Effects of the fibre components pectin, cellulose, and lignin on bile salt metabolism and biliary lipid composition in man

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SUMMARY   Randomised crossover studies in three separate groups of 10 healthy volunteers were undertaken to determine the effects of biliary lipid composition and bile salt metabolism of daily dietary supplementation for four weeks with the purified fibre components pectin (12 g/day), cellulose (15 g/day) and lignin (12 g/day). The subjects' biles were initially unsaturated with cholesterol and no significant changes in the lithogenic indices or mean percentages of cholesterol, phospholipid, or total bile acids after any of the supplements were observed. After pectin, the mean ($\pm$SD) percentage of cholic acid decreased significantly from 42·8 ($\pm$10·8) to 39·0 ($\pm$11·2), the mean ($\pm$SD) percentage of deoxycholic acid increased significantly from 18·2 ($\pm$13·7) to 25·4 ($\pm$13·5) and C^{14}-deoxycholate metabolites were raised significantly by 65%. After cellulose, the mean ($\pm$SD) percentage of chenodeoxycholic acid was increased significantly from 33·6 ($\pm$6·3) to 35·4 ($\pm$7·0), the mean ($\pm$SD) percentage of deoxycholic acid decreased significantly from 18·6 ($\pm$9·6) to 14·2 ($\pm$8·3) and C^{14}-deoxycholate metabolites halved. Lignin did not exert any significant effects. Though these results show that individual fibre components are associated with quite different effects on bile acid metabolism, in the short term no significant effect on biliary cholesterol saturation was observed in bile initially unsaturated with cholesterol. The bile acid changes most likely result from the different effects on colonic metabolism induced by the individual fibre components.

Gall stones have been included in the diseases of western civilisation and their incidence related to the intake of dietary fibre.[1][2] In subjects whose bile is initially supersaturated with cholesterol, feeding wheat bran results in a significant decrease in biliary cholesterol saturation.[3][4] The addition of bran to the diet of normal subjects, however, does not appear to alter biliary lipid composition[5-7] and the biliary lipids in vegetarians do not differ significantly from controls taking a diet relatively low in fibre.[8] Dietary fibre also appears to influence bile acid composition. Biliary deoxycholic acid (DCA) can be lowered and chenodeoxycholic acid (CDCA) increased by dietary bran supplementation[3][6] and lactulose,[9] and vegetarians have a smaller DCA pool.[10] Changes in the composition of the bile salt pool may in turn influence biliary cholesterol

saturation. Increasing the proportion of CDCA or ursodeoxycholic acid (UDCA) in the bile acid pool is associated with cholesterol desaturation of bile and gall stone dissolution[11][12] whilst DCA may have the opposite effect.[13] It has been suggested that the excessive production (or absorption) of the colonic bile acid metabolite DCA may be involved in the pathogenesis of cholesterol gall stones.[13][14] There are populations in both the South Pacific[15] and Africa,[16] however, in whom dietary fibre intakes and biliary DCA are high, yet cholesterol gall stones are rare, suggesting mechanisms other than altered DCA concentrations are also important.

It is well known that fibre from different sources has different properties[17] which in turn may have important effects on bile acid metabolism. For instance, bile acids are adsorbed to fibre[17][19] and increased bile acid binding might then enhance the faecal excretion of bile acids.[20] The anaerobic breakdown of fibre by colonic bacteria (fermentation) is also likely to have important effects on bile

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acid metabolism as a result of changes in the bacterial cell mass,[21] the pH and oxidoreduction potential in the colonic lumen[9] [22] and colonic motility.[23]

Furthermore, the fermentation process will depend on the amount and type of substrate available and the time available for its fermentation to occur.[24] It is known that the fibre component lignin for instance is not fermented to any significant extent whereas pectin is almost completely fermented.[24] [25] Cellulose on the other hand, is fermented slowly, being less complete the more rapid is gut transit.[24] [26]

In order to further evaluate the influence of dietary fibre on bile acid metabolism and the lipid composition of bile in man, we have studied the effects of a highly fermentable fraction (pectin),[25] a partially fermentable fraction (cellulose),[26] and a non-fermentable fraction (lignin).[24] [27] Changes in biliary lipid composition and bile salt metabolism after four weeks' dietary supplementation with each of these purified fibre components were investigated.

