How diet and lifestyle affect duodenal ulcers
Review of the evidence

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ABSTRACT

OBJECTIVE To demonstrate the role of diet in reducing or aggravating risk of duodenal ulcer (DU).

QUALITY OF EVIDENCE MEDLINE was searched from January 1966 to December 2001 for articles on the relationship between diet and lifestyle and DU using the key words duodenal ulcer and diet, fibre, or lifestyle. Evidence that these factors are associated with DU arose mainly from three case-control and three prospective studies (level II evidence) and from expert opinion (level III evidence).

MAIN MESSAGE A high-fibre diet appears to reduce risk of DU; soluble fibre might be associated with reduced risk also. Vitamin A intake is associated with lower risk of DU. Little evidence indicates that fat, type of fat, protein intake, or consumption of alcohol or caffeine affect the etiology of DU.

CONCLUSION A high-fibre diet, particularly if the fibre comes from fruit and vegetables, could reduce risk of DU; vitamin A might also be beneficial.

RÉSUMÉ

OBJECTIF Établir le rôle de l’alimentation comme facteur de risque de l’ulcère duodénal (UD).

QUALITÉ DES PREUVES Les articles sur la relation entre l’UD et l’alimentation ou le mode de vie ont été repérés dans MEDLINE à l’aide des mots clés duodenal ulcer et diet, fibre ou lifestyle. Les preuves d’une association entre ces facteurs et l’UD provenaient principalement de trois études de cas avec témoins, de trois études prospectives (preuves de niveau II) et d’opinions d’experts (preuves de niveau III).

PRINCIPAL MESSAGE Un régime riche en fibres semble réduire le risque d’UD; les fibres solubles pourraient aussi avoir cet effet. La prise de vitamine A est associée à un moindre risque. Il y a peu de preuves indiquant que le gras, le type de gras et les protéines alimentaires, ou encore la consommation d’alcool ou de caféine influencent l’incidence de l’UD.

CONCLUSION Un régime riche en fibres, surtout si elles proviennent des fruits et légumes, pourrait réduire le risque d’UD; la vitamine A pourrait aussi avoir cet effet.
A progressive increase in incidence of duodenal ulcer (DU) was seen in Western countries early in the 20th century; a gradual decline in incidence was noted starting in the 1960s. The decline, however, has not been uniform, and incidence of DU varies among and within Western countries. Currently, incidence of DU is increasing rapidly in most of the developing world. Despite the decline in incidence of DU in Western countries, it is still a common condition. About 300,000 new cases are diagnosed each year in the United States. It is still unclear why rates of DU declined, but the change points to the influence of non-genetic factors. Changing levels of environmental ulcerogens or antiulcerogenic factors might be important.

Several reports have provided evidence that infection with *Helicobacter pylori* increases risk of DU. Among DU patients, 75% to 100% have evidence of *H pylori* infection. Its prevalence in the general US population is about 20% among people born after 1950 and higher among older people. A similar rate of infection would be expected among Canadians. *Helicobacter pylori* infection has been shown to be a factor in relapse after healing, but whether it is the main cause of duodenal ulceration is still uncertain.

A 1994 US National Institutes of Health consensus panel recommended that initial therapy for people with DU should be elimination of *H pylori* infection. A Canadian economic analysis concluded that early eradication of *H pylori* was less costly and had the same or better outcomes than either delayed (after first recurrence) eradication or traditional drug therapy with ranitidine.

Because only a few people with *H pylori* infection eventually develop ulcers, it could be that *H pylori* infection alone is insufficient to cause ulceration and that other factors might also be important, either through synergy with *H pylori* or through a separate mechanism. Researchers have long suspected that diet is associated with DU, and dietary differences have been cited to explain the geographic variation in this disease.

Dietary factors that have been investigated include dietary fibre; fatty acids, particularly linoleic acid; vitamins C, E, and A; alcohol; and caffeine. Other lifestyle factors that might have a role in development of DU include smoking, physical activity, and body mass index. The objectives of this article are to summarize the evidence on the association between diet and lifestyle and DU and to recommend a diet that could serve as adjunct therapy to *H pylori* eradication.

