Nonalcoholic fatty liver disease is characterized by excessive fat accumulation in the liver (hepatic steatosis). Nonalcoholic steatohepatitis is characterized by steatosis, liver cell injury, and inflammation. The mechanism of nonalcoholic fatty liver disease is unknown but involves the development of insulin resistance, steatosis, inflammatory cytokines, and oxidative stress. Nonalcoholic fatty liver disease is associated with physical inactivity, obesity, and metabolic syndrome. Screening is not recommended in the general population. The diagnosis is usually made after an incidental discovery of unexplained elevation of liver enzyme levels or when steatosis is noted on imaging (e.g., ultrasonography). Patients are often asymptomatic and the physical examination is often unremarkable. No single laboratory test is diagnostic, but tests of liver function, tests for metabolic syndrome, and tests to exclude other causes of abnormal liver enzyme levels are routinely performed. Imaging studies, such as ultrasonography, computed tomography, and magnetic resonance imaging, can assess hepatic fat, measure liver and spleen size, and exclude other diseases. Liver biopsy remains the criterion standard for the diagnosis of nonalcoholic steatohepatitis. Noninvasive tests are available and may reduce the need for liver biopsy. A healthy diet, weight loss, and exercise are first-line therapeutic measures to reduce insulin resistance. There is insufficient evidence to support bariatric surgery, metformin, thiazolidinediones, bile acids, or antioxidant supplements for the treatment of nonalcoholic fatty liver disease. The long-term prognosis is not associated with an increased risk of all-cause mortality, cardiovascular disease, cancer, or liver disease. (Am Fam Physician. 2013;88(1):35-42. Copyright © 2013 American Academy of Family Physicians.)
for the progression of insulin resistance, diabetes mellitus, and cardiovascular disease. Endothelial dysfunction characterized by abnormal vascular reactivity and atherogenic cytokines may be present. Patients with these conditions may have increased nonfatal cardiovascular events, as well as coronary, cerebrovascular, and peripheral vascular diseases.

**Diagnosis**

Screening for nonalcoholic fatty liver disease is not recommended in the general population. It usually is considered after an incidental discovery of unexplained elevation of liver enzyme levels or when hepatic steatosis is noted on imaging (e.g., ultrasonography).

**HISTORY AND PHYSICAL EXAMINATION**

Patients with nonalcoholic fatty liver disease are often asymptomatic, but symptoms may include right upper quadrant pain, jaundice, and pruritus. Common causes of liver injury, such as alcohol and drug use, must be excluded. The history should explore diet, physical activity, change in weight (usually an increase, such as 40 lb [18 kg] over two to three years), and an assessment for associated conditions (e.g., diabetes, hypertension, hyperlipidemia, obesity, sleep apnea). Physicians should assess risk factors for viral hepatitis, including intravenous drug use, blood transfusions, and sexual activities. Risk factors for nonalcoholic steatohepatitis include age older than 45 years, an aspartate transaminase (AST) level greater than the alanine transaminase (ALT) level, diabetes, insulin resistance, low albumin level (less than 3.6 g per dL [36 g per L]), low platelet count (less than $100 \times 10^3$ per µL [$100 \times 10^9$ per L]), metabolic syndrome, obesity, and portal hypertension on imaging. The patient’s family history should be checked for cardiovascular and metabolic disorders, and chronic liver disease. It may be appropriate

**Table 1. Definitions of Nonalcoholic Fatty Liver Disease, Hepatic Steatosis, and Nonalcoholic Steatohepatitis**

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonalcoholic fatty liver disease</td>
<td>Fatty infiltration of the liver in the absence of other causes of steatosis, such as alcohol</td>
<td>Consider after an incidental discovery of unexplained elevation of liver enzyme levels, or when hepatic steatosis is noted on imaging (e.g., ultrasonography)</td>
</tr>
<tr>
<td>Hepatic steatosis*</td>
<td>Excessive fat accumulation in the liver Steatosis with liver cell injury and inflammation</td>
<td>Intracellular fat in more than 5% of hepatocytes</td>
</tr>
<tr>
<td>Nonalcoholic steatohepatitis*</td>
<td></td>
<td>Hepatocyte ballooning, Mallory hyaline, lymphocytic and neutrophil inflammatory infiltrate in periportal areas; hepatocyte necrosis, apoptosis, and fibrosis may be present</td>
</tr>
</tbody>
</table>

