Core Text

Part 2
Bowel nosodes & Intestinal micro-ecology

Course Text & Study Resources
by
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Bacteriology of the Bowel - Normal flora

99.9% of the normal flora consists of anaerobes numbering $10^{11}/g$ of faeces. The major bacterial groups in intestinal microflora are roughly divided into the following three groups:

1) the strict anaerobic group, including *Eubacterium*, *Bacteroides*, *Fusobacterium* and *Clostridium*,
2) the anaerobic lactic acid-producing bacteria, including *Bifidobacterium*, *Lactobacillus* and *Enterococcus*,
3) facultative anaerobic bacteria, incl. *enterobacteriaceae*. $10^8/g$ of faeces.

Table 3. Normal Faecal Flora

<table>
<thead>
<tr>
<th>ALWAYS PRESENT</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Genus</strong></td>
<td><strong>Example</strong></td>
</tr>
<tr>
<td>Bacteroides</td>
<td><em>Bacteroides fragilis</em></td>
</tr>
<tr>
<td>Clostridium</td>
<td><em>Clostridium perfringens</em></td>
</tr>
<tr>
<td>Enterococcus</td>
<td><em>Enterococcus faecalis</em></td>
</tr>
<tr>
<td>Escherichia</td>
<td><em>Escherichia coli</em></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>FREQUENTLY PRESENT</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Genus</strong></td>
<td><strong>Example</strong></td>
</tr>
<tr>
<td>Aeromonas</td>
<td><em>Aeromonas hydrophila</em></td>
</tr>
<tr>
<td>Acinetobacter</td>
<td><em>Acinetobacter baumani</em></td>
</tr>
<tr>
<td>Alcaligenes</td>
<td><em>Alcaligenes faecalis</em></td>
</tr>
<tr>
<td>Bacillus</td>
<td><em>Bacillus subtilis</em></td>
</tr>
<tr>
<td>Candida</td>
<td><em>Candida albicans</em></td>
</tr>
<tr>
<td>Corynebacterium</td>
<td><em>C. pseudodiph.</em></td>
</tr>
<tr>
<td>Enterobacter</td>
<td><em>Enterobacter aerogenes</em></td>
</tr>
<tr>
<td>Hafnia</td>
<td><em>Hafnia alvei</em></td>
</tr>
<tr>
<td>Klebsiella</td>
<td><em>Klebsiella pneumoniae</em></td>
</tr>
<tr>
<td>Neisseria</td>
<td><em>Neisseria sicca</em></td>
</tr>
<tr>
<td>Peptostreptococcus</td>
<td><em>P. asacharolyticus</em></td>
</tr>
<tr>
<td>Pseudomonas</td>
<td><em>P. aeruginosa</em></td>
</tr>
<tr>
<td>Proteus</td>
<td><em>Proteus vulgaris</em></td>
</tr>
<tr>
<td>Providencia</td>
<td><em>Providencia rettgeri</em></td>
</tr>
<tr>
<td>Sachromyes</td>
<td><em>Sachromyes cerevisiae</em></td>
</tr>
<tr>
<td>Staphylococcus</td>
<td><em>S. epidermidis</em></td>
</tr>
<tr>
<td>Streptococcus</td>
<td><em>viridans group</em></td>
</tr>
</tbody>
</table>
Bacteriology of the Bowel - Enterobacteriaceae

This is a large family of organisms containing several genera - usually associated with the intestinal tract. Many members are considered to be normal flora. The characteristics of the enterobacteriaceae are as follows:

A. Gram-negative, Rods
B. Facultative
C. Can be motile with peritrichous flagella
D. Ferment glucose
E. Oxidase negative
F. Can reduce NO₃ to NO₂
G. Produce O, K, and H antigens
   O = the outer part of LPS
   K = capsule
   H = flagella

Among the enterobacteriaceae are a number of enteric pathogens:

C Escherichia coli
C Salmonella spp.
C Yersinia enterocoltica
C Shigella spp.

Some of the enterobacteriaceae are recognised as extra-enteric pathogens:

C Escherichia coli
C Klebsiella pneumoniae
C Klebsiella oxytoca
C Proteus mirabilis
C Enterobacter aerogenes
C Enterobacter cloacae
C Serratia marscens
C Salmonella
C Citrobacter
C Edwardsiella
C Morganella
C Provedencia

¹ Frequently present in the stool of healthy subject
² E. coli subtype(s) always present - wide range of pathogenicity
Bacteriology of the Bowel -

Potentially Pathogenic Microorganisms (PPMOs)

The influence of antimicrobial agents [and systems stressors?] on the concentrations of PPMOs (potentially pathogenic micro-organisms) in the bowel is determined both by their influence on the flora that provides colonisation resistance (CR) and by their direct influence on PPMOs.

