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# Mechanisms of gastrointestinal absorption: dietary minerals and the influence of beverage ingestion

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In the absence of exogenous (dietary) factors, there are five major determinants of mineral absorption: (a) degree of solubilisation in the stomach, (b) extent of hydrolysis/endogenous ligand interaction in the small bowel and lability of this new metal species; (c) transport through the mucus layer; (d) efficiency and mode of mucosal uptake which is partly dependent on (b), although some metals may be facilitated with active processes; (e) transport into blood which, for some minerals, is regulated by intra-mucosal and systemic factors.

Addition of exogenous factors may either alter the luminal complex presented to the mucus/mucosa, or directly affect the permeability of the mucosa. Whether these effects increase or decrease absorption of the mineral depends on its normal mode and efficiency of uptake. Thus citrate increases the absorption of aluminium (normally poorly absorbed) but reduces the absorption of calcium (normally well absorbed). Finally, assessment of such effects requires the use of a validated model and should be considered in the context of real dietary situations.

## INTRODUCTION

The gastrointestinal tract is continually exposed to essential and toxic mineral elements, either from the diet or by re-circulation through the pancreatic, biliary and gastrointestinal secretions (Powell *et al.*, 1992; Table 1). Systemic absorption of such elements depends on their chemical fate within the lumen of the bowel and also their intestinal uptake. Ingested foodstuffs may significantly interfere with either of these processes, and this paper will concentrate on potential interactions between metals and some common components from beverages. Beverages are highlighted, first, because they represent by weight about 70% of our total daily oral intake; secondly, they contain components that may interfere with mineral absorption; and thirdly, they provide a better opportunity than most foodstuffs for *in-vitro* mechanistic studies.

## ANATOMY, BIOCHEMISTRY AND PHYSIOLOGY OF THE GASTROINTESTINAL TRACT

The gastrointestinal tract is a muscular tube lined by a mucous membrane that has regional variations in structure and function. The overall function of the tract, however, is to absorb nutrients while excluding as far as possible unwanted molecules.

Initial chemical and mechanical digestion occurs in the stomach and is completed in the small bowel mainly by enzymatic degradation. The proximal small intestine (duodenum and jejenum) is the site of absorption for most nutrients. Figure 1 shows a schematic diagram and high-power transmission electron micrograph of the small bowel, explaining the major anatomical features relevant to mineral absorption. Biochemical and physiological factors further influencing absorption are outlined below.

### Luminal pH

The normal stomach maintains an acid pH in the lumen which, in the fasting state is 1·5–2·0, but may increase sharply after a meal, even to pH 7, since food has a transient buffering capacity (Ovesen *et al.*, 1986). Further acid is then produced in response to the meal and the lumen is rapidly re-acidified. Most beverages have little buffering capacity. Gastric contents are emptied into the small bowel and their acidity is rapidly neutralised by pancreatic bicarbonate-based secretion into the duodenum, where it is around pH 6·5. The pH then gradually increases down to the distal small bowel so that in the terminal ileum it is about 7·5. There is then a sharp drop in pH in the first part of the large bowel (caecum) to about 5·5 which, again, becomes

**Table 1. Elemental content of gastrointestinal fluids**

| | Hepatic bile[a] ($n$ = 4) | Pancreatic juice[b] ($n$ = 5) | Supernatant from distal small bowel contents[c] ($n$ = 4) |
|---|---|---|---|
| Sodium (mM) | 145·3 ± 6·6 | 140 ± 21·4 | 105 ± 18·6 |
| Potassium (mM) | 5·93 ± 1·50 | 5·57 ± 0·61 | 8·62 ± 3·43 |
| Calcium (mM) | 2·83 ± 0·23 | 0·71 ± 0·50 | 11·9 ± 9·3 |
| Magnesium (mM) | 0·57 ± 0·26 | 0·10 ± 0·04 | 3·48 ± 3·70 |
| Zinc (μM) | 3·89 ± 3·90 | 19·3 ± 8·9 | 18·3 ± 13·8 |
| Copper (μM) | 1·10 ± 0·41 | 0·67 ± 0·66 | 5·43 ± 5·50 |

[a]From patients with biliary drains but stable liver function.
[b]From patients with pancreatic drains for chronic pancreatitis ($n$ = 3) and pancreatic ampullary tumour ($n$ = 2).
[c]From ileostomy patients without small bowel disease and with stable small bowel function.
Analyses were undertaken as before (Powell *et al.*, 1992).

gradually neutral more distally. Such values are approximate and vary between individuals. Gastric pH can profoundly affect the solubility, and hence absorption, of some minerals from the diet, such as iron (Champagne, 1989).

