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FEEDING PATTERNS, SUGAR CONSUMPTION, AND METABOLIC RISK: EXPERIMENTAL EVIDENCE WITH TRANSLATIONAL IMPLICATIONS

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ABSTRACT
High intake of simple carbohydrates and the adoption of time-restricted feeding patterns have been identified as modulators of energy metabolism and risk factors for metabolic syndrome. This study aimed to evaluate the effects of time-restricted feeding and sucrose supplementation on hepatic glycogen, lipid profile, and oxidative stress in adult rats. An experimental study was conducted with 24 male Wistar rats, randomly allocated into four groups: ad libitum diet (AD), time-restricted feeding (RT), ad libitum diet with sucrose (ADS), and time-restricted feeding with sucrose (RTS). After 30 days of intervention, serum biochemical parameters, oxidative stress markers, enzymatic activity, and hepatic glycogen concentration were assessed. Results showed that restricted feeding reduced total protein and cholesterol but led to decreased HDL-cholesterol and increased LDL-cholesterol. Sucrose supplementation elevated triglycerides and VLDL-cholesterol. All experimental groups exhibited increased oxidative stress markers, reduced endogenous antioxidant defenses, and higher gamma-glutamyl transferase activity. Hepatic glycogen was reduced in the RT and RTS groups, while alkaline phosphatase activity was elevated, suggesting hepatic impairment. In conclusion, both time-restricted feeding and sucrose intake induced dyslipidemia, oxidative stress, and hepatic alterations consistent with the metabolic syndrome phenotype. These findings highlight the importance of understanding the metabolic effects of different dietary regimens and their implications for cardiovascular disease prevention.
Keywords: Time-restricted feeding; Sucrose; Oxidative stress; Metabolic syndrome; Hepatic metabolism.

1. INTRODUCTION

Carbohydrate-rich diets play a central role in the development of obesity and are frequently associated with hyperphagia, increased fat accumulation, and the onset of metabolic disorders (Li *et al*., 2022). Epidemiological evidence indicates that visceral obesity represents a major risk factor for cardiovascular disease, particularly due to its association with insulin resistance, diabetes mellitus, and dyslipidemia - classical components of metabolic syndrome (Tian *et al*., 2025; Xu *et al*., 2025).

Metabolic syndrome is characterized by the presence of at least three clinical factors, including arterial hypertension, abdominal obesity, hyperglycemia, hypertriglyceridemia, and reduced HDL-cholesterol. Its occurrence is directly linked to increased cardiovascular mortality and the early progression of atherosclerosis (Peterseim *et al*., 2024; Zhang *et al*., 2025). Understanding the pathophysiological mechanisms that connect inadequate dietary habits with biochemical alterations is therefore essential for the prevention of these conditions (Nurkolis *et al*., 2025).

Experimental studies have demonstrated that time-restricted feeding, even without reducing total caloric intake, modifies energy metabolism (Deng *et al*., 2025). In animal models, this regimen results in changes in hepatic glycogen, lipid profile, and the production of reactive oxygen species, indicating that both prolonged fasting and excessive carbohydrate consumption can trigger oxidative stress and metabolic dysfunctions (Zanol *et al*., 2022).

Given the relevance of sucrose intake and dietary restriction as modulators of energy homeostasis and cardiovascular risk, this study aimed to evaluate the effects of time-restricted feeding and sucrose consumption on hepatic glycogen, lipid profile, and serum oxidative stress in rats subjected to an experimental protocol.

2. METHOD

2.1 Study design

This was an experimental study conducted in an animal model, aiming to evaluate the effects of time-restricted feeding and sucrose supplementation on metabolic parameters, including lipid profile, oxidative stress markers, and hepatic metabolism in adult rats.

