Enteral feeding: the effect on faecal output, the faecal microflora and SCFA concentrations

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Enteral tube feeding is common in both the hospital and community environment; however, patients can suffer alterations in faecal output that can have serious clinical sequelae. Problems associated with accurate characterisation of faecal output and definition of diarrhoea impede the comparison of research studies and prevent standardised assessment of therapeutic interventions in clinical practice. The colonic microflora may protect the patient against diarrhoea by preventing enteropathogenic infection and by producing SCFA that stimulate colonic water absorption. However, studies in healthy volunteers suggest that the composition of the enteral formula may have a negative impact on the microflora and SCFA concentrations. The addition of fructo-oligosaccharides to the enteral formula may partially prevent negative alterations to the microflora, although conclusive data from studies in patients are not yet available. Modification of the microflora with probiotics and prebiotics may hold potential in prophylaxis against diarrhoea during enteral tube feeding.

Enteral feeding

Enteral tube feeding (ETF) is a common method of nutritional support for patients who are unable to achieve their nutritional requirements through oral diet alone. Approximately 5% of the patients on general wards (Bowling, 1995), 46% of the patients on the intensive therapy unit (ITU; Hill et al. 1995) and ≤25 000 patients in the community (Elia, 2003) receive ETF at any one time. Despite the wide use of ETF, alterations in faecal output occur and diarrhoea is a common complication with a variety of negative clinical sequelae. Diarrhoea in patients on the ITU results in a reduction in the efficacy of ETF, patients receive less enteral formula, ETF may be stopped or the patient transferred to parenteral nutrition, and patients have an increased length of stay on the ITU (Montejo, 1999). The increased risk of faecal incontinence associated with diarrhoea (Bliss et al. 2000) is not only distressing for the patient and carer, but also increases the risk of infection of surgical or pressure wounds. Fluid and electrolyte abnormalities are common and the prescription of anti-diarrhoeal medication may be required (Bowling, 1995). These factors are likely to cause further distress to the patient and result in greater financial cost to the healthcare provider (Dobb, 1986). The aim of the present review is to discuss the validity and reliability of characterising faecal output and diarrhoea during ETF, the role of the colonic microflora and SCFA in its pathogenesis and the effect of the composition of the enteral formula on these characteristics.

Reporting diarrhoea during enteral tube feeding

The reported incidence of diarrhoea in patients receiving ETF ranges from 2% (Cataldi-Betcher et al. 1983) to 95% (DeMeo et al. 1998). This wide range of reported incidences is due to two main factors. First, since the pathogenesis of diarrhoea includes a number of clinical variables, the reported incidence is greatly influenced by the disease state of the patient population under investigation. For example, for patients receiving ETF those on the ITU are at greater risk of diarrhoea than those not on the ITU (Pesola et al. 1989). Second, there is no

Abbreviations: ETF, enteral tube feeding; FOS, fructo-oligosaccharides; ITU, intensive therapy unit.
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Characterising faecal output during enteral tube feeding

A variety of methods are available for the characterisation of faecal output during ETF. Whilst faecal frequency is easy to record, the accurate characterisation of faecal consistency and quantity requires experimental methods.

The experimental methods available to characterise faecal consistency include penetrometry, viscometry and lyophilisation. Penetrometry measures the penetration of a standard cone into a faecal sample (Exton-Smith et al. 1974) and data have been shown to correlate with faecal water content (Nakaji et al. 2002). Viscometry measures the force required to rotate a standard spindle in a faecal sample and data have been shown to correlate with subjective descriptors of faecal consistency (Wenzl et al. 1995). Lyophilisation of a faecal sample allows calculation of the percentage water content, a major predictor of faecal consistency (Wenzl et al. 1995).

However, each of these experimental methods has several disadvantages. Penetrometry cannot be conducted on small or liquid samples (Davies et al. 1986), viscometry cannot be conducted on formed faeces (Wenzl et al. 1995) and complete lyophilisation of a faecal sample can take up to 3 d (Bliss et al. 1999). Meanwhile, experimental characterisation of faecal weight requires the collection of the entire sample without contamination by urine and often requires the use of a bedpan or commode, or the weighing of incontinence pads (Benya et al. 1991). Most importantly, the intensive nature of these experimental methods precludes their routine use in the research or clinical setting.

