LETTER

The presence of *Sutterella* spp. in mucosa and their interactions with the epithelium suggest commensalism

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Lewis K . The presence of *Sutterella* spp. in mucosa and their interactions with the epithelium suggest commensalism. J Environ Microbiol 2022;4(1):1-2.

ABSTRACT

Although *Sutterella* species have repeatedly been linked to human illnesses such as autism, down syndrome, and Inflammatory Bowel Disease (IBD), it is still unknown how these bacteria affect health. Despite the species being so common, little is known about the interactions of Sutterella spp. with the host. In this investigation, we focused on the interaction of three well-known *Sutterella* species with the intestinal epithelium and investigated their adhesion characteristics, impact on the function of the intestinal barrier, and in vitro pro-inflammatory capability. Additionally, we looked at the relative presence and abundance of the genus *Sutterella* and *Sutterella wadsworthensis* in intestinal biopsies from healthy people and those who had Celiac Disease (CeD) or Inflammatory Bowel Disease (IBD). Our findings demonstrate a gradient of decreasing *Sutterella* spp. abundance in the duodenum of healthy people. Between the paediatric IBD or CeD patients and the healthy controls, there was no discernible change in the prevalence of *Sutterella*. *Sutterella wadsworthensis*, which preferentially bound to mucus and human extracellular matrix proteins, was able to decrease the adherence of Sutterella parvirubra, which adhered to differentiated $CaCO_2$ cells better than the other two *Sutterella* spp. The fact that only *S. wadsworthensis* caused interleukin-8 production in enterocytes suggests that the lipopolysaccharide architectures of the various species differ. However, in comparison to non-pathogenic *Escherichia coli*, its pro-inflammatory activity was relatively low. The integrity of the enterocyte monolayer *in vitro* was unaffected by *Sutterella* spp. According to our research, *Sutterella* species are common commensals with a minor pro-inflammatory potential in the human gastrointestinal tract and do not significantly contribute to the disruption of epithelial homeostasis brought on by microbiota dysbiosis and an increase in *Proteobacteria*. *Sutterella* species' capacity to cling to intestinal epithelial cells suggests that they may play an immunomodulatory role.

Key Words:*Proteobacteria; Inflammatory bowel disease; Intestinal epithelium; Celiac disease*

INTRODUCTION

Sutterella wadsworthensis, *Sutterella parvirubra*, and *Sutterella stercoricanis* are the three species of the genus that have been identified. They were isolated from human and canine faeces. *Sutterella* spp. are gram-negative, non-spore-forming rods that can thrive in anaerobic or microaerophilic environments. They have notable bile acid resistance, which may be the reason they are able to survive in the biliary tract and gut. The presence of *Sutterella* in the human microbiota is mainly unknown, and published reports are somewhat debatable. *Sutterella* spp. have been documented to be exceedingly frequent and plentiful in the intestinal mucosa of humans. Therefore, the lack of understanding is quite unexpected. First off, 86% and 71% of adults and children, respectively, have tested positive for *S. wadsworthensis*. Second, it has been discovered that both children with Celiac Disease (CeD) and healthy children have duodenal mucosa that contains *Sutterella* spp. at levels as high as 19% of the total microbiota.

In other investigations, there was no difference in the prevalence of *Sutterella* spp. between the healthy subjects and the IBD patients, despite the fact that *Sutterella* spp. have been hypothesised to be involved in the aetiology of inflammatory bowel illnesses. Additionally, *Sutterella* species have been linked to metabolic syndrome, Down syndrome, and autism. In contrast to the control group of children with merely GI dysfunction, half of the children with autism were *Sutterella*-positive, with a preponderance of either *S. wadsworthensis* or S. *stercoricanis*. The ileum and cecum contained between 2% and 7% of the total bacteria. However, a different study found no discernible difference between autistic juvenile participants with or without functional GI issues and their siblings in terms of the microbial makeup, particularly the quantity of *Sutterella*. Because of this, the findings regarding *Sutterella* spp. and human diseases are debatable, and it is yet unclear how they might play a part in the pathology and how they interact with the host.

A diversified and well-balanced bacterial colony known as a commensal colonises the mucosal surfaces of mammals without harming the host.

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Received: 2-February-2022, Manuscript No PULJEM-22-5472; Editor assigned: 4-February-2022, Pre-QC No. PULJEM-22-5472 (PQ); Reviewed: 9-February-2022, QC No. PULJEM-22-5472 (Q); Revised:11-February-2022; Manuscript No. PULJEM-22-5472 (R); Published:16-February-2022, DOI: 10.37532/puljem.22.4 (1).1-2

Commensal bacteria influence the growth of the host's innate and adaptive immune systems and encourage immunological tolerance as a part of the host-microbiota mutualism. Toll-Like Receptors (TLRs), among other pattern recognition receptors on Intestinal Epithelial Cells (IECs), are able to identify the MAMPs released by bacteria. Lipopolysaccharide (LPS), a significant MAMP generated by Gram-negative bacteria on their cell surface, triggers TLR4 signalling and thus sets off inflammatory cascades. In the Lamina Propria (LP), where the majority of immune cells do not come into contact with bacteria directly, IECs can sense MAMPs and subsequently act as transmitters of immunomodulatory signals from the microbiota to immune cells. In particular, gut commensals are required to stimulate Th17 cell development in LP, and crucially, this stimulation is reliant on bacterial adherence to IECs.

Intestinal homeostasis is maintained by the interaction of a stable, balanced microbiota with the mucosal immune system, but this balance can be readily upset by changes in the microbiota that trigger pro-inflammatory immunological reactions. Dysbiosis, which refers to an imbalance in the composition of the gut microbiota, is typically characterised by an increase in Proteobacteria, another phylum that includes Sutterella spp., and a decrease in *Firmicutes*.