**Quality of evidence**

MEDLINE was searched from January 1966 to December 2001 for articles on the relationship between diet and lifestyle and DU using the key words duodenal ulcer and diet, fibre, or lifestyle. Other articles were found from the references of the first articles generated by the search. Evidence that diet and lifestyle are associated with DU arose mainly from three case-control and three prospective studies (level II evidence) and from expert opinion (level III evidence). Other reports discussing peptic ulcer in general without making the important distinction between duodenal and gastric ulcers were not included. A few studies investigated the association between diet and DU prospectively, a method less prone to bias, but they examined associations in men only, and their results might not apply to women.

**Dietary fibre**

Fibre varies considerably in its physical properties and chemical composition. Crude fibre consists of cellulose and lignin. Dietary fibre includes crude fibre and noncellulose polysaccharides, hemicelluloses, pectins, and gums.

Dietary fibre can be classified according to its water solubility. The structural fibres, cellulose, lignin, and some hemicelluloses, are insoluble; the natural gel-forming fibres, pectins, gums,

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mucilages, and remaining hemicelluloses, are soluble.\textsuperscript{16} Table 1\textsuperscript{17} shows some of the highest contributors to total dietary fibre and fibre types by serving size as reported by the Health Professionals Follow-up Study.\textsuperscript{17}

In the past, a bland, low-fibre diet was recommended to patients with ulcers, but in the late 1970s, evidence that a higher-fibre diet was beneficial in ulcer treatment began to accumulate. A small clinical trial in India\textsuperscript{18} compared a rice diet with an unrefined wheat diet among ulcer patients over a 5-year period. Results showed that 81\% of patients on the rice diet had ulcers recur, while only 14\% of patients on the unrefined wheat diet had ulcers recur. Results of a small clinical trial\textsuperscript{19} showed that incidence of DU recurrence was lower among those who had been on a higher-fibre diet (28.2 g/d) for 6 months than among those on a lower-fibre diet (11.4 g/d). Authors of a 1990 case-control study\textsuperscript{20} attributed the protective effect not to dietary fibre, but to low intake of refined sugar because a relatively higher intake of unrefined carbohydrate might provide a gastric acid buffer. In that study, however, relative risk (RR) of ulcer disease, though not consistent or significant, tended to be reduced by both high vegetable fibre intake (highest quintile RR 0.60, 95\% confidence interval [CI] 0.23 to 1.6) and low refined sugar intake (highest quintile RR 3.7, 95\% CI 1.0 to 13.0). Separating the effects of refined sugar and fibre on nutrition-related disease is difficult in diet studies because higher-fibre diets are generally lower in refined sugar.

A prospective cohort study, the Health Professionals Follow-up Study, which looked at 51 529 US male health professionals aged 40 to 75 years in 1986,\textsuperscript{21} showed that fibre from legumes had the greatest influence on DU risk reduction (RR 0.57, 95\% CI 0.33 to 0.97). Legumes include beans, tofu, peanut butter, and nuts, which are good sources of soluble fibre. Soluble fibre reduced risk of DU significantly more than insoluble fibre did (RR 0.42; 95\% CI 0.23 to 0.75). Cereal fibre intake was associated with an upward trend in risk of DU, but this result was not statistically significant. Table 2\textsuperscript{21} summarizes the RRs associated with dietary fibre and fibre types and sources.

Data from the current study suggest that fibre of different types (soluble or insoluble) and sources (fruit, vegetable, legume, or cereal) affects risk of DU to varying, but not necessarily clinically significant, degrees. Duodenal ulcer is associated with a large acid load to the duodenum caused by several factors including increased acid output and insufficient pancreatic secretion.\textsuperscript{22} The liquid content of meals is emptied more rapidly into the

