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A Case-Control Study of Dietary Intake of Renal Stone Patients

II. Urine Biochemistry and Stone Analysis

H. M. Griffith*, B. O'Shea, M. Maguire, B. Keogh and J. P. Kevany

Department of Clinical Medicine, Meath Hospital, Dublin, and Human Nutrition Unit, Department of Community Health, Trinity College, Dublin, Ireland

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Summary. The dietary intakes of 88 renal stone cases and 88 age and sex matched controls were assessed by dietary history using a standardised questionnaire. The stone cases were divided into six subgroups established on the basis of urine biochemistry (calcium, oxalate and uric acid) and stone composition. The average intake of each group was then compared with that of their controls using standard statistical procedures. Cases with idiopathic calcium oxalate stones had significantly lower intakes of dietary fibre, non-cellulose polysaccharide, phytate, magnesium, phosphate and thiamine than controls. No significant difference in dietary intake was found between cases with high urinary calcium and uric acid and their respective controls. All cases with a high urinary oxalate had a significantly higher intake of vitamin C than controls. Our results support the belief that dietary intake is an important pre-urinary risk factor of idiopathic renal stone disease.

Key words: Dietary intake, Control study, Renal stones, Urine biochemistry, Stone composition.

Introduction

The exact cause of renal stone disease in most cases is difficult to ascertain due to its multifactorial nature. At least five urinary risk factors have been identified with the formation of the common calcium stone; increased urinary excretion of calcium, oxalate and uric acid, a high urinary pH and a reduced concentration of acid mucopolysaccharide [43]. Much research has been conducted on the dietary factors which contribute to these urinary risk factors, in particular hypercalciuria, hyperoxaluria and hyperuricosuria.

The reported incidence of hypercalciuria in renal stone patients varies widely from 20% [5] to 60% [36], the prevelance probably depending on how hypercalciuria is defined. Many studies have shown that calcium excretion is influenced by various dietary factors and therefore hypercalciuria should only be determined in relation to diet. The majority of renal stone patients have normal urinary calcium levels on a low calcium diet (2–5 mg calcium/kg/day) and above normal levels on a high calcium diet (15–20 mg calcium/kg/day) [35]. Urinary calcium excretion is also increased in normal and renal stone patients with increased protein [4, 26, 42], sodium [33], magnesium [24] and refined carbohydrate intakes [54] and with low phosphate [24] and how dietary fibre intakes [19, 51].

Changes in urinary oxalate are fifteen times more potent than urinary calcium changes in altering the saturation of urine with calcium oxalate [10] and most studies have shown that the mean level of urinary oxalate is elevated in stone patients [29]. Idiopathic renal stone formers are generally advised to reduce their oxalate intake but a number of other dietary factors can also increase urinary oxalate excretion and should be considered in patient advice. Urinary oxalate excretion is reported to increase from ingestion of oxalate precursors [18], on a low calcium, high oxalate diet [27], on a high animal protein [42] and vegetable protein intake [4], after administration of vitamin D [22], with ingestion of large doses of vitamin C [47] although many workers disagree with the last finding [11, 49] and in pyridoxine deficiency, with administration of tryptophan increasing this effect further [9].

Hyperuricosuria is a major risk factor of calcium stone disease [43]. A high purine intake increases production and excretion of uric acid [17] but most hyperuricosuric patients with calcium stones have low to normal levels of uric acid on a low purine diet [34]. Ascorbic acid supplements (>4 g/day) have also been reported to cause a significant increase in uric acid excretion in normal and hyperuricosuric patients [40]. Table 1 summarises the dietary factors associated with increases in urinary excretion of calcium, oxalate and uric acid.

Anderson [1] suggested that renal stone incidence of a community is related to its dietary structure and that if

* *Requests for reprints to:* Ms Helen M. Griffith, Research Dietitian, Department of Clinical Medicine, Meath Hospital, Dublin 8, Eire

Table 1. Relationship between dietary components and urinary risk factors in renal stone formation

Urinary risk factor	Dietary components	Nature of relationship
Hypercalciuria	Calcium	+
	Sodium	+
	Protein	+
	Magnesium	+
	Refined carbohydrates and sugars	+
	Phosphate	−
	Dietary fibre	−
	Phytate	−
Hyperoxaluria	Oxalate	+
	Oxalate precursors	+
	Calcium	−
	Purine	+
	Vitamin D	+
	Ascorbic acid	+
	Pyridoxine deficiency	+
Hyperuricosuria	Purine	+
	Ascorbic acid	+

