SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 5 | May 2025 - Peer Reviewed Journal

NON-ALCOHOLIC FATTY LIVER DISEASE ASSOCIATED WITH OBESITY: THE POTENTIAL OF ESSENTIAL PHOSPHOLIPIDS

Xudoykulova Farida Vafokulovna, Burkhanova Gulnoza Lutfilloyevna
Samarkand State Medical University

Article DOI: https://doi.org/10.36713/epra21598
DOI No: 10.36713/epra21598

ABSTRACT

The role and importance of primary care physicians (general practitioners and internists) are increasingly recognized in the diagnosis and management of patients with obesity and non-alcoholic fatty liver disease (NAFLD). This article discusses a diagnostic algorithm for obesity based on the AACE/ACE-2014 classification and explores the therapeutic potential of essential phospholipids within preventive and treatment recommendations for patients with obesity-associated NAFLD.

KEYWORDS: *obesity, diagnostic algorithm, non-alcoholic fatty liver disease (NAFLD), preventive and therapeutic recommendations, essential phospholipids, Hepagard Active®*

Over the past few decades, there has been a marked increase in the prevalence of diseases associated with changes in diet and lifestyle. A systematic analysis (1,769 reports, surveys, and studies) on the global prevalence of overweight and obesity from 1980 to 2013 clearly demonstrated a growing proportion of adults and children with a body mass index (BMI) of 25 kg/m² or more. Currently, more than one-third of adults worldwide are overweight—36.9% of men and 38% of women—while one in ten individuals is obese (BMI ≥ 30 kg/m²) [1]. Similar statistics are reported by the official WHO website: in 2014, over 1.9 billion adults were overweight, and more than 600 million were obese [2]. Given the widespread nature of obesity, the term **"globesity"** has been increasingly used in the medical literature over the past decade, underscoring the global scale and significance of this issue [1].

In the CIS Countries, the prevalence of overweight is 59.2%, and that of obesity is 24.1%; in the United States, these figures are 67.4% and 33.3%, respectively; in the United Kingdom—63.6% and 25.8% [1].

Type 2 diabetes mellitus (T2DM), cardiovascular illnesses, and gastrointestinal problems such as non-alcoholic fatty liver disease (NAFLD) and gastroesophageal reflux disease (GERD) have all significantly increased in tandem with the rise in overweight and obesity. The World Health Organization (WHO) reports that up to 23% of cases of ischemic heart disease and up to 44% of cases of type 2 diabetes are caused by overweight and obesity [2].

In most European countries, data from the **European Association for the Study of Obesity (EASO)** indicate that among adults, obesity is responsible for 80% of T2DM cases, 35% of ischemic heart disease cases, and 55% of arterial hypertension cases [3].

Obesity is an extremely costly public health issue—not only economically, but also in terms of individual and societal health, longevity, and psychological well-being [2, 4, 5].

Today, obesity is no longer viewed merely as a cosmetic issue caused by overeating or lack of self-disciplineObesity is currently recognized by the WHO and several national and international medical and scientific bodies as "adiposity-based chronic disease (ABCD)" [6], a chronic, progressive illness brought on by excessive fat accumulation as a result of numerous environmental and genetic variables. Increased body fat contributes to adipose tissue dysfunction and biomechanical stress on surrounding tissues, which results in metabolic and psychosocial health consequences, according to the American Society for Metabolic and Bariatric Surgery (ASMBS), which describes obesity as a chronic, relapsing, multifactorial neurobehavioral disease [7].

According to the pattern of fat distribution, two main types of obesity are distinguished: abdominal and gluteofemoral (see Fig. 1). In abdominal obesity, the majority of adipose tissue is located in the abdominal cavity, on the anterior abdominal wall, trunk, neck, and face (also referred to as android or male-type obesity Conversely, gluteofemoral obesity, often known as gynoid or female-type obesity, is typified by a preponderance of fat deposition in the hips and buttocks. The distribution of body fat is more uniform in mixed-type obesity.

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 5 | May 2025 - Peer Reviewed Journal

Body mass index (BMI) became the main diagnostic criterion for obesity after the World Health Organization (WHO) added it to the International Classification of Diseases (ICD) in 1950. The quantitative evaluation of obesity based on BMI is still commonly employed today (Table 1).

