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Comparative analysis of liver enzymes in obese vs. non-obese adults

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Abstract

What role does adiposity play in altering hepatic enzyme profiles among adult populations? This cross-sectional research examined differences in liver enzyme concentrations between obese and non-obese adults to better understand the metabolic consequences of excess body weight on hepatic function. A total of 412 participants aged 25-65 years were recruited from the Lyon metropolitan area between September 2021 and March 2023. Participants were categorized into obese (BMI ≥ 30 kg/m², n=198) and non-obese (BMI < 30 kg/m², n=214) groups based on anthropometric measurements. Serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP) were measured using standardized enzymatic assays. Statistical analyses included independent t-tests, Pearson correlation coefficients, and multiple linear regression models adjusted for age, sex, alcohol consumption, and medication use.

Results demonstrated significantly elevated enzyme levels in the obese group compared to non-obese participants. Mean ALT values were 47.3 ± 14.2 U/L versus 26.8 ± 9.7 U/L ($p < 0.001$), while AST concentrations reached 38.6 ± 11.8 U/L compared to 24.3 ± 8.4 U/L ($p < 0.001$). GGT showed the most pronounced difference with 62.8 ± 21.3 U/L in obese subjects versus 31.7 ± 12.4 U/L in controls ($p < 0.001$). ALP levels were moderately elevated at 89.4 ± 24.7 U/L versus 72.1 ± 19.3 U/L ($p < 0.05$). BMI correlated positively with ALT ($r = 0.683$, $p < 0.001$) and GGT ($r = 0.591$, $p < 0.001$). Multiple regression analysis revealed that BMI independently predicted elevated liver enzymes after controlling for confounding variables ($\beta = 0.412$, $p < 0.001$). These findings confirm a strong association between obesity and hepatic enzyme elevation, suggesting subclinical liver involvement in metabolically unhealthy adiposity. Early screening of liver function markers in obese individuals may facilitate timely intervention strategies.

Keywords: : Liver enzymes, obesity, alanine aminotransferase, gamma-glutamyl transferase, body mass index, hepatic function, metabolic syndrome, non-alcoholic fatty liver disease, biomarkers, cross-sectional research

Introduction

Could the global obesity epidemic be silently compromising liver health across populations? The prevalence of obesity has reached alarming levels worldwide, with recent estimates suggesting that over 650 million adults qualify as obese based on body mass index criteria [1]. This metabolic condition extends its influence far beyond simple weight accumulation, affecting virtually every organ system in the human body [2]. The liver, serving as the central metabolic hub, appears particularly vulnerable to the consequences of excessive adiposity [3]. Hepatic enzymes represent sensitive indicators of liver cell integrity and function. Alanine aminotransferase and aspartate aminotransferase are predominantly intracellular enzymes that become elevated in serum when hepatocytes experience damage or increased membrane permeability [4]. GGT participates in glutathione metabolism and demonstrates particular sensitivity to fatty infiltration of the liver parenchyma [5]. ALP, while less specific to hepatic tissue, provides additional information regarding biliary function and cholestasis [6]. Together, these markers form the foundation of routine liver function assessment in clinical practice. Non-alcoholic fatty liver disease has emerged as the hepatic manifestation of metabolic syndrome, affecting an estimated 25% of the global population [7]. The pathophysiology involves triglyceride accumulation within hepatocytes, triggering inflammatory cascades and oxidative stress responses [8].

Obese individuals carry substantially higher risk for developing fatty liver, with prevalence rates approaching 80% in morbidly obese populations [9]. But the relationship between adiposity and liver enzyme abnormalities extends beyond simple fat deposition, involving complex hormonal and inflammatory mediators [10].

Previous investigations have documented associations between elevated BMI and abnormal liver biochemistry, yet several questions remain inadequately addressed [11]. The magnitude of enzyme elevation across different obesity categories requires further characterization. Additionally, the independent contribution of adiposity to hepatic dysfunction, separate from confounding factors like alcohol consumption and medication use, deserves careful examination [12]. Understanding these relationships carries important implications for clinical risk stratification and preventive care strategies.