<table>
<thead>
<tr>
<th>FOOD</th>
<th>CONTRIBUTION TO TOTAL DIETARY FIBRE (%)</th>
<th>SERVING SIZE</th>
<th>TOTAL DIETARY FIBRE (G)</th>
<th>SOLUBLE FIBRE (G)</th>
<th>INSOLUBLE FIBRE (G)</th>
<th>HEMICELULOSE (G)</th>
<th>CELLULOSE (G)</th>
<th>LIGNIN (G)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cold cereals*</td>
<td>8.81</td>
<td>1 C (47 g)</td>
<td>6.00</td>
<td>1.04</td>
<td>4.40</td>
<td>3.29</td>
<td>0.68</td>
<td>0.42</td>
</tr>
<tr>
<td>Potatoes</td>
<td>6.36</td>
<td>1 C (210 g)</td>
<td>4.20</td>
<td>1.59</td>
<td>1.98</td>
<td>0.27</td>
<td>1.60</td>
<td>0.11</td>
</tr>
<tr>
<td>Apple</td>
<td>6.33</td>
<td>1 (138 g)</td>
<td>3.70</td>
<td>0.83</td>
<td>1.69</td>
<td>0.64</td>
<td>1.03</td>
<td>0.02</td>
</tr>
<tr>
<td>Banana</td>
<td>4.11</td>
<td>1 (114 g)</td>
<td>2.70</td>
<td>0.34</td>
<td>1.22</td>
<td>0.26</td>
<td>1.58</td>
<td>0.33</td>
</tr>
<tr>
<td>Dark bread</td>
<td>3.90</td>
<td>1 slice (25 g)</td>
<td>1.10</td>
<td>0.28</td>
<td>2.10</td>
<td>1.58</td>
<td>0.33</td>
<td>0.20</td>
</tr>
<tr>
<td>Oranges</td>
<td>3.78</td>
<td>1 (131 g)</td>
<td>3.10</td>
<td>1.96</td>
<td>0.64</td>
<td>0.03</td>
<td>0.30</td>
<td>0.31</td>
</tr>
<tr>
<td>Peas</td>
<td>3.28</td>
<td>1/2 C (80 g)</td>
<td>4.40</td>
<td>0.48</td>
<td>2.89</td>
<td>0.66</td>
<td>2.14</td>
<td>0.22</td>
</tr>
<tr>
<td>Carrots</td>
<td>2.82</td>
<td>1/2 C (78 g)</td>
<td>2.60</td>
<td>1.09</td>
<td>1.40</td>
<td>0.15</td>
<td>0.97</td>
<td>0.11</td>
</tr>
<tr>
<td>Tomato sauce</td>
<td>2.74</td>
<td>1/2 C (125 g)</td>
<td>4.20</td>
<td>0.63</td>
<td>3.15</td>
<td>0.45</td>
<td>1.97</td>
<td>0.69</td>
</tr>
<tr>
<td>Beans</td>
<td>2.69</td>
<td>1/2 C (131 g)</td>
<td>6.70</td>
<td>2.24</td>
<td>11.11</td>
<td>8.05</td>
<td>2.79</td>
<td>0.23</td>
</tr>
</tbody>
</table>

Data from Aldoori et al.\textsuperscript{17}

*Includes bran cereals, Shredded Wheat being most commonly reported.
How diet and lifestyle affect duodenal ulcers

Experimental studies\textsuperscript{26-27} have shown that large amounts of gel polysaccharides, such as guar gums and pectin, can delay gastric emptying of meals. If DU is related not only to the presence of a factor such as $H$ \textit{pylori}, but also to impaired gastrointestinal function, then additional intake of dietary fibre could contribute to reduced recurrence of DU.

In a case-control study, Grant et al\textsuperscript{30} observed that the mean percentage of linoleic acid in adipose tissue was significantly lower in men with chronic duodenal ulcer (10 ± 0.7%) than in controls (12.3 ± 0.7%). Levels of linoleic acid in adipose tissue might indicate whether the diet contains sufficient linoleic acid.

Conversely, the lack of an association between linoleic acid and reduced risk of DU observed in the Health Professionals Follow-up Study\textsuperscript{21} (RR 1.25, 95% CI 0.75 to 2.09) was attributed to the fact that research has shown that increased dietary linoleic acid intake does not increase the concentration of prostaglandin precursor, arachidonic acid, in membrane phospholipid. As prostaglandins exert a cytoprotective effect on the gastrointestinal tract, it could be this, rather than linoleic acid itself, that helps prevent DU. Additional research could determine whether linoleic acid intake has a meaningful effect on DU.

