

no resistance was noted for amoxicillin and tetracycline, while the resistance rates are as follows for other antibiotics: clarithromycin 28.6% (12/42), metronidazole 40.5% (17/42) and levofloxacin 61.9% (26/42).

Conclusions The rates of resistance to clarithromycin, metronidazole and levofloxacin are high in Filipino *H. pylori* strains. This is in contrast to the earlier antibiotic susceptibility study by Destura *et. al* in 2004 in which all isolated strains were sensitive to tested antibiotics. This new pattern of resistance indicates the decreased usefulness of the first line therapy in the Philippines and the need for other treatment regimens is emerging.

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IDENTIFICATION OF GUT MICROBIOTA AND METABOLITES SIGNATURE IN PATIENTS WITH IRRITABLE BOWEL SYNDROME

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Background Irritable bowel syndrome (IBS) is the most common gastrointestinal disorder with a prevalence of 10%~15%. The underlying mechanism of IBS is largely unknown. This study aimed to determine whether fecal metabolite and microbiota profiles could act as biomarkers for IBS and to uncover the possible gut microbe-metabolite associations.

Methods By using a gas chromatography coupled to time-of-flight mass spectrometer (GC-TOFMS) and 16S rDNA amplicon sequencing, fecal metabolites and microbiota of 15 healthy volunteers (normal control =NC) and 15 adult IBS patients were measured.

Results The IBS patients had a significantly differential metabolite profile as compared to NC group, 4 clusters with 31 metabolites were significantly up-regulated in the IBS group as compared to the NC group. Compared with the NC group, 19 microbes were significantly up-regulated and 12 microbes were down-regulated in the IBS group. The correlation matrices between IBS clinical traits and differential abundant metabolites (DAMts) or microbes (DAMbs) indicated that partial DAMts clusters were positively associated with age, the frequency of abdominal pain/abdominal discomfort and the number of bowel movements; part of DAMbs clusters was also positively associated with those above-mentioned symptoms. By correlating metabolite levels with the amount of microbial genera, a network and correlation matrix was generated to identify two DAMts/DAMbs core panels for IBS and NC groups.

Conclusions The finding of this study puts a global perspective on metabolomics and microbiota profiling in IBS patients and provides a theoretical basis for future research on the pathophysiology of IBS.

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RELATIONSHIP BETWEEN AUTISM AND GUT MICROBIOME: CURRENT STATUS AND UPDATE

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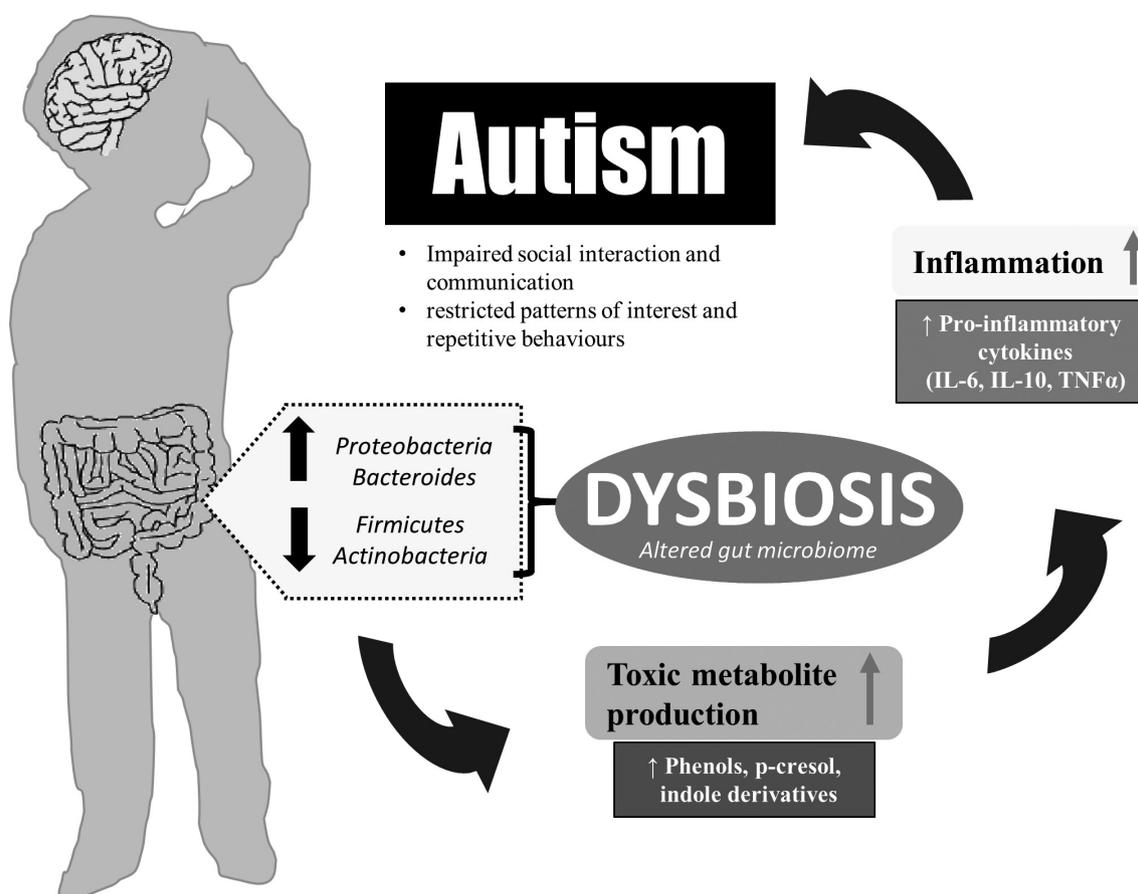
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Background Recently, many reports indicated the role of the gut microbiome in the development of autism in young children. Microbes are capable of synthesizing small molecules like fatty acids and sugars which act as signalling molecules to activate/deactivate nervous system or even trigger an inflammatory response. Thus, the current review aims to explore the role of microbes in the development of autism, summarizing data from animal models and human studies.

