Dipstick Measurements of Urinary pH have Potential for Monitoring Individual and Population Dietary Behaviors

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Abstract: The health benefits of fruit and vegetable consumption are well recognized and a recent cancer prevention report suggested modifications to diet that included reductions in red meat and increases in plant foods. However, populations in the US and UK still do not meet the 5-A-Day target for a number of reasons, including motivational issues. A readily accessible measure or biomarker of dietary change would be useful for individual and population monitoring. One potential biomarker is urine pH, which reflects the acid-base load of the diet. Dipstick measures of pH are simple and rapid and could provide a feedback mechanism during dietary change. Dietary acid-base load results from the balance between the alkaline salts prevailing in fruits and vegetables and H+ ions generated mainly from animal and cereal foods. This mini-review covers studies relating dietary acid-base load to urine pH and provides suggestions for further research.

BACKGROUND

The health benefits of high fruit and vegetable intakes are well recognized and extensive public health programmes exist to encourage increased consumption. Since obesity is an increasing societal and public health problem, and a diet containing 5 or more portions of fruit and vegetables per day (as low energy density foods) may offer protection from weight gain, this provides further incentive for adequate population consumption of fruits and vegetables [1,2]. A recent UK government report concerned with healthy body weight stated that 'everyone should eat 5 portions of fruits and vegetables per day' [3]. Also another recent cancer report, by the World Cancer Research Fund (WCRF), suggested that a reduction in red meat consumption and a diet predominating in low energy density foods, in addition to increased fruit and vegetable consumption and plant foods and moderate alcohol intake, would prevent the occurrence and reoccurrence of cancers [4].

However, populations in the US and UK still do not meet the target of 5 portions of fruit and vegetables a day and another recent UK government report found that fewer than one third of men (31%) or women (29%) consumed 5 or more portions of fruit and vegetables a day [5-7].

Despite good reason to modify diet, a number of reasons exist why individuals do not eat sufficient fruits and vegetables, and these include education, economics, accessibility and motivation, with health-related ambivalence making it psychologically difficult to maintain or make change in the face of temptation [8]. Even when individuals decide to increase fruit and vegetable consumption they are currently unable to monitor these changes or have tangible evidence of the benefits of change. It would therefore, be useful if there were a readily accessible measure or biomarker to monitor dietary change in individuals.

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Given that the growing number of government strategies and initiatives to improve diet and health and prevention of obesity are costly, monitoring of their effectiveness needs to be evaluated to ensure strategies are working. Options for monitoring for dietary change include simple self reports of food intake but there are a number of methodological issues with this, including mis-reporting of intakes [9-11]. Biological measures or biomarkers are also available and it is well established that plasma vitamin C reflects dietary intake but blood measures are intrusive, costly and not readily available to individuals [12]. However, if a simple, readily accessible biomarker of change in dietary behaviour were available, that could be used for individual or population monitoring, this would have considerable advantages.

One candidate biomarker is urine pH, which has been related to dietary acid-base load and to the more accurately measured net acid excretion (NAE) [13,14]. This mini-review covers previous intervention and epidemiological studies relating diet and synthetic compounds to urine pH and discusses whether urine pH could be a suitable biomarker for monitoring dietary change.

CONCEPTS OF ACID-BASE BALANCE AND DIET

Acid-base equilibrium in the body is maintained by 3 mechanisms; blood and tissue buffering, excretion of CO2 by the lungs and renal excretion of H+ and regeneration of HCO3 [15].

Among the known effects of metabolic acidosis are nitrogen wasting, decreased IGF-1 levels, kidney stone formation, a mild form of hypothyroidism and hyperglucocorticoidism [16]. There are also effects of acid-base metabolism on bone health, resulting from the activation of mature osteoclasts and mobilization of calcium and the corresponding inhibition of bone matrix mineralization, within a more acidic environment [17]. Four

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population studies have found a relationship between a more acidic dietary acid-base load and lower bone density in women and children [18-21].

Diet contributes to acid-base maintenance through the hepatic metabolism of the sulfur containing amino acids cysteine and methionine by generating H+ ions and reducing pH [14,22]. Metabolism of alkaline salts in fruits and vegetables, that contain carbonates and combustible organic acids and supply large amounts of Mg and K, balances the effects of H+ ions and increases pH. Dietary acid-base load is a balance between protein-rich foods such as meats, cereals and dairy foods that supply acid and fruits and vegetables that supply base precursors [14]. The kidneys are the main route of excretion of dietary H+ ions and, providing kidney function is not compromised by disease, the acid-base load of the diet is related to urine pH and net acid excretion (NAE) [22].