Methods

SUBJECTS

Three groups of 10 subjects each were investigated over an eight week period. All subjects were healthy volunteers who gave their informed consent to participate in the study, which was approved by the Wellington Hospital ethical committee in April 1981. The pectin group comprised two male medical students and eight female postgraduate nurses (mean age ±SD 30·9±7·9 years). The cellulose group was made up of four male medical students, two female medical students and four female postgraduate nurses (mean age ±SD 23·9±1·9 years). The lignin group consisted of' two male medical students, two female medical students, five female postgraduate nurses' and one female technical officer (mean age' ±SD 28·3±7·8 years).

EXPERIMENTAL DESIGN

For each group, a crossover design consisting of two periods of four weeks was used. Each subject acted as his/her own control taking either pectin, cellulose or lignin for one period only (=test period) and consuming his/her normal diet during the other period (=control period). Subjects were paired according to sex and occupation, then randomly allocated to the groups and periods, so that the number of men/women receiving the supplement first was the same as the number entering the control period first and the number of students/nurses was similarly distributed.

DIETS

Subjects were instructed to maintain their normal diet throughout the study and not to take any medication. Detailed dietary records as recommended by Bingham and Cummings were kept over the eight weeks.[28] Food scales, measuring cylinders and flasks were distributed and every item of food or drink taken was weighed or measured and entered in a note book. The dietary records were supervised by a dietician and analysed to ensure that there was no discrepancy in food patterns between the control and the test periods except for the fibre supplement. Baseline dietary fibre intakes for the two periods were obtained using standard food tables.[29] [30] Subjects were weighed immediately before the study and at the end of both the control and test periods on the same scales in light indoor clothing.

Pure pharmaceutical citrus pectin with a high methoxyl content of 9·3% on a dried basis (Yahkin Canning Company Ltd, Tel Aviv) and 99·5% pure alpha cellulose fibre derived from wood (Sigma Chemical Company Missouri) were used for the study. Pure Aspen autohydrolysis lignin, which has a high number average molecular weight (1490) and methoxyl content (16·7%), was supplied by Professor Morris Wayman and Stake Technology Ltd, Ontario. Before the test period, each subject was given a bag containing 420 g of the supplement and instructed to take a total of 15 g per day (a measure of 5 g with each meal) for 28 days. The pectin, cellulose or lignin was to be mixed with the usual food or drink taken at meal times in any way acceptable to the subjects. Any supplement left over at the end of the test period was collected and weighed. If the supply was exhausted before the end of the test period, more was distributed. The amount of pectin, cellulose and lignin taken by each subject and the side effects have been described previously.[15] Subjects experienced great difficulty in taking the full amount of pectin because of its gelling properties, whilst lignin proved to be extremely unpalatable. Mean daily intakes of both these supplements was 12 g. Cellulose was well tolerated and the full amount of 15 g/day was taken.

BILIARY SAMPLING AND ANALYSIS

At the end of both the control and test periods sampling of fasting duodenal bile was aspirated through a mercury weighted, narrow bore polyethylene tube after stimulation of the gall bladder with 30–40 U of pancreozymin-cholecystokinin (Boots Co Ltd) administered intravenously. A 5 ml sample of concentrated bile was saved and immediately divided into aliquots of 0·1–0·2 ml and stored at −18°C. Biliary lipids (cholesterol, phospholipid and total bile acid) were estimated using methods

previously described[31] and the lithogenic index calculated according to Holzbach.[32] When samples were extremely dilute they were considered inadequate for accurate analysis and excluded from this calculation as described for the National Cooperative Gallstone Study.[11] This was the case for the test period samples of subjects 5 and 7 in the pectin study and for the control period sample of subject 7 in the cellulose study. All analyses were performed in duplicate.

Individual bile acids were measured by gas liquid chromatography after samples were hydrolysed with 2N NaOH for three hours at 130°C then methylated and acetylated.[33] The gas chromatograph (Varian 3700 series) with Flame Ionisation Detector was fitted with $6' \times 2$ mm glass columns containing 3·0% AN 600 on Gas Chrom Q Mesh 100/120. Oven temperature was 270°C, injector temperature 280°C and detector temperature 290°C. The carrier gas was nitrogen at a flow rate of 40 ml/minute. Recoveries for the individual bile acids were on average 98% (coefficient of variation 5·1%).