2.2 Animals and housing conditions

A total of 24 adult male Wistar rats, with an average initial weight of 220 g, were obtained from the Central Animal Facility of UNESP – Botucatu Campus. The animals were housed individually in cages under controlled conditions of temperature (21 ± 1 °C), humidity (60 ± 5%), and a 12-hour light/dark cycle, with free access to filtered water. All procedures were conducted in accordance with current ethical guidelines for animal experimentation and were approved by the Animal Ethics Committee of UNESP - Institute of Biosciences, Botucatu Campus (Protocol No. 1183/2018).

2.3 Experimental groups

The animals were randomly assigned to four groups (n = 6 each):

- **AD (control):** standard diet ad libitum.
- **RT**: standard diet provided under time-restricted feeding (2 h daily, from 9 a.m. to 11 a.m.).
- **ADS**: standard diet ad libitum + 30% aqueous sucrose solution as drinking fluid.
- **RTS**: standard diet under time-restricted feeding (2 h daily) + 30% sucrose solution ad libitum.

The intervention lasted for a total of 30 days.

2.4 Experimental procedures

Throughout the experimental protocol, food and liquid intake (water or sucrose solution) were monitored daily. At the end of 30 days, the animals were euthanized by cervical dislocation followed by decapitation. Blood samples were collected by puncture after decapitation for serum biochemical analyses, and liver fragments were harvested for glycogen quantification using an enzymatic method.

2.5 Variables analyzed

The following parameters were assessed:

- **Serum biochemical parameters**: total proteins, glucose, triglycerides, total cholesterol, HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol, and oxidized LDL.
- **Oxidative stress markers**: lipid hydroperoxides, lipoperoxides, total antioxidant substances (TAS), reduced glutathione (GSH), oxidized glutathione (GSSG), and total reducing power.
- **Serum enzymatic activities**: gamma-glutamyl transferase (GGT), nitric oxide synthase (NOS), and alkaline phosphatase (ALP).
- **Hepatic metabolism**: hepatic glycogen concentration.

2.6 Statistical analysis

Data were expressed as mean ± standard error of the mean (SEM). Comparisons between groups were performed using two-way analysis of variance (ANOVA) with a 2×2 factorial design, followed by Tukey's post hoc test for multiple comparisons. Additional analyses were conducted, including Student's t-test for specific comparisons and Pearson's linear correlation when appropriate. A significance level of 5% ($p < 0.05$) was adopted.

3. RESULTS

The effects of time-restricted feeding and sucrose supplementation on animal metabolism were analyzed with respect to serum lipid and protein profiles, oxidative stress markers, and hepatic metabolism. The main changes observed are presented in the following three tables, organized by thematic blocks of analysis.

3.1 Serum lipid and protein profile

Total protein concentration was reduced in animals subjected to restricted feeding (RT) compared with the control group (AD), although values remained within physiological ranges. In the sucrose-supplemented group (ADS), a significant increase was observed in relation to the RTS group. Regarding the lipid profile, time-restricted feeding (RT) led to a reduction in total cholesterol, whereas sucrose supplementation (ADS and RTS) did not alter this parameter. HDL-cholesterol concentrations decreased in the RT, ADS, and RTS groups compared with the control, while LDL-cholesterol levels increased in these same groups. In addition, triglycerides and VLDL-cholesterol were higher in the ADS group, in contrast to the reduction observed in RT. These results are summarized in Table 1, which provides a comparative overview of serum protein and lipid profile values among the experimental groups.

Table 1. Concentrations of total proteins (TP), total cholesterol, HDL-cholesterol (HDL-C), LDL-cholesterol (LDL-C), triglycerides (TG), and VLDL-cholesterol (VLDL-C) in animals from the control group (AD), time-restricted feeding group (RT), sucrose-supplemented group (ADS), and time-restricted feeding with sucrose group (RTS).