Table 2. Composition of renal stones classified according to sex of subject

Stone type	Males	Females	Total
Calcium oxalate (CaOx > 75%)	8 (38%)	3 (13%)	11 (25%)
Calcium phosphate (CaP > 75%)	0	1 (4%)	1 (2%)
Mixed CaOx/CaP	12 (57%)	13 (57%)	25 (57%)
Magnesium ammonium phosphate and CaP (MAP > 10%)	1 (5%)	6 (26%)	7 (16%)
Totals	21 (100%)	23 (100%)	44 (100%)

this is so, a pathway must exist between the structure of the diet and the resultant composition of urine and stones. This study was set up to examine the relationship between dietary intake and urine and stone composition in renal stone patients. From a total sample of 88 renal stone cases, 85 cases provided at least one 24 h urine collection for biochemical analysis and 44 cases had a stone analysed. Sub-groups of cases were formed on the basis of stone and urine analysis and the nutrient content of their diet was compared with that of their matched controls using standard statistical procedures.

Patients and Methods

The study sample was divided into six subgroups established on the basis of urine and stone analysis. These subgroups included all cases with high urinary calcium, oxalate and uric acid, cases with high urinary calcium and oxalate only and cases with calcium oxalate or mixed calcium oxalate/calcium phosphate stones. The dietary intake of each case and control was assessed and analysed as described previously [16] using the same controls matched with the same cases as in the previous study.

Biochemical Analysis

Each case provided at least one 24 h urinary sample collected in an acid container (10 ml of 5 N HCl) as close to the time of diagnosis as possible while on their normal diet. Each sample was quantitatively analysed for content of calcium, oxalate and uric acid. Urinary calcium was determined by atomic absorption spectrophotometry [56]. Urinary oxalate was assayed as described by Costello and coworkers [7] with a single modification, the precipitated oxalate being extracted at 37 °C and not as described at 75 °C. Urinary uric acid was determined enzymatically using the Uricase kit supplied by Boehringer Mannheim [38].

Stones removed surgically or passed spontaneously and saved by the patient were analysed quantitatively on a routine basis and not specifically for the study. Calcium, magnesium, inorganic phosphate, oxalate and ammonia were all quantitatively determined by calorimetric methods as follows: Calcium [15], magnesium, using a Rapid-Stat kit supplied by Pierce Chemical Co., Illinois [13], inorganic phosphate [14], oxalate [21] and ammonia using Nesslers reagent [25]. Uric acid was quantitatively determined by an enzymatic method using the Uricase kit.

Statistical Analysis

Statistical analysis of the dietary intakes of the six subgroups and their matched controls included the calculation of the mean, the standard error of the mean and the application of the paired sample t-test for each of the 27 nutrients and three risk factors investigated. Results are presented as nutrient intake per individual (Whole body mass) and per kilogram of actual body weight in subjects within the normal weight for height using standard tables [30] and maximum ideal body weight ("estimated fat free mass") for overweight cases and controls using the same tables.

Results

Table 2 shows the composition of stones in relation to sex. Only 50% of cases had a stone analysed as many of them had not passed or had failed to save the stone or had not had surgery to remove a stone, up to six months after their interview. Dietary intake of calcium oxalate/calcium phosphate stone cases only were studied, as the calcium phosphate stone group had only one case while the aetiology of magnesium ammonium phosphate stones was assumed to be mainly infection and not diet related.

Table 3 shows the breakdown of cases in relation to urinalysis and sex. Cases with a urinary calcium excretion of > 7.5 mmol/day in males and > 6.25 mmol/day in females formed the high urinary calcium group (HUC). Cases with a urinary oxalate of > 0.46 mmol/day formed the high urinary oxalate group (HUO) and those with a urinary uric acid of > 4.8 mmol/day in males and > 4.5 mmol/day in females formed the high urinary acid group (HUU). Table 3 shows that 44 cases (52%) had no urinary abnormalities detected

Table 3. Urinalysis of cases classified according to sex

Urinalysis	Males (n = 48)	Females (n = 37)	Total (n = 85)
No abnormalities	18 (38%)	25 (70%)	44 (52%)
High Urinary Calcium (HUC)	9 (19%)	3 (8%)	12 (14%)
High Urinary Oxalate (HUO)	8 (17%)	8 (22%)	16 (19%)
High Urinary Uric Acid (HUU)	2 (4%)	0	2 (2%)
Mixed abnormalities:			
HUC + HUO	4 (8%)	0	4 (5%)
HUC + HUU	3 (6%)	0	3 (4%)
HUO + HUU	1 (2%)	0	1 (1%)
HUC + HUO + HUU	3 (6%)	0	3 (4%)
Total	48 (100%)	37 (100%)	85 (100%)

with respect to urinary excretion of calcium, oxalate or uric acid. Thirty cases (35%) had one abnormality only detected and 11 cases (13%) had more than one abnormality detected.