Diet acid-base load has been calculated in mEq per day using algorithms relating the ratio of protein to potassium or as PRAL (Potential Renal Acid Load) using the following equation (1):

PRAL (Potential Renal Acid Load) mEq/day = (phosphorus mg/d * 0.0366 + protein g/d * 0.4888) – (potassium mg/d * 0.0205 + calcium mg/d * 0.0125 + magnesium mg/d * 0.0263) [23].

EVIDENCE RELATING URINE PH TO DIET

Intervention Studies with Foods

The intervention studies in which diet and NAE have been related and that also measured urine pH are shown in Table **1** [13,24-31]. The dietary interventions focused mainly on either changes to the total amount of protein in the diet or on modifications of types of protein, that is, from animal to vegetable protein [13,24-28]. One study that held protein, Na, K, Ca and P constant but modified the type of protein from animal to dairy to plant sources in omnivorous, lacto-ovo-vegetarian and vegetarian diets, found increases in pH of 0.15 and 0.23 units, respectively. Two of the studies modified the amount and type of protein as well as fruits and vegetables in the diet and achieved changes of up to 1.2 pH units [13,25]. None of the studies investigated the effects of fruits and vegetables, only, on urine pH.

One study not included in the table because pH was not reported, investigated the alkalinizing effects of increasing fruit consumption on NAE in individuals fed an acidifying diet to induce hypercalcaemia [32]. Individuals were fed whey protein and either no fruit (control) or ≈ 3 (80g) portions of raw or 11 portions of cooked apple. Compared with the control group the protein-induced rise in the NAE to creatinine ratio was significantly reduced by raw fruit by ≈ 40% (P=0.041), indicating a positive effect of fruit on whey induced dietary acidification.

There appear to be few studies that have directly evaluated the relationship between the more accurately measured NAE and urine pH. However, one study in men consuming dietary protein ranging from 50g to 170g per day found a significant correlation between urine pH and NAE (r=0.83 P<0.001) [14]. The regression equation provided the following formula:

$$\text{urine pH} = -0.01*\text{NAE} + 6.9$$

Intervention Studies with Synthetic Compounds

Studies with potassium or sodium salts were also performed and although the purpose of these studies was not specifically to measure urine pH, these induced a similar range of changes in urine pH as the dietary interventions [29-31].

However, the studies described so far were small scale interventions mainly designed to estimate the effect of changes of acid-base status on markers of bone function and population estimates of the relationship between diet and urine pH have only recently been made.

Epidemiological Evidence from a Large Population Study

A recent population study found significant cross-sectional relationships between urine pH measured in casual (convenience) samples and dietary acid-base load measured as PRAL (potential renal acid load), and also with fruit and vegetables and meat consumption [33]. The study was in a middle-aged population of 22,038 men and women aged 39-78 years. pH was measured with detection sticks (dipsticks) ranging from pH 4.5 to 8.0, with 0.5 to 1.0 unit intervals. Intake of fruit and vegetables differed between the lowest and highest categories of urine pH (5.0 and 7.5 and over) by 50g in men and 68g in women, and there were also small but significant differences in meat consumption. There was also a difference of 4.2 mEq/d of PRAL between urine pH category 5.0 and 7.5 and over. The urine pH of meat-eaters differed from non-meat eaters by 0.1 unit pH, confirming a previous finding in vegetarian and omnivorous women [34]. All results were adjusted for covariates with potential to influence urine pH; age, BMI, physical activity and smoking habit. A higher plasma vitamin C (indicating high fruit and vegetable intakes) was also significantly related to a more alkaline urine (P<0.001), indicating that the associations observed between fruit and vegetable consumption and urine pH were not a chance finding.

A further finding was a gender related difference in the total intake of fruit and vegetables and urine pH (women had higher intakes than men for the same urine pH), which requires further explanation.

To compare methodologies for urine and dietary collection a validation sub-study was also performed in 363 men and women with 24-hour urine collections and data from a 7-day food diary and an FFQ (Food Frequency Questionnaire) [33]. pH in the 24-hour urine samples was measured using a pH meter. Stronger relationships were found between pH measured in the 24-hour urines and diet measured with the 7-day diary than those with the FFQ and casual urine sample that were of about twice the scale for PRAL and fruit and vegetable consumption. This suggests that the main results could have been greater if more precise methodology had been used.