	Groups			
	AD	RT	ADS	RTS
TP (g/dL)	8,5±1,0^{bc}	7,0±0,7^{a}	8,9±0,8^{c}	7,2±0,6^{ab}
Total cholesterol (mg/dL)	103,0±17,8^{a}	109,5±16,4^{a}	102,3±19,7^{a}	92,6±16,2^{a}
HDL-C (mg/dL)	51,2±4,3^{b}	34,2±9,5^{a}	37,1±9,1^{a}	36,4±9,0^{a}
LDL-C (mg/dL)	14,1±1,2^{a}	68,7±9,1^{c}	17,9±1,1^{b}	21,2±2,4^{b}
TG (mg/dL)	147,0±30,4^{b} 173,3±47,7^{b}	53,3±6,8^{a}	237,7±44,4^{c}	
VLDL-C (mg/dL)	29,4±6,0^{b}	10,6±1,3^{a}	47,5±8,8^{c}	34,6±9,5^{b}

Different letters indicate significant differences between groups, with $p < 0.05$.

3.2 Oxidative stress markers

Animals from the RT, ADS, and RTS groups showed a significant increase in lipid hydroperoxides, lipoperoxides, and oxidized LDL concentrations compared with the control group. A reduction in total antioxidant substances (TAS) was also observed, with the most pronounced decrease occurring in the ADS group. In parallel, decreased levels of reduced glutathione (GSH), oxidized glutathione (GSSG), and nitric oxide synthase (NOS) activity were noted. Conversely, serum gamma-glutamyl transferase (GGT) activity was elevated in all experimental groups, while total reducing power increased in the RT group and decreased in the RTS group. These findings are summarized in Table 2, which presents the main parameters related to oxidative stress across the experimental groups.

Table 2. Concentrations of lipid hydroperoxide (HP), thiobarbituric acid reactive substances (TBA), oxidized LDL (LDL-ox), total antioxidant substances (TAS), reduced glutathione (GSH), oxidized glutathione (GSSG), reducing power, gamma-glutamyl transferase (GGT), and nitric oxide synthase (NOS) in the serum of animals from the control group (AD), time-restricted feeding group (RT), sucrose-supplemented group (ADS), and time-restricted feeding with sucrose group (RTS)

	Groups			
	AD	RT	ADS	RTS
HP (nmol/mL)	4,4±0,1^{a}	5,2±0,5^{c}	5,6±0,3^{d}	4,8±0,2^{b}
TBA (nmol/mL)	0,17±0,03^{a}	0,23±0,02^{b}	0,23±0,03^{b}	0,27±0,02^{c}
LDL-ox (mmol/mg)	36,2±11,1^{a}	61,9±15,6^{b}	61,9±15,6^{b}	61,3±17,9^{b}
TAS (%)	66,4±6,5^{c}	56,2±3,5^{b}	51,7±3,2^{a}	59,5±1,0^{b}
GSH (nmol/mL)	13,3±0,3^{c}	12,9±0,1^{a}	13,0±0,1^{ab}	13,0±0,2^{ab}
GSSG (mmol/mL)	0,38±0,05^{c}	0,20±0,04^{a}	0,33±0,03^{b}	0,29±0,04^{b}
Reducing power	35,3±5,5^{a}	67,0±14,7^{c}	36,5±5,2^{a}	44,6±6,8^{b}
GGT (U/L)	29,1±4,6^{a}	51,9±7,5^{d}	45,7±5,7^{c}	38,9±4,3^{b}
NOS (µM)	11,6±3,3^{b}	7,9±1,5^{a}	7,4±2,0^{a}	8,6±1,4^{a}

Different letters indicate significant differences between groups, with $p < 0.05$.

3.3 Hepatic glycogen and alkaline phosphatase

Hepatic glycogen content was significantly reduced in the RT and RTS groups compared with AD and ADS, which did not differ from each other. In parallel, serum alkaline phosphatase (ALP) activity was increased in the RT, ADS, and RTS groups, suggesting that both dietary restriction and sucrose supplementation negatively affected hepatic metabolism. These results are presented in Table 3, which summarizes the mean values of hepatic glycogen and ALP activity among the experimental groups.