As in our previous paper [16], the results of all the nutrient intakes analysed for each group were too numerous to reproduce here and generally only those nutrients that show a difference of at least 0.10 level of probability between cases and controls or are of special relevance to a particular sub-group are presented. Differences of > 0.05 level of probability are not considred to be statistically significant. In all the following tables of dietary intake results, a negative mean difference indicates that the case group had a lower intake than the control group.

Table 4 shows that cases with idiopathic calcium oxalate stones had a significantly lower intake of dietary fibre, non-cellulose polysaccharide, phytate, magnesium, phosphate and thiamine, all of which have been previously associated with renal stone disease. Tables 5, 6 and 7 compare the nutrient intake of cases with at least one urinary risk factor and their matched controls, the groups generally being too small statistically to sub-divide according to sex. Table 5 shows that all cases with HUC (including those with other urinary risk factors) and cases with HUC only had no significant difference in intake between cases and controls although animal protein, fat, purine, tryptophan and panthothenic acid were consumed in greater amounts by cases with HUC only when results were presented as intake per whole body mass. Table 4 shows that all cases with HUO (including those with other urinary risk factors) and cases with HUO only had a significantly higher intake of vitamin C than their matched controls. Potassium and pyridoxine intakes were also significantly higher in the HUO only cases compared to controls. No significant differences were found between the HUU case and control group (Table 7) and those found did not reach a 0.01 level of probability.

Table 4. Differences in the average daily intake of nutrients for cases with calcium oxalate or mixed calcium oxalate/calcium phosphate stones and matched controls on whole body and estimated fat free mass basis

Nutrients (Units)	Intake per whole body mass					Intake per kg estimated fat free mass				
	Cases (n = 36)		Controls (n = 36)		mean diff.	P	Cases (n = 36)		Controls (n = 36)		mean diff.	P
x	$\bar{x}$	$2SE_{\bar{x}}$	$\bar{x}$	$2SE_{\bar{x}}$			$\bar{x}$	$2SE_{\bar{x}}$	$\bar{x}$	$2SE_{\bar{x}}$		
Total protein (g)	103.50 ± 12.47		109.60 ± 12.97		−6.10	NS	1.540 ± 0.167		1.680 ± 0.187		−0.140	NS
Animal protein (g)	70.41 ± 8.17		73.70 ± 8.13		−3.29	NS	1.075 ± 0.141		1.144 ± 0.138		−0.069	NS
Dietary fibre (g)	20.59 ± 2.07		25.00 ± 3.43		−4.41	< 0.05	0.305 ± 0.037		0.382 ± 0.054		−0.007	< 0.02
Non-cellulose polysaccharide (g)	15.51 ± 1.40		18.92 ± 2.37		−3.41	< 0.02	0.241 ± 0.026		0.292 ± 0.036		−0.051	< 0.02
Cellulose (g)	5.48 ± 0.50		6.29 ± 0.73		−0.81	NS[b]	0.086 ± 0.009		0.098 ± 0.011		−0.012	NS[b]
Lignin (g)	1.43 ± 0.27		1.83 ± 0.47		−0.40	NS[b]	0.024 ± 0.006		0.028 ± 0.007		−0.004	NS[b]
Phytate (mg)	141.50 ± 26.07		215.00 ± 47.67		−73.50	< 0.02	2.257 ± 0.446		3.353 ± 0.731		−1.096	< 0.02
Thiamine (mg)	1.65 ± 0.21		2.08 ± 0.39		−0.43	< 0.05	0.026 ± 0.004		0.031 ± 0.005		−0.005	< 0.05
Pryridoxine (mg)	1.61 ± 0.15		1.93 ± 0.28		−0.32	NS[b]	0.023 ± 0.003		0.030 ± 0.005		−0.007	NS[b]
Phosphate (g)[a]	1.58 ± 0.16		1.87 ± 0.22		−0.29	NS	0.024 ± 0.003		0.027 ± 0.003		−0.003	< 0.05
Magnesium (mg)	339.00 ± 31.80		404.03 ± 53.67		−65.03	< 0.05	5.081 ± 0.567		6.147 ± 0.817		−1.066	< 0.025