*Hepatic steatosis and nonalcoholic steatohepatitis can be distinguished only by liver biopsy and histology.
to evaluate for the presence of hereditary or less common conditions that may initially present with liver test abnormalities (Table 3). Vital signs should be obtained, including blood pressure, weight, BMI, and waist circumference. Physical examination often is unremarkable, but may include elevated blood pressure, central obesity, and hepatosplenomegaly. Figure 1 presents an algorithmic approach to the evaluation of patients with suspected nonalcoholic fatty liver disease.

**LABORATORY STUDIES**

Laboratory studies may be used to examine factors for insulin resistance and metabolic syndrome, to evaluate liver injury, and to exclude other causes of liver disease. Measures of insulin resistance include the Homeostasis Model Assessment (normal value < 3.99) and the Quantitative Insulin Sensitivity Check Index (normal value > 0.35). Online calculators are available at https://sasl.unibas.ch/11calculators-HOMA.php and https://sasl.unibas.ch/11calculators-QUICKI.php. No single laboratory test is diagnostic for nonalcoholic fatty liver disease. Liver enzyme levels have low sensitivity and specificity, and do not predict clinical outcomes.

Although elevated liver enzyme levels (i.e., AST and ALT levels) occur more commonly in patients with nonalcoholic steatohepatitis compared with hepatic steatosis, not all patients with nonalcoholic steatohepatitis have elevated AST or ALT levels. Tests to exclude viral hepatitis and hemochromatosis should be performed routinely. Additional laboratory evaluation should be considered in patients with chronically elevated liver enzyme levels or in those with a family history of cirrhosis. These tests include measurement of antinuclear antibody, smooth muscle antibody, α₁-antitrypsin, ceruloplasmin, and thyroid-stimulating hormone levels.

### Table 2. Clinical Criteria for Metabolic Syndrome

<table>
<thead>
<tr>
<th>Criteria*</th>
<th>Threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure</td>
<td>≥ 130 mm Hg systolic or ≥ 85 mm Hg diastolic or Anti hypertensive pharmacotherapy in a patient with a history of hypertension</td>
</tr>
<tr>
<td>Fasting glucose level</td>
<td>≥ 100 mg per dL (5.6 mmol per L) or Pharmacotherapy for elevated glucose level</td>
</tr>
<tr>
<td>High-density lipoprotein cholesterol level</td>
<td>&lt; 50 mg per dL (1.29 mmol per L) in women; &lt; 40 mg per dL (1.04 mmol per L) in men or Pharmacotherapy for reduced high-density lipoprotein cholesterol level</td>
</tr>
<tr>
<td>Triglyceride level</td>
<td>≥ 150 mg per dL (1.7 mmol per L) or Pharmacotherapy for elevated triglyceride level</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>≥ 35 inches (89 cm) in women or ≥ 40 inches (102 cm) in men</td>
</tr>
</tbody>
</table>

*—Meeting any three criteria constitutes a diagnosis of metabolic syndrome.


### Table 3. Differential Diagnosis of Uncommon Causes of Chronic Liver Disease

<table>
<thead>
<tr>
<th>Cause of liver disease</th>
<th>Clinical features</th>
<th>Laboratory evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td>α₁-antitrypsin deficiency</td>
<td>Hepatomegaly and elevated liver enzyme levels</td>
<td>α₁-antitrypsin level, phenotype, and liver biopsy</td>
</tr>
<tr>
<td>Autoimmune hepatitis</td>
<td>More common in women, and history of thyroid disease</td>
<td>Antinuclear antibody, smooth muscle antibody, and liver/kidney microsomal antibody tests</td>
</tr>
<tr>
<td>Hereditary hemochromatosis</td>
<td>Bronze diabetes, arthritis, congestive heart failure, impotence, and family history</td>
<td>Complete blood count, ferritin level, transferrin saturation, hemochromatosis (HFE) genetic testing, liver biopsy with staining for iron, and magnetic resonance imaging</td>
</tr>
<tr>
<td>Wilson disease</td>
<td>Neurologic and psychological presentation in addition to liver disease at young age (e.g., younger than 40 years) and family history</td>
<td>24-hour urinary copper measurement, ceruloplasmin level, liver biopsy, and genetic studies</td>
</tr>
</tbody>
</table>
Evaluation of a Patient with Suspected Nonalcoholic Fatty Liver Disease

