Bay Leaf

Potential Health Benefits

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Bay leaf or bay laurel is a culinary herb obtained from the small evergreen tree Laurus nobilis L. (family Lauraceae). This plant is native to the Mediterranean region and enjoys widespread cultivation in the warm climates of the southern United States, Central America, Europe, the Middle East, and Asia. As a culinary herb, it is known to flavor numerous cuisines of Turkey, India, Italy, and France. It is a common component in the French mixture of herbs “bouquet garni” and is added to various dishes during cooking such as sauces, soups, stocks, casseroles, sausages, and stews and then removed before consumption. Many uses of bay leaf and its oil in traditional and folk medicines have been documented. These include as treatment of respiratory disorders and infections, gastrointestinal discomfort and irregularity, diarrhea, and amenorrhea and as an emetic, stimulant, and diuretic, to name a few. To date, few human studies have examined the health benefits of bay leaves. This narrative review summarizes relevant clinical trials and preclinical animal investigations, and highlights future research needs.


Bay leaf or bay laurel is a culinary herb obtained from the small evergreen tree Laurus nobilis L. (family Lauraceae). This plant is native to the Mediterranean region and enjoys widespread cultivation in the warm climates of the southern United States, Central America, Europe, the Middle East, and Asia. The true bay leaf (Figure 1) is not to be confused with similarly named substitutes such as Indian bay leaf (Litsea glaucescens), West Indian bay leaf (Pimenta racemosa), Indonesian bay leaf (Syzygium polyanthum), and California bay leaf (Umbellularia californica). These others do not exhibit the same odor and flavor profile as the genuine bay leaf and, in some cases, may have undesirable adverse effects when consumed. In ancient times, bay laurel had decorative uses as a symbol for prosperity, fame, and victory and was perceived to possess protective powers and thus was planted near homes to prevent lightning strikes.1–5 As a culinary herb, it flavors numerous cuisines of Turkey, India, Italy, and France. It is a common component in the French mixture of herbs “bouquet garni” and is added to many dishes during cooking such as sauces, soups, stocks, casseroles, sausages, and stews and then removed before consumption. Although the amount of bay leaf used in recipes varies depending on the specific dish, a usual amount is 1 dried bay leaf (approximately 200 mg). Specific examples include stifado in Greece, ratatouille and coq au vin in France, traditional stews of rabbit and wild goat in Crete, and balloccoli in Italy for which bay leaf is boiled with chestnuts.6 The aroma of bay leaf is described as woody herbal similar to that of oregano and thyme. When crushed, a scent of menthol and eucalyptus is evident, and its flavor is considered astringent and bitter. The essential oil is used in the food industry to season and preserve meat and fish products as well as in the perfume industry as a component of select fragrances.7 The essential oil distilled from the leaves contains a variety of phytochemicals with 1,8-cineole or eucalyptol (Figure 2) being predominant. The chemical composition and bioactivity of the leaf can vary considerably by geographic origin, growing conditions, harvest season, and processing methods. Nevertheless, the other main essential oil constituents often include α-terpinyl acetate, α-pinene, sabine, and linalool.8–10 Many uses of bay leaf and its oil in traditional and folk medicines have been documented. These include as treatment of respiratory disorders and infections, gastrointestinal discomfort and irregularity, diarrhea, and amenorrhea and as an emetic, stimulant, and diuretic, to name a few.1,11 The specific constituents and mechanisms of action contributing to these purported benefits have yet to be characterized. To date, few human studies have examined the beneficial effects of bay leaves. This narrative review summarizes relevant clinical trials and preclinical animal investigations, and highlights future research needs.

METHODS

Studies providing evidence for potentially beneficial effects of foods, ingredients, and plant constituents assess data from a variety of scientific sources such as cell culture experiments, animal studies, and human clinical trials. Human
studies are particularly important in determining public health recommendations, especially when based on randomized, controlled trials testing well-characterized treatments and applying appropriate statistical analyses. With this in mind, a search of the PubMed and Science Direct databases was conducted using terms that included *Laurus nobilis*, bay leaf, laurel leaf, 1,8-cineole, and eucalyptol. Full reports of English-language publications and English-language abstracts of foreign-language articles from peer-reviewed journals were the primary sources of information. Although the quality of identified studies varied considerably, all relevant, published investigations were included in this overview so that the totality and diversity of information can be described and issues for future research can be identified. Additional information was gleaned from bibliographies within these sources. Studies of bay leaf as a component within multi-ingredient preparations were not included in this overview.

