Zeitschrift für Ernährungswissenschaft

Journal of Nutritional Sciences · Journal des Sciences de la Nutrition

Band 18 Heft 2 Juni 1979

Z. Ernährungswiss. **18**, 77–80 (1979)

ISSN 0044-264 X

Wolfson Gastrointestinal Laboratory, Gastrointestinal Unit, Western General Hospital, Edinburgh[1])
Department of Surgery, Centrallasarettet, Ostersund, Sweden[2])

The effect of dietary psyllium hydrocolloid and lignin on bile

W. G. Brydon[1]), S. Borup-Christensen[2]), W. Van der Linden[2]), and *M. A. Eastwood[1])*

With 2 tables

(Received August 10, 1978)

The ratio of the concentration of bile acids, particularly chenodeoxycholic acid to cholesterol, appears to be relevant to the appearance and disappearance of cholesterol gallstones (1, 2). Several papers (3, 4, 5) have had encouraging success with psyllium seed hydrocolloid and lignin in altering the amount of chenodeoxycholic acid and deoxycholic in the body pool or concentration in bile. *Heaton* and his colleagues have shown that bran (6) decreased the proportion of deoxycholic acid in human bile with a relative increase in the proportion of chenodeoxycholic acid. They were unable to demonstrate any such effect of lignin on biliary bile acids (7).

In this study the effects of psyllium hydrocolloid and lignin on bile composition have been investigated.

Material and methods

Two groups of Swedish patients with uncomplicated cholesterol gallstone disease were studied; one group of 10 and a second group of 11. Three quarters of both groups were female, and the age distribution was 30 to 71 years (median age 53 years). Both groups were taking their habitual diet before and during treatment with either lignin 2 g t.i.d. or psyllium hydrocolloid 10 g t.i.d. The lignin was Indulin A.T., a gift from Westvaco, Charleston, South Carolina, U.S.A.; the psyllium hydrocolloid (metamucil) was obtained from G. D. Searle & Co. Fasting samples of bile were obtained before treatment following intubation. Duodenal bile was sampled without stimulation of the gallbladder. After three weeks of treatment hepatic bile

Table 1.

No.	Storage time at − 20 ° (months)	Phospholipid P mm/l Extracted on sampling	Extracted after storage	Inorganic P mm/l Extracted on sampling	Extracted after storage
1	2	0.30	0.27	0.23	0.26
2	3	2.80	2.60	1.44	1.44
3	2	10.70	9.40	1.03	1.16
4	3	8.20	6.90	0.60	1.04
5	1	1.70	1.70	2.40	3.10
6	2	0.97	0.97	0.67	0.73
7	3	5.50	5.30	1.03	1.11

Table 2.

	Habitual diet (n = 11)		+ Psyllium hydrocolloid (n = 11)		Habitual diet (n = 10)		+ Lignin (n = 10)	
	mean	range	mean	range	mean	range	mean	range
Cholesterol mmols/l	1.4	0.1 − 2.7	4.1	0.6 − 10.5	1.4	0.3 − 4.1	7.9	3.0 − 12.8
Total bile acid mm/l	10.8	1.2 − 19.6	30.3	3.0 − 51	8.7	1.4 − 26.7	45.9	19.9 − 92.4
Lithocholic acid (% of total)	1.2	0 − 4	2.0	0.6 − 5.0	0.8	0 − 2.2	1.3	0 − 3.6
Chenodeoxycholic acid (% of total)	40	28 − 56	38	23 − 49	36	18 − 55	37	26 − 50
Deoxycholic acid (% of total)	28	4 − 53	32	4 − 54	27	3 − 50	26	8 − 42
Cholic acid (% of total)	30	16 − 46	28	13 − 57	37	23 − 79	35	21 − 51
Total bile acid/cholesterol	10.4	4.0 − 16.7	7.9	3.0 − 15.1	6.1	3.5 − 9.3	5.8	3.3 − 8.3

was sampled from the common bile duct through the cystic duct. The samples of bile so obtained were frozen on aspiration and stored at −20 °C before being packed in solid carbon dioxide in an insulated box and transported by air. The total sample was extracted twice with 10 volumes of isopropanol at 60° for 30 minutes and the extracts pooled.

The bile acid conjugates were hydrolysed and measured as methyl ketone derivatives using a modification (8) of the method of *Evrard* and *Janssen* (9) on a Pye Series 104 Gas Chromatograph; 2, 2 dimethoxy propane being used as methylating agent.

Cholesterol was measured as free cholesterol using stigmasterol as internal standard, with similar gas chromatographic conditions to those used for the separation of bile acid methyl ketones.

Phospholipid was measured using the method of *Meun* and *Smith* (10). Inorganic phophorus was measured using the method of *Kuttner* and *Lichtenstein* (11).

The effect of storage on phospholipid concentration in duodenal bile was investigated, using bile aspirated during a Lund Test Meal. Aliquots were either stored frozen at − 20° or diluted in isopropanol prior to storage at −20°. Storage time varied from 2–4 months.

