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# Intake of Nutrients, Fiber, and Sugar in Patients with Nonalcoholic Fatty Liver Disease in Comparison to Healthy Individuals

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## **ABSTRACT**

**Background:** Nonalcoholic fatty liver disease (NAFLD) is the most common cause of chronic liver disease in the world. Although some studies have been conducted about dietary intakes of these patients, but the results are inconsistent. The aim of this study was to survey all macronutrients and micronutrients included in dietary intake of these patients for better understanding the factors influencing this disease.

**Methods:** The present study is a case-control conducted in Isfahan city, Iran. The cases were recently diagnosed patients with NAFLD who identified by ultrasonography. The case (159) and control (158) individuals were matched in age and gender. Data of general characteristics and physical activity of individuals were collected through questionnaire. Dietary intake was also collected using 24 h dietary recall questionnaire.

**Results:** Waistline and body mass index for the case group were more than the control group (P < 0.05). Physical activity level in healthy individuals was more than patients with NAFLD. Dietary intake of saturated fatty acids and sugar in patients with NAFLD was more than healthy individuals (P < 0.05). Intake of total dietary fiber, folic acid, Vitamin D, zinc, and potassium in healthy individuals was more than patients with NAFLD (P < 0.05).

**Conclusions:** In total, it seems the type of dietary intake source is associated with NAFLD. Increasing saturated fatty acids and sugar and decreasing fiber, folic acid, Vitamin D, zinc, and potassium intake might play a role in the progression of this disease.

**Keywords:** Dietary intake, nonalcoholic fatty liver disease, physical activity

#### INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) is the most prevalent cause of chronic liver diseases. This disorder

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occurs because of the pathologic accumulation of fat (mainly triglycerides) in the liver. [1] Prevalence of this disease has been estimated about 10–35% of adults worldwide. It is estimated that in Iran, 7% of children and 35% of adults are affected. [2]

Until now, many studies have been surveyed the role of nutritional factors in NAFLD. Results of these

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studies have sometimes caused the researchers to suggest different nutritional reasons for this disease such that some of studies suggest that high-calorie food patterns, which lead to obesity, can also increase the risk of accumulation of lipids in liver and incidence of steatosis.<sup>[3]</sup>

On the other hand, high carbohydrate level diets can affect NAFLD by influencing on *de novo* synthesis of fatty acids and increasing blood triglycerides.<sup>[4]</sup> Results of some other studies indicates a relation between type of lipid intake and developing this disease. Such that it seems increase in receiving omega 6 to omega 3 ratio is in relation to advanced stages of the disease by activating inflammatory pathways.<sup>[5]</sup> Moreover, it seems that saturated fatty acids can damage the structure of hepatocytes by inducing apoptosis and thus advancing NAFLD.<sup>[6]</sup>

In some studies, a relation between some micronutrients and NAFLD has been suggested. Such that it seems patients with NAFLD have a lower level intake of Vitamin D and antioxidant vitamins (E and C) than healthy individuals. [7] Moreover, results from some of these studies indicated that calcium and zinc intake in patients with NAFLD was less than healthy individuals. [8]

Despite these results, the relation between dietary intake and NAFLD, in some other studies, no relation has been observed. These inconsistent results might be because of difference in studies design and aims, participant groups, and small sample size in some studies.

Because of some limitations in previous studies and also according to the fact that nutrition has an essential role in etiology of NAFLD, further investigation of relationship between diets components and this disease can help us to prevent and control it.

### **METHODS**

## Research participants

The present study is a case-control study that was approved by the Tehran University Committee and Ethics Advisory Committee, and written consent was obtained from all participants. Patients with NAFLD who admitted Isfahan fatty liver research center were chosen as the case group. In this study, according to sample size, estimation of 140 people for case and control group:

$$n_{\text{odd ratio}} = \left(\frac{1+\varphi}{\varphi}\right) \frac{\left(Z_1 - \frac{\alpha}{2} + Z_1 - \beta\right)^2}{\left(\log OR\right)^2 \pi (1-\pi)} = 140$$

And on the basis of expectancy for over- and under-reporting of dietary intakes while completing the 24-h dietary recall, 170 people were selected for each group. After calculating participant's energy intake, who receiving under 800 kcal and over 4200 kcal of energy each day were eliminated from the study. Inclusion

criteria for case group included the following: Individuals between 20 and 60 years old who were diagnosed with NAFLD after giving blood tests and performing ultrasonography, by a radiology specialist (the device used for ultrasonography was Esaot Medica, which is equipped with a convex 3.5 MHz probe).

