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Short Communication

Storage releases physiologically active content in milled maize and wheat

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Maize meal and wheat flour were stored at room temperature and humidity and separately sealed at -20 degrees Celsius. Using high resolution gas chromatography breakdown of triglycerides was measured over 12 months. Non - esterified fatty acids increased rapidly; at a higher rate when at room temperature and humidity than in cold dry conditions and to a greater degree in maize than in wheat, reaching levels sufficient to cause interference with normal physiological activity in the stomach. Maize meal underwent significant hydrolysis. The hydrolysis in maize meal was significant without oxidative loss. The content as well as the deficiencies of mono-cereal wheat or maize diets may be hazardous.

Key words: Wheat flour, maize meal, triglyceride, linoleic acid, prostaglandin E2.

INTRODUCTION

A diet heavily dependent on a single cereal may be deficient in vitamins and trace elements. The fat in milled cereal products degenerates over time (Clayton and Morrison, 1972); the additional health risks associated with this are poorly understood but may be significant for those dependent on one cereal.

There are two processes of degeneration: hydrolysis and oxidation. Hydrolysis is the process by which trigly-cerides are broken down to release non-esterified fatty acids. Though hydrolysis does not reduce the palatability of the flour, further oxidative breakdown of fatty acids is associated with bad taste and smell (Sauer, 1992).

Hydrolytic degeneration of wheat has been extensively studied (Greer et al., 1954). Little has been published about degeneration of maize (Sammon, 1999), with no description of progressive change. Hydrolysis of fats in milled maize has been put forward as a possible causative factor for oesophageal cancer in maize growing areas (Sammon, 2009).

This study compares the pattern and extent of hydrolysis of fat in milled wheat and maize under different storage conditions.

Fine maize meal and fine wheat flour were obtained from commercial millers in England, within one month of milling. Data on date of harvest was not available. Total lipids were extracted using the method of Folch et al. (1957). Free fatty acids were subsequently extracted into 0.02 N K_2CO_3, acidified with H_2SO_4 and then re-extracted into petroleum ether as described by Sukhija and Palmquist (1988). Methyl esters were prepared using diazomethane and fatty acid composition was analysed by high resolution gas chromatography. Quantification was achieved by the addition of an internal standard, heneicosanoic acid (C21:0, Sigma, UK) prior to extrac-tion.

Following this initial analysis, each sample was divided. One half of each was stored sealed at -20^{o}C, the other half at room temperature (average 21.7^{o}C), loosely covered and subject to ambient humidity (average 60.2% relative humidity) . Fatty acid extraction and analysis were carried out every month to twelve months from milling.

On initial analysis (see Figure 1), 11.3% of the fatty acid in the maize meal and 3.5% of the fatty acid in the wheat flour were present in non-esterified form.

There was progressive breakdown of fatty acids from esterified to non-esterified. The rate of hydrolysis was slow in the first three months, then progressively increased at room temperature and under conditions of air-tight cold storage.

Fatty acid profiles are shown in Table 1. Fatty acids were hydrolysed at different rates: levels of non-esterified C14:0 and C16:1 rose quickly in both flours; C18:0 and C18:1 increased more slowly in both. After twelve months

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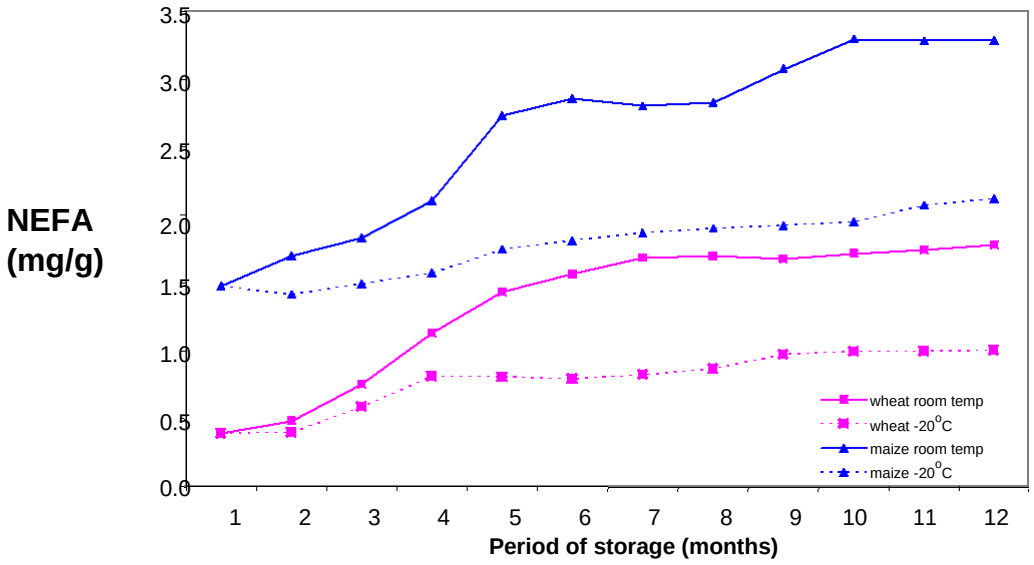

Figure 1. Release of non-esterified fatty acids in stored maize meal and wheat flour.

Table 1. Total fatty acid (FA) and non-esterified fatty acid (NEFA) composition of maize and wheat flours stored under different conditions (mg fatty acid/g flour).