M. Delmée Antibiotics

The pathogenic status of the most important enterobacteria are given below. Their corresponding bowel nosode(s) are listed. The apparent uncertainty about these is explained overleaf.

<table>
<thead>
<tr>
<th>enterobacteriaceae</th>
<th>pathogenic status</th>
<th>bowel nosode(s) (by fermentation profile)</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Shigella</em> spp.</td>
<td>EP</td>
<td>Dysenteria co.</td>
</tr>
<tr>
<td><em>Escherichia coli</em></td>
<td>EP / PPMO</td>
<td>??? Mutabile</td>
</tr>
<tr>
<td><em>Salmonella</em> spp.</td>
<td>EP</td>
<td>Gaertner ?? Mutabile</td>
</tr>
<tr>
<td><em>Klebsiella</em> spp.</td>
<td>PPMO</td>
<td>?</td>
</tr>
<tr>
<td><em>Proteus</em> spp.</td>
<td>PPMO</td>
<td>Proteus</td>
</tr>
<tr>
<td><em>Enterobacter cloacae</em></td>
<td>PPMO</td>
<td>?? Bacillus 7</td>
</tr>
<tr>
<td><em>Citrobacter</em></td>
<td>PPMO</td>
<td>? Bacillus 7</td>
</tr>
<tr>
<td><em>Edwardsiella</em> spp.</td>
<td>PPMO</td>
<td>? Morgan-p. ??? Proteus</td>
</tr>
<tr>
<td><em>Morganella</em></td>
<td>PPMO</td>
<td>Morgan-p. ? Mutabile</td>
</tr>
</tbody>
</table>

Table 4
Identification of the bowel nosodes.

Clearly, most of the information on normal bowel ecology, outlined on the previous page, was unknown when Bach and Paterson began their investigations into non-lactose fermenting bacteria. They used direct microscopy and four standard sugar-fermentation tests to distinguish between the isolated cultures.

In modern terms this methodology is inadequate on its own to properly identify the different bacterial populations. The implications of this will be discussed in the next chapter.

Table 5 below shows the fermentation characteristics of the non-lactose groups that they isolated.\(^2\) The production of acid/gas, on fermentation of glucose, lactose, saccharose and dulcitol is recorded below.

<table>
<thead>
<tr>
<th></th>
<th>18 hours</th>
<th>24-30 hours</th>
<th>24-30 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactose</td>
<td>a/g</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Glucose</td>
<td>-</td>
<td>A/G</td>
<td>A/G</td>
</tr>
<tr>
<td>Dulcitol</td>
<td>-</td>
<td>-</td>
<td>A/G</td>
</tr>
<tr>
<td>Saccharose</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>No.10 Morgan co.</td>
<td>Morg-p.</td>
<td>Morg-g.</td>
<td>Mutabile No 10</td>
</tr>
</tbody>
</table>

Table 5a.

<table>
<thead>
<tr>
<th></th>
<th>18 hours</th>
<th>24 - 30 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactose</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Glucose</td>
<td>A/G</td>
<td>A/G</td>
</tr>
<tr>
<td>Dulcitol</td>
<td>A/G</td>
<td>A/G</td>
</tr>
<tr>
<td>Saccharose</td>
<td>A/G</td>
<td>A/G</td>
</tr>
<tr>
<td>No. 7 Proteus</td>
<td>Gaertner</td>
<td>Dys. Faecalis</td>
</tr>
</tbody>
</table>

Table 5b.

The nosode was prepared by covering the pure-culture isolates with a film of sterile water for 18 hours, collecting the solution and heat-treating it in sealed tubes, and then potentising.
Limitations of Bach and Paterson’s methodology

Sugar tests with peptone water are still used to a limited extent in modern bacteriology, but the original batch of four are now considered insufficient to distinguish between pathogenic and non-pathogenic strains of bacteria (including the common bowel organism \textit{E. coli} which is known to have many thousands of biotypes.)

Modern analysts also recognise that species and biotypes merge into one another rather than forming distinct entities. Some strains of \textit{E. coli} and \textit{Shigella}, for example, are closely related antigenically and biochemically.