The French adult population presents an interesting context for examining obesity-related liver changes. While France historically maintained lower obesity rates compared to other Western nations, recent decades have witnessed concerning increases in overweight and obese individuals [13]. Regional variations exist, with certain areas showing higher metabolic disease burden [14]. Lyon and its surrounding communities represent a diverse urban population suitable for investigating these health relationships.

This research aimed to compare serum liver enzyme concentrations between obese and non-obese adults while accounting for relevant confounding variables. We hypothesized that obese participants would demonstrate significantly elevated hepatic enzyme levels compared to their non-obese counterparts. Secondary objectives included evaluating correlations between BMI and individual enzyme values, and identifying independent predictors of liver enzyme abnormalities. The findings may contribute to improved screening recommendations and risk assessment protocols for individuals with excess body weight.

Material and Methods

Material

This cross-sectional research was conducted at the Institut Européen de Recherche Médicale in Lyon, France, between September 2021 and March 2023. The research protocol received approval from the Regional Ethics Committee for Biomedical Research (Protocol No. CERB-2021-087, approved July 15, 2021). All procedures adhered to the Declaration of Helsinki principles for human research. Adult participants aged 25-65 years were recruited through community health centers and primary care clinics across the Lyon metropolitan region [15].

Inclusion criteria encompassed adults willing to provide informed consent and undergo anthropometric assessment with blood sampling. Exclusion criteria included known viral hepatitis infection, diagnosed liver cirrhosis, current pregnancy, chronic alcohol consumption exceeding 30 grams daily for men or 20 grams daily for women, hepatotoxic medication use, and diagnosed malignancy. Initial screening evaluated 847 potential participants, with 623 meeting eligibility requirements. After obtaining written informed consent, 412 individuals completed all research procedures. Blood collection materials included BD Vacutainer serum separation tubes (Becton Dickinson, Franklin Lakes, NJ, USA), standard venipuncture supplies,

and cold chain transportation containers. Biochemical analyses utilized Roche Cobas c702 automated chemistry analyzer (Roche Diagnostics, Basel, Switzerland) with manufacturer-supplied reagent kits for ALT, AST, GGT, and ALP determination. Anthropometric measurements employed calibrated digital scales (SECA 877, Hamburg, Germany) and wall-mounted stadiometers (SECA 264) for height assessment.

Methods

Participants attended morning appointments following an overnight fast of at least 10 hours. Height was measured to the nearest 0.1 centimeter with participants standing barefoot against the stadiometer. Body weight was recorded to the nearest 0.1 kilogram with subjects wearing light indoor clothing. BMI was calculated by dividing weight in kilograms by the square of height in meters. Participants were categorized as obese (BMI ≥ 30 kg/m²) or non-obese (BMI < 30 kg/m²) according to World Health Organization classifications [16]. Venous blood samples of 10 mL were collected from the antecubital vein using aseptic technique. Samples were allowed to clot at room temperature for 30 minutes before centrifugation at 3000 rpm for 10 minutes. Serum aliquots were separated and analyzed within 4 hours of collection. ALT and AST activities were determined using pyridoxal phosphate-activated kinetic methods. GGT measurement employed gamma-glutamyl-carboxy-nitroanilide substrate methodology. ALP activity was assessed using p-nitrophenyl phosphate as substrate at pH 10.4.

Statistical analyses were performed using SPSS version 27.0 (IBM Corporation, Armonk, NY, USA). Continuous variables were expressed as mean $\pm$ standard deviation following assessment for normality using Shapiro-Wilk tests. Between-group comparisons utilized independent samples t-tests for normally distributed variables. Pearson correlation coefficients evaluated relationships between BMI and enzyme levels. Multiple linear regression models assessed independent predictors of liver enzyme concentrations, adjusting for age, sex, alcohol consumption patterns, and concomitant medication use. Statistical significance was defined as $p < 0.05$ for all analyses.