## Endogenous secretions

The endogenous secretions of the bowel include saliva, gastric and pancreatic juices, bile, and the succus entericus, which is the gut mucosal secretion. Secretion into the bowel is continuous but increases in response to various stimuli. Numerous chemical constituents have been investigated in gut secretions (Lentner, 1981), but the majority of their dry weight is attributable to proteins and glycoproteins, including mucins. Specific endogenous chelators for metals such as zinc and iron have been proposed in the gut fluids (e.g. Smith *et al.*, 1969), but none definitively found.

Saliva is produced by the salivary glands in the oral cavity. The daily output is 0·5–1·5 litres, and is affected by age, sex, disease, time of day and nutritional status. Gastric juice is often contaminated by saliva and refluxed small bowel contents. Its daily output is 2–3 litres per day but may vary in disease, such as peptic ulceration; it is regulated by the interaction of nervous and hormonal stimuli. Pancreatic juice, which is also controlled by hormonal and nervous factors, has limited secretion under basal conditions, but a copious flow rapidly follows the ingestion of a meal, lasting for about three hours. Daily output is, therefore, variable but about 0·7–2·5 litres. Bile is produced by the liver and stored in the gall bladder where water and electrolytes are absorbed and mucopolysaccharides secreted. Bile flow from the gall bladder is dependent on the action of the hormone, cholecystokinin, with some input from the vagal nerve. Bile secreted into the small bowel is therefore a combination of hepatic and gallbladder bile and its daily output is about 0·6 litres. Succus entericus is the intestinal juice of the fasting state and represents an equilibrium between ingoing (absorption) and outgoing (exsorption) fluid in the intestine. The flow varies down the bowel, being 2 ml/min in the jejenum and 0·7ml/min in the ileum. Appreciable quantities of serum proteins are secreted in this intestinal juice.

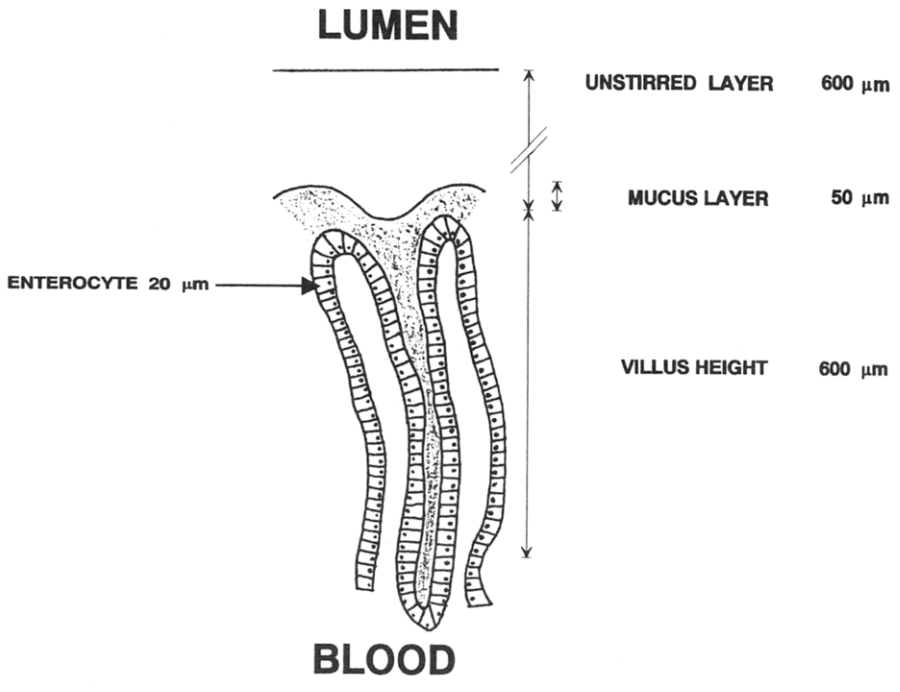
## Mucus

Mucus is produced and released mainly by the goblet cells throughout the gastrointestinal mucosa, and has two separate phases (Hunter *et al.*, 1989), namely soluble luminal mucus and an insoluble mucus gel layer adherent to the mucosal surface. Luminal mucus includes degraded mucin and is quantitatively unrelated to the amount of mucus gel adherent to the mucosal surface (Hunter *et al.*, 1989). In the antrum of the human stomach mucus has a median thickness of 180 μm (Hunter *et al.*, 1989) and about 50 μm in the small bowel.