RADIOACTIVE BILE SALT METABOLITES
Approximately 48 hours before biliary sampling, subjects were injected intravenously with 2·5 μCi of sodium taurocholate $-24C^{14}$ (Radiochemical Centre, Amersham, England) in 20 ml of sterile normal saline. The time interval between injection and sampling was noted and did not differ significantly between the control and test periods. The bile samples were prepared by first deproteinising the sample by boiling with ethanol and then separating the bile acid conjugates by thin layer chromatography, using methods previously described.[34–36] The solvent system was amylacetate, propionic acid, propan-1-ol and distilled water in the ratio of 40:30:20:10 v/v and the standard marker solution comprised glycodeoxycholate, glycocholate, taurodeoxycholate and taurocholate (Sigma Chemical Company, Missouri). After drying the chromatography plates, adequate separation of the bile acid conjugates was checked by brief exposure to iodine

vapour. The plates were then marked out in 5 mm strips and scraped into counting vials containing a 2:1 mixture of a toluene based scintillation fluid and Triton X-100. Radioactivity was measured in a Philips PW4700 liquid scintillation counter and the counts corrected by using sodium glycocholate $-1^{14}C$ solution (Radiochemical Centre, Amersham, England) as an internal standard. The corrected counts per minute (cpm) for each bile salt fraction were expressed as a percentage of the total radioactivity of the sample. All samples were studied in duplicate. Results were analysed in terms of total C^{14}-metabolites of sodium taurocholate $-24C^{14}$ (=glycocholate and taurodeoxycholate and glycodeoxycholate) and C^{14}-deoxycholate conjugates. In this study, 2·5 μCi ^{14}C was used on each occasion, equivalent to approximately 0·1 mrads radiation exposure on the test day.

STATISTICS
The results for the test and control samples were compared using the Student's t test for paired data, or for non-parametric data, the Wilcoxon's test. The observed probability value (p) was taken as significant at the 0·05 level. Correlation coefficients were calculated and their statistical significance determined using a two tailed t test.

Results

DIETS
Dietary data is given in Table 1. Apart from the fibre supplement, there were no statistically significant differences between the control or test periods for either dietary intakes or body weight.

BILIARY LIPID COMPOSITION
As shown in Table 2, the mean percentages of biliary cholesterol, phospholipid and total bile acids were not altered significantly by pectin, cellulose or lignin supplementation. Likewise, no significant changes in the lithogenic indices were found after any of the supplements. Total biliary lipid concen-

Table 1   *Mean (±SD) daily dietary intakes for pectin, cellulose and lignin groups during control and test periods (excluding fibre supplement)*

	Pectin		Cellulose		Lignin	
	Control period	Test period	Control period	Test period	Control period	Test period
Total energy (MJ)	8·1± 2·0	8·1± 2·5	9·7± 4·2	9·0± 3·6	8·0± 1·8	7·6± 1·5
Protein (g)	69·8±17·1	71·1±24·9	78·2± 31·3	74·9± 23·1	68·6±15·3	63·2±12·3
Fat (g)	86·3±37·6	87·9±28·7	103·9± 39·6	94·4± 34·5	82·7±24·2	77·8±20·1
Carbohydrate (g)	207·6±50·7	216·1±68·9	254·8±127·1	255·1±116·3	219·4±54·1	211·0±53·3
Alcohol (g)	9·8± 7·6	6·9± 7·0	15·6± 18·1	5·1± 7·1	5·5± 6·0	9·7±16·6
Fibre (g)	18·3± 3·6	22·9± 8·3	21·7± 6·9	24·2± 15·8	21·6± 5·1	20·3± 5·6

Table 2   *Mean (±SD) percentage of biliary cholesterol phospholipid and total bile acids and lithogenic indices for control and test samples of pectin, cellulose and lignin studies.*

Biliary lipid composition	Pectin		Cellulose		Lignin	
	Control period	Test period	Control period	Test period	Control period	Test period
Cholesterol	6·8	6·4	6·1	5·3	5·3	5·4
(%)	±1·4	±2·3	±2·2	±1·6	±1·2	±1·9
Phospholipid	24·7	27·6	25·9	22·9	23·4	22·5
(%)	±4·8	±7·5	±5·6	±4·5	±5·3	±3·9
Total bile acids	68·5	66·0	68·0	71·7	71·3	72·1
(%)	±5·3	±8·1	±7·3	±5·5	±5·3	±4·9
Lithogenic index	1·00	0·89	0·82	0·80	0·81	0·80
(Holzbach)	±0·18	±0·22	±0·19	±0·23	±0·21	±0·25

trations did not vary significantly between experimental periods.

