

## Faecal microbiota of individuals with autism spectrum disorder

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### Abstract

Many children with autism spectrum disorders (ASDs) suffer from gastrointestinal problems such as diarrhoea, constipation and abdominal pain. Such symptoms may be due to a disruption of the indigenous gut microbiota promoting the overgrowth of potentially pathogenic microorganisms. These observations have stimulated investigations into possible abnormalities of intestinal microbiota in autistic patients. The purpose of the present study was to determine if a relationship exists between ASD severity (mild – severe) and gastrointestinal (GI) microbial populations. The faecal microbiota of 22 male and 6 female participants with ASDs (aged 2-14 years) were analyzed by standard microbial culture methods and compared within-group (based on ASD severity) and with a standard laboratory reference range. Comparisons between children with mild ASD and those with moderate to severe ASD, as well as comparisons to a neurotypical control group previously reported, revealed that no significant differences appear to exist in the composition of the gut microbiota. Nevertheless, examination of each individual's gut microbial composition showed 10 cases of unusual findings which means 1 out of 3 cases have unusual microbiota. Our data do not support consistent GI microbial abnormalities in ASD children, but the findings do suggest that aberrations may be found in a minority of ASD children. Further studies are required to determine the possible association between the microbiota and gastrointestinal dysfunctions in a subset of children with both ASD and gastrointestinal problems.

**Keywords:** *Microbiota; ASD; gastrointestinal tract; bacteria*

### Introduction

Autism spectrum disorders (ASD) are behaviorally defined developmental disorders (including autistic and Asperger's disorder) with a wide range of behaviours. Although the etiology of ASD is unknown, data suggest that autism may have multiple etiologies with both

genetic and environmental contributions (Herbert, 2010). This diverse etiology may explain the spectrum of observed behaviours seen in affected individuals. Nevertheless, recent studies have suggested gastrointestinal (GI) disorders and associated symptoms are commonly reported in individuals with ASD, but key issues such as the prevalence and best treatment of these conditions are incompletely understood (Buie, Campbell et al., 2010; Buie, Fuchs et al., 2010; Galiatsatos, Gologan, & Lamoureux, 2009; Gondalia, Palombo, Knowles, & Austin, 2010). The clinical picture of GI disturbance includes constipation, diarrhoea, foul-smelling stools, gaseousness, abdominal bloating, signs of abdominal discomfort, and decreased intestinal carbohydrate digestive enzyme activity (Horvath, Papadimitriou, Rabsztyl, Drachenberg, & Tildon, 1999). Nevertheless, the incidence and nature of GI symptoms in those with ASD remains somewhat uncertain and, even, controversial (Ibrahim, Voigt, Katusic, Weaver, & Barbaresi, 2009; Kuddo & Nelson, 2003; Molloy & Manning-Courtney, 2003).

Reasons to consider that microorganisms may be involved in autism include the following; gastrointestinal symptoms are common at ASD onset and often persist, antimicrobials (e.g., oral vancomycin) may lead to a clear-cut response and relapse may occur when the vancomycin is discontinued, and some patients have responded to several courses of vancomycin and relapsed each time it was discontinued. Vancomycin has proven effective in reducing autistic symptomatology and is known to be highly effective against Gram-positive organisms. When given orally, vancomycin is poorly absorbed by the gut, so any effect seen with its use is likely due to its action on intestinal bacteria (Finegold, 2008; Sandler et al., 2000).

One hypothesis is that the toxins produced by microorganisms may play an important role in the cause of ASD, in particular, lipopolysaccharides (LPS), the bacterial toxins from Gram-negative bacteria (Gondalia

et al., 2010). LPS induces a depressive syndrome, characterized by anhedonia, anorexia, body weight loss, and reduced locomotor, exploratory, and social behaviours (Marvel, Chen, Badr, Gaykema, & Goehler, 2004; Singal, Tirkey, Pilkhwil, & Chopra, 2006). LPS toxicity works synergistically with mercury and other heavy metal poisonings to expand damage (Rumbeiha, Fitzgerald, Braselton, Roth, & Kaneene, 2000). Interestingly, mercury is arguably a suspect as an environmental trigger in some etiological theories of ASD (Austin, 2008; Bernard, Enayati, Redwood, Roger, & Binstock, 2001; Palmer, Blanchard, Stein, Mandell, & Miller, 2006; Palmer, Blanchard, & Wood, 2009).