Inclusion criteria for control group included the following: Volunteers who were the neighbors of case group and were same as them in age and sex. Blood tests and ultrasonography were performed on them and their wellbeing in case of NAFLD was approved (not suffering from any stages of hepatic steatosis). Exclusion criteria included the following: Alcohol consumption, following special diets 2 months before the study, regular use (at least continues for 1 week) of any nutritional supplements in the last 6 months, pregnant or breastfeeding women, individuals with type B or C hepatitis and diagnosed Wilson's disease, use of drugs effective on liver, and biochemical biomarkers.

Data about age and sex were recorded by researcher by means of general information questionnaire. Physical activity rate was estimated by short form of International Physical Activity Questionnaire. [9] Based on instructions of this questionnaire, cases of the study were classified into three physical activity levels. Low-level physical activity category: If the individual does not meet the criteria for intermediate- or high-level categories. Intermediate-level physical activity category: If the individual meets one of these criteria: Three days or more, each day at least 20 min of intense physical activity or five days or more, each day at least 30 min of intermediate physical activity or walking. High-level physical activity category: If the individual meets one of these criteria: Having at least three days of intense physical activity which reaches the minimum of 1500 MET-min/week or seven days a week, any combination of walking, intermediate, or intense activity, if the total score of physical activity reaches a minimum of 3000 MET-min/week.

## **Nutritional intake assessments**

For determining the nutritional intake, a 24 h dietary recall questionnaire was filled for every individual. Then, foods were converted into their ingredients, and their amounts were calculated into grams and were encoded. Then, the amounts of energy, macronutrients, and micronutrients were calculated by entering the data to NUT4 software Nutritionist 4 (Version7; N-squared computing, OR USA) which was modified for Iranian items were used.

## Statistical data analysis

After collecting, data were analyzed using SPSS software version 16 (SPSS Inc., Chicago, IL, USA). Quantitative and qualitative data were presented as mean ± standard deviation (SD) and frequency and percentages, respectively. Kolmogrov–Smirnov was used to examine the normal distribution of variables. Log transformation

was conducted for no normally distributed variables. The relationship of the disease status with qualitative variables was assessed by Chi-square and comparing the quantitative data between groups was conducted using independent *t*-test and analysis of covariance as appropriate.

## **RESULTS**

# Demographic variables and physical activity

General and anthropometric information collected from healthy and patients are shown in Table 1. There is no difference between two groups in frequency of two genders, average of age, height, and weight (P > 0.05). However, the body mass index (BMI) was higher in the case group (P < 0.05). Physical activity comparisons show that patients are less active than healthy individuals (P < 0.05).

# Energy and macronutrients intake

Mean  $\pm$  SD of total energy and macronutrients intake between case and control groups is shown in Table 2. No difference in these variables was observed between two groups (P > 0.05). Intake of saturated fat in patients with NAFLD was more than healthy individuals (P < 0.05). Other dietary lipids intake in case and control groups had no difference (P > 0.05).

Sugar intake had a statistically significant difference between two groups such that it was denoted that sugar consumption in patients with NAFLD is more than healthy individuals (P < 0.05). Mean  $\pm$  SD of total dietary fiber (TDF) in healthy individuals was more than patients with NAFLD (P < 0.05), but no difference was observed in soluble dietary fiber and insoluble dietary fiber (P > 0.05) [Table 3].

# Vitamins and minerals intake

In Table 4, intake of vitamins and minerals after adjustment of energy intake between two groups are shown. Mean  $\pm$  SD of dietary intake of folic acid and Vitamin D in healthy individuals are more than patients with NAFLD (P < 0.05). Moreover, it was declared that intake of potassium and zinc in healthy individuals is higher than patients with NAFLD (P < 0.05). No other difference was observed in intake of other vitamins and minerals in two groups (P > 0.05).