BILIARY BILE ACID COMPOSITION
The effects of pectin, cellulose and lignin on the percentages of individual bile acids are shown in Table 3. Pectin was associated with significant decreases in CA and total primary bile acids whilst DCA and the total secondary bile acids were significantly increased.

Cellulose on the other hand, was associated with a significant increase in CDCA whilst DCA, LCA and the total secondary bile acids were significantly decreased.

In the lignin group, no significant changes were seen in the percentages of individual bile acids after supplementation.

BILIARY LIPID AND BILE ACID CORRELATIONS
Correlation coefficients were calculated between the

lithogenic indices and the proportion of CA, CDCA and DCA in the bile. No significant correlations were found.

Correlation coefficients between the main individual bile acids were also calculated and are given in Table 4. A significant negative correlation was found between biliary CA and DCA in all groups. Although a negative correlation was also seen between CDCA and DCA, this was not as marked and only one correlation was statistically significant.

TAUROCHOLATE − 24C^{14} METABOLITES
These results are shown in Table 5. After pectin, C^{14}-deoxycholate metabolites of taurocholate −24C^{14} were significantly increased ($p < 0.05$) whilst no significant changes in the total C^{14}-metabolites were observed. In the cellulose group, there were no significant changes though overall C^{14}-deoxycholate metabolites were halved. Lignin did not alter any of the metabolites.

Table 3   *Mean (±SD) percentages of biliary CA, CDCA, DCA, LCA, UDCA, primary bile acids and secondary bile acids for control and test samples of pectin, cellulose and lignin groups*

Biliary bile acid composition (%)	Pectin		Cellulose		Lignin	
	Control period	Test period	Control period	Test period	Control period	Test period
CA	42·8	39·0*	43·8	48·2	47·1	49·0
	±10·8	±11·2	±11·1	±8·3	±9·9	±8·6
CDCA	35·3	32·4	33·6	35·4*	36·3	35·4
	±8·7	±9·8	±6·3	±7·0	±7·6	±6·5
DCA	18·2	25·4*	18·6	14·2*	13·7	12·6
	±13·7	±13·5	±9·6	±8·3	±12·9	±8·3
LCA	1·2	1·6	1·6	0·9*	1·5	1·2
	±0·6	±1·2	±0·6	±0·6	±1·0	±0·8
UDCA	1·8	1·6	2·4	1·4	1·4	1·8
	±2·4	±2·8	±2·9	±1·5	±1·1	±2·0
Primary	78·1	71·4*	77·4	85·5	83·5	84·3
	±13·6	±14·4	±11·5	±9·4	±13·0	±9·5
Secondary	20·1	27·0*	20·2	15·1*	15·2	13·8
	±14·0	±13·7	±10·0	±8·6	±13·0	±8·3

* Significant differences between control and test samples at the 0·05% level.

Table 4 *Correlation coefficients between the percentages of biliary bile acids, CA and DCA: between CDCA and DCA: and between CA and CDCA for the pectin, cellulose and lignin groups*

	CA vs DCA		CDCA vs DCA		CA vs CDCA	
	Control period	Test period	Control period	Test period	Control period	Test period
Pectin	−0·73*	−0·73*	−0·63	−0·62	−0·04	−0·06
Cellulose	−0·75*	−0·64*	−0·45	−0·57	−0·22	−0·26
Lignin	−0·77†	−0·68*	−0·70*	−0·50	0·09	−0·24

* $p<0.05$. † $p<0.01$.

Discussion

The results of the present study in healthy volunteers with unsaturated bile show that dietary supplementation for a month with certain purified fibre components is associated with significant effects on bile acid composition and metabolism of the bile salt pool whilst biliary lipid composition is not altered. Pectin lowered the percentages of primary bile acids CA and CDCA whilst increasing the secondary bile acid DCA, whereas cellulose exerted the opposite effect, and lignin appeared to have no effect. C^{14}-deoxycholate metabolites of taurocholate $24C^{14}$- were more than doubled after pectin, halved following cellulose and unaltered by lignin. It is accepted, however, that the effects of the fibre supplements, especially on biliary lipids, may not have been fully apparent in just four weeks and that bile which is unsaturated with cholesterol is unlikely to become even less saturated after fibre ingestion.[6] Generalisations about the highly purified components used in this study should be treated with caution because the properties of fibre components vary according to their source and method of preparation.[17]