Table 3. Hepatic glycogen concentration and alkaline phosphatase (ALP) activity in the serum of animals from the control group (AD), time-restricted feeding group (RT), sucrose-supplemented group (ADS), and time-restricted feeding with sucrose group (RTS).

	Groups			
	AD	RT	ADS	RTS
Hepatic glycogen (mg/g tissue)	34,71±8,29[b] 26,08±7,40[a]	23,61±6,93[a]	34,55±5,78[b]	
ALP (U/L)	739,8±183,1[a] 1532±316,7[b]	1340±217,8[b]	1546±461,2[b]	

Different letters indicate significant differences between groups, with $p < 0.05$.

4. DISCUSSION

The results of this study demonstrate that both time-restricted feeding and sucrose supplementation induced significant metabolic alterations in adult rats, characterized by dyslipidemia, oxidative stress, and hepatic changes. These alterations are closely related to mechanisms described in the literature regarding the development of metabolic syndrome and its cardiovascular complications, reinforcing the translational relevance of the experimental models used (Masenga *et al*., 2023; Islam *et al*., 2024).

4.1 Dyslipidemia and metabolic syndrome

Short-term daily dietary restriction reduced total cholesterol concentration but was associated with decreased HDL-cholesterol and increased LDL-cholesterol, indicating an unfavorable lipid profile. A similar pattern was observed in sucrose-supplemented groups, in which triglycerides and VLDL-cholesterol increased, resembling the metabolic profile commonly found in individuals with metabolic syndrome. Previous studies in both humans and animals have shown that smaller, denser LDL particles are highly atherogenic, contributing to elevated cardiovascular risk (Herman & Birnbaum, 2021; Vekic *et al*., 2022).

Chronic consumption of refined carbohydrates, such as sucrose, promotes hepatic synthesis of triglycerides, which are transported by very-low-density

lipoproteins (VLDL-C), thereby intensifying lipid deposition in adipose tissue and promoting visceral obesity (Hunealt *et al*., 2023). Hypertriglyceridemia and the concomitant reduction in HDL-C observed in this study represent central elements of the at-risk metabolic phenotype. This profile, described in various populations, has been associated with early atherosclerosis and the progression of cardiovascular disease (Shateri *et al*., 2024; Clemente-Suárez *et al*., 2022).

The increase in serum alkaline phosphatase (ALP) activity in the RT, ADS, and RTS groups suggests hepatic repercussions of both dietary restriction and excessive carbohydrate intake. Alterations in hepatic serum enzymes are recognized as markers of cellular damage, and their elevation supports the hypothesis that inadequate dietary strategies may compromise metabolic homeostasis (Thakur *et al*., 2023; Cho & Choi, 2021). Taken together, these findings reinforce the interdependence between diet, lipid metabolism, and metabolic syndrome risk (Bo *et al*., 2024).

4.2 Oxidative stress and hepatic alterations

In addition to dyslipidemia, this study demonstrated increased oxidative stress markers in animals subjected to dietary restriction, sucrose supplementation, and their combination. Increased lipid hydroperoxides, lipoperoxides, and oxidized LDL were accompanied by reduced total antioxidant substances, suggesting an imbalance between reactive oxygen species and antioxidant defense mechanisms. This condition is widely recognized as a triggering factor for atherosclerosis and endothelial dysfunction (Arias-Chávez *et al*., 2023; Sidorova *et al*., 2025; Batty, Bennett & Yu, 2022).

Reductions in glutathione concentrations (GSH and GSSG) and nitric oxide synthase (NOS) activity indicate impaired endogenous antioxidant defenses. Glutathione depletion, in particular, is directly associated with the activation of gamma-glutamyl transferase (GGT), as observed in this experiment. Previous studies have reported the relationship between oxidative stress, increased serum GGT, and metabolic diseases, including obesity and diabetes mellitus (Mitrić & Castellano, 2023). These findings support the hypothesis that oxidative overload plays a central role in the pathophysiology of metabolic syndrome.