# DISCUSSION

Our study results indicated that waistline, hipline, and BMI in the case group are higher than control group. The results of Hashemi Kani *et al.* study indicate that patients with NAFLD have higher average of weight, waistline, and BMI than healthy individuals.<sup>[7]</sup> Moreover, Capristo *et al.* (by using dual-energy X-ray) found that weight and body fat percentage of patients with NAFLD are more than healthy individuals.<sup>[10]</sup>

Table 1: Comparison of demographic, anthropometric, and physical activity data between nonalcoholic fatty liver disease and control groups

Variable	Meai	Р	
	NAFLD (n=159)	Control (n=158)	
Sex#			
Woman	91 (57.2)	90 (56.9)	0.9
Man	68 (42.7)	68 (43.0)	
Age (year) <sup>¥</sup>	$47.8 \pm 9.2$	$48.0 \pm 9.1$	0.9
Weight (kg) <sup>¥</sup>	$79.8 \pm 12.3$	$77.4 \pm 10.3$	0.1
Height (cm)*	$163.4 \pm 8.2$	$163.1 \pm 8.2$	0.9
BMI (kg/m²)**	$32.2 \pm 1.4$	$29.1 \pm 2.8$	0.02
Physical activity#*			
Low	93 (58.4)	72 (45.5)	
Moderate	63 (39.6)	78 (49.4)	0.04
High	3 (1.9)	8 (5.1)	

\*BMI=Body mass index, NAFLD group versus control group with independent Student's *t*-test, \*Chi-square. NAFLD=Nonalcoholic fatty liver disease, \*Significant differences observed between two groups (*P*<0.05)

Table 2: Comparison of energy and macronutrients intake between nonalcoholic fatty liver disease and control groups

Variable	Mean±SD		P
	NAFLD (n=159)	Control (n=158)	
Energy (kcal)*	1985.5±748.9	1972.2±719.9	0.8
Carbohydrate (g)**	$269.0 \pm 134.2$	$274.7 \pm 122.5$	0.7
Protein (g)**	$75.8 \pm 30.2$	$77.7 \pm 31.4$	0.5
Fat (g)**	55.3±31.1	$53.5 \pm 30.2$	0.6
Percentage of carbohydrate intake*	$59.1 \pm 10.8$	59.5±10.6	0.7
Percentage of protein intake*	15.5±4.1	16.0±4.2	0.3
Percentage of fat intake*	$25.3 \pm 10.4$	$24.4 \pm 10.2$	0.4
Food weight (g)**	$2156.5 \pm 1115.4$	$2035.6 \pm 1064.5$	0.8

\*NAFLD group versus control group with ANCOVA adjusted for age and sex,
\*\*NAFLD group versus control group with ANCOVA adjusted for energy intake, age,
and sex. ANCOVA=Analysis of covariance, NAFLD=Nonalcoholic fatty liver disease,
SFA=Saturated fatty acid, MUFA=Monounsaturated fatty acid, PUFA=Polyunsaturated
fatty acid, EPA=Eicosapentaenoic acid, DHA=Docosahexaenoic acid

As the results suggest, physical activity level in healthy participants is more than NAFLD patients. An overview of the epidemiological evidence reported lower chance of diagnosis with NAFLD in individuals who exercise two times per week compared to sedentary people. [111] According to the present study, consumption of saturated fatty acids in patients is more than healthy individuals. In studies conducted by Musso *et al.* and Zelber-Sagi *et al.*, it was shown that consumption of saturated fat sources such as red meat has a direct relation with NAFLD. [6,12] Glucose-dependent insulinotropic polypeptide has a role in lipid metabolism as an intermediate polypeptide. Increase of this polypeptide because of triglyceride accumulation in liver induces NAFLD. [13,14]

Table 3: Comparison of dietary carbohydrates and dietary fiber intake between nonalcoholic fatty liver disease and control groups

Variable	Mear	P	
	NAFLD (n=159)	Control (n=158)	
Glucose (g)	15.9±11.6	14.6±11.1	0.8
Galactose (g)	$2.6 \pm 3.2$	$2.0 \pm 2.6$	0.9
Fructose (g)	$17.5 \pm 16.9$	$16.5 \pm 11.9$	0.5
Sucrose (g)	$28.2 \pm 45.6$	$25.2 \pm 24.3$	0.4
Lactose (g)	$7.8 \pm 9.0$	$7.8 \pm 9.1$	0.9
Maltose (g)	$2.2 \pm 1.9$	$1.5 \pm 1.1$	0.9
Sugar (g)*	$99.2 \pm 47.5$	$89.1 \pm 43.8$	0.04
Cholesterol (mg)	$130.1 \pm 115.4$	$127.4 \pm 116.6$	8.0
SFA (g)*	$14.4 \pm 9.1$	$12.2 \pm 8.1$	0.01
MUFA (g)	$14.7 \pm 12.4$	$15.9 \pm 11.4$	0.3
PUFA (g)	$17.3 \pm 12.7$	$15.7 \pm 12.8$	0.2
Oleic acid (g)	$11.2 \pm 9.9$	$11.2 \pm 10.5$	0.9
Linoleic acid (g)	$12.1 \pm 12.1$	$11.5 \pm 13.2$	0.6
Linolenic acid (g)	$0.3 \pm 0.2$	$0.2 \pm 0.2$	0.7
EPA (g)	$0.002 \pm 0.006$	$0.003 \pm 0.007$	0.3
DHA (g)	$0.006 \pm 0.014$	$0.007 \pm 0.015$	0.4
TDF (g)*	$31.2 \pm 18.9$	$35.3 \pm 17.2$	0.04
SDF (g)*	$0.6 \pm 0.5$	$0.8 \pm 0.5$	0.09
ISDF (g)*	$4.2 \pm 4.2$	$5.0 \pm 4.3$	0.08