Methods Referring to the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines, searches were performed in three databases (PubMed, ScienceDirect, Web of knowledge and; database inception to 31/12/2018) using ‘microbiome’ OR ‘microbiota’ combined with ‘autism’ as MeSH terms. All the titles and abstracts retrieved were screened based on the inclusion and exclusion criteria. Studies reporting gut microbiome data in relation to the development of autism were included. Studies without gut microbiome data and/or reports on the evaluation on autism were excluded, along with reviews, conference abstracts, case studies, and comments.

Results Out of the 2237 articles were accessed, seven studies were eligible for the qualitative analysis according to the inclusion criteria. Two studies described the murine model of autism, while the remaining five studies focused on children. One study revealed that there are no significant differences in gut microbiome among the three groups—severe, mild and healthy children, while three studies indicated that there is a higher abundance of *Proteobacteria* and *Bacteroides* in autistic children, with a reduced population of *Firmicutes* and *Actinobacteria* (figure 1). One study highlighted that higher *Clostridiaceae* species present in autistic children which explain for the development of autism in children as these species could produce toxic metabolic products (e.g., phenols, p-cresol, indole derivatives). Similar results were also observed in autistic animal models - there is increase abundance of *Bacteroides*, *Parabacteroides*, *Sutterella*, *Dehalobacterium* and *Oscillospira* genera.

Conclusions Altogether, these results revealed a positive correlation between dysbiosis and autism. Microbiome alterations may contribute to the development of autism, particularly via the production of toxic bacterial metabolites and alteration in immune function. By keeping the ‘healthy’ gut bacteria in



Abstract IDDF2019-ABS-0321 Figure 1 Three studies indicated that there is higher abundance of proteobacteria and bacteroides in autistic children with reduced population of firmicutes and actinobacteria

check, these efforts could improve intestinal and mental health, easing and reducing autistic behaviour in children.

IDDF2019-ABS-0322 **DISSECTING THE GUT AND SKIN: BUDDING ASSOCIATION BETWEEN GUT MICROBIOME IN THE DEVELOPMENT TO PSORIASIS?**

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Background Microbiome consists of normal bacteria flora that resides in the human gut and are key contributors to host metabolism. This symbiotic bond evolves throughout our entire life, from birth to old age, and is the result of different environmental influences. Recently, reports revealed dysbiosis of gut microbiome could relate to psoriasis occurrence. Psoriasis - chronic immune-mediated inflammatory disease affects around 2–4% of the global population. This systematic review aims to reveal the dysbiosis of the gut microbiome and the occurrence of psoriasis.

Methods A thorough search was conducted using predefined MeSH terms ‘gut’, ‘microbiome’ or ‘microbiota’ and ‘psoriasis’ in 3 databases (Pubmed, Medline, ScienceDirect; from database inception to December 2018). All titles and abstracts obtained were screened based inclusion and exclusion criteria. Studies reporting gut microbiome data in relation to gut microbiome effects were included. Studies without gut microbiome data and/or psoriasis were excluded along with conference abstracts, reviews, systematic reviews, meta-analyses, and comments.

Results The gut microbiome is able to control the imiquimod-induced skin inflammation by altering the T cell response, thus affects the pathogenesis of psoriasis. Studies clearly reported the dysbiosis microbiome of psoriasis patients compared to healthy persons. Psoriatic gut microbiome in a few studies revealed an increase presence of *Faecalibacterium* and a decrease of *Bacteroides*. Another study reported a high abundance of *Bacteroidia* in the microbiome of its psoriasis patients, suggesting the potential pathogen that promotes the pathogenesis of psoriasis. As one of the common normal intestinal bacteria in healthy individuals, lower levels of *Faecalibacterium prausnitzii* have been observed in patients with Crohn’s disease, obesity, metabolic syndrome and major depressive diseases. However, in psoriatic patients, *Faecalibacterium prausnitzii* have been abundant in the gut microbiota - a similar pattern seen in infants with eczema and atopic dermatitis. Probiotics containing normal flora is essential for individuals to maintain a healthy gut microbiota.