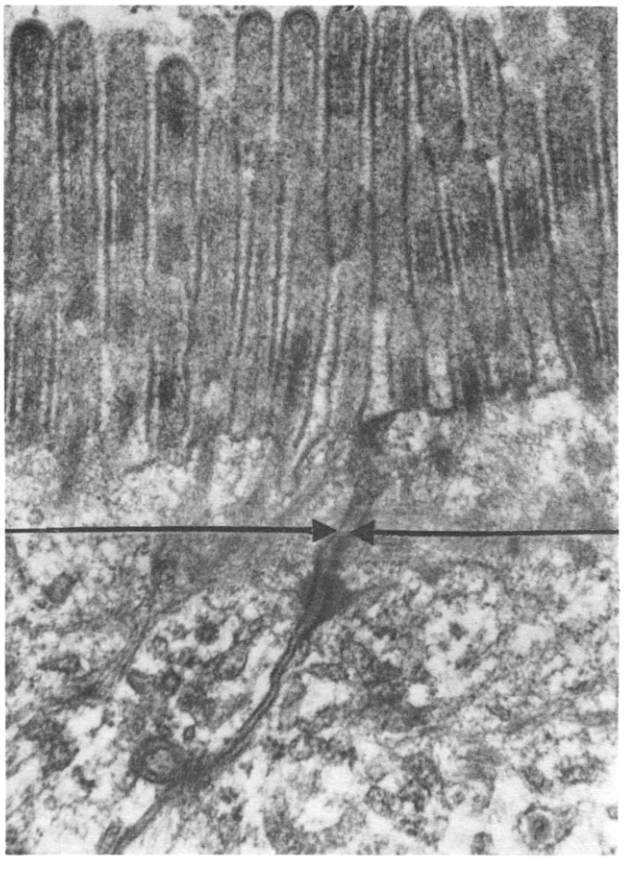
Throughout the bowel this layer is a defence barrier and probably also a transport medium (Guth & Engelhardt, 1989). Mucus is made up of large, heavily glycosylated proteins (mucins) that have molecular weights of 2–20 million Daltons and are biochemically distinct from mucopolysaccharides (Rhodes, 1989). The mucin molecules have a protein core with oligosaccharide side-chains, always oxygen linked by *N*-acetyl galactosamine on to serine or threonine (Rhodes, 1989). Further sugars attach to the galactosamine, such as *N*-acetyl neuraminic acid (sialic acid), fructose, galactose or *N*-acetyl glucosamine. The mean chain length is about 15 sugar residues and there is considerable variation in their linkage giving great heterogeneity to the oligosaccharide side-chain (Rhodes, 1989). Mucus is a complex secretion, and although it is largely made up of the mucus glycoproteins, some non-mucin components have been identified in both human gastric mucus and bronchial secretions, including polysaccharides, lipid, secretory IgA and lactoferrin (Clamp & Creeth, 1984).

## Transit times

Food in the stomach dramatically reduces the rate of gastric emptying. Thus, the mean gastric residence time




(a)



(b)

**Fig. 1.** During digestion, luminal contents are propelled towards villi of the small bowel mucosa (a) and first encounter an unstirred water layer of 40–600 $\mu$m functional thickness and then a mucus layer (shaded area) of approximately 50 $\mu$m thickness. Absorption of nutrients may be through the membrane of the small bowel absorptive cells (enterocytes) or between junctional complexes of adjacent cells. (b) An electron micrograph (×40,000) of adjacent areas of two enterocytes. The dividing junctional complex is apparent (arrows), through which paracellular permeation may occur. However, the short length of junctional complex immediately below the free surface is, as above, tightly bound and almost impermeable under normal conditions and is termed the tight junction or zonula occludens. The surface of each cell is composed of a 'brush border' of long microvilli, between and over which are luminally protruding membrance surface proteins (glycocalyx or fuzzy coat) and mucus. The cell surface may also surround materials and then invaginate; thus some minerals could be internalised by this process of endocytosis.