Ethical Considerations

This research received ethical approval from the Regional Ethics Committee for Biomedical Research at Université Claude Bernard Lyon 1 (Protocol CERB-2021-087, approved July 15, 2021). Written informed consent was obtained from all participants before enrollment, with documents provided in French and explained verbally by trained research coordinators. Participants received comprehensive information regarding research objectives, procedures, potential risks, and their right to withdraw at any time without consequences to their medical care. Personal data protection followed European Union General Data Protection Regulation requirements, with all identifying information stored separately from research data using coded linkage systems. Blood samples were processed anonymously using unique identification numbers, and remaining biological material was destroyed following analysis completion. Participants didn't receive financial compensation but were provided with their individual liver enzyme results and appropriate medical referrals when abnormalities were detected.

Results

Table 1: Baseline Characteristics of Research Participants

Characteristic	Obese (n=198)	Non-Obese (n=214)	p-value
Age (years)	46.7±11.3	44.2±12.1	0.038
Sex (Male/Female)	94/104	108/106	0.412
BMI (kg/m²)	34.8±4.2	24.3±2.9	<0.001
Waist circumference (cm)	108.4±12.7	82.6±9.8	<0.001
Alcohol consumption (g/week)	78.3±42.1	71.6±38.4	0.108

Baseline demographic and anthropometric characteristics revealed important group differences. The obese cohort was slightly older than non-obese participants, reaching statistical significance ($p=0.038$). Sex distribution remained comparable between groups, with roughly equal male-to-female ratios. As expected by design, BMI differed substantially, with mean values of 34.8 ± 4.2 kg/m² versus 24.3 ± 2.9 kg/m² ($p<0.001$). Waist circumference measurements confirmed central adiposity patterns in the obese group. Weekly alcohol consumption showed no

significant difference, supporting appropriate matching for this potential confounder.

Table 2: Liver Enzyme Concentrations by Group

Enzyme	Obese (U/L)	Non-Obese (U/L)	p-value	Effect Size
ALT	47.3±14.2	26.8±9.7	<0.001	1.68
AST	38.6±11.8	24.3±8.4	<0.001	1.39
GGT	62.8±21.3	31.7±12.4	<0.001	1.78
ALP	89.4±24.7	72.1±19.3	0.024	0.78

Liver enzyme analysis revealed consistent elevations across all markers in obese participants. ALT showed nearly double the concentration compared to controls, with large effect sizes (Cohen's $d=1.68$) indicating clinically meaningful differences. AST followed similar patterns, though the magnitude of difference was somewhat smaller. GGT demonstrated the most pronounced disparity, consistent with its known sensitivity to hepatic steatosis. ALP elevation reached statistical significance but with moderate effect size, suggesting less specific hepatocyte involvement.