Table 1
WHO Obesity BMI Classification (2004)

Category of Weight	BMI (kg/m2)
Underweight	< 18.5
Weight range: normal	18.5–24.9
Overweight	25–29.9 C

However, a number of prospective studies examining the relationship between obesity and elevated BMI and death rates have produced conflicting findings, with some suggesting that people with overweight or mild obesity have higher survival rates than people with normal or low BMI. The necessity of additional criteria to evaluate obesity in clinical practice is therefore a topic of continuous discussion.

For instance, the American Association of Clinical Endocrinologists/American College of Endocrinology's AACE/ACE-2014 recommendations [8] advise determining the clinical significance of obesity by looking at whether or not obesity-related disorders, illnesses, or consequences are present (Table 2).

Table 2
AACE/ACE-2014 Obesity Classification [8]

Obesity Stage	BMI	Obesity-Related Conditions / Comorbidities
Overweight	≥ 25 kg/m²	No obesity-associated conditions, diseases, or complications
Stage 0 Obesity	≥ 30 kg/m²	No obesity-associated conditions, diseases, or complications
Stage 1 Obesity	≥ 25 kg/m²	One or more mild-to-moderate obesity-associated conditions, diseases, or complications
Stage 2 Obesity	≥ 25 kg/m²	One or more severe obesity-associated conditions, diseases, or complications

Obesity-Related Conditions and Comorbidities include:

Metabolic syndrome (MetS)
Impaired glucose tolerance
Type 2 diabetes mellitus
Dyslipidemia
Arterial hypertension (AH)
Non-alcoholic fatty liver disease (NAFLD)
Gastroesophageal reflux disease (GERD)
Obstructive sleep apnea
Osteoarthritis
Difficulty or inability to engage in physical activity
Polycystic ovary syndrome (PCOS)
Stress and urge urinary incontinence
Psychological disorders / stigmatization [8]

The **Diagnostic Algorithm for obesity** based on the AACE/ACE-2014 classification includes **four consecutive steps**.
Step One: The primary care physician (general practitioner or internist) performs **anthropometric screening**, which includes the **measurement of BMI and waist circumference (WC)**.

Step Two: Based on clinical examination and routine laboratory and instrumental testing, the primary care physician conducts a **screening for obesity-associated conditions or complications**.

Step Three: If any such conditions or complications are detected, a **further evaluation** should be performed jointly with a **relevant specialist** to assess the **severity of the changes**.

Step Four: Treatment and prevention recommendations for patients with **Stage 1 and Stage 2 obesity** should be provided not only by the primary care physician but also by an **endocrinologist and appropriate specialist(s)**.

A focus on potential obesity-related health risks—rather than merely stating BMI—offers a more proactive and preventive approach. This is especially important for patients with a BMI ≥ 25 kg/m² who already exhibit obesity-associated conditions or complications. At the same time, a somewhat more lenient approach toward individuals with BMI ≥ 30 kg/m² but no current comorbidities (i.e.,

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 5 | May 2025 - Peer Reviewed Journal

Stage 0 obesity) should be viewed cautiously. The designation of Stage 0 does not ensure that these problems would not arise later on or advance to Stage 1 or Stage 2.

However, a positive clinical status (i.e., no consequences despite high BMI) should encourage the patient to follow preventative guidelines to lower the chance of developing obesity-related health problems in the future.

One of the most common medical diseases linked to obesity is Metabolic Syndrome (MS). Increased visceral fat mass, impaired peripheral tissue insulin sensitivity, and hyperinsulinemia are the hallmarks of multiple sclerosis. Arterial hypertension (AH) develops as a result of these alterations, which also cause disruptions in the metabolism of carbohydrates, fats, and purines.

Central (abdominal) obesity, which is characterized by a waist circumference (WC) of more than 80 cm for women and more than 94 cm for males, is the main diagnostic criterion for multiple sclerosis.

Additional Criteria for MS include
Blood pressure (BP) ≥140/90 mmHg or ongoing antihypertensive therapy;
Elevated triglyceride levels (≥1.7 mmol/L);
Reduced HDL cholesterol (HDL-C): <1.0 mmol/L in men, <1.2 mmol/L in women;
Increased LDL cholesterol (LDL-C) >3.0 mmol/L;

Impaired Glucose Tolerance (IGT) – elevated 2-hour plasma glucose after oral glucose tolerance test (OGTT) with 75 g of anhydrous glucose: ≥7.8 and <11.1 mmol/L, with fasting plasma glucose (FPG) <7.0 mmol/L;

Impaired Fasting Glucose (IFG) – elevated FPG ≥6.1 and <7.0 mmol/L, with 2-hour plasma glucose during OGTT <7.8 mmol/L;

