HSP90 Renal structural damage
Tsitsimpikou et al., 2016	DECA (4 mg/Kg/2× per week) (10 mg/Kg/2× per week)	Male Rabbits	↑ Urea e Creatinine ↑ TBARS ↓ GSH Renal structural damage
Dornelles et al., 2017	BU e ST (1,25 mg/Kg) (2,5 mg/Kg) (5 mg/Kg)	Male Wistar	↑ EROs e TBARS ↓ GSH e T-SH
Kahal and Allem, 2018	DECA (30 mg/Kg/week)	Male mice	Renal structural damage
Tofighi et al., 2018	DECA (10 mg/Kg)	Male Wistar	↑ Cistatin C e Urea ↓ Creatinine Clearance ↑ Gene expression of nephrin and podocin ↑ 8-OHdG

- Aumento do **peso e volume renal**
- Aumento do volume de tubos contorcidos **distais e proximais**
- Aumento de **Cistatina C**

# Alterações renais (clínico)

Article	Treatment	Human	Results
<a href="#">Kantarci et al., 2018</a>	Testosterone enanthate DECA Methandrostenolone BU	Young Athletes	↑ BUN e Creatinine ↑ Renal Tissue Volume Renal structural damage  Focal segmental glomerulosclerosis Nephroangiosclerosis Chronic interstitial nephritis
<a href="#">El-Reshaid et al., 2018</a>	Testosterone injections up to 250 mg/day Multiple AAS	Adult males	Acute interstitial nephritis Nephrocalcinosis with chronic interstitial nephritis
<a href="#">Ali et al., 2020</a>	Testosterone propionate Nandrolone twice a week 250	Adult males	Focal segmental glomerulosclerosis Membranous glomerulonephritis

- Aumento do **volume renal**
- Nefrites
- FSGS

# Complicações do sistema reprodutor

**Table 1. Adverse Effects of AAS**

ORGAN/APPARATUS	ADVERSE EFFECTS OF ANABOLIC STEROIDS
Reproductive	<b>Male:</b>  decreased reproductive hormones testicular atrophy oligospermia/azoospermia impotence prostatic hypertrophy prostatic carcinoma gynecomastia priapism
	<b>Female:</b>  menstrual irregularities clitoral hypertrophy uterine atrophy breast atrophy teratogenicity