of fasting individuals is increased from 0·5 to more than 14·5 h by frequent intake of food (Davis *et al.*, 1984; Mojaverian *et al.*, 1985). Intestinal transit time may be independent of the dietary state (Davis *et al.*, 1984), but the size of the species in the lumen of the bowel is important, for in several studies tablets or capsules have a median small intestinal transit time of 8 h, compared to 3–4 h for smaller species or liquids (Wilson *et al.*, 1984; Ambrecht *et al.*, 1986). It is difficult to describe the effects of transit times on absorption of minerals without implicating other factors, such as pH and diet. Nevertheless, the greater the time spent in the stomach and bowel, probably the better the opportunity of dissolving and absorbing an ingested mineral.

### Unstirred water layer and microclimate pH

The unstirred water layer of the lumen of the bowel arose as a theoretical concept, that as molecules from the thoroughly mixed central bulk phase of the intestinal lumen are propelled towards the mucosa they encounter layers of water that are progressively less mixed. Ultimately, diffusion becomes the sole means of molecular movement. There is no absolute demarcation between these layers, but the functional thickness (40-600 $\mu$m in the small bowel) can be measured in a number of tissues (Wilson & Dietschy, 1974). The unstirred water layer lies just above, and probably becomes an integral part of, the mucus layer, and it may act particularly as a rate-limiting factor in the diffusion of molecules that do not have active transport mechanisms (Wilson & Dietschy, 1974).

In a layer on the surface of the cells of the bowel mucosa is a pH microclimate; significantly different from the pH of the lumen and maintained more constant (Rechkemmer *et al.*, 1986). It is also not exactly defined, but is probably an integral part of the mucus layer on the gut mucosa. It is measurable by lowering a micro-electrode onto the area of gut tissue until a depression is seen (Lucas & Blair, 1978). Mechanisms for control of the microclimate are not understood, although the mucus–bicarbonate barrier plays a major role in the stomach and probably the intestine (Rechkemmer *et al.*, 1986). The microclimate was considered to be acidic (Lucas & Blair, 1978) compared to the lumen, but more recent work has not supported this (Rechkemmer *et al.*, 1986), perhaps partly due to differences in electrodes or due to measurements made *in vitro* and *in vivo*. Furthermore, differences between species are apparent; thus using the same *in-vitro* methods, the microclimate pH of the proximal jejenum in man was 5·93 ± 0·5 (mean ± SD) and in the rat 6·48 ± 0·09 (Lucas & Blair, 1978). From different *in-vivo* studies in the rat, the following microclimate pH values have been recorded: stomach 6·68 ± 0·71 (Ross *et al.*, 1981); proximal jejenum 6·1 ± 0·1 (Lucas, 1983) or 7·16 ± 0·11 (Rechkemmer *et al.*, 1986); distal ileum 7·05 ± 0·05 (Lucas, 1983). The intra-study variation suggests a tight control of this microclimate. Factors affecting this microclimate include reduced blood flow and ingestion of metabolisable sugars (Daniel & Rehner, 1986), both of which reduce the pH. Under certain circumstances, this microclimate pH may be more important than luminal pH, as shown for the absorption of short-chain fatty acids (Engelhardt & Rechkemmer, 1984). Effects of microclimate pH on mineral absorption are not known, but since pH affects both charge and relative stability of metal–ligand complexes, then this should be further considered.