Combined IFG/IGT – FPG ≥6.1 and <7.0 mmol/L together with 2-hour plasma glucose during OGTT ≥7.8 and <11.1 mmol/L.
A definitive diagnosis of MS is made when at least **three criteria** are present: **one primary and two additional** [9].
Nearly all patients with MS exhibit hepatic steatosis, and approximately half present with steatohepatitis, which positions **Non-**

Alcoholic Fatty Liver Disease (NAFLD) as the **hepatic manifestation of MS** [10–12].
Currently, the concept of NAFLD is well-defined and includes a spectrum of liver disorders:
Hepatic steatosis (fatty liver),
Steatosis with hepatocellular inflammation and injury (**Non-Alcoholic or Metabolic Steatohepatitis – NASH**), and
Fibrosis, with potential progression to cirrhosis.

Primary NAFLD is typically associated with obesity and endogenous disorders of lipid and carbohydrate metabolism. NAFLD is significantly more prevalent in patients with obesity and dysmetabolic conditions: identified in **90% of individuals with obesity** [13], those with **dyslipidemia** [14,15], and in **three out of five** patients with **type 2 diabetes mellitus (T2DM)** [16,17]. NAFLD is considered an **independent risk factor** for cardiovascular diseases (CVDs) [18,19], T2DM, chronic kidney disease (CKD), and colorectal cancer [12,20].

Epidemiological studies over the past decade demonstrate that NAFLD is among the most prevalent gastroenterological diseases in the United States and Western Europe. In the general adult population of industrialized nations, the prevalence of NAFLD varies between 20–35% (average ~25%) across epidemiological studies, and may reach up to 45% in certain ethnic groups, such as individuals of Hispanic origin [21]. NAFLD prevalence increases with age [18].

As part of the **National Health and Nutrition Examination Surveys (NHANES)** research program, the prevalence of **chronic liver diseases (CLD)** in the United States was studied from 1988 to 2008 [22]. During this period, the prevalence of hepatitis B, hepatitis C, and alcoholic hepatitis remained nearly unchanged, while the proportion of **Non-Alcoholic Fatty Liver Disease (NAFLD)** among CLD cases increased from **46.8% to 75.1%**. This rise in NAFLD prevalence paralleled the increasing rates of **obesity**, **insulin resistance**, **type 2 diabetes mellitus (T2DM)**, and **arterial hypertension (AH)**.

About 10% of patients with non-alcoholic steatohepatitis (NAFLD), or 2-3% of the adult population, have NASH. Women between the ages of 40 and 50 are more likely to be diagnosed with NASH, accounting for 60–75% of cases [23]. An epidemiological study carried out in the UK found that 12% of patients with NASH proceeded to liver cirrhosis within 8 years, whereas 2% of patients with simple steatosis acquired liver cirrhosis over 15–20 years.

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 5 | May 2025 - Peer Reviewed Journal

Large cohort studies of patients with **cryptogenic liver cirrhosis**, which included analysis of comorbidities and risk factors, suggest that in many cases (**up to 60–80%**), "cirrhosis of unknown etiology" may actually develop against the background of **undiagnosed NASH**.

In Russia, data from a large epidemiological study (**DIREG_L_01903**, 2007), led by academician V.T. Ivashkin and involving over 30,000 outpatients seen by primary care physicians, reported NAFLD in 27% of patients. Among them, non-alcoholic hepatic steatosis was detected in 80.3%, steatohepatitis in 16.8%, and cirrhosis in 2.9% of cases. Notably, only 1% of individuals were aware of having this disease [10, 24].

At the end of 2015, experts from the Russian Gastroenterological Association (RGA)**,** the Russian Society for the Study of the Liver (ROSSL)**,** and the National Internet Society of Internal Medicine Specialists summarized the findings of the DIREG2 nationwide epidemiological study on NAFLD prevalence, conducted across 16 cities in Russia between 2013 and 2014**.** The study involved over 50,000 patients and more than 1,000 gastroenterologists, general practitioners, and pediatricians providing outpatient care. NAFLD was diagnosed in 37.3% of study participants. The rise in disease prevalence was particularly notable among younger, working-age individuals (18–39 years). Key risk factors for NAFLD development included: BMI >27 kg/m², obesity, T2DM, and hypercholesterolemia. Risk factors were identified in 92% of patients with NAFLD [25, 26].