Definitive typing of bacteria today may involve a computerised analysis of several hundred biochemical test results and genetic ‘fingerprinting.’

If we restrict ourselves to the results of Bach and Paterson’s fermentation tests \cite{2}, we can see that there is considerable uncertainty concerning the bacteriology of the nosodes. The microscopy and naked eye appearance of the colonies, go a little way to reducing this uncertainty.

<table>
<thead>
<tr>
<th>Nosode</th>
<th>Bacteria conforming to Bach and Paterson’s test types</th>
</tr>
</thead>
<tbody>
<tr>
<td>\textit{B. Morgan}</td>
<td>Morganella morganii, Proteus mirabilis (4% sucrose +ve), Salmonella subgenus IV, Aeromonas salmonicida, Edwardsiella tarda, Escherischia blattae, Haffnia alvei (7% are lactose +ve)</td>
</tr>
<tr>
<td>\textit{B. Gaertner}</td>
<td>Salmonella paratyphi A, Salmonella subgenus I &amp; II, Salmonella cholerasuis (27% are dulcitol positive)</td>
</tr>
<tr>
<td>\textit{B. No. 7}</td>
<td>Citrobacter koseri (45% dulcitol +ve, 85% lactose +ve, 45% sucrose +ve), Enterobacter cloacae (12% dulcitol +ve, 76 lactose +ve)</td>
</tr>
<tr>
<td>\textit{B. Proteus}</td>
<td>Edwardsiella hoshinae, E. tarda biogroup 1, Obesumbacterium proteus biogroup 2, Proteus myofaciens, Proteus penneri, Proteus vulgaris biogroup 2.</td>
</tr>
<tr>
<td>\textit{B. Mutabile}</td>
<td>Morganella morganii (lactose +ve 3%), Salmonella subgenus III (lactose 33% +ve), Salmonella subgenus III (lactose +ve 33%), Salmonella subgenus IV (lactose -ve)</td>
</tr>
<tr>
<td>\textit{B. Dysenteriae}</td>
<td>Shigella dysenteriae, Shigella flexneri, Shigella boydii, Salmonella gallinarium, Salmonella typhus</td>
</tr>
<tr>
<td>\textit{B. Faecalis}</td>
<td>An enterococcus ?, Bacillus faecalis alcaligenes, ? Acinetobacter</td>
</tr>
<tr>
<td>\textit{B. No. 10}</td>
<td>?</td>
</tr>
</tbody>
</table>

Table 6 - reproduced from Alexander, M. \textit{Reidentifying the bowel nosodes} BHJ April 1998 Vol 77. pp. 67-71
Micro-ecology of the Human GI Tract - Introduction

An estimated $10^{14}$ living bacteria encompassing more than 400 species are present in the human gut. This represents a very complex milieu which is metabolically active. Wide variation occurs because the intestinal ecosystem has a close relationship with the host and which is perpetually in contact with xenobiotics, mainly food and drugs.

The microflora of different regions of the GI-tract:

The stomach normally contains a small number of predominantly Gram-positive and aerobic bacteria and the small intestine represents a transitional zone between the scarce population of the stomach and the high density population of the colon.

In the proximal small bowel bacterial concentration and pattern of microflora are similar to that of the stomach, while in distal ileum gram-negative begin to outnumber the gram-positive and bacterial concentration increases significantly.

The small intestine contains only $10^3$ to $10^5$ bacteria per gram of luminal content. The forceful peristalsis exceeds the bacterial rate of multiplication and only bacteria which adhere to the mucosa can persist.

The most dramatic change occurs across of ileo-cecal valve, with the total number of micro-organisms increasing up to one million fold, and anaerobes outnumbering aerobes in the ratio of 1000:1.

In the colon, peristalsis is slower and bacterial population reaches immense numbers: $10^9$ to $10^{11}$ per gram. Concentration of nutrients are poor in the lumen but richer near the epithelial cells where mucus and micronutrients are concentrated. Bacterial factors promoting colonisation of the mucus layer result in enhanced metabolism and multiplication.

Development of the human intestinal microflora:

The human foetus is devoid of bacteria before birth. After birth it rapidly becomes colonised. By the end of the second year, the composition of the child's microflora resembles closely that of the adult. The composition of the adult flora changes with age.