The microbiological ecosystem of the gut is complex and not completely understood, but appears to be of key importance in health and disease (Gibson & Roberfroid, 1995) and is often conceptualized as an essential "organ" in providing nourishment, regulating epithelial development, instructing innate immunity and a broad range of other activities. Intestinal bacteria secrete both detrimental and beneficial compounds and overgrowth of certain species of bacteria or other changes to the normal intestinal microbiota have been reported with ASD (Finegold et al., 2002; Yap et al., 2010). The treatment of GI dysfunction in ASD with anti- or probiotics has been proposed as a way to regulate intestinal microbiota and subsequently improve symptom severity in ASD (Sandler et al., 2000; Tuohy, Probert, Smejkal, & Gibson, 2003). The variable results reported are not surprising given the lack of consistent evidence as to the specific microbial adjustment that is desirable. While many studies have compared the faecal microbiota of individuals with ASD to controls (Finegold et al., 2002; Parracho, Bingham, Gibson, & McCartney, 2005; Song, Liu, & Finegold, 2004), only one has examined the relationship between faecal microbiology and ASD severity (Finegold et al., 2010). The purpose of the present study was, therefore, to determine if a relationship exists between ASD severity (mild – severe) and GI microbial populations. Such an association would be consistent with a dose-response relationship whereby GI microflora aberrations are considered to play a role in the aetiology of the disorder or, alternatively at least, a mechanistic role related to symptom severity.

## Method

### Participants

Twenty-eight individuals aged 2-14 years ( $M = 4.8$ ,  $SD = 3.3$ ; 22 male and 6 female) with ASD participated in this study. None of the participants were taking antibiotics within 14 days prior to the sampling. Dietary intake was not controlled and so this likely varied across individuals. The participants were recruited from consecutive patients presenting to the private

psychology practice of the fourth author. The study was approved by the Human Research Ethics Committee of Swinburne University of Technology, and informed consent was given by parents on behalf of their children who participated in the study.

### Faecal sampling and analysis

Faecal microbial analysis by standard microbial culture methods was carried out by a commercial laboratory, Bioscreen Medical ([www.bioscreenmedical.com](http://www.bioscreenmedical.com)). Participants were provided with a sample collection kit containing a specimen container (lid with two holes), an air-locked plastic bag, two gel freezer packs and an AnaeroGen Compact foil sachet (Oxoid, UK) to create anaerobic conditions inside the plastic bag.

Faecal samples were collected in specimen containers with a scoop attached to the lid. Participants were instructed to collect enough faeces to fill at least 1/3 of the scoop with faeces from used toilet paper. Participants were instructed to screw the lid on firmly, place the container in the plastic bag along with an opened AnaeroGen Compact foil sachet and seal the bag to make it airtight. For transport to the laboratory, samples were sealed in a polystyrene box together with two freezer packs.

### Statistical methods

Faecal microbial analysis was carried out to identify the presence and quantities of bacteria (aerobes and anaerobes), and the yeast, *Candida*. Analysis of variance was used to examine difference between mild and severe ASD groups. Data analysis was also carried out between the ASD groups as a whole and the commercial laboratory reference ranges. Various groups of organisms were also analysed for possible significant associations and data from this study were compared with that from published control group data (Finegold, Flora, Attebery, & Sutter, 1975) as well as to the laboratory reference ranges. Data were analyzed using the Statistical Package for Social Sciences (SPSS).

## Results & Discussion

Participants were divided into two groups: those with moderate to severe ASD ( $n = 16$ ) and those with mild ASD ( $n = 12$ ) (Table 1). The severity category was based on CARS (Childhood Autism Rating Scale) assessments conducted by registered psychiatrists or psychologists and the child's score reported by parents' of participants. Scores of less than 37 are considered mild and those at or above 37 are considered moderate – severe (Rellini, Tortolani, Trillo, Carbone, & Montecchi, 2004). Details on the specific bacteria recovered with ranges and converted mean counts are given in Table 2. These counts represent a conversion from the means obtained after log transformation of the

data values. The log transformation was used because of extremely skewed distributions of some organisms, which reflected relatively small sample numbers. All counts are based on weight of dry stool.

Analyses of variance revealed no significant differences between the two ASD severity groups on mean levels for any of the analytes (see Table 1). Furthermore, no relationships were evident between the levels of any bacterial populations examined across age and gender. Inspection of each individual case revealed some aberrant results, but not in a consistent or common enough pattern to significantly affect the mean group levels. Ten ASD children appeared to show more variation in microbial profile than the other 18 ASD children (see Table 3). Given the lack of associations between GI microflora population levels and autism severity we decided to compare our total sample means with the normal laboratory reference ranges as well as means reported for neurotypical individuals in a previously published study (Finegold et al., 1975).