A major **clinical feature of NAFLD** (including steatosis and NASH) is its **asymptomatic or mildly symptomatic presentation**. NAFLD symptoms are **nonspecific** and merely reflect the **presence of liver damage**, without correlating with the **severity** of the disease [27, 28].

Asthenia (general weakness, increased fatigue, etc.) is the **most common syndrome** observed in patients with hepatic steatosis and NASH. According to the literature, **"asymptomatic" hepatomegaly** may be detected in up to **75%** of patients with NASH during physical examination. Other symptoms typical of **chronic liver diseases** are often **absent** in most patients.

Metabolic syndrome (MS) is characterized by an increase in **visceral fat mass**, decreased **insulin sensitivity** in peripheral tissues, and **hyperinsulinemia**, which lead to disorders in **carbohydrate, lipid, and purine metabolism**, as well as **arterial hypertension**. The **average age** of NAFLD patients at the time of diagnosis is **45–50 years**. Most individuals with hepatic steatosis and NASH have **excess body weight**, exceeding the ideal by **10–40%** (**BMI >30 kg/m²**). In **primary NAFLD**, clinical symptoms are often related to **coexisting disturbances in carbohydrate and lipid metabolism**.

A **common component of NAFLD's clinical presentation** is the presence of **functional biliary tract disorders** (gallbladder and bile duct dysfunctions) — up to **30% of patients** report **pain and discomfort in the right upper quadrant**, especially **after eating**. **Laboratory tests may reveal cytolysis and cholestasis syndromes** [29, 30].

Cytolysis syndrome in NAFLD (non-alcoholic fatty liver disease) manifests as a 2–3-fold increase in serum alanine aminotransferase (ALT) activity and a 2–10-fold increase in aspartate aminotransferase (AST) activity compared to normal levels. However, the AST/ALT ratio does not allow for a clear distinction between alcoholic hepatitis and NASH (non-alcoholic steatohepatitis). An AST/ALT ratio greater than 3 is observed in approximately 32% of patients with NASH, and a ratio above 1 is seen in about 40% of patients.

Cholestasis syndrome: Hyperbilirubinemia, ranging from 25–35 µmol/L, is present in 12–17% of cases; more often, serum bilirubin levels remain within the normal range. Alkaline phosphatase (ALP) activity is moderately elevated in 40–60% of patients. In 30–60% of NASH patients, elevated levels of ALP and gamma-glutamyl transpeptidase (GGT) may be observed, usually not exceeding twice the upper limit of normal.

Mesenchymal-Inflammatory Syndrome, or inflammation syndrome, is not typical for NASH. However, hypergammaglobulinemia can be found in 13–30% of patients. Antinuclear antibodies (ANA) at titers of 1:40 to 1:320 are present in 40% of patients, while smooth muscle antibodies are usually absent.

Hepatodepression Syndrome is also uncharacteristic for NASH. Hepatocellular failure typically develops only with the progression to liver cirrhosis. However, hypoalbuminemia in NASH may be observed in patients with diabetic nephropathy.

Laboratory signs of **disordered carbohydrate metabolism** (e.g., elevated blood glucose or impaired glucose tolerance) and **lipid metabolism abnormalities** (e.g., hypercholesterolemia and hypertriglyceridemia) are frequently detected. **Hematologic abnormalities** are not characteristic of NASH, except in cases where **hypersplenism** develops due to cirrhosis.

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 5 | May 2025 - Peer Reviewed Journal

Imaging methods (ultrasound, CT, MRI) can confirm hepatomegaly, provide indirect assessment of the degree of hepatic steatosis, and detect the development of portal hypertension. **Radionuclide scanning** with sulfur colloid labeled with technetium-99m (^{99m}Tc) allows visualization of focal steatosis as defects in isotope uptake. However, imaging techniques cannot reliably differentiate between simple hepatic steatosis and NASH. **Esophagogastroduodenoscopy (EGD)** can identify esophageal varices during the progression of steatohepatitis to liver cirrhosis [31].

NAFLD is Most Often Detected Incidentally, typically when abdominal ultrasound reveals signs of hepatic steatosis. Ultrasound features of diffuse fatty liver disease include:

1. Diffusely hyperechoic ("bright") liver parenchyma;
2. Increased liver echogenicity relative to the kidneys;
3. Blurred and smoothed vascular structures;
4. Distal attenuation of the ultrasound signal.

In some cases, against the background of fatty infiltration, areas of **reduced echogenicity** may be visualized, corresponding to **unaffected liver parenchyma** [32, 33].