Disturbances in the intestinal microflora:

Although the composition of the GI-tract flora is fairly stable in healthy persons, it can be altered by many factors such as antibiotics, emotional stress, surgical operations of the GI-tract, disorders of peristalsis, inflammatory bowel diseases, cancer, disorders of the cellular and humoral immune system and age.
Micro-ecology of the Human GI Tract - Research models

The modern concept of the intestinal ecosystem is largely based on studies by Schoedler and Dubos (1964). Various additional models have evolved over the past 30 years, through the efforts of several research teams in different parts of the world (Luckey-Savage-Freter-Midtvedt-Raibaud-Ducluzau etc).

Studies, based on the use of germfree animals and gnotobiology as well as new culture techniques for anaerobic bacteria have provided research models and have led to a number of new concepts, as well as opening up several new avenues for future research.

Regulation within the Intestinal Ecosystem

Our bacterial population is maintained by a surface immunity based on processes of:

- recognition
- biofeedback (immunological and biochemical)
- local environmental shift involving synergy / inhibition
- modulation of the competitive balance between sub-populations

Recognition in the gastrointestinal tract involves the gut-associated lymphatic tissue (GALT) which ‘learns’ by pinocytosis and perabsorption of bacteria and bacterial products.

The stability (and protective capacity) of the immune system as a whole is dependant on the maintenance of dynamic equilibrium within the healthy gut.

Disturbances of Symbiotic Equilibrium

If gut symbiosis is rendered chaotic, under the influence of toxins, pathogens, drugs, or psycho-neuro-endocrine stress, then immunological chaos will manifest not only in the GI tract but elsewhere giving rise to a globally lowered resistance to opportunistic infection.

Bacterial sub-populations change in their relative numbers and locations with changes in the environment. In the 1950s Baumgaertel suggested that deleterious changes in the environment (‘milieu’ or ‘terrain’) could result in coliforms degenerating into toxic variants.

Neustaedter found that enterobacteria subtypes (usually E. coli), which ferment lactose very slowly, were clearly associated with chronic illness.

Haenel introduced the terms of eubiosis and dysbiosis. The term eubiosis is used to describe balanced steady conditions between the microflora and the host, whereas dysbiosis means a significant shift away from eubiosis.

Various dysbiotic states are recognised. Most typically they manifest as repression of bifido-bacteria by tribes of coliforms, enterobacteria, and especially an increase of enterococci, proteus, yeasts and clostridia.
Micro-ecology of the Human GI Tract - Eubiosis

In the eubiotic state faecal flora is:

- 90% obligate anaerobic *bifido-bacteria* and *bacteroides*,
- 10% obligate aerobic bacteria (*coli*, enterococci, lactobacilli),
- less than 1% of other enterobacteria
  (*proteus*, clostridiae, staphylococci, aerobic spores and yeasts).

There is evidence to indicate that patterns of illness (distinct from illness caused by obvious infection by enteropathogens) are associated with characteristic shifts in the microflora.

For example: increase in *Group D Streptococci* in faeces of ulcerative colitis patients. In other studies it has been shown that colonic flora changes in composition when the patient is under stress.

For many years it has been standard practice in stool microbiology to identify the relative populations of obligate and facultative anaerobes. Identification of enterobacteria (particularly *E. coli*) was also based on differentiation of lactose fermenting degree (1-3).

**Group 1**
(very good fermentors) are frequently found in healthy subjects.

**Group 3**
(inhibited or delayed fermentation) are frequently found in sick patients.

This has some consistency with the findings of Bach and Paterson although they focussed on the balance between good fermentors and non-lactose fermentors (NLF). Their singular emphasis on the importance of non-lactose fermenters (NLF) is not fully consistent with observations of Seelig and Neustaedter, who did not particularly associate NLF with illness.

Bach and Paterson, however, were able to demonstrate changes in the bowel flora under the influence of homeopathic treatment. They found that, in chronic cases, the bowel flora changed temporarily after homoeopathic treatment, to a state which they normally associated with illness (increased NLF).

At first glance this seems to be a paradoxical response to treatment. One possible explanation for the phenomenon may concern changes in the surface adherancy of specific subpopulations, their displacement and their expulsion via the stool.