The liver, due to its regulatory function in energy metabolism, is particularly vulnerable to these alterations. The reduction in hepatic glycogen observed in animals subjected to restricted feeding reflects the mobilization of energy reserves during prolonged fasting, whereas the concomitant increase in ALP suggests tissue impairment. This interaction between hepatic dysfunction and oxidative stress supports the notion that both excessive sucrose intake and time-limited dietary restriction constitute risk factors for hepatic integrity and systemic metabolic balance (Schuster *et al*., 2024; Reis-Costa *et al*., 2024; Huneault *et al*., 2023). In summary, the findings indicate that different dietary regimens induce alterations consistent with the metabolic syndrome phenotype, including dyslipidemia, oxidative stress, and signs of hepatic impairment, highlighting the need to better understand the metabolic effects of inadequate dietary patterns (Islam *et al*., 2024).

5. FINAL CONSIDERATIONS

This study aimed to evaluate the effects of time-restricted feeding and sucrose supplementation on hepatic glycogen, lipid profile, and serum oxidative stress in adult rats. The results showed that both restriction and high intake of simple carbohydrates induced dyslipidemia, reduced HDL-cholesterol, and increased LDL-cholesterol, in addition to changes in oxidative stress markers and hepatic integrity. These findings reinforce the relationship between inadequate dietary patterns and the development of conditions compatible with metabolic syndrome and increased cardiovascular risk.

As a contribution, this study provides experimental evidence that helps elucidate the biochemical mechanisms through which sucrose intake and time-limited dietary restriction interfere with lipid and oxidative metabolism. The data presented here broaden the discussion on the influence of different dietary regimens on metabolic balance and may inform cardiovascular health prevention strategies with potential translational application.

However, some limitations should be considered. The use of an animal model restricts direct extrapolation of the results to human populations, as physiological differences may influence metabolic responses. Furthermore, the experimental protocol had a relatively short duration, which did not allow assessment of long-term effects. Future research in animal models with prolonged intervention periods, as well as controlled clinical studies, will be necessary to confirm and expand these findings.

REFERENCES

ARIAS-CHÁVEZ, D. J. *et al*. Combined fructose and sucrose consumption from an early age aggravates cardiac oxidative damage and causes a dilated cardiomyopathy in SHR rats. **Journal of Clinical Biochemistry and Nutrition**, v. 73, n. 3, p. 205-213, 2023. DOI: https://doi.org/10.3164/jcbn.23-2. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC10636576/. Accessed on: 25 Aug. 2025.

BATTY, M.; BENNETT, M. R.; YU, E. The role of oxidative stress in atherosclerosis. **Cells**, v. 11, n. 23, p. 3843, 30 Nov. 2022. DOI: https://10.3390/cells11233843. Available at: https://pubmed.ncbi.nlm.nih.gov/36497101/. Accessed on: 21 Sept. 2025.

BO, T. *et al*. Hepatic selective insulin resistance at the intersection of insulin signaling and metabolic dysfunction-associated steatotic liver disease. **Cell Metabolism**, v. 36, n. 5, p. 947-968, 2024. DOI: https://doi.org/10.1016/j.cmet.2024.04.006. Available at: https://www.cell.com/cell-metabolism/fulltext/S1550-4131(24)00194-6. Accessed on: 25 Aug. 2025.

CHO, Y. A.; CHOI, J. H. Association between carbohydrate intake and the prevalence of metabolic syndrome in Korean women. **Nutrients**, v. 13, n. 9, p.

3098, 2021. DOI: https://doi.org/10.3390/nu13093098. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC8465012/. Accessed on: 24 Aug. 2025.