\*Significant differences observed between two groups (P<0.05). NAFLD group versus control group with ANCOVA adjusted for energy intake, age, sex.TDF=Total dietary fiber, SDF=Soluble dietary fiber, ISDF=Insoluble dietary fiber, SFA=Saturated fatty acid, MUFA=Monounsaturated fatty acid, PUFA=Polyunsaturated fatty acid, EPA=Eicosapentaenoic acid, DHA=Docosahexaenoic acid, ANCOVA=Analysis of covariance, NAFLD=Nonalcoholic fatty liver disease, SD=Standard deviation

Intake of sugar in patients with NAFLD is higher than healthy individuals but no difference in other carbohydrates was observed. Until now, a few studies have surveyed type of consuming carbohydrate in NAFLD patients. In the study of Yoshari *et al.* showed direct relation between weekly intake of fructose-rich sources and NAFLD.<sup>[15]</sup> In two other studies conducted by Zelber-Sagi *et al.*<sup>[12]</sup> and Toshimitsu *et al.*,<sup>[8]</sup> it was indicated that the consumption of food sources containing simple carbohydrates is in direct relation with developing this disease. According to the conducted studies using high levels of sugar can lead to advance in steatosis by increasing *de novo* synthesis of free fatty acids and lipid accumulation in hepatic tissue.<sup>[16,17]</sup>

In our study, TDF intake in healthy individuals is higher than patients with NAFLD. In two studies conducted by Cortez-Pinto *et al.*<sup>[18]</sup> and Kim *et al.*,<sup>[19]</sup> it was found that healthy people use more fiber-rich foods such as vegetables than patients with NAFLD. It seems intake of dietary fiber can be a protective factor against NAFLD because by increasing dietary fiber intake, blood LDL, fat accumulation in body, and resistance to insulin are decreased.<sup>[20]</sup>

Results of our study indicated that dietary intake of Vitamin D was higher in control group than NAFLD.

Table 4: Comparison of vitamins and minerals intake between nonalcoholic fatty liver disease and control groups