In view of the impractical nature of experimental methods, nurses routinely characterise faecal output visually. The reliability of visual characterisation of faecal consistency has been measured in a number of studies. The reported agreement beyond chance between nurses visually characterising faecal consistency ranges from fair (k 0.25; Allen et al. 1994) to substantial (k 0.68; Bliss et al. 2001), although none of these patients was receiving ETF (Landis & Koch, 1977). The validity of visual characterisation of faecal weight has been measured in a simulated study in which thirty-four nurses estimated the weight of common patient inputs and outputs (Daffurn et al. 1994). The mean estimated weight of 100 g faeces in a bedpan was 135 (sd 59) g, indicating a systematic overestimation of actual faecal weight.

Experimental methods of characterising faecal output are accurate but impractical, whereas visual characterisation is practical but inaccurate. This situation presents problems for monitoring faecal output in both the research and clinical setting. A variety of charts have been developed to improve visual characterisation of faecal output. The Bristol Stool Chart (Lewis & Heaton, 1997) incorporates verbal and pictorial descriptors of faecal consistency; however, it does not include other important faecal characteristics and has not been validated in patients receiving ETF. A chart has been developed recently that incorporates verbal and pictorial descriptors of faecal frequency, consistency and quantity to assist in the accurate visual characterisation of faecal output in patients.
receiving ETF (Fig. 1; Whelan et al. 2001b). The chart includes four categories for faecal consistency: hard and formed; soft and formed; loose and unformed; liquid; and three categories for faecal weight: <100 g; between 100 and 200 g; >200 g. Faecal frequency is incorporated by recording each time that faeces are voided. The chart has been validated in a questionnaire survey of health professionals and a clinical study of thirty-six patients starting ETF (Whelan et al. 2001b). When two different nurses used the chart to independently characterise fifty-nine faecal samples, the agreement beyond chance was almost perfect for faecal consistency (95%; \(\kappa = 0.91\)) and substantial for faecal weight (83%; \(\kappa = 0.75\)). In addition, 83% of faecal samples were assigned to the correct faecal-weight category (\(\kappa = 0.75\)). The chart can assist in the accurate visual characterisation of faecal output in the research and clinical setting.

**Pathogenesis of diarrhoea during enteral tube feeding**

The pathogenesis of diarrhoea in patients receiving ETF has been extensively investigated and a number of mechanisms have been identified. First, microbial contamination of the enteral formula with >10^5 colony-forming units/ml is associated with diarrhoea (Okuma et al. 2000) and can occur due to non-sterile handling of the feeding system (Beattie & Anderton, 1999). Second, the risk of infection with *Clostridium difficile*, a Gram-positive enteropathogen, which can cause diarrhoea, pseudomembranous colitis and fulminant colitis (Mylonakis et al. 2001), is threefold greater in patients receiving ETF than matched patients not receiving ETF (Bliss et al. 1998). Third, intragastric infusion of enteral formula stimulates an abnormal secretion of fluid into the lumen of the ascending colon (Bowling et al. 1994). Fourth, the prescription of antibiotics increases the risk of diarrhoea (Keohane et al. 1984; Guenter et al. 1991) as a result of their deleterious effect on the colonic microflora (Levy, 2000). Several other mechanisms for diarrhoea during ETF have been established, including drug therapy and hypoalbuminaemia, and have been thoroughly reviewed elsewhere (Bowling, 1995). However, the role of the colonic microflora remains an area of increasing interest.
Colonic microflora

The gastrointestinal tract contains a complex microbial ecosystem of >500 different species of bacteria. The numbers and type of bacteria vary between the sections, with $10^7$ colony-forming units/g stomach contents, rising to $10^{12}$ colony-forming units/g in the colon (Steer et al. 2000). The indigenous microflora can inhibit the growth of enteropathogens in the colonic lumen by competing for nutrients and space and by producing antimicrobials (Gibson & Wang, 1994; Lievin et al. 2000) and acids (Wang & Gibson, 1993), a process termed colonisation inhibition. The microflora can also inhibit adhesion of enteropathogens to the colonoocytes via steric and chemical hindrance of receptor sites (Bernet-Camard et al. 1997), a process termed competitive exclusion.