CLEMENTE-SUÁREZ, V. J. *et al*. The burden of carbohydrates in health and disease. **Nutrients**, v. 14, n. 18, p. 3809, 2022. DOI: https://doi.org/10.3390/nu14183809. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC9505863/. Accessed on: 24 Aug. 2025.

DENG, J. *et al*. Effect of time-restricted feeding and caloric restriction in metabolic associated fatty liver disease in male rats. **Nutrition & Metabolism** (London), v. 22, n. 14, 2025. Available at: https://nutritionandmetabolism.biomedcentral.com/articles/10.1186/s12986-025-00906-3. Accessed on: 20 Aug. 2025.

HERMAN, M. A.; BIRNBAUM, M. J. Molecular aspects of fructose metabolism and metabolic disease. **Cell Metabolism**, v. 33, n. 12, p. 2329-2354, 2021. DOI: https://doi.org/10.1016/j.cmet.2021.09.010. Available at: https://www.cell.com/cell-metabolism/fulltext/S1550-4131(21)00411-9. Accessed on: 23 Aug. 2025.

HUNEAULT, H. E. *et al*. The impact and burden of dietary sugars on the liver. **Hepatology Communications**, v. 7, n. 11, e0297, 2023. DOI: https://doi.org/10.1097/HC9.0000000000000297. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC10629746/. Accessed on: 24 Aug. 2025.

ISLAM, M. S. *et al*. The interplay of factors in metabolic syndrome: understanding its roots and complexity. **Molecular Medicine**, v. 30, p. 279, 2024. DOI: https://doi.org/10.1186/s10020-024-01019-y. Available at: https://molmed.biomedcentral.com/articles/10.1186/s10020-024-01019-y. Accessed on: 20 Aug. 2025.

LI, M. *et al*. Trends in insulin resistance: insights into mechanisms and therapeutic strategy. **Signal Transduction and Targeted Therapy**, v. 7, p. 216, 2022. DOI: https://doi.org/10.1038/s41392-022-01073-0. Available at: https://www.nature.com/articles/s41392-022-01073-0. Accessed on: 20 Aug. 2025.

MASENGA, S. K. *et al*. Mechanisms of oxidative stress in metabolic syndrome. **International Journal of Molecular Sciences**, v. 24, n. 9, p. 7898, 2023. DOI: https://doi.org/10.3390/ijms24097898. Available at: https://pubmed.ncbi.nlm.nih.gov/37175603/. Accessed on: 22 Aug. 2025.

MITRIĆ, A.; CASTELLANO, I. Targeting gamma-glutamyl transpeptidase: a pleiotropic enzyme involved in glutathione metabolism and in the control of redox homeostasis. **Free Radical Biology and Medicine**, v. 208, p. 672-683, 2023. DOI: https://doi.org/10.1016/j.freeradbiomed.2023.09.020. Available at:

https://www.sciencedirect.com/science/article/pii/S0891584923003710. Accessed on: 25 Aug. 2025.

NURKOLIS, F. *et al*. New insight on dietary strategies to increase insulin sensitivity and reduce diabetes prevalence: an expert perspective and recommendation. **Discover Food**, v. 5, p. 136, 2025. DOI: https://doi.org/10.1007/s44187-025-00422-6. Available at: https://link.springer.com/article/10.1007/s44187-025-00422-6. Accessed on: 21 Aug. 2025.

PETRIDI, F. *et al*. Effects of early and late time-restricted feeding on parameters of metabolic health: an explorative literature assessment. **Nutrients**, v. 16, n. 11, p. 1721, 2024. DOI: https://doi.org/10.3390/nu16111721. Available at: https://www.mdpi.com/2072-6643/16/11/1721. Accessed on: 23 Aug. 2025.

PETERSEIM, C. M. *et al*. Metabolic syndrome: an updated review on diagnosis and treatment for primary care clinicians. **Journal of Primary Care & Community Health**, v. 15, p. 21501319241309168, 2024. DOI: https://doi.org/10.1177/21501319241309168. Available at: https://pubmed.ncbi.nlm.nih.gov/39714021/. Accessed on: 20 Aug. 2025.