Variable	Mean±SD		P
	NAFLD (n=159)	Control (n=158)	
Vitamin A (RE)	717.6±984.0	836.4±1290.4	0.4
$\beta$ carotene ( $\mu$ g)	$970.8 \pm 1438.8$	$1087.2 \pm 2051.8$	0.5
Vitamin E (mg)	$12.0 \pm 14.9$	$12.2 \pm 15.7$	0.1
$\alpha$ tocopherol (mg)	$5.9 \pm 5.9$	$5.6 \pm 4.2$	0.5
Vitamin K (μg)	$139.3 \pm 371.0$	$135.6 \pm 376.2$	0.9
Vitamin D (μg)*	$0.4 \pm 1.1$	$0.7 \pm 1.3$	0.03
Thiamine (mg)	$1.7 \pm 0.9$	$1.0 \pm 0.8$	0.9
Riboflavin (mg)	$1.6 \pm 0.8$	$1.8 \pm 0.7$	0.8
Niacin (mg)	$20.7 \pm 12.1$	$19.4 \pm 11.2$	0.8
Pantothenic acid (mg)	$4.2 \pm 2.5$	$4.0 \pm 2.1$	0.8
Pyridoxine (mg)	$1.6 \pm 0.9$	$1.8 \pm 0.8$	0.6
Folic acid (µg)*	$366.3 \pm 20.3$	411.6±199.5	0.04
Vitamin B12 (μg)	$3.7 \pm 18.1$	$4.1 \pm 18.9$	0.9
Biotin (μg)	$14.9 \pm 10.7$	$15.3 \pm 12.8$	0.9
Vitamin C (mg)	$167.2 \pm 110.0$	$176.2 \pm 158.9$	0.5
Sodium (mg)	$1923.2 \pm 1075.9$	$2022.1 \pm 1092.1$	0.9
Iron (mg)	$15.4 \pm 8.6$	$16.5 \pm 9.7$	0.8
Magnesium (mg)	$352.8 \pm 156.8$	$374.4 \pm 165.2$	0.9
Zinc (mg)*	$9.4 \pm 4.3$	$10.6 \pm 4.5$	0.02
Manganese (mg)	$2.8 \pm 2.1$	$3.2 \pm 2.8$	0.9
Fluoride (µg)	11.154.2±9587.5	$11325.1 \pm 9243.5$	0.8
lodine (µg)	$0.1 \pm 0.5$	$0.1 \pm 0.4$	0.9
Potassium (mg)*	$3108.9 \pm 1359.2$	$3468.6 \pm 1258.9$	0.02
Calcium (mg)	$820.4 \pm 395.6$	$861.0 \pm 432.3$	0.2
Phosphorus (mg)	$1456.5 \pm 637.7$	$1522.8 \pm 629.5$	0.2
Copper (mg)	$1.7 \pm 0.9$	$1.6 \pm 0.8$	0.7
Selenium (mg)	$0.1 \pm 0.1$	$0.1 \pm 0.1$	0.9

\*Significant differences observed between two groups (*P*<0.05). NAFLD group versus control group with ANCOVA adjusted for energy intake, age, sex.ANCOVA=Analysis of covariance, NAFLD=Nonalcoholic fatty liver disease, SD=Standard deviation

Also in a case-control study conducted by Targher, the results indicated that Vitamin D intake in patients with NAFLD was much lower than healthy individuals. It is declared that deficiency Vitamin D has a relation with lipid accumulation outside of hepatocytes and development of NAFLD.<sup>[21]</sup>

Results of the presents study show that healthy individuals receive more folic acid, potassium, and zinc than patients with NAFLD. In study of Toshimitsu *et al.*, it was found that decrease in intake of zinc has a direct relation with the intensity of stages of this disease. [8] Studies indicate that some antioxidants in diet (such as zinc) can regulate hepatic aminotransferases and also prevent lipid accumulation in the liver of patients with NAFLD. [22] On the basis of our information from studies conducted until now, there was no relation observed between potassium and development of NAFLD.

Limitations of this study are using ultrasonography for diagnosing NAFLD because this method has much lower

accuracy than fibro-scan or liver biopsy methods. Also using only one 24 h dietary recall questionnaire is of other limitations of the present study. Strength, however, is using case-control nature of the study that is better than cross-sectional designs and provides stronger evidence regarding risk factors. Also comparing all of the dietary macronutrients and micronutrients between two groups of patients with NAFLD and control group which were matched for age and sex was conducted for the first time. In our study the cases were recently diagnosed patients with NAFLD.

## **CONCLUSIONS**

In total, it seems that in etiology of NAFLD what is more important than essential macronutrients intake ratio, is the type of intake sources. Such that according to our study results, individuals with NAFLD consume more amounts of sugar and saturated fatty acids and less amount of sources containing fiber (soluble and insoluble) in comparison to healthy individuals. Also it seems that low intake of Vitamin D, folic acid, zinc, and potassium are associated with the development of this disease.

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## **Conflicts of interest**

There are no conflicts of interest.

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## **REFERENCES**

- McCullough AJ. The clinical features, diagnosis and natural history of nonalcoholic fatty liver disease. Clin Liver Dis 2004;8:521-33.
- Alavian SM, Mohammad-Alizadeh AH, Esna-Ashari F, Ardalan G, Hajarizadeh B. Non-alcoholic fatty liver disease prevalence among school-aged children and adolescents in Iran and its association with biochemical and anthropometric measures. Liver Int 2009;29:159-63.
- Caldwell SH, Oelsner DH, Iezzoni JC, Hespenheide EE, Battle EH, Driscoll CJ.
  Cryptogenic cirrhosis: Clinical characterization and risk factors for underlying
  disease. Hepatology 1999;29:664-9.
- Donnelly KL, Smith Cl, Schwarzenberg SJ, Jessurun J, Boldt MD, Parks EJ.
   Sources of fatty acids stored in liver and secreted via lipoproteins in patients