## FATE OF MINERALS IN THE GASTROINTESTINAL TRACT

The fate of dietary aluminium as it traverses the gastrointestinal tract was discussed in a recent review (Powell & Thompson, 1993). The mechanisms involved are common to many ingested cations. Briefly, some solubilisation occurs in the acid environment of the stomach, the extent of which will depend on the gastric pH, the element and its ingested form. Following gastric emptying into the more neutral environment of the small bowel there is a strong tendency for many cations, such as aluminium, copper, iron and zinc, to hydrolyse and form polymeric hydroxy-ion species. However, it should be stressed that although no specific metal chelators have been found in the bowel secretions, many inorganic and organic molecules with ligand properties are endogenously secreted into the lumen. Such ligands include albumin (Oppenheim, 1970; Clemente *et al.*, 1971), citrate (Piper *et al.*, 1967), lactate (Piper *et al.*, 1967; Powell *et al.*, 1990), lactoferrin (Dipaola & Mandel, 1980; Nicolai *et al.*, 1984), phosphate (Powell & Thompson, 1993), pyruvate (Piper *et al.*, 1967) and, most importantly, soluble mucins (Powell & Thompson, 1993). The luminal chemistry of dietary elements remains poorly studied, but it is likely that these ligands interfere significantly with metal ion hydrolysis and so prevent gross precipitation. The resulting hydroxy–metal–ligand complex will be more strongly dissociative than the metal–hydroxide, so that, even if polymerisation occurs, a labile and potentially available form of the metal is still present in the gut lumen. This has been well shown for the hydroxy–ferric–fructose system (Bates *et al.*, 1972) and also explains why strongly hydrolytic metals such as copper and zinc can still be well absorbed. In addition to this mechanism, mucus appears to play an important role. Firstly, soluble mucus may bind and stabilise the growth of hydroxy–metal polymers, so taking the role of endogenous ligand. Thus 'gastroferrin', which is probably gastrically degraded mucin, stabilises the formation of iron or chromium colloids at neutral pH values (Rudzki *et al.*, 1973). Secondly, the mucosally adherent layer of mucus has a large capacity for uptake of metals, probably by direct binding with metal ions that have remained unpolymerised and also by interaction with the hydroxy–metal–ligand polymers. The mucus layer may therefore partly regulate metal absorption, such that strongly bound and kinetically

slow elements (e.g. aluminium) traverse the layer slowly towards the mucosa, compared to poorly bound and labile elements (e.g. calcium).

At the brush border (Fig. 1(b)) of the enterocyte (absorptive cell), a number of mechanisms allow the uptake of minerals. These are illustrated in Fig. 1 and typically include facilitated and non-facilitated transcellular diffusion, paracellular permeation through the junctional complex, and endocytosis. They are not mutually exclusive and often all occur for the same element. The predominant type of absorption, however, may depend on the mineral species presented to the mucosa, although if the mineral complex is rapidly dissociative then speciation is of less importance. Some minerals, such as aluminium and bismuth, do not undergo facilitated (active) absorptive processes and partly for this reason are poorly absorbed, largely by paracellular permeation (Powell & Thompson, 1993). The ingested form of the mineral, binding to mucus, kinetics of metal–ligand exchange processes, and for hydrolytic ions the dissociative capacity of hydroxy–mineral–ligand polymers, are also all important determinants of the final mucosal uptake. Finally, the transport of minerals from the bowel mucosa into the blood often depends on a number of regulatory intracellular and systemic factors. These are particularly important for the regulation of essential elements, for example, calcium (vitamin D status), iron (intra-enterocyte transferrin/iron-ferritin stores), and copper and zinc (intra-enterocyte metallothionein).

## INFLUENCE OF EXOGENOUS FACTORS ON MINERAL ABSORPTION

The absorption of non-essential metals from the gastrointestinal tract may be either promoted or inhibited by concomitant ingestion of foodstuffs. In contrast, essential metals may have their absorption decreased, but rarely increased, mainly because *in vivo*, the regulatory mucosal factors outlined above are overriding. Such effects on mineral absorption are either due to competitive binding or mucosal alterations.

### Competitive binding

The normal processes of mineral absorption involve (hydroxy)–metal–ligand interactions, mucus binding and mucosal uptake. These are all competitive processes, so metal ions may compete for the same binding site, and binding molecules for the same metal ion. Thus, in absorption experiments, indirect metal-metal interactions are commonly observed. There is some evidence for the stimulation of trace element absorption by the macro elements, such as calcium and magnesium (Akedalu & Heaton, 1992), but competitive inhibition is more usual, particularly from chemically similar elements (Flanagan, 1989). The prior administration of one element, or restricting certain essential elements may also have a subsequent effect on the absorption of other metals, due to inhibition or upregulation of systemic and/or mucosal regulatory factors. However the ubiquity of dietary elements and their presence in endogenous secretions, means that under normal dietary conditions in the western world, metal-metal interactions are continuous and physiological; such processes have been reviewed elsewhere (Flanagan, 1989).