Electron microscopy has revealed the intimacy of the surface adhesion of many organisms on the microvilli. Some species have highly adapted surface protein structures which facilitate adhesion, in some cases with a degree of damage to the microvillous structure of the endothelium. An appropriate homeopathic stimulus may ‘loosen’ their grip on the endothelium and allow safer organisms to populate the vacated areas.
Homeopathic Concepts and The Influence of Remedies

In the following section we will consider Paterson’s observations of the changes in the bowel flora and reconsider the interpretation of these observations. Before we examine current ideas on intestinal dysbiosis we should consider the 20th century perspective. One of the most lucid historical sources on the subject is Thomas Dishington’s paper The Pathogenesis of Dysentery and the Proving of the Nosode Dys. co. 13

Thomas Dishington

A. Statement summarising the homeopathic disease concept

Disease is not an entity to be expelled from the body, but a dynamic error in the life forces, an unbalance in the vital functions that can be corrected only by the recuperative power within the living cells themselves. - Dishington 13

A1 A reinterpretation and expansion

Disease is not an entity to be expelled from the body, but a [systems-disturbance] that can be corrected only by [engaging the intrinsic potential for self correction] within the living [systems] themselves, [and eliminating any extrinsic factors] which push the system towards chaos - My brackets

* What Dishington refers to as the ‘vicious cycle of [the] disease process’ and which might also be termed block to cure.

B. Disturbed bacteria-host symbiosis is a systems disturbance

The bacteria cannot be looked upon as the sole causation of the disease, but are present because of the dynamic error within the life of the patient, and because they are closely related to that dynamic error they are within themselves the reflex of the host and they are the carriers of infection from the host. - Dishington 15

B1 The non-lactose fermenting populations in the stool of some ill patients are intrinsic to their systems-disturbance. The profile of the organism(s) present is specific to the illness state.

C. The autologous vaccine or nosode can deliver the systems-information required to correct the disturbance.

A nosode therefore made up from, say 100 non-lactose fermenting bacilli of one group ... contains the ... reflex of one hundred patients with that [systems-disturbance]. - Dishington 13

C1 Bach and Paterson supported the use of polyvalent nosodes derived from many individual cultures, from many patients. The strengths and weaknesses of this are discussed in the next section.
The Influence of the homeopathic remedy on bowel microecology

Bach and Paterson demonstrated that the bacterial profile of the stool showed patterns of correspondence with the homeopathic remedy used. For example, if you give a patient homoeopathic Sulphur he/she will be observed to yield Morgan. 21

John Paterson
Between 1927 and 1932, Paterson cultured 8000 stool specimens and he published some preliminary findings. 21 During this time Paterson observed: ‘Homoeopathic potencies are capable of completely altering the bacterial flora of the bowel, and this fact has been demonstrated in many hundreds of cases.’ Paterson, J. Potentized Drug and its Action on the Bowel Flora.

After treatment with homoeopathic Sulphur or Calcarea, patients were found to have increased numbers of ‘Morgan’ in the stool. Patients treated with Lycopodium clavatum yielded a ‘Morgan type’ organism with a different fermentation profile, which was given the name Morgan-gaertner. 21

‘Here, on a bacteriological basis, are found these three remedies in association under one main type, which however, can be subdivided. Into one subgroup comes sulphur and calcium, while in the other Lycopodium stands alone.’ 21

This ‘sub-typing’ of Morgan (Bach) into Morgan pure and Morgan gaertner, arose as a result of remedy experimentation. (See fermentation profiles on page 1.22.)

Over the next few years 12,000 specimens were collected from medical attendees and analysed. Using the bacteriological methods of that time the six groups of non-lactose fermenting bacilli were isolated (table 7) and ‘identified’ in the stools of homeopathically treated patients.

Bacterial adherence

“Recent developments indicate that some adhesive bacteria are able to recruit a variety of structurally diverse host proteins, adhesive glycoproteins, growth factors and cytokines, by initially binding heparin and functionally similar sulphated polysaccharides to their surfaces, whence they serve as non-specific, secondary recruiting sites for other host molecules.”

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<table>
<thead>
<tr>
<th>Organism</th>
<th>%</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. Morgan pure</td>
<td>5.68</td>
<td>14.82</td>
</tr>
<tr>
<td>B. Morgan-gaertner</td>
<td>3.27</td>
<td></td>
</tr>
<tr>
<td>B. Morgan “X”</td>
<td>2.73</td>
<td></td>
</tr>
<tr>
<td>Unclassified ‘hybrid’</td>
<td>3.14</td>
<td></td>
</tr>
<tr>
<td>B. Proteus pure</td>
<td>2.27</td>
<td>3.37</td>
</tr>
<tr>
<td>B. Proteus hybrid</td>
<td>1.46</td>
<td></td>
</tr>
</tbody>
</table>

Table 7

Why do remedies increase the stool numbers of certain organisms?