Liver histological examination remains the gold standard in diagnosing NAFLD (non-alcoholic fatty liver disease). However, without taking into account the **patient's history** (specifically, ruling out alcohol intake in a hepatotoxic dose—more than 20 mL of ethanol per day), it is impossible to differentiate between alcoholic hepatitis and NASH (non-alcoholic steatohepatitis) based solely on histological findings.

According to the **Bologna Conference (2009) recommendations**, liver biopsy is advised for NAFLD patients in the following scenarios:

- Motivated Patient;
- presence of **type 2 diabetes mellitus**;
- **abdominal (android) obesity**;
- **thrombocytopenia** (platelet count ≤140 x 10⁹/L);
- **insulin resistance**;
- any indirect signs of liver cirrhosis [34].

If serum aminotransferase levels are within the normal range, liver biopsy is not indicated.
From the above epidemiological data, an important practical conclusion can be drawn: **every third to fourth patient visiting a general practitioner or internist in Russian outpatient clinics requires preventive treatment for hepatic steatosis** [10, 25, 26].

Pathogenetically justified treatment of NAFLD associated with obesity includes **lifestyle modifications**, encompassing **balanced nutrition and regular, moderate physical activity**.
There is currently **no universally accepted pharmacological treatment** for NAFLD. However, this does not preclude the use of medications in **adjunct to dietary and lifestyle recommendations**.
Early intervention, ideally during the **steatosis stage**, is advisable. Most patients with obesity-associated NAFLD benefit from a treatment approach that:

- stabilizes **hepatocyte membranes**,
- provides **antioxidant protection**,
- includes **immunomodulatory agents** with anti-inflammatory properties, and
- addresses **biliary tract dysfunction** [27, 29, 31, 35–37].

In this context, the **use of essential phospholipids (EPLs)** as part of combination therapy is well justified.
EPLs are preparations with a high content of purified **phosphatidylcholine** derived from soybeans. A key component, **1,2-dilinoleoyl-phosphatidylcholine (DLPC)**, differs from regular phospholipids by having an additional linoleic acid molecule at the first position. This feature enables it to **fill membrane defects**, thereby increasing **membrane flexibility and fluidity** [35, 36, 38].
EPLs are **structural and functional units of cell membranes**, ensuring their **plasticity and high functional activity**. The therapeutic efficacy of DLPC in NAFLD is due to its ability to:

- **block lipid peroxidation (LPO)**,
- exert **membrane-stabilizing** and **antioxidant effects**.

Thanks to its **polyunsaturated bonds**, DLPC can **integrate into cell membranes** and **replace damaged phospholipids**, which may have been affected by **oxidative stress, cytokines, alcohol**, or **environmental toxins**.

Thus, **liver biopsy remains the diagnostic cornerstone** for NAFLD, but histological findings must always be interpreted in light of clinical history, particularly regarding alcohol intake.

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 5 | May 2025 - Peer Reviewed Journal

DLPC-based therapy works through **at least two mechanisms** to counteract **membranopathy** resulting from oxidative stress and to systemically reduce the **pro-inflammatory state** of the body. In the liver, DLPC:

- activates **triglyceride lipase**,
- improves mitochondrial and endoplasmic reticulum function,
- normalizes **lipid metabolism and excretion**.

With **long-term use**, DLPC suppresses inflammation and fibrogenesis, and **prevents hepatocyte apoptosis** [39–41].
Exogenous essential phospholipids (EPLs), introduced into the body as part of a medicinal product, have a **positive effect** on **lipid and protein metabolism**, **liver detoxification function**, and on the **restoration and maintenance of hepatocyte cellular structure**. They help **suppress fatty degeneration** and **fibrous tissue formation** in the liver [35, 36, 38, 43–45].

The rationale for using **EPL-containing preparations** in NAFLD therapy lies in their **experimentally proven** [35] **comprehensive action**: restoration of **cell membranes**, **antioxidant**, **anti-inflammatory**, and **antifibrotic** effects. Additionally, **phosphatidylcholine levels** are significantly **reduced in NAFLD patients** compared to healthy individuals [45].

On the Russian pharmaceutical market, **combined formulations** are gaining attention, particularly those in which **EPLs are combined with other active ingredients** that enhance hepatoprotective effects. One such **combined hepatoprotective supplement** is **Hepagard Active®** (Eurasian Patent No. EA19268, dated 28.02.2014), which contains **EPLs**, **L-carnitine**, and **vitamin E**.

