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Do Chili Peppers Cause Ulcers? - A Burning Question

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Damage to the epithelial lining of the stomach can be caused by a number of etiologic factors including *Helicobacter pylori* infection, mucosal ischemia, stress, overproduction of histamine, pepsin, gastrin, etc. which ultimately lead to hypersecretion of acid (Lynn and Brooks, 1991). It is also clear that ulcers can be caused by excessive consumption of alcohol or aspirin (Marotta and Floch, 1991). Thus, it is not surprising that the ingestion of chili peppers and other spices has been suspected of contributing to ulcer disease for many years, because people assume that the burning sensation they cause in the mouth is accompanied by a comparable effect on the stomach. Although studies on the relationship between diet and ulcers within cultures that consume considerable amounts of chili (e.g. Indian or Thai) are limited, it has been suggested for example that ulcers are less prevalent in Northern Nigeria than in Southern regions of that country because of the greater use of red pepper in the diet of the latter (Solanke, 1973). The purpose of this paper is to critically evaluate some of the current literature pertaining to this question.

The active principle in red chili peppers, paprika and some other hot spices which imparts the pungent flavor is capsaicin (8-methyl-N-vanillyl-6-noneamide). This noxious substance is capable of stimulating a wide variety of primary afferent neurons, and a great deal is now known about the receptors and ionic channels that are activated by capsaicin (Maggi, 1991). In cutaneous nerves, for example, capsaicin is a powerful irritant which depolarizes pain fibers and elicits an axon reflex due to the influx of Na<sup>+</sup> and Ca<sup>++</sup> ions. It is of interest that although the stomach is richly innervated by capsaicin-sensitive afferent nerves, direct application of capsaicin to the stomach does not generally produce a conscious perception of gastric pain. A number of studies have examined the effects of capsaicin on the gastric mucosa in humans and these are reviewed below.

As early as 1951, Sanchez-Palomera investigated the effects of chili pepper on acid secretion in the stomach. In his study, as well as in all of the ones to be discussed subsequently, the patients were intubated with a nasogastric tube to permit the direct instillation of the agent into the stomach. This was necessary in order to avoid triggering a 'cephalic phase' of gastric acid secretion which would have likely occurred if the spice had been given by mouth. After analyzing samples of gastric fluid collected for two hours after intragastric administration of 1 gram red pepper, this study found no significant difference in the acidity of the secretions compared to samples from control patients in which only distilled water was administered into the stomach. Solanke (1973) injected pH balanced suspensions of 4 grams of red pepper in water into the stomachs of patients with ulcers. In this study, the red pepper elicited a statistically significant increase in gastric acid output compared to distilled water. The difference between the two results may be attributable to the greater quantity of pepper employed in the latter study as well as the use of ulcer patients rather than normal subjects. It is relevant to point out that 3 grams of red chili powder is the approximate amount consumed daily in India, whereas this represents a vastly greater amount than would be found in even a 'spicy' American diet and somewhat lower than that consumed daily in Western regions of Nigeria. It is also likely that the actual content of capsaicin per gram of chili powder (approximately 0.14%) may vary from one type of chili pepper to the next.

After the instillation of 1.5 grams of red pepper into the stomachs of healthy volunteers which had fasted overnight, Myers et al. (1987) reported a small but significant increase in gastric acid and pepsin secretion, and also an increase in the DNA content of the gastric washes compared to controls. The increase in DNA was believed to reflect mucosal exfoliation of cells. None of the parameters were significantly different from those obtained from patients that had been given 625 mg of aspirin. After intragastric instillation of 3 grams of red pepper in normal subjects with varying histories of chili consumption, Viranuvatti et al. (1972) used fiberoptic gastroscopy to assess the reaction of the gastric mucosa to the pepper. 13 of the 20 subjects showed no detectable change during the 15 minutes of observation, 3 showed mild edema and/or hyperemia, 3 displayed moderate reactions of mucosal edema and microbleeding, and one subject exhibited overt hemorrhage. There was no correlation between the prior history of chili consumption and the mucosal reaction observed. Apart from the question of whether or not chili peppers contribute to the pathogenesis of ulcers, other studies have examined the effect of chili ingestion on the healing of ulcers. Schneider et al. (1956) observed that the ingestion of 0.5 grams of chili pepper with each meal did not appreciably alter the time course of healing of duodenal ulcer nor did it increase the symptomatology of "heartburn" although it produced marked hyperemia of the gastric mucosa when administered directly without food. Similarly, Kumar et al. (1984) reported that the rate of duodenal ulcer healing was no different (as assessed by endoscopy) in 25 patients who consumed 3 grams of chili pepper per day from that of 25 ulcer patients who maintained a normal bland diet.

Based on the literature discussed above, the following conclusions may be drawn. Large doses of chili pepper instilled directly into the stomach of a normal fasting subject generally produce a relatively mild response consisting of a minor increase in gastric acid secretion and hyperemia. Although some individual variation may exist from one patient to the next, the degree of the response in general suggests that it is unlikely for consumption of chili peppers in the diet to cause ulcers per se. Since chili peppers are almost never consumed alone on an empty stomach, it is quite possible that the results described above represent an exaggerated response to a somewhat artificial condition. However, one cannot entirely rule out the possibility that chronic consumption of large amounts of peppers may exacerbate the development of ulcers in patients who are already predisposed to the disease due to other factors such as gastritis, alcohol consumption, etc. On the other hand, some investigators have gone so far as to suggest that chronic treatment with capsaicin may actually produce a cytoprotective response of the gastric mucosa to inhibit ulcer formation (reviewed by Maggi, 1990). This observation has been obtained in several experimental studies, and awaits substantiation in a more clinically relevant model.

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