Patient presents with suspected nonalcoholic fatty liver disease

Obtain history and perform physical examination
Calculate body mass index and measure waist circumference
Perform initial laboratory tests: AST, ALT, alkaline phosphatase, total protein, albumin, total bilirubin, hepatitis B surface antigen, hepatitis C antibody, ferritin, iron, fasting glucose, A1C, lipid panel, and low-density lipoprotein cholesterol levels; prothrombin time

Obtain liver imaging (e.g., ultrasonography, computed tomography, magnetic resonance imaging)
Perform subsequent laboratory tests: AST, ALT, alkaline phosphatase, total protein, albumin, total bilirubin and insulin levels
Consider additional testing in patients with chronically elevated liver enzyme levels or a family history of cirrhosis: antinuclear antibody, smooth muscle antibody, α1-antitrypsin, ceruloplasmin, thyroid-stimulating hormone levels

Treat underlying medical disorders (e.g., diabetes mellitus, hyperlipidemia, hypertension, sleep apnea) and measure AST and ALT levels every three to four months

Initiate lifestyle changes (e.g., healthy diet, weight loss, exercise)

Follow up in six to 12 months

Normalization of AST and ALT levels, and improvement of steatosis on ultrasonography?

No

Perform noninvasive test for fibrosis; consider additional imaging of liver

Negative

Continue routine follow-up

Positive

Refer to gastroenterology

Perform liver biopsy

Steatosis

Continue routine follow-up

Nonalcoholic steatohepatitis

Advanced fibrosis or cirrhosis?

No

Consider experimental pharmacologic treatment in randomized controlled trials

Screen for varices and hepatocellular cancer

Yes

Evaluate for liver transplant

Yes

No

NONINVASIVE TESTS FOR LIVER FIBROSIS

In response to inflammatory cytokines and liver injury, collagen deposition occurs in the liver and results in fibrosis. Noninvasive tests for fibrosis may reduce the need for liver biopsy in patients with nonalcoholic fatty liver disease. A commercially available combination of serologic markers of fibrosis has a sensitivity of 47% and specificity of 90% for determining advanced fibrosis. A validation study in 285 patients with morbid obesity found that noninvasive biomarkers
were accurate for predicting advanced fibrosis.\textsuperscript{21} Other tests include magnetic resonance elastography and various other scoring systems (Table 5).\textsuperscript{22-26}

**LIVER BIOPSY**

Although liver biopsy may not affect treatment decisions, it remains the criterion standard for diagnosis and determination of steatosis, as well as the grade of inflammation and the stage of fibrosis.\textsuperscript{2,13} Liver biopsy is the only test that distinguishes hepatic steatosis from non-alcoholic steatohepatitis, but its routine use is controversial in persons with nonalcoholic fatty liver disease. Liver biopsy should be considered in atypical clinical situations (e.g., patients with normal BMI or highly elevated liver enzyme levels). Hepatic steatosis is defined as the presence of intracellular fat in more than 5% of hepatocytes. Findings in nonalcoholic steatohepatitis include hepatocyte ballooning, Mallory hyaline, and mixed...

