Milling of wheat, maize and rice: Effects on fibre and lipid content and health

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Milling has different effects on the bran and germ. In the case of wheat the two come apart separately. They can be separated by sieving and are stable for a period of time without further treatment. In the case of maize and rice the bran and germ come away together and the resultant bruising releases the lipases which interact with the oil content leading, if left untreated, to early rancidity of the combined germ and bran. Thus wholemeal wheat flour has a stable shelf life for a variable period of time, but the only satisfactory way to eat whole maize is either on the cob or home-pounded and cooked on the same day. Rice can only be eaten in the unrefined state as brown or unmilled rice. Milled rice undergoes changes during storage. During the milling of rice some of the lipase present in the bran enters the endosperm and as the rice is stored it reacts with a small amount of oil present in the rice grain. Some say that this results in an improvement in taste. The resulting lipolysis results in the formation of free fatty acids followed by a process of peroxidation that produces ketoaldehydes.

Experiments on animal peptic ulcer models have shown that this results in an improvement in taste. These were not present in the refined staple foods in areas with low prevalence of duodenal ulceration. However, evidence is increasing to suggest that it rapidly becomes ulcerogenic. Similar experiments have shown that freshly milled rice bran is protective, but that it rapidly becomes ulcerogenic. Thus milled rice is not only deprived of gastroprotective lipids but also, on storage, becomes ulcerogenic, which is a possible factor in the high prevalence of duodenal ulceration in milled rice-eating countries.

With the discovery of Helicobacter pylori there has been much emphasis on its being the prime cause of duodenal ulceration. However, evidence is increasing to suggest that it may be a secondary infection affecting chronicity. Moreover, it should be remembered that many other factors have been shown to be associated with duodenal ulceration. These include familial tendency, acute anxiety as in the Second World War, cigarette smoking and the introduction of roller milling. Of these factors, the latter two greatly increased at the beginning of the twentieth century, which is the time when the epidemic of duodenal ulceration began. A suggestive feature about smoking is that it results in an increase in the parietal cell mass and therefore in an increase in the maximal ability of the stomach to secrete acid, which itself is so strongly associated with duodenal ulceration. Which of these factors are truly aetiological and which are confounding factors that happened to be increasing at the same time remain unknown. It is important to keep an open mind.

The results of experiments on animal peptic ulcer models, however, strongly support the possibility that the loss of certain protective lipids, resulting from the milling of staple carbohydrate foods, may be an important factor. More needs to be known about the nature and action of these lipids.

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