Table 1  
*Mean values of faecal microbiota examined for different groups (mild vs. moderate-severe) of ASD individuals*

	Mild ASD group (n = 12)	Moderate to severe ASD group (n = 16)	P-value
Total bacterial count	10.26	10.36	.62
Total aerobe count	7.31	7.37	.83
Total anaerobe count	10.26	10.35	.64
<i>Candida</i>	2.48	3.98	.07
<i>Escherichia coli</i>	6.44	6.69	.55
<i>Enterococcus</i> spp.	6.79	6.51	.58
<i>Staphylococcus</i> spp.	5.62	5.63	.98
<i>Streptococcus</i> spp.	6.71	6.73	.96
<i>Bifidobacterium</i> spp.	9.14	9.38	.52
<i>Clostridium</i> spp.	7.24	7.47	.79

Anecdotal reports by parents and several recent reports have provided evidence that ASD children suffer from disturbed GI tract function (Buie, Campbell et al., 2010; Buie, Fuchs et al., 2010). Nevertheless, comprehensive assessments of GI abnormalities in ASD children are not widely or routinely performed. In the

present study, comparisons between children with mild ASD and those with moderate to severe ASD (Table 1), as well as comparisons to a neurotypical control group (Table 2), revealed that significant differences did not appear to exist in the composition of the gut microbiota. However, examination of each individual's gut microbial composition showed occasional cases of unusual findings. For example, in four ASD participants, we found low numbers of total bacterial and total aerobes (patients 1, 2, 7 and 9). *Escherichia coli* levels were lower than the normal range in ASD patients 2, 4 and 5, while other aerobic bacteria (*Citrobacter* spp., *Enterobacter* spp., *Staphylococcus* and *Streptococcus* spp.) were higher than the normal range in two ASD patients and *Bifidobacterium* were significantly higher than the normal range. *Clostridium* spp was higher in patient 8 (see Table 3).

There are several important limitations to this study that should be acknowledged. One is the small sample size, and the resultant limitations to generalisability of the findings and statistical power. Also, we did not utilise a control group but, rather, compared our clinical data to previously published data and laboratory reference ranges for normal control populations. A further limitation was our single time-point sampling which does not allow for determining stability of microbial populations over time. Finally, we did not control for dietary differences amongst our participants. Obviously, it would be ideal to experimentally manipulate dietary intake to remove its influence on GI microbiology.

The kind of individual aberrations found in our sample are consistent with the current consensus that GI dysfunction does occur more frequently in ASD than non-ASD children (Buie, Campbell et al., 2010), but possibly only in a minority of individuals. It is critical, therefore, for future research to attempt to identify this minority *a priori* in order to conduct more advanced testing beyond group mean comparisons which our study show may dilute (at a group level) any aberrations apparent in the minority of children. Furthermore, future researchers should cast a wider net in their search for microbial correlates in ASD. All studies conducted to date have examined only a fraction of the microbial species known to inhabit the gut, both pathogenic and commensal. Additionally, microbial species beyond bacteria should be examined, including viruses, fungi and protozoa. If microbial correlates to the GI dysfunction seen in ASD can be identified, promising opportunities for influencing the clinical course of the GI disturbance through appropriate intervention may be developed, such as pro- or antibiotic therapy or dietary intervention.

Table 2

*Comparison of bacterial counts between individuals with an ASD, control group and laboratory reference range*

	Lab ref (n = 177) <sup>a</sup>		Total ASD (n = 28)		Control <sup>b</sup> (n = 25)		
	Range	n	Range <sup>c</sup>	Converted mean	n	Range <sup>v</sup>	Converted mean
Total bacterial count							
Total bacterial count	11-12	28	9-12	10.32	25	-	10.55
Total aerobic count	7-8	28	5-9	7.35	25	-	9.92
Total anaerobic count	8-12	28	9-12	10.31	25	-	11.58
<i>Candida</i>	< 4	18	0-6	3.49	-	-	3.39
Facultative Gram negative bacteria							
<i>Escherichia coli</i>	7-8	27	4-8	6.59	23	4-11	8.10
<i>Klebsiella</i> spp.	<5	3	4-7	5.48	12	4-10	4.81
<i>Citrobacter</i> spp.	<6	0	-	-	1	8	2.76
<i>Enterobacter</i> spp.	<6	4	5-7	6.24	1	4	2.17
Facultative Gram positive bacteria							
<i>Enterococcus</i> spp.	<6	9	6-7	6.41	-	-	-
<i>Staphylococcus</i> spp.	<5	8	5-7	5.63	9	4-10	4.44
<i>Streptococcus</i> spp.	<6	25	5-9	6.73	-	-	-
Alpha-haemolytic <i>Streptococcus</i>	<6	14	4-8	6.66	9	5-11	3.91
Beta-haemolytic <i>Streptococcus</i>	<6	5	4-6	5.17	-	-	-
Non-haemolytic <i>Streptococcus</i>	<6	22	4-9	6.43	-	-	-
Anaerobic Gram negative bacteria							
<i>Bacteroides</i>	8-12	27	9-11	9.93	10	-	9.63
<i>Prevotella</i> spp.	<10	19	9-11	9.73	-	-	-
<i>Porphyromonas</i> spp.	<9	2	9-10	9.80	-	-	-
<i>Eubacterium</i>	<9	7	9-11	9.67	-	-	-
Anaerobic Gram positive bacteria							
<i>Eubacterium</i>	<9	0	-	-	-	-	-
<i>Bifidobacterium</i> spp.	7-9	22	7-10	9.29	10	-	8.31
<i>Lactobacillus</i> spp.	6-8	8	7-10	8.24	10	-	6.90
<i>Clostridium</i> spp.	<9	28	0-10	9.71	-	-	-
<i>Peptostreptococcus</i> spp.	<5	1	0-10	9.71	-	-	5.91

