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• EDITORIAL •

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

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During the last thirty years the main interest in the medical consequences of milling of staple carbohydrate foods, particularly wheat and maize, has been in its effect on the fibre content as a result of the milling. The late nineteenth and early twentieth centuries in the West saw great changes in milling processes, from stone milling using water or wind power, to increasingly sophisticated roller milling, with an increasing loss of fibre in the process. In the 1970s and onwards there was an enhanced interest in possible diseases which could be related to the loss of fibre in the diet. At one time the list included diverticulitis, appendicitis, varicose veins, deep vein thrombosis, carcinoma of the colon, Crohn' s disease, ulcerative colitis, irritable bowel syndrome, peptic ulcer, gall stones, hiatus hernia and gastro-oesophageal reflux, disorders of lipid metabolism and coronary heart disease! Over the course of time medical evidence has narrowed this list down to a much smaller number, of which the most important are diverticular disease and carcinoma of the colon.

The effect of fibre on peptic ulcer disease was attributed to its buffering effect on acid secretion. There seemed to be a relationship between the fibre content of staple diet and the geographical prevalence of duodenal ulceration. The prevalence was lower in populations using unrefined wheat, millets or maize with a high fibre content and higher in populations using refined wheat or maize flour or milled rice with low fibre contents[1-4]. There were, however, abnormalities which did not fit in with this pattern, such as the high prevalence of duodenal ulcer in the Highlands of Ethiopia, where the staple food was unrefined teff (Eragrastosis abyssinica) with a high fibre content[4]. A possible explanation of this abnormality may lie in the lipid content of teff (see next paragraph). However, in addition, acid secretion studies showed that, whilst fibre had an initial buffering effect on gastric acidity, the resulting antral stimulation led to a higher acid output[5].

A further effect of milling was on the lipid content of staple carbohydrate foods. Experiments on animal peptic ulcer models showed that the lipids present in the unrefined staple foods in areas with low prevalence of duodenal ulcer had a gastroprotective effect against ulceration and also promoted ulcer healing. These were not present in the refined staple foods of the areas with high duodenal ulcer prevalence[6-9].

Lipids are found in both the bran and the germ of staple carbohydrate foods. In wheat and rice more lipids are found in the bran, but in maize the bulk lies in the predominantly large germ. Lipases are also present principally in the germ.

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 the latter are ulcerogenic. Similar experiments have shown that freshly milled rice bran is protective, but that it rapidly becomes ulcerogenic[10,11]. 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[12,13]. 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[14], which itself is so strongly associated with duodenal ulceration[15]. 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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