[a] normally expressed in mg
[b] < 0.10 level of probability

Table 5. Differences in the average daily intake of nutrients for high urinary calcium cases and matched controls on whole body and estimated fat free mass basis

Nutrients (Units)	Intake per whole body mass				Intake per kg estimated fat free mass			
	$\bar{x}$ $2SE_{\bar{x}}$	$\bar{x}$ $2SE_{\bar{x}}$	mean diff.	*P*	$\bar{x}$ $2SE_{\bar{x}}$	$\bar{x}$ $2SE_{\bar{x}}$	mean diff.	*P*
(A)	Cases (n = 22)	Controls (n = 22)			Cases (n = 22)	Controls (n = 22)		
Dietary fibre (g)	20.84 ± 3.84	24.23 ± 5.67	−3.39	NS	0.299 ± 0.059	0.366 ± 0.090	−0.067	NS
Total protein (g)	113.73 ± 13.23	111.91 ± 18.93	1.82	NS	1.667 ± 0.200	1.704 ± 0.337	−0.037	NS
Animal protein (g)	79.64 ± 10.39	75.14 ± 14.50	4.50	NS	1.164 ± 0.163	1.121 ± 0.169	0.043	NS
Calcium (g)[a]	1.13 ± 0.18	1.23 ± 0.28	−0.10	NS	0.016 ± 0.003	0.019 ± 0.003	−0.003	NS
Phosphate (g)[a]	1.79 ± 0.24	1.84 ± 0.35	−0.05	NS	0.025 ± 0.004	0.028 ± 0.006	−0.003	NS
(B)	Cases (n = 12)	Controls (n = 12)			Cases (n = 12)	Controls (n = 12)		
Dietary fibre (g)	18.41 ± 3.34	23.65 ± 9.21	−5.24	NS	0.274 ± 0.045	0.350 ± 0.131	−0.076	NS
Total protein (g)	119.42 ± 17.49	98.58 ± 19.94	20.84	NS	1.748 ± 0.027	1.502 ± 0.324	0.246	NS
Animal protein (g)	86.42 ± 14.30	67.38 ± 13.63	19.04	NS[b]	1.270 ± 0.231	1.011 ± 0.214	0.259	NS
Calcium (g)[a]	1.16 ± 0.20	1.07 ± 0.23	0.09	NS	0.016 ± 0.004	0.016 ± 0.004	0.000	NS
Phosphate (g)[a]	1.83 ± 0.33	1.66 ± 0.45	0.17	NS	0.027 ± 0.005	0.025 ± 0.007	0.002	NS
Fat (g)	145.30 ± 25.78	108.35 ± 20.32	36.95	NS[b]	2.144 ± 0.370	1.774 ± 0.520	0.370	NS
Purine (mg)	163.08 ± 37.51	129.08 ± 40.63	34.00	NS[b]	2.621 ± 0.652	1.953 ± 0.601	0.668	NS
Tryptophan (g)	1.55 ± 0.24	1.25 ± 0.26	0.30	NS[b]	0.023 ± 0.004	0.019 ± 0.005	0.004	NS
Pantothenic acid (mg)	6.56 ± 1.16	5.76 ± 1.63	0.80	NS[b]	0.103 ± 0.027	0.085 ± 0.023	0.018	NS

(A) = All cases with high urinary calcium including those with other urinary abnormalities
(B) = Cases with only high urinary calcium

[a] normally expressed in mg
[b] 0.10 level of probability

Table 6. Differences in the average daily intake of nutrients for high urinary oxalate cases and matched controls on whole body and estimated fat free mass basis