Results

The effect of storage on biliary phospholipid can be seen in table 1. The mean fall in phospholipid concentration was 7 %. This fall is much less than reported by *Murison* (12) who found losses of as much as 50 % in 4 weeks. The inorganic phosphorus increased in some of these samples. In some of the more dilute biles, the phospholipid concentration expressed as a molar percentage fraction of the total bile acids plus cholesterol plus phospholipid was very low, i.e. 2–10%. In these samples the inorganic phosphorus levels in the bile were in general higher (2–6 mmols/l) than in others (0.1–1.5 mmols/l). It was felt that there had been a loss of phospholipid phosphorus in some samples and so it was thought to be inappropriate to report phospholipid concentrations or use triangular coordinate analysis in this study.

Biliary bile acid and cholesterol concentrations on the different dietary regimes are shown in table 2. The increased concentration of cholesterol, total bile acids and individual bile acids after treatment reflect the sampling site. Paired data were analysed using Wilcoxon's matched pairs signed rank test. There is no effect of either psyllium hydrocolloid or lignin on the lithocholic, chenodeoxycholic, deoxycholic or cholic acid content of the bile. The ratio of bile acid to cholesterol is uninfluenced by treatment.

Discussion

Our results for phospholipids underlines the warning by *Murison* and his colleagues (12). The loss of phospholipid phosphorus in the more dilute biles may be caused by biliary alkaline phosphatases in the duodenum. To an extent the large losses observed by *Murison* (12) can be overcome by using isopropanol rather than the Folch extraction method. The Folch extraction method fails to extract the more polar lysolecithin, which is a decomposition product which results from storage.

Previous authors have reported alterations in bile acid patterns by dietary psyllium hydrocolloid and lignin in animals and humans (3, 4, 5). Such effects have not been demonstrated in the group studied here. This is in accord in part with the results of *Heaton* (7) for lignin.

The reasons may be methodological: we used gas liquid chromatography whereas the other authors used thin layer chromatography to separate the two bile acids chenodeoxycholic and deoxycholic acid. Clear separation of these two is important and may be easier using gas liquid chromatography. There may be species differences with different biliary lipid metabolism in man, the rat and the hamster. In the animal experiments the lignin and psyllium seed husk were given as 4–5% of the diet. It is unlikely that 6 g of lignin or 30 g of ispaghula reach that proportion of the diets in our subjects. However, *Beher* (4) obtained effects on the deoxycholic acid pool with 12 g/day.

Whereas other authors have been able to modify the biliary bile acid composition using lignin and psyllium seed husk, we have been unable to achieve such change.

Summary

Animal experiments suggest that supplementing the diet with either psyllium seed husk or lignin alters the ratio of deoxycholic acid to chenodeoxycholic in bile. In this study dosages of psyllium seed husk or lignin acceptable to patients with gallstones do not appear to alter the relative amounts of cholesterol, or individual bile acids in the bile.

Zusammenfassung

Die Ergebnisse von Experimenten mit Tieren führen zu der Feststellung, daß ein Fütterungszusatz von entweder Psyllium-Samenkernen oder Lignin das Verhältnis von Desoxycholsäure zu Chenodesoxycholsäure in der Galle ändert. In dieser Untersuchung scheint bei Patienten mit Gallensteinen eine akzeptable Dosis von Psyllium-Samenkernen oder Lignin die relativen Mengen von in der Galle befindlichem Cholesterin oder den einzeln verschiedenen Gallensäuren nicht zu ändern.

References

1. *Admirand, W. H., D. M. Small,* J. Clin. Invest. **47,** 1043 (1968). – 2. *Danzinger, R. K., A. F. Hofmann, L. J. Schoenfield, J. L. Thistle.* New Engl. J. Med. **286,** 1(1972). – 3. *Beher, W. T., K. K. Casazza,* Proc. Soc. Exp. Biol. Med. **136,** 253 (1971). – 4. *Beher, W. T., B. M. Schuman, M. A. Black, G. J. Lin, K. K. Casazza,* Henry Ford Hosp. Med. J. **21,** 21 (1973). – 5. *Bergman, F., W. van der Linden,* Z. Ernährungswiss. **14,** 218 (1975). – 6. *Pomare, E. W., K. W. Heaton,* Brit. Med. J. 1973/**IV,** 262. – 7. *Heaton, K. W., S.T. Heaton, R. E. Barry,* Scand. J. Gastroenterology **6,** 281 (1971). – 8. *Mitchell, W. D., J. M. Findlay, R.J. Prescott, M. A. Eastwood, D. B. Horn,* Gut **14,** 348 (1973). – 9. *Evrard, E., G. Janssen,* J. Lipid Research **9,** 226 (1968). – 10. *Meun, D. H. C., K. C. Smith, Analytical Biochemistry* **26,** 364 (1968). – 11. *Kuttner, T., L. Lichtenstein,* J. Biol. Chem. **86,** 671 (1930). – 12. *Murison, J., D. Festi, P. E. Ross, I. A. D. Bouchier,* Clin. Chim. Acta **68,** 159 (1976).

Authors' address:

W. G. Brydon, Wolfson Gastrointestinal Laboratory, Gastrointestinal Unit, Western General Hospital, Edinburgh, EH 4 2 XU