- **L-carnitine** is a **lipotropic compound** that accelerates **fatty acid utilization** by transporting fatty acids into mitochondria, where they undergo **β-oxidation**.
  The inclusion of L-carnitine in EPL-based combination therapy **enhances the liver's protein-synthesizing and detoxification functions** [46, 47].
- **Vitamin E**, a **universal antioxidant**, acts **synergistically** with EPLs by protecting hepatocyte membranes from damage by **free radicals**, thus helping maintain **hepatic cell integrity** [48].
-

This **combined composition** of **Hepagard Active®** provides **hepatoprotective and lipotropic effects**.
Clinical observations have demonstrated the **efficacy** of this supplement in patients with **metabolic syndrome** [49–53]. The product has also shown a **modulating effect** on **carbohydrate and lipid metabolism**, a **significant reduction in leptin levels**, and, in **overweight individuals**, a **reduction in adipose tissue mass**.

Course-based administration of the combined supplement containing **EPLs**, **vitamin E**, and **L-carnitine** (Hepagard Active®), at **1 capsule 3 times daily for one month or more**, supports liver function—**both metabolically and detoxically**—and contributes to **lower cholesterol and leptin levels**.

The **innovative formulation** of Hepagard Active® also helps **prevent hepatic steatosis**, particularly in **comorbid patients** (with **obesity, type 2 diabetes mellitus**, etc.).

The effects of **Hepagard Active®** have been confirmed in **numerous clinical studies and reviews** [29, 30, 49–61].
The use of **Hepagard Active®** for a **2-month period** in women with **overweight and obesity** was associated with **reduction in body weight and obesity grade**, as well as **decreased waist circumference**. This was accompanied by a favorable **modulatory effect on lipid metabolism parameters** (triglycerides, total cholesterol, and low-density lipoprotein cholesterol) and **carbohydrate metabolism** indicators.

Bioimpedance analysis of body composition showed a **statistically significant decrease** in **body fat percentage** and **visceral fat**. Quality of life assessments in individuals who completed a **rehabilitation program including Hepagard Active®** revealed a **marked positive impact** on parameters such as **"vitality," "emotional responses," and "sleep,"** indicating a significant **improvement in overall well-being** and **emotional status** [51–53].

It is well established that during **prolonged fasting**, the most significant deficiency in the body is **lecithin**, which contributes to **impaired liver function**. **Hepagard Active®** contains essential components that are critical during periods of **fasting**, when intake of such nutrients is limited.

The use of **essential phospholipids** (such as those in Hepagard Active®) during the **recovery phase post-fasting** facilitates a **gentle transition** and **accelerates the restoration of liver functions** [51, 59].

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 5 | May 2025 - Peer Reviewed Journal

Administration of **Hepagard Active®** to patients with **NAFLD** at a dose of **1 capsule 3 times daily with meals for 3 months** has demonstrated:

- **Improvement in hepatobiliary tract function**,
- **Normalization of the lipid profile**,
- **Reduction in body weight**, and
- **Improvement in quality of life** indicators [49–51].

Hepagard Active® intake also results in:

- **Improved gallbladder contractility** [57],
- **Positive changes in lipid peroxidation markers and immune homeostasis**,
- **Normalization of cholesterol metabolism**, and
- **Beneficial effects on physical and psychological aspects of quality of life** [51–53].

Thus, in **obese individuals**, particularly those with associated conditions such as **metabolic syndrome (MS)** and **NAFLD**, it is clinically reasonable to include **EPL-containing preparations** (such as **Hepagard Active®**) as part of a **comprehensive therapeutic and preventive approach**.

