

Variations in IBD's Gut Microbiome Across Borders

Hakan Alagozlu*

Department of Drug Metabolism and Pharmacokinetics, Universiti Sains, Malaysia

Introduction

It would be pertinent in a broader context to discover biomarkers for the diagnosis of inflammatory bowel disease (IBD). International research on these microbial indicators is still lacking, though. We used available DNA shotgun meta genomic data to explore taxonomic macrobiotic differences in IBD. We used sequence data from our prior Spanish CD and UC cohort, download sequence data from a Chinese CD cohort and download taxonomic and functional profiling tables from a USA CD and UC cohort for this purpose. The primary explanatory factors of micro biome changes at the global level were geographic location and illness phenotype.

Description

Even though they were recruited from various nations and had varying degrees of illness severity, CD patients may have a remarkably similar microbial taxonomic makeup. Geographic location, disease activity status and other environmental factors are significant contributors to microbiota changes in IBD, according to our study. We therefore highly advise future IBD studies to take these aspects into account in order to find internationally reliable and consistent biomarkers [1].

IBDs, which include Crohn's disease (CD) and ulcerative colitis (UC), are multifactorial, chronic gastrointestinal tract disorders that have been linked to changes in the gut flora. IBD, especially in CD patients, has consistently been linked to decreased microbial community richness and diversity, as well as decreased numbers of beneficial microbes and increased numbers of potentially dangerous bacterial species. However, a variety of lifestyle or environmental factors, including geography, ethnicity, medicines and dietary habits, may make it difficult to interpret and repeat microbiome investigations. This is particularly problematic when studying small cohort sizes because these factors may hide the impact of the disease [2].

The relative abundance of *Enterococcus*, *Fusobacterium*, *Streptococcus*, *Escherichia coli* and *Ruminococcus gnavus* has frequently and reliably been found to be positively associated with IBD, while the relative abundance of *Faecali bacterium* and members of roseburia has typically and consistently been found to be negatively associated with IBD. However, certain bacterial taxa identified as IBD biomarkers were also discovered in type 2 diabetes and other chronic diseases (T2D). For instance, the specificity of the available microbiome profiles for disease discriminating has been questioned in light of a relationship between an enrichment in *Christensenellaceae* and *escherichia coli* and CD and T2D-associated dysbiosis.

It is well known that there is a connection between the microbiota and the pathogenesis of IBD, but little is known about how geography and the

environment it affects can affect the makeup of the microbiota in IBD. This is the first study that, to our knowledge, uses DNA shotgun metagenomic to analyze variations in the gut microbiota among IBD patients on three different continents [3]. Geographical variations could be a result of host genetic, nutritional and behavioral variations.

Following prior studies, we found that geographic location was a significant driver of microbiome variation in both IBD and HC after analyzing the impact of the metadata factors on the microbiome. Stewart et al. discovered that environmental factors, such as geographic location and household exposures, were significant determinants of the microbiome structure in a large multicenter longitudinal study on early life. In a more recent international microbiome investigation, Clooney et al. found that the location followed by the presence or absence of a CD diagnosis had the biggest influence on the microbiota. In addition, He et al. described the gut microbiomes of 7009 healthy people from 14 districts of a Chinese province and discovered that, among other factors, host location showed the highest relationships with microbiome variability [4].

In terms of dysbiosis, we have demonstrated that changes in the make-up of the faecal microbiome were more evident in CD than in UC, which is consistent with other studies. According to Scanlan et al., the temporal stability of dominating species was noticeably lower in CD than in HC. According to Morgan et al., the IBD population in general and CD in particular were linked to a dysbiosis that was manifested by alterations in the firmicutes and proteobacteria phyla. In order to create integrated longitudinal molecular profiles of host and microbial activity during disease, Lloyd-Price et al. studied 132 individuals for a period of one year. Participants with CD or UC had a disproportionate number of samples in the dysbiotic set, with 24.3% and 11.6% of their samples, respectively, being labelled as such.

Escherichia coli was the most abundant species and *Ruminococcus bromii* and *Ruminococcus bicirculans* were the considerably most reduced microbial species in CD compared with HC based on illness phenotype and merging the Spanish and USA cohorts. Of the two, only the latter is consistent with prior findings fascinatingly, Fang et al. discovered that the strain identified by the metagenomic technique was identical to known pathogenic adherent-invasive *E.coli* strains when they performed a strain level investigation of *E. coli* in CD. Additionally, it has been demonstrated that *Ruminococcus bromii* can break down dietary-resistant starches, but *ruminococcus bicirculans* can use plant glucans. These two bacterial species may be important in the metabolism of a plant-based diet and the preservation of gut homeostasis, but they are absent from CD.

Disease activity status did not correlate with particular microorganisms in either the CD or the UC groups. The fact that other factors like age and BMI had a bigger impact on microbiome variation than illness severity scores could be used to explain this finding. In this regard, we discovered reports with conflicting findings that suggested clinical activity had an effect. In a sizable paediatric CD cohort, Gevers identified an axis defined by an elevated abundance of bacteria, such as Enterobacteriaceae, Pasteurellaceae, Veillonellaceae and Fusobacteriaceae and a decreased abundance of Erysipelotrichales, Bacteroidales and Clostridiales, that strongly correlated with disease status [5].

Conclusion

Over 16S rRNA sequencing, shotgun metagenomic analysis has a few advantages. 16S rRNA sequencing has been frequently employed to study the relationship between gut microbiota and IBD since it is an affordable tool for taxonomic characterization. This method has a number of drawbacks due to the heterogeneity of the rRNA operon copy number across the bacterial kingdom

*Address for Correspondence: Hakan Alagozlu, Department of Drug Metabolism and Pharmacokinetics, Universiti Sains, Malaysia; E-mail: hakanalagozlu12@yahoo.com

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and the lack of truly universal primers for PCR amplification. Additionally, this technique results in taxonomic resolution mostly up to the genus level due to the short size of the 16S rRNA gene. In this study, taxonomic profiling is accomplished using single copy marker gene databases rather than the shotgun metagenomic technique, which entails random DNA sequencing from the entire content of a clinical sample.

Conflict of Interest

None.

References

1. Bernstein, Charles N. and Jessica D. Forbes. "Gut microbiome in inflammatory bowel disease and other chronic immune-mediated inflammatory diseases." *Inflamm Intest Dis* 2 (2017): 116-123.
2. Franzosa, Eric A., Alexandra Sirota-Madi, Julian Avila-Pacheco and Nadine Fornelos, et al. "Gut microbiome structure and metabolic activity in inflammatory bowel disease." *Nat Microbiol* 4 (2019): 293-305.
3. Halfvarson, Jonas, Colin J. Brislawn, Regina Lamendella and Yoshiki Vázquez-Baeza, et al. "Dynamics of the human gut microbiome in inflammatory bowel disease." *Nat Microbiol* 2 (2017): 1-7.
4. Morgan, Xochitl C., Timothy L. Tickle, Harry Sokol and Dirk Gevers, et al. "Dysfunction of the intestinal microbiome in inflammatory bowel disease and treatment." *Genome Biol* 13 (2012): 1-18.
5. Nishida, Atsushi, Ryo Inoue, Osamu Inatomi and Shigeki Bamba, et al. "Gut microbiota in the pathogenesis of inflammatory bowel disease." *Clin J Gastroenterol* 11 (2018): 1-10.

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