Table 2. Effect of dietary fibre sources and types on relative risk of duodenal ulcer: Comparing highest to lowest quintiles.

<table>
<thead>
<tr>
<th>FIBRE SOURCE OR TYPE</th>
<th>RELATIVE RISK*</th>
<th>95% CONFIDENCE INTERVAL</th>
<th>MULTIVARIATE RELATIVE RISK*</th>
<th>95% CONFIDENCE INTERVAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total dietary fibre</td>
<td>0.56</td>
<td>0.32-0.99</td>
<td>0.55</td>
<td>0.31-0.96</td>
</tr>
<tr>
<td>Total crude fibre</td>
<td>0.48</td>
<td>0.26-0.87</td>
<td>0.50</td>
<td>0.28-0.90</td>
</tr>
<tr>
<td>Soluble fibre</td>
<td>0.42</td>
<td>0.23-0.75</td>
<td>0.40</td>
<td>0.22-0.74</td>
</tr>
<tr>
<td>Insoluble fibre</td>
<td>0.61</td>
<td>0.35-1.05</td>
<td>0.59</td>
<td>0.34-1.03</td>
</tr>
<tr>
<td>Fruit fibre</td>
<td>0.57</td>
<td>0.31-1.05</td>
<td>0.62</td>
<td>0.35-1.11</td>
</tr>
<tr>
<td>Vegetable fibre</td>
<td>0.68</td>
<td>0.41-1.13</td>
<td>0.70</td>
<td>0.42-1.17</td>
</tr>
<tr>
<td>Leguminous fibre</td>
<td>0.57</td>
<td>0.33-0.97</td>
<td>0.56</td>
<td>0.33-0.96</td>
</tr>
<tr>
<td>Cereal fibre</td>
<td>1.43</td>
<td>0.81-2.52</td>
<td>1.38</td>
<td>0.78-2.46</td>
</tr>
</tbody>
</table>

Data from Aldoori et al.\textsuperscript{21} *Adjusted for age and total energy.
\textsuperscript{*}Adjusted for age, body mass index, smoking, and use of nonsteroidal anti-inflammatory drugs and acetylsalicylic acid.

Fatty acids

Experimental data from Hollander and Tarnawski\textsuperscript{28} demonstrated that the ability of the gastroduodenal mucosa to resist injury or accelerate its healing might be related to dietary intake of linoleic acid. Linoleic acid is the body’s main source of arachidonic acid, the precursor to prostaglandins, which exhibit cytoprotective activity in the duodenum. The main dietary sources of linoleic acid are vegetable and seed oils; vegetable oils replaced lard in Western diets in the latter half of the 20th century. Using observational data, Hollander and Tarnawski suggested that this could partly explain the decreased incidence of DU.\textsuperscript{29}

Vitamin A

Table 3\textsuperscript{21} shows a summary of micronutrients’ effect on risk of DU from the Health Professionals Follow-up Study. Aldoori and colleagues\textsuperscript{21} found an inverse association between vitamin A and risk of DU (RR 0.39, 95% CI 0.20 to 0.76 for highest quintile of intake, or 26 769 IU/d). Effect on the RR of DU from plant sources of vitamin A (measured as carotenoids, and

Table 3. Effect of micronutrients on relative risk of duodenal ulcer: Comparing highest to lowest quintiles.

<table>
<thead>
<tr>
<th>MICRONUTRIENT</th>
<th>RELATIVE RISK*</th>
<th>95% CONFIDENCE INTERVAL</th>
<th>MULTIVARIATE RELATIVE RISK*</th>
<th>95% CONFIDENCE INTERVAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin E</td>
<td>0.60</td>
<td>0.35-1.02</td>
<td>0.59</td>
<td>0.35-1.01</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>0.39</td>
<td>0.20-0.76</td>
<td>0.46</td>
<td>0.23-0.91</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>1.00</td>
<td>0.58-1.72</td>
<td>1.10</td>
<td>0.62-1.93</td>
</tr>
<tr>
<td>Folic acid</td>
<td>0.64</td>
<td>0.38-1.09</td>
<td>0.70</td>
<td>0.40-1.23</td>
</tr>
<tr>
<td>Vitamin B\textsubscript{1}</td>
<td>0.60</td>
<td>0.34-1.06</td>
<td>0.62</td>
<td>0.34-1.11</td>
</tr>
<tr>
<td>Vitamin B\textsubscript{2}</td>
<td>0.55</td>
<td>0.31-0.96</td>
<td>0.54</td>
<td>0.31-0.97</td>
</tr>
<tr>
<td>Potassium</td>
<td>0.41</td>
<td>0.23-0.72</td>
<td>0.55</td>
<td>0.30-1.02</td>
</tr>
</tbody>
</table>