- with nonalcoholic fatty liver disease. J Clin Invest 2005;115:1343-51.
- Parker HM, Johnson NA, Burdon CA, Cohn JS, O'Connor HT, George J. Omega-3 supplementation and non-alcoholic fatty liver disease: A systematic review and meta-analysis. J Hepatol 2012;56:944-51.
- Musso G, Gambino R, Pacini G, De Michieli F, Cassader M. Prolonged saturated fat-induced, glucose-dependent insulinotropic polypeptide elevation is associated with adipokine imbalance and liver injury in nonalcoholic steatohepatitis: Dysregulated enteroadipocyte axis as a novel feature of fatty liver. Am J Clin Nutr 2009;89:558-67.
- Hashemi Kani A, Alavian M, Esmaillzadeh A, Adibi P, Azadbakht L. Dietary quality indices and biochemical parameters among patients with non-alcoholic fatty liver disease. Hepatitis 2013;4:90-3.
- Toshimitsu K, Matsuura B, Ohkubo I, Niiya T, Furukawa S, Hiasa Y, et al. Dietary habits and nutrient intake in non-alcoholic steatohepatitis. Nutrition 2008;24:1097-102.
- Craig CL, Marshall AL, Sjöström M, Bauman AE, Booth ML, Ainsworth BE, et al. International physical activity questionnaire: 12-country reliability and validity. Med Sci Sports Exerc 2003;35:1381-95.
- Capristo E, Miele L, Forgione A, Vero V, Farnetti S, Mingrone G, et al. Nutritional aspects in patients with non-alcoholic steatohepatitis (NASH). Eur Rev Med Pharmacol Sci 2005;9:265-8.
- Nimer A. Nutrition and physical activity in NAFLD. An overview of the epidemiological evidence. World J Gastroenterol 2011;17:3377-89.
- Zelber-Sagi S, Nitzan-Kaluski D, Goldsmith R, Webb M, Blendis L, Halpern Z, et al. Long term nutritional intake and the risk for non-alcoholic fatty liver disease (NAFLD): A population based study. J Hepatol 2007;47:711-7.
- Kim SJ, Nian C, McIntosh CH. Resistin is a key mediator of glucose-dependent insulinotropic polypeptide (GIP) stimulation of lipoprotein lipase (LPL) activity in adipocytes. J Biol Chem 2007;282:34139-47.
- Hansotia T, Maida A, Flock G, Yamada Y, Tsukiyama K, Seino Y, et al. Extrapancreatic incretin receptors modulate glucose homeostasis, body weight, and energy expenditure. J Clin Invest 2007;117:143-52.
- Yoshari N, Ebrahimi M, Asghari M. Dietary fructose association with lipid profile in non-alcoholic fatty liver disease. Babol Med J 2014;2:23-30.
- Clarke SD. Polyunsaturated fatty acid regulation of gene transcription: A molecular mechanism to improve the metabolic syndrome. J Nutr 2001;131:1129-32.
- Haque M, Sanyal AJ. The metabolic abnormalities associated with non-alcoholic fatty liver disease. Best Pract Res Clin Gastroenterol 2002;16:709-31.
- Cortez-Pinto H, Jesus L, Barros H, Lopes C, Moura MC, Camilo ME. How different is the dietary pattern in non-alcoholic steatohepatitis patients? Clin Nutr 2006;25:816-23.
- Kim CH, Kallman JB, Bai C, Pawloski L, Gewa C, Arsalla A, et al. Nutritional assessments of patients with non-alcoholic fatty liver disease. Obes Surg 2010;20:154-60.
- Leach NV, Dronca E, Vesa SC, Sampelean DP, Craciun EC, Lupsor M, et al.
   Serum homocysteine levels, oxidative stress and cardiovascular risk in non-alcoholic steatohepatitis. Eur J Intern Med 2014;25:762-7.
- Zhou QG, Hou FF, Guo ZJ, Liang M, Wang GB, Zhang X. 1,25-Dihydroxyvitamin D improved the free fatty-acid-induced insulin resistance in cultured C2C12 cells. Diabetes Metab Res Rev 2008;24:459-64.
- Guo CH, Chen PC, Ko WS. Status of essential trace minerals and oxidative stress in viral hepatitis C patients with nonalcoholic fatty liver disease. Int J Med Sci 2013;10:730-7.