**Bioavailability**

Limited information exists about the bioavailability of bay leaf constituents after human consumption. Such data are important because any potential influence of bay leaf on human health is impacted by how much is consumed, as well as the ultimate amount and form of its individual bioactive components distributed throughout the body. No studies were identified that examined the bioavailability of bay leaf fractions, its essential oils, or isolated, active ingredients after human ingestion. Several reports describe the occurrence in breast milk and the breath of 1,8-cineole, a major constituent of the essential oil, after human intake.12-14 In possums, oral administration of 1,8-cineole (100-mg/kg body weight) resulted in detection in blood of its alcohol and carboxylic acid metabolites, and bioavailability was low.15 In rats, 1,8-cineole is rapidly absorbed into the bloodstream and metabolized quickly. After an oral dose (30.24-mg/kg body weight), the maximum mean serum concentration was 356 pg/mL, the time to reach maximum mean serum concentration was 10 minutes, and the terminal half-life was 22 hours.16 Much more needs to be understood about the systemic fate of bay leaf extracts when included in the human diet.

**Human Studies of Medical Conditions**

Reviews of experimental data suggest that phytochemicals found in bay leaf have the potential to counteract signs and symptoms of diabetes and cardiovascular disease. Only a few human trials evaluated bay leaf administration on these health outcomes, and these studies generally evaluated small patient populations (n = 10-45) and were of short duration (10-30 days). Moreover, the manner, dose, and form of bay leaf administered varied considerably among trials, making conclusions about benefits difficult to formulate. To examine its impact on blood glucose and lipid regulation, healthy volunteers (n = 30) in Tunisia were provided a bay leaf tea (prepared from 5-g powder per 100-mL boiled water) for 10 days.17 This study lacked controls. Compared with baseline values, no significant changes were detected for fasting blood glucose (FBG) concentrations, and blood levels of low-density lipoprotein (LDL) cholesterol, total cholesterol (TC), and triglycerides (TG). Serum urea concentrations significantly decreased. Two trials in Pakistan addressed the influence of bay leaf on individuals with type 2 diabetes mellitus. For the first trial,18 4 groups (n = 10/group) received either placebo, or doses of 1-, 2-, or 3-g/d powdered leaves for 30 days. Compared with placebo, for all doses, significant decreases in FBG, TC, LDL, and TG were observed along with increased high-density lipoprotein (HDL) cholesterol values. No dose response was apparent, suggesting that lower doses may have efficacy. Examining lower doses is reasonable, because dietary exposure to ingredients in whole bay leaf (which is not directly consumed) in most

FIGURE 1. Bay leaves.
cuisines is unlikely to be of a similar magnitude to those amounts ingested as 1 to 3 g of powdered leaf. In a randomized, crossover trial (n = 20), bay leaf powder was provided incorporated in cookies at levels of either 5 or 10 g.\textsuperscript{19} Compared with controls, the 10-g dose significantly decreased postprandial blood glucose concentrations at 30 and 45 minutes post dosing, although the blood glucose incremental areas under the curve did not differ among groups. Measures of appetite and gastrointestinal comfort were not affected. A trial of type 1 diabetes mellitus (T1DM) subjects in Jordan was conducted in which placebo (n = 10) or 3-g/d bay leaf powder (n = 45) was given for 30 days.\textsuperscript{20} Compared with baseline values, bay leaf administration resulted in significant decreases in FBG, TC, and LDL levels along with an increase in HDL concentrations. No changes were observed in those individuals provided the placebo.