Data from Aldoori et al.\textsuperscript{21} *Adjusted for age and total energy.
\textsuperscript{*}Adjusted for age, energy-adjusted dietary fibre, body mass index, smoking, and use of nonsteroidal anti-inflammatory drugs and acetylsalicylic acid.
specifically, β-carotene) was attenuated (carotenenoids: RR 0.69, 95% CI 0.35 to 1.34; β-carotene: RR 0.60, 95% CI 0.30 to 1.22), but dietary retinal, excluding supplements and multivitamins, was still inversely associated with risk of DU (RR 0.50, 95% CI 0.26 to 0.93 for the highest quintile). Dietary sources of retinal include milk products, eggs, and organ meats.

**Vitamins C and E**
Overall, clinical study results show that concentrations of vitamin C are lower in people with gastric disorders than in those with normal endoscopic findings. A possible association between vitamin C and risk of DU was examined in the Health Professionals Follow-up Study, but no association was found. In the same study, vitamin E was inversely associated with risk of DU, but the association was attenuated when vitamin A was included in the analysis.

**Alcohol and coffee**
Alcohol and coffee (with or without caffeine) have been identified as risk factors for DU because they stimulate acid secretion, but findings have been inconsistent. A prospective study of 7624 Hawaiian men of Japanese descent, born between 1900 and 1919, showed that neither total alcohol nor consumption of particular kinds of alcohol were associated with risk of DU. Among health professionals, none of whom were heavy drinkers, alcohol consumption of more than 30 g/d (one drink contains roughly 15 g) had little effect on risk of DU. Moderate alcohol consumption might not affect risk of ulcer; alcohol abuse might cause gastritis and other disorders.

Little association was observed between caffeine, caffeine-containing beverages (coffee, tea), and decaffeinated coffee and risk of DU among health professionals.

**Other lifestyle factors**
**Smoking.** Smoking might lead to greater risk of gastric ulcer than DU. The very high risk reported in an earlier study could be explained by failure to adjust for confounding variables. In the Health Professionals Follow-up Study, current smoking was not significantly associated with higher risk of DU (RR 1.24; 95% CI 0.71 to 2.16), but smoking might delay healing of an ulcer, and quitting smoking might help prevent recurrence of ulcers.

**Physical activity and body mass index.** Findings about the influence of physical activity on risk of DU are inconsistent and range from no effect to a beneficial effect. A case-control study by Katschinski and colleagues showed that the proportion of patients with DU who had highly active jobs was 38%. The association between physical activity and DU persisted even when social class and smoking status were considered. No information was collected about physical activity outside work.

In a prospective study, Cheng et al found that both moderately active and active men had a 45% to 60% reduced risk of DU. Cross-sectional data from the British Regional Heart Study indicate that body mass index and the proportion of obese people tends to fall with increasing exercise, so the effect of body mass index on risk of DU would likely be similar to the effect of physical activity.
Conclusion

Dietary fibre might have a role in preventing formation or recurrence of DU. Fibre types (soluble or insoluble) and sources (fruit, vegetables, legumes, or cereal) affect risk of DU to varying degrees. Foods high in soluble fibre, such as oranges, carrots, and beans, seem to be more effective in reducing risk of DU. Data on the effect of diet and lifestyle on risk of DU are sparse, particularly regarding women, and more research is needed. Vitamin A might also have a protective effect against DU. Other dietary and lifestyle factors have a limited effect on risk of DU, but quitting smoking might help with ulcer healing and preventing ulcer recurrence. Dietary advice for patients with DU should emphasize increased fibre and adequate consumption of, for example, green leafy vegetables. Patients who smoke should quit.

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Competing interests
None declared

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References