Note: <sup>a</sup>Bioscreen (2009); <sup>b</sup>Finegold et al (1975); <sup>c</sup>Log<sub>10</sub> no. of organisms per g of faeces (rounded off to nearest log in the case of range)

Table 3

Microbiological data from examination of faecal samples obtained from 10 selected children with abnormal microbiota compared with a control group

Organism isolated	Microbiological data, by patient number										
	Control group converted mean <sup>a,b</sup>	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8	Patient 9	Patient 10
Total bacterial count	10.55	9.61	8.98	10.28	10.51	10.26	10.28	9.91	11.18	9.36	10.43
Total aerobe count	9.92	8.84	5.00	7.18	7.43	5.96	8.57	6.73	6.58	6.18	8.20
Total anaerobe count	11.58	9.58	8.98	10.28	10.51	10.26	10.28	9.91	11.18	9.36	10.43
<i>Candida</i>	3.39	6.08	.	.	.	.	.	.	4.85	.	4.46
<i>Escherichia coli</i>	8.10	6.94	4.08	7.18	5.90	4.87	6.97	6.61	6.08	6.11	.
<i>Klebsiella</i> spp.	4.81	.	.	-	6.51	4.20	.	.	.	.	.
<i>Enterococcus</i> spp.	-	7.11	.	-	.	.	.	.	5.36	.	.
<i>Staphylococcus</i> spp.	4.44	6.48	.	-	5.20	.	5.57	.	.	.	4.88
<i>Streptococcus</i> spp.	-	8.82	4.49	4.79	7.36	5.91	8.56	6.15	.	5.32	8.20
<i>Bifidobacterium</i> spp.	8.31	8.83	7.32	.	8.75	9.20	.	9.43	.	7.92	.
<i>Lactobacillus</i> spp.	6.90	7.83	.	.	-	-	9.67	-	-	.	8.94
<i>Clostridium</i> spp	-	4.61	6.79	9.96	9.90	4.61	5.82	9.15	10.23	6.49	9.83

Note: <sup>a</sup>Finegold et al (1975); <sup>b</sup>Log<sub>10</sub> no. of organisms per g of faeces (rounded off to nearest log in the case of range)

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### Research Profile

Ms. Shakuntla Gondalia is a Microbiologist and her current research interests includes the role of gastrointestinal microflora in autism, identify the major differences in microflora populations amongst autistic children, and to determine if intervention designed to normalise the population leads to improvements in autistic symptoms and molecular genetics of extrimophiles.

Associate Professor Enzo Palombo is head of the Environmental Biotechnology Centre in the Faculty of Life and Social Sciences, Swinburne University of Technology. His current research interests include the molecular biology of human enteric viruses, expression of viral proteins, the identification of bio-active compounds from medicinal plants and mushrooms and environmental microbiology.

Dr Simon Knowles is a psychologist and Senior Lecturer in the Faculty of Life and Social Sciences, Swinburne University of Technology. Simon's main research focus involves clinical and biological exploration of the mechanisms underlying functional gut syndromes such as Irritable Bowel Syndrome, psychopathology and clinical interventions associated with chronic illness, psychoimmunity and the brain-gut-axis, stress and oral and gut bacteria.

Associate Professor David Austin is a clinical psychologist and Associate Professor in the Faculty of Life and Social Sciences, Swinburne University of Technology, and the founding Director of SABRI. He is recognised both nationally and internationally for his work on the development, delivery, and evaluation of internet-based treatments for psychological disorders, and has authored over 30 papers published in international peer-reviewed journals.