### Table 4. Imaging Tests for the Evaluation of Nonalcoholic Fatty Liver Disease

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
<th>LR+*</th>
<th>LR–*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contrast-enhanced CT</td>
<td>84 to 87</td>
<td>75 to 86</td>
<td>59 to 72</td>
<td>92 to 94</td>
<td>1.4</td>
<td>0.06 to 0.09</td>
</tr>
<tr>
<td>Magnetic resonance imaging</td>
<td>96</td>
<td>93</td>
<td>85</td>
<td>98</td>
<td>5.9</td>
<td>0.02</td>
</tr>
<tr>
<td>Ultrasonography</td>
<td>60 to 100</td>
<td>77 to 95</td>
<td>52 to 89</td>
<td>82 to 100</td>
<td>1.1</td>
<td>0 to 0.2</td>
</tr>
<tr>
<td>Unenhanced CT</td>
<td>88 to 95</td>
<td>90 to 99</td>
<td>79 to 98</td>
<td>95 to 98</td>
<td>3.7</td>
<td>0.02 to 0.05</td>
</tr>
</tbody>
</table>
| CT = computed tomography; LR– = negative likelihood ratio; LR+ = positive likelihood ratio; NPV = negative predictive value; PPV = positive predictive value.

*—Calculated using a prevalence of 30%.

Information from references 17 through 19.

### Table 5. Noninvasive Tests for Advanced Fibrosis in Patients with Nonalcoholic Fatty Liver Disease

<table>
<thead>
<tr>
<th>Test</th>
<th>Cutoff</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
<th>LR+*</th>
<th>LR–*</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST/ALT ratio\textsuperscript{22}</td>
<td>0.8</td>
<td>74</td>
<td>78</td>
<td>59</td>
<td>88</td>
<td>1.4</td>
<td>0.1</td>
</tr>
<tr>
<td>AST/platelet count ratio\textsuperscript{22}</td>
<td>1</td>
<td>27</td>
<td>89</td>
<td>51</td>
<td>49</td>
<td>1.1</td>
<td>0.4</td>
</tr>
<tr>
<td>BARD score\textsuperscript{22}</td>
<td>2</td>
<td>89</td>
<td>44</td>
<td>41</td>
<td>90</td>
<td>0.7</td>
<td>0.1</td>
</tr>
<tr>
<td>Enhanced liver fibrosis panel\textsuperscript{23}</td>
<td>10.5</td>
<td>100</td>
<td>98</td>
<td>96</td>
<td>100</td>
<td>21</td>
<td>0 to ∞</td>
</tr>
<tr>
<td>FIB-4\textsuperscript{22}</td>
<td>1.3</td>
<td>85</td>
<td>65</td>
<td>51</td>
<td>91</td>
<td>1.0</td>
<td>0.1</td>
</tr>
<tr>
<td>FibroTest (FibroSure)\textsuperscript{24}</td>
<td>0.7</td>
<td>15</td>
<td>98</td>
<td>76</td>
<td>73</td>
<td>3.2</td>
<td>0.3</td>
</tr>
<tr>
<td>Fibrometer\textsuperscript{25}</td>
<td>0.8</td>
<td>79</td>
<td>96</td>
<td>89</td>
<td>91</td>
<td>8.5</td>
<td>0.09</td>
</tr>
<tr>
<td>Magnetic resonance elastography\textsuperscript{26}</td>
<td>2.74 kPa</td>
<td>94</td>
<td>73</td>
<td>60</td>
<td>97</td>
<td>1.5</td>
<td>0.04</td>
</tr>
<tr>
<td>Nonalcoholic fatty liver disease fibrosis score\textsuperscript{22}</td>
<td>–1.5</td>
<td>78</td>
<td>58</td>
<td>44</td>
<td>86</td>
<td>0.8</td>
<td>0.2</td>
</tr>
</tbody>
</table>

ALT = alanine transaminase; AST = aspartate transaminase; BARD = body mass index, AST/ALT ratio, diabetes mellitus; kPa = kilopascal; LR– = negative likelihood ratio; LR+ = positive likelihood ratio; NPV = negative predictive value; PPV = positive predictive value.

*—Calculated using a prevalence of 30%.