OTHER INFLUENCES ON URINE PH

In addition to diet and the known metabolic disturbances that affect NAE and urine pH (diabetic ketoacidosis, renal failure and diuretic medication), age and body composition are also important.

Table 1. Intervention studies designed to measure effects of modifications to intake of Acid-base load on renal net acid excretion (NAE) that also measured urine pH

Intervention	N (% women)	Author, date (reference number)	Details of intervention	Urine pH (*change*) units
Dietary interventions				
Normal to high protein diet with carbohydrate restriction	10 (70%)	Reddy, 2002, [24]	91g protein and 285g carbohydrate to 164g protein and 19g carbohydrate	6.09 to 5.56 (***0.53***)
Diets designed to be acid then alkaline inducing	8 (0%)	Buclin, 2001 [25]	Protein 99g to 92g, K 75 to150 mmol/d, meat with some vegetables to no meat with FNVEG.	6.1 to 7.3 (***1.2***)
Low protein (LP), moderate (MP) and high protein (HP) with changes to protein type and fruit and vegetable consumption	6 (50%)	Remer, 1994 [13]	Protein 49g (LP), 95g (MP), 120g (HP). LP lactovegetarian (LV) with 21 portions FNVEG, MP 8.8 portions FNVEG, LP 2.9 portions FNVEG.	6.7 (LV) to 6.0 to 5.5 (***0.3, 1.2***)
Low protein to high protein with Ca and P held constant	8 (50%)	Trilok, 1989 [26]	50g to 106g protein (F), 50g to 120g (M). HP diet = LP + casein, lactalbumin, wheat gluten and dried egg whites	F 6.67 to 6.24 (***0.43***), M 6.07 to 5.81 (***0.26***)
Changes to protein type with Na, K, Ca and P held constant	15 (53%)	Breslau, 1988 [27]	75g protein changed from animal (A) to lacto-ovo vegetarian (LV) to vegetarian (V)	6.17(A) to 6.32(LV) (***0.15***) to 6.55(V) (***0.23***)
Low protein to high protein with Ca and P held constant	6 (100%)	Lutz, 1984 [28]	44g (LP) to 102g protein (HP)	6.0 (LP) to 5.2 (HP) (***0.8***)
Interventions with synthetic compounds				
Potassium citrate intervention	30 (100%)	Marangella, 2004 [29]	Potassium citrate 1mEq/kg body weight	6.11 to 6.33 (***0.22***)
Potassium bicarbonate intervention	201 (100%)	Frassetto, 2005 [30]	$KHCO_3$ 30, 60 and 90 mmol/d	6.57 to 7.00 (***0.43***), 6.5 to 7.01 (***0.51***), 6.34 to 7.20 (***0.86***)
Potassium bicarbonate intervention	9 (0%)	Lemann, 1989 [31]	$KHCO_3$ 61 mmol/d	6.00 to 6.68 (***0.68***)
Sodium bicarbonate intervention	6 (100%)	Lutz, 1984 [28]	$NaHCO_3$ 70 mEq/d plus 102g protein (HP)	5.3 (HP only) to 6.9 (HP+$NaHCO_3$) (***1.6***)

Abbreviations: LP low protein, MP medium protein, HP high protein. FNVEG – fruit and vegetables. m = men; f = women

Clinical Conditions

In active clinical conditions such as diabetic ketoacidosis or renal failure, measurement of urine pH to indicate dietary change is unlikely to be informative. The effect of dietary interventions on those receiving diuretic medication would require investigation.

It has recently been observed that urine pH is more acidic in those with type 2 diabetes than in normal volunteers [35]. A more acidic urine pH has also been observed in studies of the metabolic syndrome, gout and uric acid stone formers [36-38].

Body Size

Endogenous acids contribute to the total acid load in the body and production of organic acids is related to weight, BMI (body mass index), muscle mass and body surface area [13,33,39-41]. Endogenous acids comprise a wide spectrum of single analytes, including citric acid, oxalic acid, malic acid, succinic acid and lactic acid as well as the anionic amino acids, glutamic acid and aspartic acid [39,40]. A method of estimating organic acid production (OAest) involves using the equation (2)

OAest (mEq/d) = individual body surface area X 41/1.73 [23]

(body surface area m^2 = 0.007184 – height (cm) 0.725 – weight (kg) 0.425).