REFERENCE

1. *Khayrieva M. F., Karomatov I. D. Shafran v profilaktike i lechenii metabolicheskogo sindroma (obzor literatury) //Biologiya i integrativnaya meditsina. – 2018. – №. 7. – S. 112-119.*
2. *Banzaraksheev, Vitaliy Gambalovich. "Fitoterapiya i fitoprofilaktika narusheniy lipidnogo obmena (obzor literatury)" Vestnik Buryatskogo gosudarstvennogo universiteta. Meditsina i farmatsiya 12 (2012): 77-80.*
3. *Gabbasova L. V. i dr. Fitoterapiya pri gepatobiliarnykh zabolevaniyakh //Meditsinskiy vestnik Bashkortostana. – 2009. – T. 4. – №. 5. – S. 74-77.*
4. *Skvortsov V. V., Lun'kov M. V., Levitan B. N. Kombinirovannaya fitoterapiya pri khronicheskoy alkogol'noy bolezni pecheni //Meditsinskiy sovet. – 2020. – №. 15. – S. 97-103.*
5. *Karomatov I. D., Rakhmatova D. Lekarstvennoe rastenie-barbaris //Biologiya i integrativnaya meditsina. – 2019. – №. 1 (29). – S. 197-220.*
6. *Korsun E. V. Metafilaktikazabolevaniy pecheni sredstvami fitoterapii //Vestnik Buryatskogo gosudarstvennogo universiteta. Meditsina i farmatsiya. – 2008. – №. 12. – S. 12-16.*
7. *Ubeeva E. A., Nikolaev S. M., Ubeeva I. P. Osnovnye napravleniya fitoterapii zabolevaniĭ pecheni //Vestnik Buryatskogo gosudarstvennogo universiteta. Meditsina i farmatsiya. – 2017. – №. 3. – S. 3-9.*
8. *Ubeeva I. P., Verlan N. V., Nikolaev S. M. Primenenie lekarstvennykh rasteniĭ, obladayushchikh sedativnym deĭstviem v lechenii zabolevaniĭ nervnoĭ sistemy //Vestnik Buryatskogo gosudarstvennogo universiteta. Meditsina i farmatsiya. – 2017. – №. 3. – S. 15-21.*
9. *Khudoykulova F. V. i dr. THE STRUCTURE, AGE FEATURES, AND FUNCTIONS OF HORMONES //PEDAGOG. – 2023. – T. 1. – №. 5. – S. 681-688.*
10. *KhUDOYKULOVA F. V., MAVLYaNOVA Z. F. JIGARNING NOALKOGOL YOG'XASTALLIGIGA ZAMONAVIY QARASHLAR //ZhURNAL BIOMEDITsINY I PRAKTIKI. – 2022. – T. 7. – №. 4.*
11. *Khudoykulova F. V. i dr. the structure, age features, and functions of hormones. pedagog, 1 (5), 681-688. – 2023.*
12. *Khudoykulova F. V. i dr. THE STRUCTURE, AGE FEATURES, AND FUNCTIONS OF HORMONES //PEDAGOG. – 2023. – T. 1. – №. 5. – S. 681-688.*
13. *Vafokulovna K. F. NO ALCOHOL OF THE LIVER DIAGNOSTIC AND TREATMENT OF OBESITY DISEASE MODERN OBJECTIVES //Conference Zone. – 2022. – S. 600-605.*
14. *Statsenko E. S. i dr. AKTUAL''NYE VOPROSY DIETOTERAPII V LEChENII NEALKOGOL''NOY ZhIROVOY BOLEZNI PEChENI //Vestnik Volgogradskogo gosudarstvennogo meditsinskogo universiteta. – 2022. – T. 19. – №. 1. – S. 3-8.*
15. *Andreev K. A. i dr. Priverzhennost' modifikatsii obraza zhizni pri nealkogol'noy zhirovoy bolezni pecheni //Byulleten' sibirskoy meditsiny. – 2021. – T. 20. – №. 4. – S. 112-122.*
16. *Gubergrits N. B. i dr. Sovremennye predstavleniya o pitanii i fizicheskoy aktivnosti v lechenii nealkogol'noy zhirovoy bolezni pecheni //Eksperimental'naya i klinicheskaya gastroenterologiya. – 2018. – №. 2 (150). – S. 100-109.*
17. *Karomatov I. D., Togboev K. T. Unabi perspektivnoe i lechebnoprofilakticheskoe sredstvo //Biologiya i integrativnaya meditsina. – 2017. – №. 6. – S. 165-182.*
18. *Banzaraksheev V. G. i dr. Fitokorrektsiya aterogennoĭ dislipidemii pri eksperimental'nom povrezhdenii pecheni //Vestnik Buryatskogo gosudarstvennogo universiteta. Meditsina i farmatsiya. – 2012. – №. 2. – S. 83-87.*
19. *Tazhibaeva F. R. Differentsirovannyy podkhod k sindromu vnutripechenochnogo kholestaza i puti ego razresheniya //Sinergiya. – 2016. – №. 2. – S. 100-106.*
20. *Khayrieva M. F., Karomatov I. D. Gretskiy orekh i metabolicheskie narusheniya (obzor literatury) //Biologiya i integrativnaya meditsina. – 2018. – №. 8. – S. 29-41.*
21. *Shtrigol' S. Yu. i dr. Lekarstvennye rasteniya, pochki i obmen mochevoy kisloty. – 2014.*