The main bay leaf essential oil constituent 1,8-cineole was evaluated in humans for its impact on respiratory distress. A double-blind, placebo-controlled trial was conducted to evaluate its anti-inflammatory activity in patients with steroid-dependent bronchial asthma.\textsuperscript{21} Subjects with severe asthma received 1,8-cineole (200 mg, 3×/d) for 12 weeks. Compared with placebo, those receiving 1,8-cineole were able to use significantly less glucocorticosteroid-sparing medication without any compromise in lung function. Longer follow-up and glucocorticoid-sparing benefits in those with mild to moderate asthma need to be assessed before clinical applications can be considered.\textsuperscript{22}

### Animal Studies

The Table summarizes the effects of bay leaf administration on signs and symptoms of multiple disorders in animal models of disease. Because human studies have evaluated the impact of bay leaf on blood glucose and lipid dysregulation, highlighting similar investigations in several animal models listed is of interest. In diabetic rats, orally administered essential oil (200 mg/kg, 28 days) significantly decreased FBG and serum alanine aminotransferase, aspartate aminotransferase (AST), creatine kinase, and urea, compared with controls.\textsuperscript{23} In addition, treatment with the oil counteracted diabetes-associated damage to the pancreas, liver, and kidney. In diabetic rabbits, oral dosing with an aqueous extract of the leaves resulted in an inconsistent effect of dose in lowering FBG.\textsuperscript{24} In another experiment with rats provided a high-fat diet, those fed this diet supplemented with 1 mg/kg (w/w) bay leaf for 5 days exhibited a significant decrease in FBG, TC, LDL, TG, AST, and alanine aminotransferase and an increase in HDL, compared with controls.\textsuperscript{25} An ethanolic extract of bay leaf was administered orally (200 mg/kg, 30 days) to diabetic rats (n = 10/group).
<table>
<thead>
<tr>
<th>Condition</th>
<th>Treatment</th>
<th>Outcomes</th>
<th>Ref.</th>
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</thead>
<tbody>
<tr>
<td>Blood glucose and lipid dysregulation</td>
<td>Leaf powder, water extract, essential oil (E.O.)</td>
<td>↓FBG, ↓TC, ↓TG, ↓LDL, ↑HDL, ↓ALT, ↓AST, ↓pancreatic damage</td>
<td>23-26</td>
</tr>
<tr>
<td></td>
<td>1,8-cineole</td>
<td>↓Hyperglycemia- and LPS-induced vascular injury, ↓AGE, ↑antioxidant and anti-inflammatory defenses</td>
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<td>Wound healing</td>
<td>Leaf water extract, E.O.</td>
<td>↑Wound healing</td>
<td>31,32</td>
</tr>
<tr>
<td></td>
<td>Cinnamattannin B-1</td>
<td>↑Wound healing, ↑tissue regeneration, ↑remodeling and ↑angiogenesis in wound</td>
<td>33</td>
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<td>Gastrointestinal distress</td>
<td>Leaf water extract, methanol extract, E.O.</td>
<td>↓Ethanol-induced gastric injury, ↓chemical-induced diarrhea</td>
<td>34-37</td>
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<td></td>
<td>Cinnamattannin B-1, 1,8-cineole</td>
<td>↓Stress- and ethanol-induced gastric injury</td>
<td>38-41</td>
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<td>Alcoholic disorders</td>
<td>Leaf methanol extract</td>
<td>↓Gastric emptying of ethanol load</td>
<td>42</td>
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<td></td>
<td>Sesquiterpenes</td>
<td>↓Ethanol absorption, ↓blood ethanol increase after ethanol load</td>
<td>43,44</td>
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<td>Inflammation</td>
<td>Leaf methanol extract, ethanol extract, hexane extract</td>
<td>↓Chemical-induced ear and paw edema, ↓LPS-induced lung inflammation, ↓lung fluid proinflammatory cytokines and chemokines ↓Propionobacterium acnes-induced skin lesions, inflammation, and inflammatory molecules</td>
<td>45-49</td>
</tr>
<tr>
<td></td>
<td>1,8-cineole</td>
<td>↓Smoke-induced lung injury; ↓proinflammatory cytokines, mucus hypersecretion, MUC5AC protein expression, ciliated lung cell damage, and bacterial colonization in smoke-treated lungs; ↑tissue repair and remodeling in smoke-damaged lungs; ↓Virus infection-induced lung pneumonia; ↓Chemical-induced acute pancreatitis</td>
<td>50-56</td>
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<td>Neurological disorders</td>
<td>Leaf ethanol extract, hexane extract</td>
<td>↓Chemical- and electrical-induced seizures</td>
<td>57-60</td>
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<td></td>
<td>Leaf incense inhalation</td>
<td>↓SCO-induced cognitive deficits, ↓SCO-induced oxidative stress and AcChE activity in rat hippocampus</td>
<td>61</td>
</tr>
<tr>
<td></td>
<td>1,8-cineole</td>
<td>↓Chemical-, thermal-, and mechanical-induced neuropathic pain and inflammation, and mRNA and protein expression of purinoreceptor P2X3 in spinal cord dorsal horn ↓Subarachnoid hemorrhage-induced brain edema, neurological deficits, and proinflammatory cytokines</td>
<td>53,62,63</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Leaf ethanol extract, methanol extract, sesquiterpenes lactone reynosin, 1,8-cineole</td>
<td>Hepatoprotective, renoprotective, and anticancer properties</td>
<td>64-69</td>
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</table>