Does homeopathic sulphur alter the bacterial ability to ‘recruit’ adhesive glycoproteins by altering their ability to bind sulphated polysaccharides? Do populations become less adherent and increase in the luminal content as a result? (Resulting in Paterson’s observation of increased stool numbers.)

If homoeopathic remedies can alter the surface adherence of any population of bowel organism, or if it can alter any aspect of their gene expression, viz. enzyme function or substrate utilisation, there will be systems consequences.
PROPOSITION
If a homoeopathic remedy can alter the surface adherence of any population of bowel organism, or if it can alter any aspect of their gene expression, there will be changes in enzymal activity, substrate utilisation, leading to tertiary systems-effects.

Here are some extracts from current reviews of bowel ecology:
[my underlining of some key points]

1. Metabolic activities of flora and enzyme expression
To get an idea about the role of a given organism in the human gastrointestinal tract it is necessary to know its metabolic capabilities. This requires either growing this organism in the laboratory and studying its metabolism in vitro or developing methods for the in situ-detection of bacterial activities.
Growing a microorganism in a medium may be a difficult task if the optimal growth conditions are not known and the organism under study does not grow on conventional media. However, even if growth conditions can be found, it is not quite clear whether the activities observed in vitro are also relevant for the in vivo situation.
One of the major tasks in future research will therefore be directed to the development of methods for monitoring enzyme expression and activity in situ at the cellular level.
Therefore, classical microbiological techniques and molecular based techniques are not mutually exclusive but complementary.
M. BLAUT Assessment of bacteria in the gut microbial ecosystem

2. Bacteria/bacteria interactions
Major substrates of the intestinal flora are dietary constituents that escape digestion in the small intestine and endogenous sources such as mucins and proteins from sloughed cells and the host’s digestive enzymes. The availability of substrates is a major factor for the development of bacterial communities. Interactions between bacterial population groups are largely based on cross feeding which means that one bacterial population group utilises certain degradation products formed by other bacteria as substrates. The variety of potential substrates formed during microbial breakdown of the primary substrates increases during the transformation of polymeric substrates to oligomers and monomers and other cleavage products.
M. BLAUT Assessment of bacteria in the gut microbial ecosystem
3. Multispecies communities - Biofilms
Other types of interactions between bacteria may involve the formation of antibacterial substances inhibitory to the growth of certain bacterial groups. In such a situation, the ecological niche opened by the inhibition of one group of bacteria may facilitate the establishment of another bacterial group which has the same substrate spectrum but is not susceptible to the antimicrobial compound. The ability of one species to survive in this habitat may depend on its ability to establish as a member of a multispecies community as present for example in so called biofilms. This in turn may depend on the presence of partner organisms required for attachment.
M. BLAUT Assessment of bacteria in the gut microbial ecosystem

4. Cell to Cell Signalling
The formation of multispecies assemblages as encountered in biofilms would be expected to require cell-cell communication to occur. Exchange of information between bacteria may be brought about by the formation of signal molecules that are released into the environment and can be sensed by other bacteria. Such signals may influence the behaviour and metabolic capabilities of bacteria by way of modulating gene expression.
In spite of the large number of bacteria and species present in the human intestine only very few examples of cell-cell signalling have been discovered. It can be assumed that most cases of cell-cell communication in this habitat have not yet been discovered.
M. BLAUT Assessment of bacteria in the gut microbial ecosystem

5. DNA transfer
Bacteria may not only influence other bacteria by the release of signal molecules, but also by the transfer of genetic material between bacteria. Although the principal mechanisms of DNA transfer are known it is not clear to which extent gene transfer occurs between bacteria in the gastrointestinal tract.
Gene transfer in the gastrointestinal is important because it has possible implications for the spread of antibiotic resistances and the ability of bacteria to adapt to environmental changes.
M. BLAUT Assessment of bacteria in the gut microbial ecosystem
6. Regional availability
The large intestinal microbiota is characteristically viewed as being a homogeneous entity, yet the proximal colon and distal bowel differ markedly in relation to their nutritional availabilities and physicochemical attributes.

G.T. Macfarlane Colonic ecosystem

7. Implications of non-culturable organisms
Except in very broad terms, little is known of the metabolic relationships that exist between individual bacterial communities in the colon, or of the ecology and multicellular organisation of the microbiota.

Moreover, a number of molecular studies have suggested that only a small fraction of bacterial species in natural communities, such as the gut, are culturable, thus, while we can readily determine that the ecosystem contains considerable numbers of phylogenetically and physiologically different bacteria, the relative population sizes and types of non-culturable organisms present in the microbiota are largely unknown.