Nutrients (Units)	Intake per whole body mass				Intake per kg estimated fat free mass			
	$\bar{x}$ $2SE_{\bar{x}}$	$\bar{x}$ $2SE_{\bar{x}}$	mean diff.	*P*	$\bar{x}$ $2SE_{\bar{x}}$	$\bar{x}$ $2SE_{\bar{x}}$	mean diff.	*P*
(A)	Cases (n = 24)	Controls (n = 24)			Cases (n = 24)	Controls (n = 24)		
Oxalate (mg)	234.33 ± 38.53	211.33 ± 27.70	23.00	NS	3.809 ± 0.772	3.107 ± 0.441	0.702	NS
Vitamin C (mg)	109.00 ± 28.41	72.00 ± 13.72	37.00	< 0.02	1.770 ± 0.453	1.140 ± 0.249	0.630	< 0.02
Thiamine (mg)	1.77 ± 0.29	1.75 ± 0.30	0.02	NS	0.028 ± 0.005	0.028 ± 0.006	0.000	NS
(B)	Cases (n = 16)	Controls (n = 16)			Cases (n = 16)	Controls (n = 16)		
Oxalate (mg)	258.56 ± 49.08	207.63 ± 37.82	50.93	NS	3.823 ± 0.861	3.231 ± 0.631	0.597	NS
Vitamin C (mg)	105.20 ± 26.48	68.07 ± 16.45	37.13	< 0.02	1.779 ± 0.525	1.121 ± 0.325	0.658	< 0.02
Potassium (g)[a]	4.48 ± 0.62	3.48 ± 0.52	1.00	NS[b]	0.074 ± 0.012	0.057 ± 0.011	0.017	< 0.05
Pyridoxine (mg)	1.90 ± 0.30	1.47 ± 0.27	0.43	NS[b]	0.031 ± 0.006	0.024 ± 0.006	0.007	< 0.05
Thiamine (mg)	1.92 ± 0.39	1.61 ± 0.38	0.31	NS	0.029 ± 0.007	0.026 ± 0.007	0.003	NS
Cellulose (g)	6.20 ± 0.71	4.96 ± 1.07	1.24	NS[b]	0.094 ± 0.017	0.076 ± 0.015	0.018	NS[b]

(A) = All cases with high urinary oxalate including those with other urinary abnormalities
(B) = Cases with only high urinary oxalate

[a] normally expressed in mg
[b] < 0.10 level of probability

Table 7. Differences in the average daily intake of nutrients for high urinary uric acid cases and matched controls on whole body and estimated fat free mass basis

Nutrients (Units)	Intake per whole body mass				Intake per kg estimated fat free mass			
	Cases (n = 9)	Controls (n = 9)	mean diff.	*P*	Cases (n = 9)	Controls (n = 9)	mean diff.	*P*
	$\bar{x}$ $2SE_{\bar{x}}$	$\bar{x}$ $2SE_{\bar{x}}$			$\bar{x}$ $2SE_{\bar{x}}$	$\bar{x}$ $2SE_{\bar{x}}$		
Total protein (g)	101.41 ± 22.0	115.20 ± 27.60	−13.79	NS	1.39 ± 0.33	1.53 ± 0.41	−0.14	NS
Animal protein (g)	70.89 ± 17.13	79.00 ± 18.47	−8.11	NS	0.86 ± 0.31	1.05 ± 0.28	−0.19	NS
Purine (mg)	158.10 ± 50.20	142.11 ± 37.00	15.99	NS	2.20 ± 0.75	1.89 ± 0.55	0.31	NS

Discussion

All the nutrient differences found between the calcium oxalate stone group and their controls (Table 4) have previously been associated with renal stone formation. A low dietary fibre intake has been associated with a high stone incidence [2, 44] but our findings in this respect differ from another similar study [39] where no significant difference in dietary fibre intakes were found between renal stone cases and age and sex matched controls. Details of stone type of their cases were not reported however and the probable mixture of stone types could have influenced the results. Robertson and co-workers [45] reported that the prevelance of renal stones in vegetarians was 40–60% of that predicted for groups of individuals taken from the general population and matched for age, sex and social class. This finding they attributed to the lower intake of animal protein in the vegetarian group but as vegetarians in Britain eat about twice the national average amount of fibre (41 g/ day) [12], it may also have influenced the low prevelance in the vegetarian group.

The lower phytic acid intake in the case group supports the studies of Modlin [31, 32] who found an association between a high stone incidence and a low phytic acid intake. He hypothesised that a reduction in dietary phytate and probable concommitent reduction of urinary phosphorylated inositols (Inhibitors of stone formation) could be an important factor in the formation of renal stones. Phytic acid may also reduce calcium absorption by forming calcium phytate complexes, not readily absorbed in the intestine [19, 51].