Information from references 22 through 26.
lymphocytic and neutrophilic inflammatory infiltrate in perivenular areas.\textsuperscript{27} In addition, hepatocyte necrosis, apoptosis, and fibrosis may be present, and can be graded and staged.\textsuperscript{27,28} However, liver biopsy poses moderate risk of complications and is invasive, subject to sampling error, and expensive.\textsuperscript{29}

**Treatment**

The goals of therapy include the prevention or reversal of hepatic injury and fibrosis.\textsuperscript{2} Trials examining therapeutic modalities have evaluated disease-oriented outcomes (e.g., reduction in liver enzyme levels, decrease in fibrosis) but have not addressed patient-oriented outcomes (e.g., reduction in morbidity and mortality). Comorbid conditions, such as diabetes, hyperlipidemia, hypertension, or sleep apnea, should be addressed and treated appropriately. Statins are not contraindicated in patients with nonalcoholic fatty liver disease, and the risk of hepatotoxicity in these patients is not increased compared with the general population\textsuperscript{2} (Table 6\textsuperscript{13,30-33}).

**Exercise and Weight Loss**

A healthy diet, weight loss, and exercise are first-line therapeutic measures to reduce insulin resistance in patients with nonalcoholic fatty liver disease.\textsuperscript{2} Although there is no established treatment, a healthy low-fat diet may have benefits independent of weight loss. A modest weight loss of 5% to 10% can result in normalization of AST levels.\textsuperscript{2} A meta-analysis of 49 randomized controlled trials found that weight loss was safe in patients with nonalcoholic fatty liver disease, and it improved liver histology.\textsuperscript{30} Patients should be

---

**Table 6. Treatment Options for Nonalcoholic Fatty Liver Disease**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Liver enzyme levels</th>
<th>Liver histology</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angiotensin receptor blockers</td>
<td>Improved</td>
<td>Improved</td>
<td>Meta-analysis of one RCT; limit use to patients with nonalcoholic fatty liver disease and hypertension\textsuperscript{30}</td>
</tr>
<tr>
<td>Antioxidant supplements (vitamin E, N-acetylcysteine, and oral betaine glucuronate)</td>
<td>Inconsistent</td>
<td>Inconsistent</td>
<td>Systematic review, meta-analysis, and one RCT\textsuperscript{10-32}</td>
</tr>
<tr>
<td>Bariatric surgery</td>
<td>Data not available</td>
<td>Data not available</td>
<td>Systematic review; no RCTs or quasi-RCTs\textsuperscript{30}</td>
</tr>
<tr>
<td>Exercise</td>
<td>Inconsistent</td>
<td>Improved</td>
<td>Meta-analysis\textsuperscript{10}</td>
</tr>
<tr>
<td>Fibrates</td>
<td>Improved</td>
<td>Not effective</td>
<td>Meta-analysis of one RCT\textsuperscript{10}</td>
</tr>
<tr>
<td>l-carnitine</td>
<td>Improved</td>
<td>Not effective</td>
<td>Meta-analysis of one RCT\textsuperscript{10}</td>
</tr>
<tr>
<td>Metformin (Glucophage)</td>
<td>Improved</td>
<td>Inconsistent</td>
<td>Meta-analysis of six RCTs\textsuperscript{30}</td>
</tr>
<tr>
<td>Omega-3 fatty acids</td>
<td>Improved</td>
<td>No effect</td>
<td>Little evidence of effectiveness\textsuperscript{13}</td>
</tr>
<tr>
<td>Orlistat (Xenical)</td>
<td>Improved</td>
<td>Improved</td>
<td>Meta-analysis\textsuperscript{10}</td>
</tr>
<tr>
<td>Pentoxifylline (Trental)</td>
<td>Improved</td>
<td>Inconsistent</td>
<td>Meta-analysis and systematic review\textsuperscript{10,33}</td>
</tr>
<tr>
<td>Simvastatin (Zocor)</td>
<td>Improved</td>
<td>Inconsistent</td>
<td>Meta-analysis\textsuperscript{30}</td>
</tr>
<tr>
<td>Thiazolidinediones (pioglitazone [Actos] and rosiglitazone [Avandia])</td>
<td>Improved</td>
<td>Inconsistent</td>
<td>Meta-analysis of nine RCTs; associated with weight gain\textsuperscript{10,32}</td>
</tr>
<tr>
<td>Ursodeoxycholic acid</td>
<td>Improved</td>
<td>Inconsistent</td>
<td>Meta-analysis of six RCTs\textsuperscript{30}</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Improved</td>
<td>Improved</td>
<td>Meta-analysis\textsuperscript{10}</td>
</tr>
</tbody>
</table>

\textsuperscript{RCT} = randomized controlled trial.