Pectin is a gel forming component of fibre which is almost completely metabolised in the human gut,

Table 5 *Men (±SD) percentage biliary total C^{14}-metabolites and C^{14}-deoxycholate metabolites 48 hours after administration of taurocholate −24C^{14}*

	Total C^{14}-metabolites (%)		C^{14}-deoxycholate metabolites	
	Control period	Test period	Control period	Test period
Pectin	42·4	52·2	16·2	26·8*
	±16·6	±15·8	±12·0	±19·7
Cellulose	42·4	36·6	12·8	6·7
	±20·4	±23·2	±13·7	±6·0
Lignin	41·9	43·6	9·3	10·0
	±16·7	±13·0	±6·7	±5·7

* $p<0.05$

apparently by bacterial fermentation.[24 25 37] It has been previously shown that the ingestion of fermentable carbohydrate results in a larger colonic bacterial cell mass.[21] If this bacterial cell mass were in turn associated with a parallel increase in metabolic activity – for example, 7α-dehydroxylation – a rise in the production of the secondary bile acids could occur. The results of our study are consistent with this proposed mechanism as pectin was associated with a significant increase in both the percentage of biliary DCA and the recirculation of C^{14}-deoxycholate metabolites. The percentage of LCA was not significantly increased, but this may reflect the low solubility of this bile acid[12] with less being absorbed and recirculated. Alternatively less CDA may have been available for 7α-dehydroxylation, perhaps as a result of it being more strongly bound to faecal residues than would have been the case with CA.[18 19] It could be argued that with increased fermentation of pectin and presumably increased short chain fatty acid production, the pH of the large bowel contents may fall resulting in less 7α-dehydroxylation.[9] In spite of this theoretical possibility, however, faecal pH was not significantly altered after the feeding of pectin in these subjects,[23] though faecal pH might not necessarily reflect caecal pH.

Could the gelling effects of pectin have contributed to the observed changes in bile acid metabolism? If pectin were to form gels in the small intestine it is possible the absorption of compounds trapped within it might be inhibited. Pectin binds bile acids *in vitro*[38] although the exact mechanism is yet to be determined. Increased small intestinal trapping of bile acids by pectin could enhance the delivery of bile acids bound to pectin into the colon. Here the pectin would undergo fermentation thus releasing its trapped bile acids, which in the case of CA and CDCA, if not further bound by the colonic luminal residues, would be available for 7α-dehydroxylation. The increased DCA and C^{14}-deoxycholate metabolites in bile could therefore have resulted from relatively more CA being available for 7α-dehydroxylation. Although CDCA is

likely to have undergone similar 7α-dehydroxylation, relatively less may have been available as this bile acid would be more tightly bound to colonic luminal residues than would be the case with CA.[18] [19] Thus the bile acid changes induced by pectin, whereby more significant effects were observed for CA and its metabolite DCA than for CDCA and its metabolite LCA, could be explained by differences in the availability of the respective primary bile acids for 7α-dehydroxylation as a result of differences in their binding to colonic luminal residues. This explanation is supported by correlations between the individual bile acids whereby DCA was negatively correlated with both CA and CDCA, the correlation being stronger for CA.

The effect of cellulose on bile acid metabolism, whereby the CDCA is raised and the DCA depressed, is quite different from that of pectin. Preferential binding of secondary bile acids by the cellulose could account for this effect, but similar changes were not seen after lignin supplementation and it is lignin rather than cellulose which has been shown *in vitro* to have the greater bile acid binding capacity.[18] Alteration in intestinal transit time is an alternative explanation. Cellulose generally decreases intestinal transit time (though not invariably) and this would mean less time for the absorption of bile acids and also less time for 7α-dehydroxylation to occur. In the present study intestinal transit time was shortened from 55 to 40 hours[23] but there was no correlation between intestinal transit time and biliary bile acid composition after cellulose. Thus alterations in transit cannot be wholly responsible for the effect of cellulose. Cellulose is partly fermentable[39] so some changes in bacterial cell mass are possible, but likely to be in the direction of an increase rather than a reduction. But cellulose also significantly lowered faecal pH from 6·4 to 6·1.[23] At this level bacterial 7α-dehydroxylation could be significantly inhibited.[40] [41] Any effect of a greater bacterial cell mass would be negated by the pH effect. Indeed, the taurocholate $-24C^{14}$ metabolite study revealed that C^{14}-deoxycholate metabolites were halved after cellulose. The increase in biliary CDCA, albeit minor, could be explained by the lifting of a specific inhibition of CDCA synthesis by DCA[42] or by reduction in the competitive inhibition of CDCA absorption by DCA.[12]