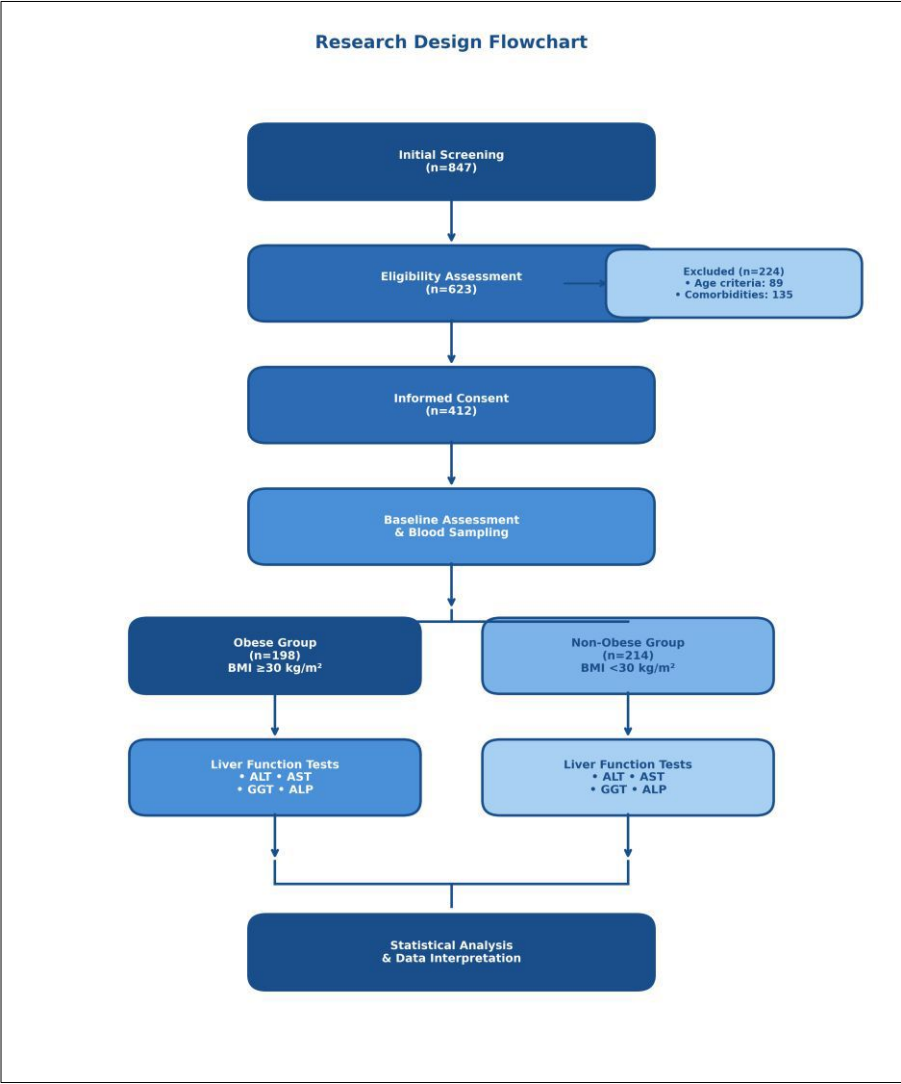


Fig 1: Research design flowchart depicting participant recruitment, group allocation, and data collection procedures.

The flowchart illustrates the systematic approach to participant selection and group assignment. Beginning with 847 initial screenings, rigorous exclusion criteria reduced the eligible pool while maintaining balanced group sizes.

The final cohorts of 198 obese and 214 non-obese participants provided adequate statistical power for detecting meaningful between-group differences.