REIS-COSTA, A. *et al*. The effects of long-term high fat and/or high sugar feeding on sources of postprandial hepatic glycogen and triglyceride synthesis in mice. **Nutrients**, v. 16, n. 14, p. 2186, 2024. DOI: https://doi.org/10.3390/nu16142186. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC11279633/. Accessed on: 26 Aug. 2025.

SCHUSTER, K. *et al*. Starvation in mice induces liver damage associated with autophagy. **Nutrients**, v. 16, p. 1191, 2024. DOI: https://doi.org/10.3390/nu16081191. Available at: https://www.mdpi.com/2072-6643/16/8/1191. Accessed on: 26 Aug. 2025.

SHATERI, Z. *et al*. The association between carbohydrate quality index and conventional risk factors of cardiovascular diseases in an Iranian adult population. **BMC Research Notes**, v. 17, n. 1, p. 243, 2024. DOI: https://doi.org/10.1186/s13104-024-06897-3. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC11370298/. Accessed on: 24 Aug. 2025.

SIDOROVA, Y. *et al*. The study of approaches to modeling oxidative stress in male Wistar rats: the comparative analysis of diet-induced, chemically induced, and physiologically induced models. **International Journal of Molecular Sciences**, v. 26, p. 6872, 2025. DOI: https://doi.org/10.3390/ijms26146872. Available at: https://www.mdpi.com/1422-0067/26/14/6872. Accessed on: 25 Aug. 2025.

TIAN, W. *et al*. The effects of low-carbohydrate diet on glucose and lipid metabolism in overweight or obese patients with T2DM: a meta-analysis of randomized controlled trials. **Frontiers in Nutrition**, v. 11, p. 1516086, 6 Jan. 2025. DOI: https://doi.org/10.3389/fnut.2024.1516086. Available at: https://pubmed.ncbi.nlm.nih.gov/39834467/. Accessed on: 20 Aug. 2025.

THAKUR, S. *et al*. Biomarkers of hepatic toxicity: an overview. **Current Therapeutic Research, Clinical and Experimental**, v. 100, p. 100737, 2024. DOI: https://doi.org/10.1016/j.curtheres.2024.100737. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC11163176/. Accessed on: 25 Aug. 2025.

VEKIC, J. *et al*. Atherosclerosis development and progression: the role of atherogenic small, dense LDL. **Medicina** (Kaunas), v. 58, n. 2, p. 299, 2022. DOI: https://doi.org/10.3390/medicina58020299. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC8877621/. Accessed on: 23 Aug. 2025.

XU, S. *et al*. Correlation of visceral adiposity index and dietary profile with cardiovascular disease based on decision tree modeling: a cross-sectional study of NHANES. **European Journal of Medical Research**, v. 30, n. 1, p. 123, 22 Feb. 2025. DOI: https://doi.org/10.1186/s40001-025-02340-w. Available at: https://pubmed.ncbi.nlm.nih.gov/39987443/. Accessed on: 20 Aug. 2025.

ZANOL, J. F. *et al*. High-refined carbohydrate diet alters different metabolic functions in female rats. **Molecular and Cellular Endocrinology**, v. 558, p. 111774, 1 Dec. 2022. Available at: https://www.sciencedirect.com/science/article/pii/S0303720722002222. Accessed on: 20 Aug. 2025.

ZHANG, Z. *et al*. The new definition of metabolic syndrome including hyperuricemia improves its prognostic value: results from NHANES database. **BMC Cardiovascular Disorders**, v. 25, p. 93, 2025. DOI: https://doi.org/10.1186/s12872-025-04529-7. Available at: https://bmccardiovascdisord.biomedcentral.com/articles/10.1186/s12872-025-04529-7. Accessed on: 20 Aug. 2025.