The effects on mineral absorption of specific dietary ligands have also been studied, such as polyphenols, phytate and citrate. It is often considered that high-molecular-weight ligands (> 500 Da) reduce absorption of minerals, whereas low-molecular-weight ligands increase absorption and more so if an uncharged metal–ligand complex is formed. However, first, the lability of the new complex needs to be considered. Secondly, there is a negatively charged mucus layer to traverse, which is orders of magnitude thicker than the lipophilic absorptive cell membrane, thirdly there are regulatory mucosal factors for the essential elements and fourthly, the ligand may alter mucosal permeability (below). For example, the low-molecular-weight citrate ligand markedly increases absorption of aluminium (Powell & Thompson, 1993) but decreases that of calcium (Rumenapf & Schwille, 1987), demonstrating the complexity of such ligand effects. Furthermore, ligand interactions need consideration under suitable dietary conditions, which often are not reflected by animal work or single human dosing experiments. Polyphenols in tea are such an example.

Polyphenols are present in a number of plant foods (Sanderson, 1972; Mehanso *et al.*, 1987; Managan, 1988) and in the Western world exposure to man is mainly through tea drinking, since polyphenols account for approximately 30% dry weight of freshly picked tea (Sanderson, 1972). Polyphenols are potent metal binders facilitated largely by the galloyl sub-structure (Brune *et al.*, 1989) and favour interactions with $M^{3+}$ (e.g. $Fe^{3+}$, $Al^{3+}$). Thus, it has been found with challenge studies that tea drinking may markedly reduce the absorption of iron (Disler *et al.*, 1975; Reddy & Cook, 1991) due to luminal complexation of the metal by large non-absorbable polyphenols. Rodent models for this should be treated with caution since tea-induced inhibition of iron uptake could not be reproduced in adult rats (Reddy & Cook, 1991) but has been reported in suckling or weanling rats (Fairweather-Trait *et al.*, 1991). Furthermore, in man or immature rodents these data should not be over-interpreted. First, cooked haem iron which is a small but readily available source of dietary iron, is unaffected by the co-ingestion of polyphenols from tea (Disler *et al.*, 1975). Secondly, the complex mixture of promoters and inhibitors of iron absorption, within the typical Western diet have an 'averaging' effect on iron availability (Cook *et al.*, 1991). Thirdly, the capacity to absorb iron increases with decreasing iron status. Thus ligand effects on mineral availability are exaggerated from such single dosing experiments, and only under extreme circumstances is tea likely to affect significantly dietary iron availability. Outside of the Western world, diet may have a marked

effect on iron availability, which is due, not to beverage ingestion, but to less varied diets, such as those high in phytate (Siegenberg *et al.*, 1991) or polyphenol-containing vegetables (Tuntawiroon *et al.*, 1991).

Brewed tea contains 2–6 mg litre$^{-1}$ aluminium making this a major dietary source of the metal (Owen *et al.*, 1992). Although this aluminium is partly bound to the non-absorbable polyphenols (Baxter *et al.*, 1989), increased urinary excretion of aluminium was reported after tea drinking (Koch *et al.*, 1988). This is surprising, since aluminium is so poorly absorbed (Powell & Thompson, 1993) and polyphenols are expected to further reduce $M^{3+}$ absorption. Furthermore, work with weanling rats could not demonstrate increased aluminium absorption with tea drinking, and two recent *in-vitro* studies showed that only a small percentage of aluminium from tea was potentially available (Owen *et al.*, 1992; Powell *et al.*, 1993). The paradox is partly explained by the misuse of urinary analysis for aluminium absorption (Koch *et al.*, 1988), without accounting for the increase in urinary output with tea drinking (Powell *et al.*, 1993). Thus, whether aluminium ingested in tea is at all available in the context of a varied Western diet remains to be established.

Hence the effects of luminal complexation on ingested minerals must be considered with respect to the normal physiological situation, in the context of a typical diet and using suitable models for assessment.