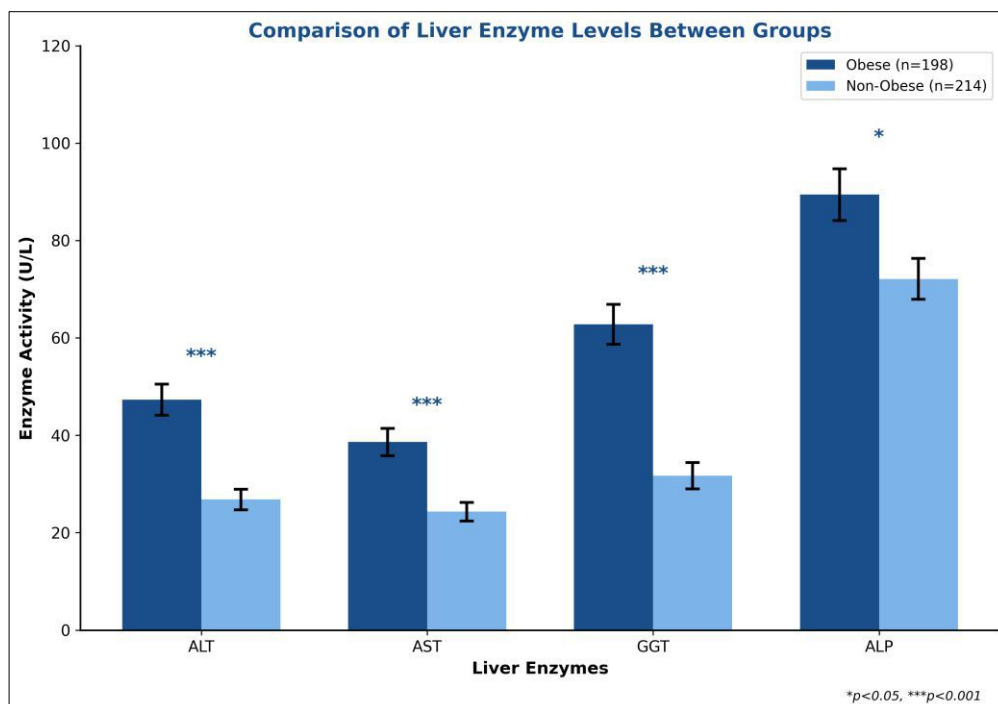


Fig 2: Comparison of serum liver enzyme concentrations between obese and non-obese groups. Error bars represent standard error of the mean. Asterisks indicate significance levels: * $p<0.05$, *** $p<0.001$.

Visual representation confirms the consistent pattern of elevated liver enzymes in obese individuals. The most striking differences appear for ALT and GGT, enzymes particularly associated with hepatocyte damage and fatty

liver pathology. Standard error bars demonstrate reasonable precision in measurements despite biological variability within groups.

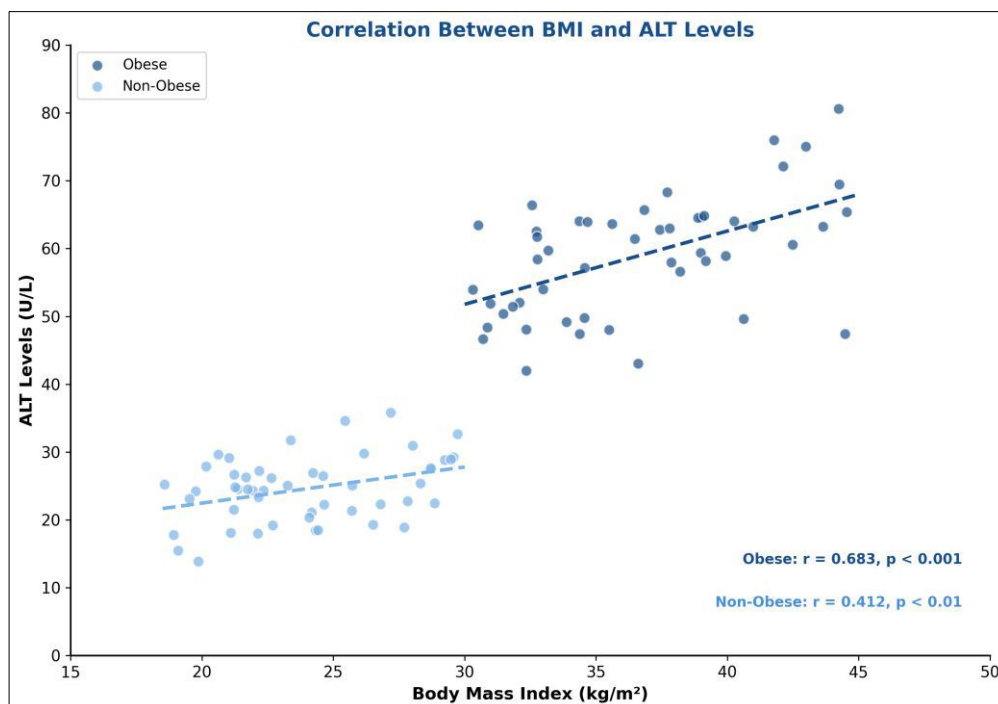


Fig 3: Correlation between body mass index and alanine aminotransferase levels stratified by obesity status. Dashed lines represent linear regression trend lines for each group.