The colonic microflora may be able to prevent enteropathogenic infection through their potential to exert colonisation inhibition and competitive exclusion. In patients receiving ETF the increased risk of enteropathogenic infection may therefore result from negative alterations to the microflora, predisposing the patient to diarrhoea. In view of the involvement of enteropathogens in the pathogenesis of diarrhoea during ETF, the effect of consumption of enteral formulas on the faecal microflora has been investigated in a series of studies in healthy subjects (Table 1).

The results of these studies are conflicting, with some demonstrating large reductions (Winitz et al. 1970) and some demonstrating no significant changes (Attebery et al. 1972; Crowther et al. 1973; Bounous & Devroede, 1974; Bornside & Cohn, 1975) to the major genera of the faecal microflora when healthy subjects change from normal diet to a fibre-free ‘low-residue’ enteral formula. These earlier studies have major methodological weaknesses, including sample sizes that were so small they precluded statistical analysis (Winitz et al. 1970; Attebery et al. 1972; Crowther et al. 1973). Furthermore, the aim of these studies was often to investigate the efficacy of low-residue enteral formulas for preoperative bowel preparation. Consequently, subjects often received an enema (Winitz et al. 1970) or laxatives (Bornside & Cohn, 1975), or both (Attebery et al. 1972) before starting consumption of the enteral formula, a procedure that does not accurately model ETF.

Despite their major limitations, these early experiments indicated that the carbohydrate source in the enteral formula might affect the extent to which alterations occur to the microflora. Winitz et al. (1970) showed that replacing glucose in enteral formulas with sucrose prevented a decrease in the concentrations of coliforms and bacteroides. Indeed, some carbohydrates may even be able to stimulate the growth of beneficial bacterial groups, a phenomenon termed the ‘prebiotic concept’. A prebiotic is defined as a ‘non-digestible food ingredient that beneficially affects the host by selectively stimulating the growth and/or activity of one or a limited number of bacteria in the colon, and thus improving host health’ (Gibson & Roberfroid, 1995). The most extensively studied prebiotics are fructo-oligosaccharides (FOS), which are polymers of glucose and fructose monomers in varying ratios. FOS are found naturally in artichokes, chicory, leeks, asparagus, onions

<table>
<thead>
<tr>
<th>Reference</th>
<th>ETF formula</th>
<th>Sample size</th>
<th>Consumption period (d)</th>
<th>Effect on major genera of faecal microflora</th>
</tr>
</thead>
<tbody>
<tr>
<td>Winitz et al. (1970)</td>
<td>Elemental (glucose)</td>
<td>4</td>
<td>13</td>
<td>Decrease: Total bacteria, bacteroides, clostridia, coliforms, enterococci, lactobacilli</td>
</tr>
<tr>
<td></td>
<td>Elemental (glucose)</td>
<td>8</td>
<td>4</td>
<td>Decrease: Total bacteria, bacteroides, clostridia, coliforms, enterococci, lactobacilli</td>
</tr>
<tr>
<td></td>
<td>Elemental (sucrose)</td>
<td>8</td>
<td>4</td>
<td>Decrease: Total bacteria, clostridia, enterococci, lactobacilli</td>
</tr>
<tr>
<td>Attebery et al. (1972)</td>
<td>Elemental (glucose)</td>
<td>3</td>
<td>8–10</td>
<td>No change: Bacteroides, coliforms</td>
</tr>
<tr>
<td>Crowther et al. (1973)</td>
<td>Elemental</td>
<td>3</td>
<td>10</td>
<td>Decrease: Enterococci</td>
</tr>
<tr>
<td>Bornside &amp; Cohn (1975)</td>
<td>Elemental (dextrin)</td>
<td>10</td>
<td>7</td>
<td>No change: Total bacteria, anaerobes, aerobes, bacteroides, colistridia, bifidobacteria</td>
</tr>
<tr>
<td>Garleb et al. (1996)</td>
<td>Low residue</td>
<td>9</td>
<td>14</td>
<td>Decrease: Bifidobacteria</td>
</tr>
<tr>
<td></td>
<td>Low residue + FOS (5 g/l)</td>
<td>9</td>
<td>14</td>
<td>No change: Bifidobacteria</td>
</tr>
<tr>
<td></td>
<td>Low residue + FOS (10 g/l)</td>
<td>9</td>
<td>14</td>
<td>Increase: Bifidobacteria</td>
</tr>
<tr>
<td>Whelan et al. (2003a)</td>
<td>Low residue</td>
<td>10</td>
<td>14</td>
<td>No change: Total bacteria</td>
</tr>
<tr>
<td></td>
<td>Fibre (15 g/l) + FOS (5 g/l)</td>
<td>10</td>
<td>14</td>
<td>Decrease: Total bacteria, clostridia</td>
</tr>
</tbody>
</table>