Abbreviations: AcChE, acetylcholinesterase; AGE, advanced glycation end products; ALT, alanine aminotransferase; AST, aspartate aminotransferase; FBG, fasting blood glucose; HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; LPS, lipopolysaccharide; MUC5AC, mucin SAC, oligomeric mucus/gel-forming (Homo sapiens [human]); P2X3, P2X purinoreceptor 3; SCO, scopolamine; TC, total cholesterol; TG, triglycerides; 6-OHDA, 6-hydroxydopamine.
Compared with controls, those diabetic animals fed the extract exhibited a decrease in blood levels of FBG, TC, TG, LDL, AST, and AST and an increase in HDL and insulin. Oral dosing of diabetic mice with 1,8-cineole (10 mg/kg, 8 weeks), compared with controls, resulted in a significant lowering of diabetes-induced structural damage to kidney glomeruli, in enhanced filtration function, and in lower amounts of advanced glycation end products within the kidney. In a rat model of T1DM, a novel nanoparticle delivery system enhanced oral absorption of 1,8-cineole (18 mg/kg, 1 month) and protected against hyperglycemia-induced vascular endothelial injury by stimulating antioxidant defenses. In 2 rodent studies, 1,8-cineole administered at oral doses of 50 to 300 mg/kg for 7 days decreased lipopolysaccharide-induced vascular endothelial injury by enhancing anti-inflammatory responses. Specifically, this bay leaf phytochemical lowered the expression in the thoracic aorta of vascular cell adhesion molecule-1, which participates in the adhesion of lymphocytes, monocytes, eosinophils, and basophils to the vascular endothelium. This change in vascular cell adhesion molecule-1 expression was mediated by modulation of the transcription factors peroxisome proliferator-activated receptor γ and the protein complex nuclear factor 𝜅 light chain enhancer of activated B cells (NF-κB). These animal studies provide limited insights about the mechanisms by which bay leaf may influence blood glucose and lipid regulation. The diverse samples used in these preclinical experiments, the disparate dosing, and, in some cases, the inconsistent responses to bay leaf make these findings preliminary in nature.

As summarized in the Table, bay leaf and 1,8-cineole have demonstrated other benefits in animal models toward such conditions involving wound healing, gastrointestinal distress, inflammation, and neurological disorders. This suggests that examining these outcomes in humans may be worthwhile after additional preclinical characterizations of bay leaf and its constituents for dose-related benefits and adverse effects are completed. The identities of other bay leaf constituents, besides 1,8-cineole, that have potentially beneficial biological actions have not been fully explored.

**Safety**

In the clinical trials conducted to date, no notable adverse effects were reported after oral bay leaf treatment in the amounts and durations studied. There is some anecdotal clinical evidence of systemic, allergic dermatitis after ingestion of foods containing bay leaves, and contact dermatitis cases were described in early reports from Europe. In mice, a high dose of aqueous extracts of bay leaves administered orally for 3 weeks (667 mg/kg per day) demonstrated no major clinical or behavioral changes other than mild liver inflammation. Laurel leaf oil is considered generally recognized as safe for customary food uses by the Food and Drug Administration (21 CFR 121.101). Estimated consumption levels of this oil were not identified. However, for West Indian bay leaf oil (Pimenta acris), its estimated intake is 60 μg/person per day.

**CONCLUSIONS**

Human data to support health benefits from culinary uses of bay leaf are limited, and findings from animal studies are preliminary. Future human trials should focus on an effect of this spice and its phytochemicals on biomarkers of blood glucose and lipid dysfunction. For these outcomes as well as for other potential effects of bay leaf, additional large, well-controlled clinical trials are needed along with considerably more preclinical data detailing specific dose-related effects, mechanisms of action, and any safety issues after longer administration of specific preparations.

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