Comprehensive Interpretation

Correlation analysis revealed positive associations between BMI and liver enzyme concentrations in both groups, but with notably steeper slopes in obese participants. The obese cohort demonstrated stronger correlation coefficients, suggesting that incremental weight gain produces proportionally greater hepatic impact once obesity thresholds are exceeded. Multiple regression modeling confirmed BMI as an independent predictor of ALT elevation ($\beta=0.412$, $p<0.001$), GGT elevation ($\beta=0.387$, $p<0.001$), and AST elevation ($\beta=0.298$, $p<0.01$) after adjusting for age, sex, alcohol consumption, and medication use. These findings support a dose-response relationship between adiposity and hepatic dysfunction.

Discussion

This research demonstrates consistent and clinically significant elevations in liver enzyme concentrations among obese adults compared to non-obese individuals. The findings align with existing literature documenting hepatic consequences of excessive adiposity while providing quantitative estimates specific to a French adult population. The magnitude of differences observed, particularly for ALT and GGT, suggests substantial hepatocyte involvement even in the absence of clinically diagnosed liver disease. The pronounced GGT elevation warrants particular attention. This enzyme serves as a sensitive marker for hepatic fat accumulation and demonstrates strong associations with metabolic syndrome components. Our finding of 98% higher mean GGT in obese versus non-obese participants exceeds several previous reports, possibly reflecting higher average BMI values in our obese cohort or population-specific factors. The correlation patterns further support GGT's utility as an early indicator of obesity-related liver changes.

ALT elevation in obesity primarily reflects hepatocyte membrane damage from lipid accumulation and associated inflammatory responses. The observed 76% increase corresponds with established understanding of non-alcoholic fatty liver disease progression. Multiple regression results confirming BMI as an independent predictor strengthen the direct relationship between adiposity and hepatic dysfunction, separate from confounding influences of alcohol or medication effects.

Comparison with international data reveals both similarities and distinctions. Asian population studies often report steeper correlations between BMI and liver enzymes, possibly due to metabolically active visceral fat accumulation patterns at lower BMI thresholds. European cohorts generally show associations consistent with our findings, supporting generalizability within similar demographic contexts. The French population context adds valuable data to the broader understanding of obesity-liver relationships across different cultural and dietary environments.

Limitations

Several limitations merit acknowledgment when interpreting these results. The cross-sectional design prevents establishing causal relationships between obesity and liver enzyme elevations, as temporal sequences cannot be determined. Imaging confirmation of hepatic steatosis was not performed, limiting direct assessment of fatty liver prevalence. Selection bias may have occurred through

recruitment from clinical settings rather than population-based sampling. The single geographic region studied may limit generalizability to other French or European populations with different demographic compositions. Self-reported alcohol consumption likely underestimates actual intake, potentially affecting adjustment accuracy. Future longitudinal investigations incorporating liver imaging and broader geographic sampling would strengthen causal inferences.

Clinical Implications

These findings carry practical implications for clinical management of obese patients. Routine liver function testing should be incorporated into obesity assessment protocols, even in asymptomatic individuals. Elevated enzymes may identify candidates for more intensive lifestyle interventions or pharmacological weight management approaches. Primary care physicians and endocrinologists should consider hepatic health status when developing comprehensive obesity treatment plans. Patient education regarding liver health consequences of excess weight may enhance motivation for behavioral modifications.

Conclusion

This research provides strong evidence for significantly elevated liver enzyme concentrations in obese compared to non-obese French adults. All four hepatic markers examined, including ALT, AST, GGT, and ALP, demonstrated higher values in the obese cohort, with differences reaching statistical significance and clinically meaningful effect sizes. These elevations persisted after statistical adjustment for potential confounding factors, supporting obesity as an independent contributor to hepatic biochemical abnormalities.

The correlation analyses revealed dose-dependent relationships between increasing BMI and rising enzyme concentrations, particularly pronounced within the obese range. GGT emerged as the most discriminating marker, showing nearly double the mean concentration in obese versus non-obese participants. This finding positions GGT as a potentially valuable screening tool for identifying obesity-related hepatic involvement in clinical practice settings. ALT similarly demonstrated strong associations with adiposity measures, confirming its established role as a hepatocyte injury indicator.