## Mucosal alterations

The intestinal mucosa, including mucus layer, microclimate pH and unstirred water layer, is subject to permeability changes following exposure to certain chemicals. For example ingestion of metabolisable sugars such as glucose markedly alters microclimate pH (Daniel & Rehner, 1986), although this effect on mineral absorption remains unexamined. Glucose may also increase permeability of the mucosal tight junction, which is a part of the junctional complex between intestinal cells (Fig. 1). The tight junction sub-structure probably consists of high-tensile protein strands that pass from individual, and between adjacent mucosal cells (Sanderson & Walker, 1993). Sodium-coupled solute transport (e.g. glucose and some amino acids) triggers contraction of cytoskeletal elements of the enterocyte, inducing condensation of these intercellular protein microfilaments and, in the rat, this opens the tight junction (Sanderson & Walker, 1993). The calculated effective pore size of the opened tight junction is 5 nm (normally 0·5–1 nm in man) allowing for example the paracellular passage of an eleven-amino-acid-containing polypeptide (Sanderson & Walker, 1993). However, this effect has not been shown in man nor examined with respect to mineral absorption, although elements largely absorbed through the paracellular route (e.g. aluminium and bismuth) could be investigated. Indeed, a definite method of opening the tight junction structure does increase aluminium absorption some 10- to 50-fold (Powell & Thompson, 1993). Certain ligands, such as citrate and EDTA, can effectively chelate intercellular calcium, which in addition to the high-tensile protein strands, is required for integrity of the tight junction structure. Thus proximal small bowel tissue treated with citrate or EDTA shows a prolonged reduction in electrical resistance, which is indicative of a significant fall in tight-junction integrity (Froment *et al.*, 1989). A more empirical study demonstrated that ruthenium red, which is normally an impermeable dye, can traverse the bowel tissue following pre-treatment with citrate (Froment *et al.*, 1989). Transmission electron micrographs showed that this is paracellular movement through tight junctions, rendered permeable by citrate. In this case the tight junctions surrounding goblet cells (mucus-secreting cells) appeared most susceptible.

Citrate is a common food component, that, owing to its large effect on aluminium absorption, has received much attention (Powell & Thompson 1993). Surprisingly, despite an increase this century in consumption of citrus products in Britain, total naturally occurring citrate intake has not increased (Table 2). This is largely due to a corresponding decrease in the intake of potatoes (Table 2), which even now account for one-quarter of naturally occurring citrate in a detailed estimate of the British diet (Table 3). In addition, sodium citrate and citric acid as the food additive acidulants E371 and E330 are added to a typical British diet at 1·1g/person/day, when based on a *per capita* intake (MAFF,1993). These are ubiquitous additives in hundreds of consumables and in soft drinks may be present, as consumed, at several grams/litre.

Thus, the significance for mineral absorption of some

**Table 2. Trends in naturally occurring citric acid content of the British diet**

| | Estimated intake of citric acid (g/day) and main contributors to intake (%) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Food | 1909–13 | 1924–28 | 1942 | 1952 | 1962 | 1972 | 1982 | 1992 |
| Milk, cream and cheese | 24 | 24 | 28 | 33 | 36 | 36 | 32 | 28 |
| Potatoes | 50 | 49 | 45 | 39 | 33 | 31 | 28 | 25 |
| Citrus fruit | 0 | 0 | 2 | 4 | 5 | 6 | 6 | 6 |
| Fruit juice | 0 | 0 | 0 | 0 | 1 | 2 | 8 | 14 |
| All other foods[a] | 26 | 27 | 26 | 23 | 24 | 25 | 26 | 26 |
| Total intake (g/day) | 2·5 | 2·4 | 2·5 | 2·8 | 2·6 | 2·6 | 2·5 | 2·5 |

[a]Includes sugars and preserves, other fruit and vegetables, cereal products, soft drinks, confectionery and alcoholic drinks.
Source: MAFF estimates.

**Table 3. Naturally occurring citric acid content of the British diet, 1992**

| Food | Estimated citric acid intake (mg/day) | % of intake |
|---|---|---|
| All milk, cream and cheese | 700 | 28 |
| Total sugars and preserves | 30 | 1 |
| Total vegetables and products of which | 910 | 36 |
| — potatoes and products | 630 | 25 |
| — fresh green vegetables | 100 | 4 |
| — tomatoes | 90 | 4 |
| Total fruit and products of which | 640 | 26 |
| — citrus fruit | 160 | 6 |
| — fruit juice | 360 | 14 |
| Total cereals | 60 | 2 |
| Soft drinks | 110 | 4 |
| Confectionery | 0 | 0 |
| Alcoholic drinks | 50 | 2 |
| Total | 2500 | 100 |

Source: MAFF estimates.

high-glucose and/or high-citrate drinks (e.g. Lucozade) remains to be established. Again, in the context of normal varied Western diets such anticipated effects of increased paracellular permeability may be reduced. Finally it is possible that this route could be exploited to increase absorption of essential minerals (e.g. iron) since intracellular regulation is bypassed.

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