Many studies have shown a lower urinary magnesium excretion in stone formers compared to stone free controls [53, 57] although others have not found this to be so [55]. Magnesium increases the capacity to keep calcium oxalate in solution [56], and has been found to be effective in preventing the recurrence of calcium oxalate stones [23, 28]. A low dietary phosphate has been shown to increase urinary calcium excretion [24] and phosphate supplements have been reported to cause a significant reduction in the recurrence rate of calcium stones [8] possibly by increasing the excretion of pyrophosphate. It is difficult to say if any real significance can be attached to the lower thiamine intake in cases, as thiamine, dietary fibre and phytic acid have many common food sources and therefore it may simply be a secondary effect of a low dietary fibre and phytic acid intake.

One dietary study of idiopathic calcium oxalate stone formers [42] showed that stone formers consumed significantly more animal protein than controls, while our study showed no significant difference in this respect. There are two possible reasons for these different results. Firstly, they studied only male stone formers, while our study was of both sexes (20 males and 16 females). Our previous paper [16] showed that male and female stone formers had completely different trends in dietary consumption compared to sex matched controls. Secondly, they selected controls from laboratory and medical staff, some of whom may have been more aware of the detrimental effects of a high animal protein diet than the general population and consequently have altered their intakes, resulting in a lower mean intake of animal protein.

The limitations of studying urinary risk factors using a single 24 h urine collection have been reported [48]. However, the number of our cases presenting with one or more urinary risk factors (Table 3) agrees well with the findings of another study [39] where at least three 24 h collections were included for analysis per patient. The percentage of predominately calcium oxalate stones was lower and the percentage of mixed calcium oxalate/calcium oxalate stones was higher in our study (Table 2) than that of other workers [37, 46]. As urinary pH was not measured it was not possible to say whether this is due to a real difference in the prevailing urinary pH of Irish renal stone patients arising from differences in diet, or merely due to the small sample and low percentage of stones actually available for analysis.

We found no significant differences in dietary intake between HUC cases and controls, but differences may have been concealed due to the small number of HUC cases studied. Other workers found no significant difference in the dietary calcium intakes of hypercalciuric stone formers and controls [50].

The significantly higher intake of vitamin C in the HUO case group was surprising. Although high doses of vitamin C have been associated with hyperoxaluria, the mean vitamin C intake of our HUO cases (107 mg/day) was not excessive compared to the amounts reported to cause hyperoxaluria i.e. > 1 g/day [3, 47]. In normal individuals 40% of dietary vitamin C undergoes a non-enzymatic conversion to oxalate [29] and certain individuals may be even more efficient at converting dietary vitamin C to oxalate than normal [3]. The in vitro conversion of vitamin C to oxalate can occur in assay procedures which involve the acidification of the urine with HCL and heating in a boiling water bath for 30 min [11]. In our assay, the oxalate was extracted at 37 °C and using this procedure, volunteers who ingested 8 g, of vitamin C daily did not show an increased urinary oxalate [11]. Changes in urinary oxalate concentration have one of the greatest effects in increasing the risk of stone incidence in a population [41] which suggests that the increase in urinary oxalate that might occur from only moderate intakes of vitamin C (80–140 mg/day) could be important in renal stone formation.

The higher pyridoxine and potassium intake found in the HUO group has not been reported elsewhere but the latter was not unexpected, as many rich sources of vitamin C are also rich sources of potassium. Although HUO cases did on average have higher dietary oxalate intakes than controls, this difference was not statistically significant, a finding also reported by Hodgkinson [20].

No significant difference in dietary intakes was found between HUU cases and controls and this differs from the findings of Coe and co-workers [6] who reported a significant difference in purine intake between ten hyperuricosuric

patients and five matched controls. It is possible that some usually normouricosuric cases were included in our sample (five cases had only one 24 h uric acid estimated) and this may have diluted the difference in dietary intake found.

Further dietary research on larger groups of idiopathic renal stone patients and more detailed studies of patients grouped according to underlying aetiological factors would seem worthwhile. The advantage of studying cases according to presence of urinary risk factors is demonstrated in the differences in vitamin C intake found between cases and controls. This difference was not evident when these HUO cases were studied as part of the total group. The findings presented in this and our previous paper [16] support the belief that diet is an important pre-urinary risk factor of idiopathic renal stone disease.

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H. M. Griffith
Research Dietitian
Department of Clinical Medicine
Meath Hospital
Dublin 8
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