Total NEAP (net endogenous acid production) is the sum of PRAL (outlined in equation (1)) and estimated organic acids (outlined in equation (2)). It follows that in health, individual excretion of organic acids will be a constant related to body composition, with greater excretion of endogenous organic acids in those with greater BMI (body mass index).

Age

The age related decline in renal function is well documented with known increases in blood H+ ions and the potential for less efficient excretion of H+ as individuals age

[42,43]. Therefore, urine pH and NAE would be less likely to be strongly related to diet acid-base load in older individuals. In one small intervention study of 6 women, mean urinary pH was consistently more alkaline in the 2 women aged up to 40 years than the 4 aged 59 to 62 years, at any point in the intervention [28].

DISCUSSION

To date the dietary intervention studies have been small and mainly designed to establish the effects of diet on markers of bone function and also to determine the effect of modifying protein consumption rather than the alkalizing effects of fruit and vegetables. Urine pH was usually measured in 24-hour urine samples and not with dipsticks. The epidemiological study established that relationships between dietary acid-base load, fruit and vegetables and meats and urine pH exist within a Western type diet, at the population level, even when measured with a casual urine samples and dipsticks. The smaller associated sub-study indicated that a greater scale of relationship would have been found using more accurate methods. However, the use of casual urines and dipsticks for relating and evaluating changes in dietary behaviors within individuals and at the population level requires further investigation.

A previous study compared NAE and urine pH, measured by dipsticks, but was designed to compare the pH in first morning, fasting, urine sample and NAE [44]. The correlation between NAE and pH was reasonably substantial but was not statistically significant, leading the authors to conclude that urine pH would not be useful. However, there was a significant correlation of -0.71 ($P<0.005$) with NAE measured in the overnight collection and the morning pH sample and a significant correlation with titratable acid in both the overnight and morning collections. This study did not test the relationship with NAE and non-fasting urine pH, which would be more relevant if pH was being used to detect dietary change.

Issues with Urine pH and Future Research Directions

As diet, age, BMI and some clinical conditions influence urine pH and as urine pH varies throughout the day and from day to day, the intra-individual ranges of urine pH need to be established [25]. The optimum time of day for measurement of urine pH when related to diet needs to be established. The influence and scale of change associated with dietary modification in fruit and vegetable consumption needs to be established, within individuals. Although intra-individual variability in urine pH could be considered a disadvantage, it could also be a benefit since it would allow individuals to monitor dietary change and receive feedback.

The reliability, repeatability and potential error associated with dipstick measures requires further investigation. The use of dipsticks that measure urine pH with 0.25 unit differences should be tested. More research on the relationship between pH in casual and 24-hour urine samples is also needed. It would be essential to understand the variability in response to fruit and vegetable intakes in individuals of different age groups and body size and to establish whether there are gender differences. To date little research on diet and urine pH has been done in children [14].

Collection and measurement of saliva samples is more convenient than urine samples and so the relationship between urine pH and pH measured in saliva needs investigating. Since food consumption affects the composition of saliva, it would be important to establish how long after meals pH measures should be made. As with urine, the reliability, repeatability and error with dipstick measures would need to be evaluated.

So, given the known beneficial effects of increased fruit and vegetable consumption on health and the potential for obesity prevention, it would be worthwhile further investigating the use of urine pH for individual monitoring of increased fruit and vegetable consumption in the usual Western type diet. Given that body size and age also relate to urine pH and NAE, it is likely that it is the within person change in urine pH with diet, rather than the absolute value that would be important when monitoring change. The fact that urine pH is also affected by modifications in meat consumption would mean that urine pH could be suitable for monitoring the dietary changes recommended by the WCRF report. Indeed, the intervention study that modified protein type from meat to soya protein (omnivorous to lacto-ovo vegetarian), and kept protein and mineral intake constant, found a measurable increase in urine pH [27].

CONCLUSION

Larger well designed dietary intervention studies are required with variable quantities of fruit and vegetables, and also with meat, that take into account age, gender and body size to establish whether casual urine pH measured with dipsticks has utility for individual and population dietary monitoring. If the results of these studies provide evidence of sufficient intra-individual variability in urine pH with diet, it is conceivable that monitoring urine pH could be used to provide feedback to individuals and act as a motivational tool to encourage change in individual eating behaviors and also to monitor population dietary initiatives.

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