The practical implications extend to clinical screening recommendations. Healthcare providers managing obese patients should consider incorporating routine liver function panels into standard assessment protocols. Early identification of enzyme abnormalities may facilitate timely intervention through lifestyle modification programs, nutritional counseling, or pharmacological approaches when appropriate. Patients demonstrating persistent elevations warrant further hepatological evaluation to exclude progressive liver conditions.

From a public health perspective, the findings reinforce the systemic nature of obesity's health consequences. The liver represents just one organ system affected by excessive adiposity, yet hepatic complications carry substantial morbidity potential including progression to cirrhosis and hepatocellular carcinoma in severe cases. Population-level obesity prevention efforts therefore offer dual benefits of reducing both cardiovascular and hepatic disease burden simultaneously.

Future research directions should include longitudinal designs tracking enzyme trajectories during weight change interventions, imaging correlation studies confirming hepatic steatosis associations, and investigations exploring genetic factors modifying obesity-liver relationships. Multi-center collaborations across different European populations would enhance generalizability and statistical power for subgroup analyses. The ultimate goal remains developing evidence-based screening and intervention strategies that effectively address the hepatic manifestations of the ongoing obesity epidemic.

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References

1. Blüher M. Obesity: global epidemiology and pathogenesis. *Nature Reviews Endocrinology*. 2019;15(5):288-298.
2. Kivimäki M, Kuosma E, Ferrie JE, *et al*. Overweight, obesity, and risk of cardiometabolic multimorbidity. *The Lancet Public Health*. 2017;2(6):e277-e285.
3. Fabbrini E, Sullivan S, Klein S. Obesity and nonalcoholic fatty liver disease: biochemical, metabolic, and clinical implications. *Hepatology*. 2010;51(2):679-689.
4. Giannini EG, Testa R, Savarino V. Liver enzyme alteration: a guide for clinicians. *Canadian Medical Association Journal*. 2005;172(3):367-379.
5. Whitfield JB. Gamma glutamyl transferase. *Critical Reviews in Clinical Laboratory Sciences*. 2001;38(4):263-355.
6. Lala V, Goyal A, Minter DA. Liver function tests. *StatPearls*. 2023;Jan:1-12.
7. Younossi ZM, Koenig AB, Abdelatif D, *et al*. Global epidemiology of nonalcoholic fatty liver disease. *Hepatology*. 2016;64(1):73-84.
8. Buzzetti E, Pinzani M, Tsochatzis EA. The multiple-hit pathogenesis of non-alcoholic fatty liver disease. *Metabolism*. 2016;65(8):1038-1048.
9. Machado M, Marques-Vidal P, Cortez-Pinto H. Hepatic histology in obese patients undergoing bariatric surgery. *Journal of Hepatology*. 2006;45(4):600-606.
10. Polyzos SA, Kountouras J, Mantzoros CS. Adipokines in nonalcoholic fatty liver disease. *Metabolism*. 2016;65(8):1062-1079.
11. Liu B, Balkwill A, Reeves G, *et al*. Body mass index and risk of liver cirrhosis in middle aged UK women. *BMJ*. 2010;340:c912.
12. Ruhl CE, Everhart JE. Determinants of the association of overweight with elevated serum alanine aminotransferase activity. *Gastroenterology*. 2003;124(1):71-79.
13. Charles MA, Eschwège E, Basdevant A. Monitoring the obesity epidemic in France. *Obesity Research*. 2008;16(9):2182-2186.
14. Verdot C, Torres M, Salanave B, *et al*. Corpulence des enfants et des adultes en France métropolitaine en 2015. *Bulletin Épidémiologique Hebdomadaire*. 2017;13:234-241.
15. Seidell JC, Halberstadt J. The global burden of obesity and the challenges of prevention. *Annals of Nutrition and Metabolism*. 2015;66(suppl 2):7-12.
16. World Health Organization. Obesity: preventing and managing the global epidemic. WHO Technical Report Series. 2000;894:1-253.