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 5 | May 2025 - Peer Reviewed Journal

22. *Mikhaylenko L. V. Kompleksnoe primenenie fitoterapii i mineral'nykh vod dlya korrektsii metabolicheskogo sindroma na etape sanatorno-kurortnogo lecheniya : dis. – Rossiyskiy nauchnyy tsentr vosstanovitel'noy meditsiny i kurortologii, 2011.*
23. *Cherkashina E. A. Aktual'nye voprosy diagnostiki i lecheniya nealkogol'noy zhirovoy bolezni pecheni //Meditsinskiy sovet. – 2015. – №. 4. – S. 67-71.*
24. *Livzan M. A. i dr. Nealkogol'naya zhirovaya bolezn' pecheni u lits s abdominal'nym tipom ozhireniya //Dokazatel'naya gastroenterologiya. – 2014. – T. 3. – №. 4. – S. 8-14.*
25. *Lazebnik L. B. i dr. Gipolipidemicheskaya terapiya u bol'nykh s nealkogol'noy zhirovoy bolezn'yu pecheni //CardioSomatika. – 2010. – T. 1. – №. 1. – S. 38-45.*
26. *Tsukanov V. V., Vasyutin A. V., Tonkikh Yu. L. Sovremennye printsipy vedeniya patsientov s nealkogol'noy zhirovoy bolezn'yu pecheni //Doktor. Ru. – 2019. – №. 3 (158). – S. 11-14.*
27. *Komshilova K. A., Troshina E. A. Ozhirenie i nealkogol'naya zhirovaya bolezn' pecheni: metabolicheskie riski i ikh korrektsiya //Ozhirenie i metabolizm. – 2015. – T. 12. – №. 2. – S. 35-39.*
28. *Nikonov E. L., Aksenov V. A. Sovremennye podkhody k diagnostike i lecheniyu nealkogol'noy zhirovoy bolezni pecheni //Profilakticheskaya meditsina. – 2018. – T. 21. – №. 3. – S. 62-69.*
29. *Salikhova S. i dr. Parametry mineral'noy plotnosti kostnoy tkani pri nealkogol'noy zhirovoy bolezni pecheni //Zhurnal vestnik vracha. – 2014. – T. 1. – №. 3. – S. 182-184.*
30. *Marchesini G. Klinicheskie rekomendatsii EASL-EASD-EASO po diagnostike i lecheniyu nealkogol'noy zhirovoy bolezni pecheni //Journal of hepatology. – 2016. – T. 2. – №. 5-P. – S. 1388-1402.*
31. *Egamova M., Mavlyanova Z., Burkhanova G. Primenenie lechebnoy fizkul'tury pri detskikh tserebral'nykh paralichakh v domashnikh usloviyakh //Zhurnal vestnik vracha. – 2018. – T. 1. – №. 2. – S. 114-117.*
32. *Kamalova Y., Sobirova S., Mavlanova Z. Therapeutic gymnastics as an important part of facial nerve neuritis rehabilitation //InterConf. – 2021.*
33. *Tarasova L. V., Tsyganova Yu. V. Rannyaya diagnostika nealkogol'noy zhirovoy bolezni pecheni: rol' biomarkerov i kompleksnykh indeksov nealkogol'nogo steatoza pecheni //Eksperimental'naya i klinicheskaya gastroenterologiya. – 2023. – №. 8. – S. 27-36.*
34. *Mills S. et al. Principles and practice of phytotherapy. Modern herbal medicine. – Churchill Livingstone, 2000.*
35. *Heinrich M. et al. Fundamentals of Pharmacognosy and Phytotherapy: Fundamentals of Pharmacognosy and Phytotherapy E-Book. – Elsevier Health Sciences, 2017.*
36. *Daniyal M. et al. Prevalence and current therapy in chronic liver disorders //Inflammopharmacology. – 2019. – T. 27. – S. 213-231.*
37. *Al-Khazraji K. A. et al. Role of Liv. 52 in non-infectious chronic liver disease //Glob. J. Health Sci. – 2022. – T. 14. – S. 76-92.*
38. *Lamiya A., Bello B., Adda D. K. Biochemistry of Non Infectious Hepatitis-A Review.*
39. *Aidonojie P. A. et al. A Facile Study concerning the Legal Issues and Challenges of Herbal Medicine in Nigeria //The Indonesian Journal of International Clinical Legal Education. – 2022. – T. 4. – №. 4.*