ETF, enteral tube feeding; FOS, fructo-oligosaccharides.
and garlic (Van Loo et al. 1995). When given as a supplement to the normal diet of human subjects they have been shown to increase the concentration of faecal bifidobacteria and reduce clostridia (Gibson et al. 1995).

FOS have been added to enteral formulas for some time because of their bifidogenic capabilities and their inhibition of potentially-pathogenic bacteria (Wolf et al. 2003). Two studies have investigated the effect of fortification of enteral formulas with FOS on the faecal microflora of healthy subjects. In a prospective randomised double-blind trial consumption of a low-residue enteral formula caused a reduction in faecal bifidobacteria, whereas fortification with 5 g FOS/l prevented this reduction and fortification with 10 g FOS/l caused an increase in bifidobacteria (Garleb et al. 1996). In a prospective randomised double-blind cross-over trial fluorescent in situ hybridisation, a highly-specific nucleotide probe method (Blaut et al. 2002), was used to quantify the effect of enteral formulas on the faecal microflora of healthy subjects (Whelan et al. 2003a). Consumption of a low-residue enteral formula resulted in a 52% reduction in the concentration of total faecal bacteria, but without marked changes to the concentrations of bifidobacteria, clostridia and bacteroides. However, fortification of the formula with 15 g mixed fibre and 5 g FOS/l resulted in an increase in the concentration of bifidobacteria and a reduction in clostridia (Whelan et al. 2003a).

The results of these recent studies clearly suggest that low-residue enteral formulas cause negative alterations to the faecal microflora in healthy subjects, which can be partially prevented by fortification with FOS. However, alterations to the microflora may also affect colonic fermentation and SCFA production.

Colonic fermentation and SCFA production

The colonic microflora ferment polysaccharides, oligosaccharides, proteins, peptides and glycoproteins to produce SCFA (Macfarlane & Macfarlane, 2003). The carbohydrates account for the majority of SCFA production and reach the colon in the form of dietary NSP and oligosaccharides that are resistant to digestion in the upper gastrointestinal tract, or as disaccharides in conditions such as lactose malabsorption (Cook & Sellin, 1998). Acetate, propionate and butyrate constitute approximately 90% of the SCFA produced by the microflora, with valerate, hexanoate, isobutyrate and isovalerate being produced in smaller amounts (Cook & Sellin, 1998). In vitro studies suggest that the extent of fermentation of the carbohydrate affects not only the amount but also the pattern of SCFA production. SCFA production rates for fibres commonly used in enteral formulas were compared in vitro using faecal inoculum from healthy subjects (Kapadia et al. 1995). Poorly-fermentable oat husk did not result in a change in the production of total SCFA, acetate, propionate or butyrate, whereas highly-fermentable soybean oligosaccharide increased their production (Kapadia et al. 1995). Approximately 95% of the SCFA produced in the colon are then absorbed and metabolised in the colonocytes, liver, muscle and brain tissue (Topping & Clifton, 2001). SCFA absorption occurs by diffusion, anion-exchange or carrier-mediated exchange, resulting in the concomitant absorption of water and Na (Tyagi et al. 2002). Interestingly, the abnormal secretion of fluid into the colon of subjects receiving ETF is reversed during an infusion of SCFA into the caecum (Bowling et al. 1993).