	Initial analysis		NEFA at Room Temperature		NEFA at -20^{0}C	
	Total FA	**NEFA**	**3 months**	**12 months**	**3 months**	**12 months**
Maize meal						
C14:0	0.009	0.002	0.002	0.008	0.002	0.006
C16:0	1.536	0.238	0.286	0.401	0.264	0.336
C16:1	0.023	0.003	0.005	0.009	0.004	0.005
C18:0	0.282	0.020	0.020	0.048	0.019	0.028
C18:1	3.515	0.275	0.319	0.750	0.248	0.365
C18:2n-6	7.145	0.870	1.070	1.913	0.855	1.229
C18:3n-3	0.280	0.049	0.056	0.102	0.054	0.07
Total	13.041[§]	1.476	1.831	3.287	1.493	2.118
Wheat flour						
C14:0	0.017	0.001	0.002	0.008	0.003	0.007
C16:0	1.917	0.089	0.112	0.443	0.110	0.161
C16:1	0.020	0.001	0.002	0.004	0.002	0.003
C18:0	0.091	0.002	0.004	0.013	0.004	0.004
C18:1	1.196	0.023	0.194	0.198	0.043	0.099
C18:2n-6	7.044	0.236	0.471	0.999	0.353	0.588
C18:3n-3	0.568	0.018	0.046	0.071	0.039	0.084
Total	11.104[§]	0.391	0.758	1.778	0.587	1.003

[§] Total fatty acid after 12 months storage was 13.116 and 10.748 mg/g flour for maize and wheat respectively, when stored at -20^{0}C; 12.976 and 9.897 mg/g flour when stored at room temperature.

storage at room temperature and relative humidity there was 191 mg non-esterified linoleic acid per 100 g maize flour and 100 mg non-esterified linoleic acid per 100 g wheat flour.

The expected pattern of progressive hydrolysis occurred in maize and wheat flour stored at room temperature. However hydrolysis was also present at low temperature and under airtight conditions. Non-esterified fatty acid release was greater and evident earlier in maize than in wheat.

Maize meal stored for 12 months at room temperature and humidity had 25.2% of its fatty acid in non-esterified

form, which was a marked increase from the 1% normally found in whole maize at maturation.

Dietary maize products are associated with significant physiological effects: Schepp et al. (1988) showed that a high linoleic acid diet, based on corn oil, caused increased intra-gastric prostaglandin E2 (PGE2) production and suppressed intra-gastric acid secretion in rats. The influence of linoleic acid in the triglyceride form as found in corn oil and in whole maize, appears to be slow. Non-esterified linoleic acid has a more immediate effect and is effective in much smaller quantities. Nakaya et al. (2001) showed that labelled non-esterified linoleic acid added to rat gastric mucosa cells was converted to arachidonic acid and PGE2 concentration increased in a time and dose-dependent manner.

191 mg of non-esterified linoleic acid is available per 100 g of maize meal, and 100 mg of non-esterified linoleic acid per 100 g of wheat flour, providing the precursor for a physiologically significant amount of arachidonic acid and consequently PGE2.

Significant health effects are most likely to be found where maize meal or wheat flour is the dietary form predominantly used. Ingestion of milled maize products is associated with heartburn and regurgitation in Transkei, South Africa (Sammon, 1994).

Squamous cancer of the oesophagus occurs worldwide in endemic numbers only in communities which depend on either maize or wheat. Within a community almost entirely dependent on maize, oesophageal cancer is strongly associated with the regular consumption of maize in the meal form (van Rensburg, 1985). The physiological changes associated with consumption of degenerating maize meal may be a factor in the aetiology of this disease.

There was little loss of total fatty acid (signifying oxidative degeneration) in either maize or wheat under cold conditions. In the flours stored at room temperature a loss of 11% of total fatty acid from the wheat flour was recorded, but only 0.5% loss of total fatty acid from the maize meal. The ability of maize meal to suffer a high percentage hydrolysis (25.7%) without significant oxidative change is of concern, since physiologically significant hydrolysis may occur in maize meal without the warning that a rancid taste provides.

REFERENCES

Clayton TA, Morrison WR (1972). Changes in flour lipids during the storage of wheat flour. J. Sci. Food. Agric. 23: 721-736

Folch J, Lees M, Sloane-Stanley GH (1957). A simple method for the isolation and purification of total lipids from animal tissues. J Biol. Chem. 226: 497-509

Greer EN, Jones CR, Moran T (1954). The quality of flour stored for periods up to 27 years. Cereal Chem. 31(6): 439-450

Nakaya A, Wakabayashi H, Imamura L, Fukuta K, Makimoto S, Naganuma K, Orihara T, Minemura M, Shimizu Y, Nagasawa T, Hamazaki T, Watanabe A (2001). Helicobacter pylori alters n-6 fatty acid metabolism and prostaglandin E2 synthesis in rat mucosal cells. J. Gastroenterol Hepatol. 16(11): 1197-2005

Sammon AM. 1994. Heartburn in Transkei - Letter to the editor. Dis Esophagus. 7: 220

Sammon AM (1999). Maize meal, non-esterified linoleic acid, and endemic cancer of the esophagus – preliminary findings. Prostaglandins and other lipid mediators. 57(2-3): 167-71

Sammon AM (2009). Squamous cancer of the oesophagus in Africa. Published at scoafrica.org Sammon. England.

Sauer DB (1992). Storage of Cereal Grains and Their Products. Fourth Edition. American Association of Cereal Chemists. St. Paul, Minnesota, USA.

Schepp W, Steffen B, Ruoff HJ, Schusdzairra V, Classen M (1988). Modulation of rat gastric mucosal prostaglandin E2 release by dietary linoleic acid: effects on gastric acid secretion and stress-induced mucosal damage. Gastroenterol. 95(1): 18-25.

Sukhija PS, Palmquist DL (1988). Rapid method for determination of total fatty acid content and composition of feedstuffs and feces. J Agric Food Chem. 36: 1202-1206.

van Rensburg SJ, Bradshaw ES, Bradshaw D, Rose EF (1985). Oesophageal cancer in Zulu men, South Africa: A case-control study. Br. J. Cancer. 51: 399-405.