Information from references 13, and 30 through 33.
Nonalcoholic Fatty Liver Disease

PHARMACOLOGIC THERAPY

Three Cochrane reviews found insufficient evidence to support the use of bile acids (e.g., ursodeoxycholic acid), antioxidant supplements, metformin (Glucophage), or thiazolidinediones in the absence of diabetes in patients with nonalcoholic fatty liver disease. Another meta-analysis of 49 randomized controlled trials found that thiazolidinediones (especially pioglitazone [Actos]) improved steatosis and inflammation but were associated with a weight increase of 4.5 to 11 lb (2 to 5 kg) in 66% to 75% of patients and edema in 4% to 10% of patients. This meta-analysis also found conflicting and heterogenous results for metformin, simvastatin (Zocor), antioxidants, pentoxifylline (Trental), telmisartan (Micards), and L-carnitine.

A randomized controlled trial of 247 adults with nonalcoholic steatohepatitis found improvement in AST and ALT levels with the use of vitamin E and pioglitazone but no improvement in fibrosis. Fibrates, statins, and omega-3 fatty acids produce modest improvement in AST levels but do not offer advantages over weight loss and increased physical activity. A systematic review of two randomized trials and four prospective cohort studies found that pentoxifylline reduced AST and ALT levels, although the study did not examine improvements in patient morbidity or mortality.

Prevention

There are no specific studies addressing the prevention of nonalcoholic fatty liver disease; however, because obesity and physical inactivity are strongly correlated with the condition, it is reasonable to expect that increasing physical activity and encouraging weight loss would be helpful. Patients with nonalcoholic fatty liver disease should be immunized for hepatitis A and B, and be encouraged to limit alcohol use to prevent the development of alcohol-induced liver disease.

Prognosis

A large prospective cohort study in the United States involving 11,371 adults found that nonalcoholic fatty liver disease was not associated with an increased risk of all-cause mortality, cardiovascular disease, cancer, or liver disease. Although hepatic steatosis rarely progresses to cirrhosis, 15% to 30% of patients with nonalcoholic steatohepatitis progress to advanced fibrosis, and 12% to 35% with advanced fibrosis progress to cirrhosis. Patients with cirrhosis should be monitored for signs of portal hypertension, disease progression, and hepatocellular carcinoma.

Data Sources: A PubMed search was completed in Clinical Queries using the key search terms nonalcoholic fatty liver disease, hepatic steatosis, nonalcoholic steatohepatitis, pathogenesis, diagnosis, and treatment. The search included meta-analyses, randomized controlled trials, clinical trials, and reviews. We also searched the Agency for Healthcare Research and Quality evidence reports, Clinical Evidence, the Cochrane database, Essential Evidence Plus, the National Guideline Clearinghouse, and DynaMed. Search date: April 4, 2011.

The Authors

THAD WILKINS, MD, is a professor in the Department of Family Medicine at Georgia Regents University in Augusta.

ALTAF TADKOD, MD, is a family physician in private practice at Barrow Regional Medical Center in Winder, Ga. At the time this article was written, Dr. Tadkod was a third-year resident in the Department of Family Medicine at Georgia Regents University.

IRYNA HEPBURN, MD, is a gastroenterologist in private practice at the Good Samaritan Digestive Health Specialists in Lebanon, Pa. At the time this article was written, Dr. Hepburn was a gastroenterology fellow at the Georgia Regents University.

ROBERT R. SCHADE, MD, is a professor at the University of Pittsburgh (Pa.) Medical Center Presbyterian. At the time this article was written, Dr. Schade was a professor in the Department of Medicine, the chief of the Division of Gastroenterology and Hepatology, and the medical director of the Special Procedures/Endoscopy Unit at Georgia Regents University.

Address correspondence to Thad Wilkins, MD, Georgia Regents University, 1120 15th St., HB-4032, Augusta, GA 30912 (e-mail: jwilkins@gru.edu). Reprints are not available from the authors.

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