In view of the ability of SCFA to stimulate colonic water absorption during ETF, the effect on SCFA production of changing from a normal diet to consuming enteral formula as the only source of nutrition has been investigated in three experiments in healthy subjects (Table 2). Low-residue enteral formula caused a reduction in the concentration of total faecal SCFA in two studies (Zimmaro et al. 1989; Whelan et al. 2003a), a reduction in acetate and propionate in one study (Whelan et al. 2003a) and a reduction in butyrate in all three studies (Zimmaro et al. 1989; Lampe et al. 1992; Whelan et al. 2003a). Fortification of the formula with a fibre source still caused a reduction in butyrate (Lampe et al. 1992; Whelan et al. 2003a).

### Table 2. Published studies investigating the effect of changing from a normal diet to enteral formula as the only source of nutrition on SCFA concentrations in healthy human subjects

<table>
<thead>
<tr>
<th>Reference</th>
<th>ETF formula</th>
<th>Sample size</th>
<th>Consumption period (d)</th>
<th>Effect on SCFA concentrations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zimmaro et al. (1989)</td>
<td>Low residue</td>
<td>13</td>
<td>8</td>
<td>Decrease: Total SCFA, butyrate</td>
</tr>
<tr>
<td>Lampe et al. (1992)</td>
<td>Low residue</td>
<td>11</td>
<td>18</td>
<td>Decrease: Butyrate</td>
</tr>
<tr>
<td></td>
<td>Fibre (15 g/d modified guar)</td>
<td>11</td>
<td>18</td>
<td>No change: Total SCFA, acetate, propionate</td>
</tr>
<tr>
<td></td>
<td>Fibre (15 g/d soya polysaccharide)</td>
<td>11</td>
<td>18</td>
<td>Decrease: Butyrate</td>
</tr>
<tr>
<td>Whelan et al. (2003a)</td>
<td>Low residue</td>
<td>10</td>
<td>14</td>
<td>Decrease: Total SCFA, acetate, propionate, butyrate</td>
</tr>
<tr>
<td></td>
<td>Fibre (15 g/l mixed) + FOS (5 g/l)</td>
<td>10</td>
<td>14</td>
<td>No change: Isobutyrate, valerate, isovalerate, butyrate</td>
</tr>
</tbody>
</table>

ETF, enteral tube feeding; FOS, fructo-oligosaccharides.
Whelan et al. (2003a), but in one study it also prevented
the reduction in total SCFA, acetate and propionate
(Whelan et al. 2003a). The differences in the results of
these studies may reflect the fibre source in the enteral
formula and also the sample collection method used.
Zimmaro et al. (1989) and Lampe et al. (1992) both
measured the concentration of SCFA in samples of colonic
fluid collected from orally-ingested dialysis bags; however,
the effect of colonic transit time on the absorption of
SCFA into the dialysis bags is unknown. Whelan et al.
(2003a) measured the faecal concentration of SCFA;
however, this measurement reflects only the amount
remaining in the faeces following colonic absorption.
Furthermore, colonic absorption of SCFA is higher when
transit time is longer and, since low-residue enteral for-
formula increases the transit time through the gastrointestinal
tract (Silk et al. 2001), it is possible that the reduction in
faecal SCFA reflects an increase in absorption rather than a
reduced production per se. In the future, novel stable-
isotope methods using \([1-\text{\textsuperscript{13}C}]\text{acetate\) to measure endo-
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(Poueteau et al. 2003).