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8. Adherent vs. Non-adherent Populations
In the large gut, individual bacterial species and assemblages of microorganisms exist in a multiplicity of different microhabitats and metabolic niches, on the mucosa and in the mucus layer, as well as in the gut lumen. Examination of intestinal material by scanning electron microscopy and fluorescent light microscopy shows that most of the bacteria are not freely dispersed, but occur in clumps, and in aggregates attached to plant cell structures and other solids.

With respect to the numerically predominant culturable species, bacteria attached to surfaces in the gut lumen appear to be phylogenetically similar but physiologically distinct from non-adherent populations.

These adherent organisms are more directly involved in the breakdown of complex insoluble polymers than unattached bacteria, which provides a competitive advantage in the ecosystem. Close spatial relationships between bacterial cells growing on surfaces are important in other ways, particularly in relation to metabolic communication between different groups of microorganisms in the microbiota.

Furthermore, they are ecologically significant in that they minimise potential growth limiting effects on secondary cross-feeding populations, that are associated with mass transfer resistance...

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9. Relationship between mucosal populations and health

In healthy people, mucosal populations are more difficult to study than faecal bacteria due to difficulty in gaining access to the bowel, and has restricted studies on these communities.

Consequently, little information is available concerning the composition, metabolism and health-related significance of bacteria growing at, or near the mucosal surface.

G.T. Macfarlane *Colonic ecosystem*

See also: S. Swift *Cell-cell communication MEHD*
Clinical Implications: draft appendix to Part 2 Systems & Symbiosis - Text Under Review

The uncertainty concerning the precise bacteriological identity of the nosodes has very significant implications for their future within medicine. It may be clinically vital that the Bach and Paterson's nosode preparations were polyvalent. i.e. prepared compositely from many samples, sourced from different patients.

It is highly likely that these collated bacterial groups include a variety of subtypes. So, for example, Dys. co. is a composite from more than one hundred patients and probably contains all three Shigella subspecies. Which means it may have the potential to correct the immunological consequences arising from most instances of Shigella infection.

The other important consideration is the possibility that the self perpetuating nature of intestinal dysbiosis hinges on the ecological relationships between organisms (Biofilms)

These relationships, if pathological, might be positively altered by potencies of their constituent organisms. The nosodes, in their present form may, therefore, represent systems stimuli that are much more complex than would be available from pure culture preparations. (compare Bacillimum with Tuberculimum).

The fermentation profile of the bacterial groups shown above may have a significant bearing on symptomatology, particularly the regional GI symptoms linked with each nosode and possibly the associated food predilections and intolerances. Consequently one organism within the composite nosode will fit the bacteriological status of the patient, although it was selected on the basis of the symptomatology of its fermentation group.

In the chronic case the original pathogen (if it ever existed) may no longer be identifiable in the stool. The resulting dysbiosis may be complex or difficult to define and may only be correctable with a composite nosode. The ideal response to the situation would be to create and administer an autonosode, since this would reflect the exact nature of the dysbiosis. Fortunately, the use of the indicated Bach - Paterson nosode, whose composite nature is best reflected in the symptomatology of the patient, often seems to be effective in these patients.

Although there are clinical advantages arising from the (probably) composite nature of the bowel nosodes, there are serious scientific disadvantages, particularly concerning the reproducability of the nosodes and the interpretation of clinical data.

It would be impossible to reproduce the existing bowel nosodes on the basis of Bach and Paterson's methodology (as it is with many other nosodes and sarcodes in general homeopathic use). The current stocks appear to be active, given the unequivocal clinical responses which they still evoke. However, their clinical relevance might diminish over time with the emergence of new pathogens, sub-pathogens and commensals. Changes in diet, bacterial mutation and the widespread use of antibiotics may also change the flora with which we coexist and, correspondingly, the nature of intestinal dysbiosis.
Certain bacterial groups (for which nosodes do currently exist) have not yet been extensively examined from a homeopathic perspective. For example, does helicobacter pylori have an associated mind picture? What is the symptom-picture of campylobacter induced dysbiosis, does it contain rheumatological and skin symptoms?

Clearly, a significant discipline has been opened up which warrants further scientific enquiry. It would be possible today to develop a new array of specific nosodes, whose bacteriological identity is clear and whose clinical value could be assessed over time.

