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DISSECTING THE GUT AND BRAIN: POTENTIAL LINKS BETWEEN GUT MICROBIOTA IN DEVELOPMENT OF ALZHEIMER'S DISEASE?

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Background Recently researchers speculated that the gut microbiota might contribute to the development of chronic diseases like Alzheimer's disease (AD), given that some bacteria are capable of synthesising amyloids and inducing inflammation via production of endotoxins. Thus, this study aims to understand the role of gut microbiota in AD development, obtaining clues on the regulation of gut microbes to prevent and tackle AD.

Methods Following the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines, searches were performed in 4 databases (PubMed, Medline, ScienceDirect, EBSCO; database inception to February 2018) using 'gut', 'microbiome' combined with 'Alzheimer's' or 'amyloid' as MeSH terms. All titles and abstracts retrieved were screened based on the inclusion and

exclusion criteria. Studies reporting gut microbiome data in relation to the development of AD were included. Studies without gut microbiome data and/or AD biomarker(s) were excluded, along with reviews, conference abstracts, systematic reviews, meta-analyses, comments, and letters to the editor.

Results After removing duplicate records, a total of 155 articles were accessed, which resulted in 70 articles excluded based on their titles and abstracts. Seven out of 85 studies were eligible for the qualitative analysis according to the inclusion criteria. Among these, six studies involve murine model, while one study was conducted in humans. Using transgenic murine model of AD, all studies reported a remarkable shift in the microbiota composition as compared to controls, with significant changes in phyla like *Firmicutes* and *Bacteroidetes*. The high abundance of pro-inflammation bacteria like *Proteobacteria* may induce immunological reactions and neuroinflammation, which are known aetiology of AD. Complementing with *in vivo* studies, Cattaneo et al. (2017) recorded higher abundance of pro-inflammatory bacteria (e.g. *Escherichia/Shigella*, *Pseudomonas aeruginosa*) and lower distribution of anti-inflammatory bacteria (e.g. *Bacillus fragilis*, *Eubacterium rectale*, *Eubacterium hallii*, *Faecalibacterium prausnitzii* and *Bacteroides fragilis*) in amyloid-positive patients as compared to healthy subjects.

Conclusions As depicted in figure 1: The dysbiosis of the gut microbiota plays a significant role in the development of AD. Evidence showed the high abundance of pro-inflammatory bacteria with a reduced population of anti-inflammatory bacteria could promote neuroinflammation, which can exacerbate amyloid beta plaques formation.

Abstract IDDF2018-ABS-0239 Figure 1