Healthy subjects are frequently used in studies inves-
tigating the effect of diet on the faecal microflora and
SCFA concentrations, because they otherwise have a stable
microflora (Bornsind, 1978). However, whilst they can
accurately model the effect of the composition of the
enteral formula on the faecal microflora they are not a true
reflection of patients receiving ETF. Patients receiving
ETF frequently receive a variety of medications, such as
antibiotics, which have gross effects on the faecal micro-
flora (Levy, 2000) and SCFA concentrations (Claussen et al.
1991). Furthermore, patients frequently have comorbidities
that are known to have altered faecal microflora (such as
Crohn’s disease; Seksk et al. 2003) and SCFA concen-
trations (such as patients following surgery on the gastro-
intestinal tract; Scheppach et al. 1989). Age is also known
to influence the faecal microflora (Hopkins et al. 2001), yet
healthy subjects recruited to trials of enteral formula
consumption are often much younger than patients receiv-
ing ETF. Despite the need for parallel studies in patients,
there have been only three studies that have investigated
the faecal microflora and SCFA concentrations in those
receiving ETF (Table 3).

Table 3. Published studies investigating the effect of enteral tube feeding (ETF) on the faecal microflora and SCFA concentrations of patients

<table>
<thead>
<tr>
<th>Reference</th>
<th>Design</th>
<th>Sample size</th>
<th>ETF formula</th>
<th>Effect on faecal microflora</th>
<th>Effect on SCFA concentrations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sobotka et al. (1997)</td>
<td>Prospective, cross-over trial</td>
<td>Nine patients starting ETF</td>
<td>Low residue v. 15 g FOS/l Low residue</td>
<td>Not measured</td>
<td>No change: Total SCFA, acetate, propionate, butyrate</td>
</tr>
<tr>
<td>Schneider et al. (2000)</td>
<td>Cross-sectional with control</td>
<td>Eight patients on long-term ETF v. ten controls</td>
<td>Decreased: Anaerobes No difference: Total bacteria, bacteroides Increased: Aerobes</td>
<td>No change: Total SCFA, acetate, propionate, butyrate No difference: Total SCFA, acetate, propionate, butyrate</td>
<td></td>
</tr>
<tr>
<td>Nakao et al. (2002)</td>
<td>Prospective, observational</td>
<td>Twenty patients with diarrhoea on ETF 28 g soluble fibre/d</td>
<td>Decrease: Aerobes No change: Total bacteria, anaerobes</td>
<td>No change: Butyrate Increase: Total SCFA, acetate, propionate</td>
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FOS, fructo-oligosaccharides.

Faecal SCFA concentrations were measured in a prospec-
tive non-randomised cross-over trial in nine patients at
baseline, after 7 d of ETF with a low-residue formula and
after a further 7 d of ETF with an enteral formula fortified
with 15 g FOS/l (Sobotka et al. 1997). There were no signi-
ficant changes to the concentrations of total SCFA, acetate,
propionate or butyrate following either low-residue ETF or
FOS-fortified ETF. However, analysis of the individual
data shows that some patients had large increases, some
had large reductions and some had stable concentrations of
SCFA.

In a cross-sectional study Schneider et al. (2000) demon-
strated lower concentrations of faecal anaerobes and higher
concentrations of aerobes in eight patients recei-
v ing long-term low-residue ETF compared with healthy
controls, despite no differences in faecal bacteroides,
clostridia and bifidobacteria. However, the difference in
anaerobes : aerobes may be explained by the patients being
older than the healthy controls (57 years v. 30 years), a
factor known to have similar effects on the microflora
(Hopkins et al. 2001). Despite the different microbial
profile, there were no differences between patients and
healthy controls in the concentrations of total SCFA,
acetate, propionate or butyrate. However, there were large
inter-individual variations in concentrations and only small
sample sizes.

