Impact of CFTR modulation with Ivacaftor on Gut Microbiota and Intestinal Inflammation

Chee Y. Ooi^{1,2,3 *#}, Saad A. Syed^{4,5,6#}, Laura Rossi^{4,5,6}, Millie Garg¹, Bronwen Needham¹, Julie Avolio⁷, Kelsey Young⁷, Michael G. Surette^{4,5,6}, Tanja Gonska^{7,8}

¹ School of Women's and Children's Health, Medicine, The University of New South Wales, Sydney, NSW, Australia

² Molecular and Integrative Cystic Fibrosis (miCF) Research Centre, Sydney Children's Hospital, Randwick, NSW, Australia

³ Department of Gastroenterology, Sydney Children's Hospital, Randwick, NSW, Australia

⁴Department of Medicine, McMaster University, Hamilton, Ontario, Canada

⁵Department of Biochemistry & Biomedical Sciences, McMaster University, Hamilton, ON, Canada

⁶Farncombe Family Digestive Health Research Institute, McMaster University, Hamilton, ON, Canada

⁷Department of Paediatrics, Division of Gastroenterology, Hepatology and Nutrition, The Hospital for Sick Children, Toronto, Ontario, Canada

⁸Translational Medicine, Research Institute, The Hospital for Sick Children, Toronto, Ontario, Canada

*Corresponding author

[#]Co-first authors

SUPPLEMENTAL FIGURE LEGENDS

Figure S1: Gut microbiome communities do not appear different with taxonomic summaries (A) by treatment (B) or sex (C), and do not differ in species richness (Observed Species) (D) or evenness (Shannon Diversity) (E) following treatment.

Figure S2: Gut microbiome weakly cluster by geographic location (Sydney, Australia vs. <u>Toronto, Canada).</u>

Figure S1





Figure S2