In the present study dietary supplementation of lignin did not change biliary bile acid composition. This is in accord with the hamster studies using the same high molecular weight, high methoxyl auto-hydrolysed lignin.[43] The animal study showed that dietary supplements with this lignin, on a 5% w/w basis caused an increase in faecal bile acid excretion without altering biliary bile acid composition. In the

hamsters, however, any tendency to cholesterol saturation secondary to a lithogenic diet was counteracted by the lignin, a trend not seen in the present study. It is probable that a 12 g supplement of lignin to the human diet is not equivalent to the 5% w/w composition of the hamster diet. It may be that massive doses of lignin are required before a bile acid sequestrant effect comparable with cholestyramine is evident.[27] [44] As 12 g/day was the maximal tolerated dose of lignin in this study it may be difficult to achieve such an effect in man. The lack of an effect of lignin on bile salt metabolites though makes it even less likely that the changes in biliary acid composition seen after cereal fibre supplementation are due to the adsorption of bile acids by the lignin.

Alteration in the percentages of individual bile acids in the bile may be important with respect to the biliary cholesterol saturation. In a large study of patients with gall stones, the concentration of biliary cholesterol showed a weak negative correlation with the proportion of CDCA and a weak positive correlation with the percentage of DCA.[11] A high percentage of DCA has previously been related to lithogenicity[45] and experimental manipulation of the proportion of DCA in bile has usually altered the cholesterol saturation in the same direction[3] [13] [14] [46] though not invariably so.[47] In the present study there were no significant changes in the percentages of biliary cholesterol or the lithogenic index despite alterations in the proportions of CDCA and DCA after both pectin and cellulose supplementation. Nor were there any significant correlations between biliary lipid and the bile acid proportions. This probably reflects the rather modest changes in the percentages of the individual bile acids. Cellulose did, however, tend to lower the highest percentages of biliary cholesterol as well as increasing CDCA and decreasing DCA. Furthermore, in a single subject taking 15 g cellulose for a three month period the cholesterol saturation fell by 29% over an extended period (six months) so it remains possible that cellulose might exert a favourable influence on supersaturated bile in the long term. Longer term studies would be required to clarify this suggestion. Significant effects on the cholesterol saturation of bile also may have been missed by the study having been performed with normal volunteers. In previous studies bran supplementation has been associated with a reduction in biliary cholesterol saturation in subjects with supersaturated bile,[3] [4] but is without effect when biliary cholesterol saturation is normal initially.

The findings in this study help to clarify the apparently diverse effects of fibre on biliary bile acid composition reported previously. The low biliary

Table 6 *Properties of pectin, cellulose and lignin and proposed mechanisms of action*

	Pectin	Cellulose	Lignin
Fermentability	High	Intermediate	Negligible
Effect on stool characteristics	Negligible	↑ wt, ↓ ITT, ↓ pH	Negligible
Effect on bile acid composition	↑ 2° bile acids	↓ 2° bile acids	No effect
Proposed mechanism	Bacterial metabolic activity promoted by increased bacterial mass; ? increased delivery bile acids into colon	Bacterial metabolic activity reduced by low pH, rapid transit	Bacterial metabolic activity not affected

Wt=24 hour wet stool weight; ITT=intestinal transit time; 2°=secondary

DCA concentration seen in some vegetarians,[10] in Nigerians[48] and after bran supplements,[3] [6] are likely to be because of the high cellulose component of these diets, altering stool characteristics in such a way that secondary bile acid produciton or reabsorption is diminished. In contrast, highly fermentable diets would be expected to increase rather than decrease the amount of secondary bile acids present as long as 7α-dehydroxylation is not inhibited. This would explain the results of biliary bile acid composition reported in South Pacific[15] and African populations[16] where large amounts of fermentable fibre are consumed. These hypotheses are summarised in Table 6.

Finally, as individual fibre components have differing metabolic effects, care should be taken in interpreting studies reporting the effects of total dietary fibre on biliary lipid composition and bile acid metabolism.

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