The faecal microflora and SCFA concentrations of
twenty patients with diarrhoea during ETF who were
treated with \(\leq 28\) g galactomannan were measured in
a prospective observational study (Nakao et al. 2002). Follow-
ing 4 weeks of treatment, diarrhoea had resolved in all patients and there was a reduction in the concen-
tration of faecal aerobes. However, whether the change in
microflora was a result of treatment with galacto-
mannan, the resolution of diarrhoea or the extended
period of ETF is unclear. Following treatment there
were also increases in the concentrations of faecal
SCFA, acetate and propionate. However, faecal water
content decreased with the resolution of diarrhoea and,
since the data for SCFA were expressed per g wet faeces,
the reported increase in SCFA concen-
tration was likely to be due to less-dilute faeces rather

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The faecal microflora and SCFA concentrations of
twenty patients with diarrhoea during ETF who were
treated with \(\leq 28\) g galactomannan were measured in
a prospective observational study (Nakao et al. 2002). Follow-
ing 4 weeks of treatment, diarrhoea had resolved in all patients and there was a reduction in the concen-
tration of faecal aerobes. However, whether the change in
microflora was a result of treatment with galacto-
mannan, the resolution of diarrhoea or the extended
period of ETF is unclear. Following treatment there
were also increases in the concentrations of faecal
SCFA, acetate and propionate. However, faecal water
content decreased with the resolution of diarrhoea and,
since the data for SCFA were expressed per g wet faeces,
the reported increase in SCFA concen-
tration was likely to be due to less-dilute faeces rather
than an increase in production per se (Whelan et al. 2002).

The studies of patients receiving ETF indicate a potential imbalance between faecal anaerobes and aerobes. This imbalance is likely to be associated with a variety of factors, including antibiotic prescription and alterations in faecal output. However, studies in healthy subjects indicate that the composition of the enteral formula may also be a factor. There is a need for larger longitudinal studies investigating the faecal microflora in patients receiving ETF.

Profiling the microflora of patients may elucidate factors associated with the incidence of diarrhoea. This procedure has already been undertaken in patients with *C. difficile*-associated diarrhoea that are known to have reduced microbial diversity, lower concentrations of bacteroides and bifidobacteria and higher concentrations of enterobacteria and lactobacilli (Hopkins et al. 2001). If the faecal microflora of patients receiving ETF are altered, their ability to exert colonisation inhibition and competitive exclusion may be reduced. This outcome would not only indicate a mechanism for the increased risk of *C. difficile* infection in patients receiving ETF (Bliss et al. 2000), but would also assist the prediction of patients at greatest risk of developing diarrhoea and *C. difficile*-associated diarrhoea. However, the most appropriate management strategy for patients with an altered microflora is unclear. No prospective randomised controlled trials of the use of probiotics to prevent or treat diarrhoea in patients receiving ETF have been published (Whelan et al. 2001a). An alternative strategy is to provide patients with exogenous bacteria, or ‘probiotics’. The revised definition of a probiotic is ‘a preparation of or a product containing viable, defined micro-organisms in sufficient numbers, which alter the microflora (by implantation or colonisation) in a compartment of the host and by that exert beneficial effects in this host’ (Schrezenmeir & de Vrese, 2001). Two prospective randomised controlled trials have investigated the effect of probiotic prophylaxis against diarrhoea during ETF (Whelan et al. 2001a). The use of lactobacilli did not affect the incidence of diarrhoea in forty-one patients starting ETF (Heimburger et al. 1994), whereas the use of *Saccharomyces boulardii* in 128 patients on the ITU starting ETF reduced the number of patient days with diarrhoea by 25% (Bleichner et al. 1997). Investigation of other probiotic strains and their use in different patient populations is required in order to elicit the efficacy of probiotics in preventing diarrhoea during ETF.

**Conclusion**

Diarrhoea is a common and serious complication of ETF. Problems associated with the definition of diarrhoea and the accurate characterisation of faeces have impeded the comparison of research studies and prevented the standardised assessment of therapeutic interventions in clinical practice. Microflora and SCFA in the colon may protect the patient against diarrhoea. However, studies in healthy volunteers suggest that the composition of the enteral formula may have a negative impact on the microflora and SCFA concentrations, which may be modulated by the use of prebiotic carbohydrates. Conclusive data in patients receiving ETF are not yet available.

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**References**


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