Paradoxically, a purely bacteriological/scientific approach might yield reproduceable nosodes but their remedy pictures may be much less detailed and of lower clinical value than the ones we have. Unless, of course, samples were derived from (and readministered to) homeopathically assessed patients and composite remedies were created on the basis of linked symptomatology, as well as linked bacteriology.

**Dysbiosis and pathology**

Aetiological observations have very important implications for homeopathic treatment. Might they ultimately provide us with a nosode for Crohn's Disease?

Aetiological associations between illnesses and triggers, causes and contributory factors have been made for centuries. The pathological consequences of infection are still coming to light. In some cases the primary observations have been made at a surprisingly early date and are still the subject of investigation and controversy. One such observation concerns the possible association between Crohn's Disease and Mycobacterium paratuberculosis.

*M. paratuberculosis* was first isolated and described in 1895 by Johne & Frothingham who were investigating an infiltration of the intestinal tract of cattle with chronic diarrhoea. In *æxxxxxxx',* a microbiology book of 18XX the following statement appears:

Mycobacteria are classified according to the host species, i.e. human, bovine, murine, avian and piscine. They are strict aerobes which are difficult to culture. Mycobacteria are Gram-positive, albeit technically difficult to stain. Morphologically they are acid-fast rods that do not produce flagella, capsules or spores and require special growth media due to their specific nutritional requirements.

More recent studies have confirmed that M. paratuberculosis is related to the M. avium group since it has more antigens in common with M. avium than with either M. tuberculosis, or M. bovis.

Mycobacteria include slow-growing and fast-growing species; the slow-growing organisms are considered to be causal agents for some chronic infections. *M. paratuberculosis* belongs to the slow-growing category.
Johne's disease is a veterinary condition of ruminants caused by *M. paratuberculosis*. It is a chronic, granulomatous enteritis which gives rise to debilitating diarrhoea, and weight loss. Johne's disease is a very widespread infection of domestic animals and occurs throughout the world, most commonly in cattle, but also in sheep and goats. *M. paratuberculosis* is resistant to drying and appears to be capable of protracted survival in faeces on pasture land. Although Johne's disease can also affect many wildlife species it is not currently classed as a zoonosis.

**ASSOCIATION WITH CROHN'S DISEASE**

Crohn's disease was first described by Crohn and colleagues in 1932. Over the years, many theories regarding its aetiology have been suggested including: allergic reaction, auto-immune disease, defective mucosal barrier, dietary factors, measles virus and, of course, *M. paratuberculosis*.

However, it was Dalziel in 1913 who first suggested a possible association between *M. paratuberculosis* and a chronic intestinal enteritis in humans. He described several patients with chronic intestinal enteritis, and drew parallels with the recently described disease in cattle called paratuberculosis (Johne's disease). He suggested that "the histological characteristics of the two diseases were so similar as to justify a proposition that they may be the same".

To date, the aetiology of Crohn's disease still eludes the scientific community. However, investigators keep coming back to the question of whether or not Crohn's disease could be a mycobacterial disease. There is evidence both for and against this theory. For a comprehensive review of this complex subject readers are referred to review papers by Chiodini (1989) and Thompson (1994).

It was Chiodini and his co-workers who, in 1984, successfully cultured the first two strains of M. paratuberculosis in the USA from patients with Crohn's disease. Since then this organism has been sporadically cultured from humans with Crohn's disease and a total of 10 isolates of M. paratuberculosis had been cultured from patients with Crohn's disease in the USA, Australia, the Netherlands and France (Chiodini, 1989).

When subjected to restriction fragment length polymorphism (RFLP) molecular typing, isolates of *M. paratuberculosis* from patients with Crohn's disease have been shown to be of the bovine type rather than of ovine-caprine origin, as their RFLP patterns were indistinguishable from those of strains isolated from cattle (François, Krishnamoorthy & Elion, 1997). However, it must be stressed that relatively few strains were examined; only 4 human isolates were compared with 8 bovine strains and 1 from a caprine source.

In early 1998, Hermon-Taylor and colleagues published the case history of a boy in whom Crohn's disease was diagnosed five years after developing enlarged lymph nodes on his neck at 7 years of age. *M. paratuberculosis* was detected by PCR in biopsies, both from the enlarged lymph nodes and the inflamed intestine five years later. The boy was treated with the antimicrobials rifabutin and clarithromycin for an extended period and became completely asymptomatic.

This report has been viewed by some as the first documented case of *M. paratuberculosis* causing disease in a human being. Others insist that this single case fails to prove the link between *M. paratuberculosis* and Crohn's disease.