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Data Article

Specialized pro-resolving mediator lipidome and 16S rRNA bacterial microbiome data associated with human chronic rhinosinusitis

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ABSTRACT

Chronic rhinosinusitis (CRS) is a clinical syndrome defined by symptoms including nasal congestion, facial pain and pressure, anosmia, and rhinorrhea lasting more than 12 weeks. Several mechanistically distinct processes lead to the development of clinical symptoms in CRS including innate immune dysfunction, dysregulated eicosanoid metabolism and perturbations in host-microbiome interactions [1]. We developed a database comprised of patient demographic information, lipid mediator metabolomic profiles, and 16S bacterial rRNA gene sequence data from 66 patients undergoing endoscopic sinus surgery. Briefly, ethmoid sinus tissue and middle meatal swabs were collected from patients, including non-CRS controls, CRS with polyps (CRSwNP), and CRS without polyps (CRSsNP). Lipid mediator pathways from arachidonic acid (AA) and docosahexanoic acid (DHA) were analyzed by

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liquid chromatography/tandem mass spectrometry. Bacterial taxa were profiled in parallel by 16S rRNA gene sequencing. This database provides a useful compendium of AA/DHA metabolomic profiles and associated bacterial microbiota in patients with varying disease subtypes, demographics, and risk factors/comorbidities.

Specifications Table

Subject	Otorhinolaryngology and Facial Plastic Surgery
Specific subject area	Innate immunity in patients with chronic rhinosinusitis. Identifying associations between bacterial microbiome and lipid metabolomics in patients with distinct chronic rhinosinusitis subtypes.
Type of data	Table (Datasets 1–3, Excel Spreadsheets) Fastq formatted DNA sequence data (Dataset 4)
How data were acquired	HPLC-MS/MS tandem mass spectrometry, C18 solid phase extraction, 16S rRNA microbiome sequencing and associated instrumentation. [2]
Data format	Raw (Datasets 1, 2, 4) Analyzed (Dataset 3)
Parameters for data collection	Sinonasal swabs and tissue specimens were collected from 66 human subjects undergoing endoscopic sinus surgery. 32 lipid mediators were quantified in tissue specimens using HPLC-MS/MS tandem mass spectrometry. Bacterial 16S rRNA genes were PCR amplified from swab-associated DNA, subjected to high-throughput DNA sequencing, and sequences annotated using SINA/SILVA.
Description of data collection	Preparation of tissue specimens for targeted HPLC-MS/MS tandem mass spectrometry of lipid mediator panel, extraction of DNA from sinonasal swabs, 16S rRNA gene PCR amplification (V1V2 region), Illumina MiSeq paired-end sequencing, and taxonomic annotation.
Data source location	Institution: University of Colorado School of Medicine Department of Medicine 12,700 E. 19th Ave, B168 Aurora, Colorado Country: United States of America 39°44′43.5″N 104°50′26.7″W
Data accessibility	With the article (Datasets 1–3). Public repository (Dataset 4). Repository name: NCBI Sequence Read Archive Data identification number: PRJNA678776 URL: https://www.ncbi.nlm.nih.gov/sra/PRJNA678776
Related research article	Co-submission: Vickery, T.W., et al., *Altered tissue specialized pro-resolving mediators in chronic rhinosinusitis.* Prostaglandins Leukot Essent Fatty Acids, 2021. 164: p. 102,218.

Value of the Data

- The persistent inflammation observed in chronic rhinosinusitis is thought to result from dysregulated innate immunity including perturbations in lipid mediator metabolism and dysbiosis of the surface microbiome. Despite these observations, relationships between lipid mediator production and microbiome composition have not been analyzed together or in association with disease phenotypes. The databases provided in this publication includes detailed lipidomics profiles in conjunction with microbiome sequences and patient data.
- These data will be of particular interest to investigators interested in the relationship between acute and chronic upper airway inflammation and host-microbial interactions.
- This database allows for comparison of patient specific factors including medical comorbidities with chronic rhinosinusitis disease phenotypes in relationship to microbiome com-

position and lipid mediator pathway regulation. The combined dataset will allow for identification of new associations between patient specific factors, disease state, microbiome composition and lipid metabolism which may identify new therapeutic targets for specific subsets of CRS patients.

1. Data Description

The symptoms associated with CRS such as facial pain/pressure, nasal congestion, rhinorrhea, and anosmia lasting more than 12 weeks represent a common clinical endpoint to several mechanistically distinct pathologic processes. Many hypotheses exist about the pathogenesis of CRS, which include exposure to Alternaria fungi in the environment, deleterious effects of staphylococcal enterotoxin on upper airway epithelia, dysfunctional host-microbiome interactions, disturbances in innate immune function and dysregulated eicosanoid metabolism [1]. Because so many factors have been implicated in the development of clinical symptoms, efforts to classify disease phenotypes have proved challenging. The presence or absence of nasal polyps and/or aspirin exacerbated respiratory disease (AERD) are frequently used to separate patients into distinct "endotypes". Further efforts have been made to categorize endotypes based on profiling of immunologic factors [3–5] and immune phenotypes, most notably on the basis of predominant helper T-cell types (Th1 vs. Th2) [1,6]. Despite these efforts to classify CRS, individual patients are increasingly thought to represent their own endotypes, each manifesting a unique constellation of demographics, medical comorbidities, environmental exposures, surface microbiome, and metabolomic profile. To better define CRS endotypes, we collected demographic information, complete 16S rRNA microbiome profile, and lipidomic signatures from surgical sinus tissues from 66 patients with chronic sinusitis [2]. Researchers interested in understanding associations between inflammatory lipid mediators, bacterial microbiota, and patient-specific risk factors will be able to use these data for further analysis. Patient demographic information, key disease measures such as sino-nasal outcome test (SNOT-22) scores, and medical comorbidities (i.e., smoking, asthma) were collected for all 66 patients included in study. (Supplement Table 1)

Prior studies examining inflammatory lipid mediators utilized enzyme-linked immunosorbent assays (ELISA) to quantify prostaglandins, leukotrienes and lipoxins from patients with sinusitis. These studies demonstrated that CRSwNP is characterized by high levels of cysteinyl leukotrienes and lipoxin A4 and low levels of prostaglandin E2 [7]. We utilized liquid chromatography tandem mass spectrometry (LC-MS/MS) approach to evaluate arachidonic acid and docosahexaenoic acid metabolism in each patient's surgical sinus tissue. Tissue was collected at the time of harvest in the operating room and immediately snap-frozen in liquid nitrogen. The tissue was then gently homogenized in methanol and lipid mediators extracted on a C18 column. Lipids (Supplement Table 2) were then analyzed using a lipid mediator quantitation method encompassing 32 lipid mediators and 9 internal standards for the 66 patients specified in Supplement Table 1.

Perturbations in the surface microbiome within the paranasal sinuses have been previously shown to differ between healthy patients and those with CRS. For example, bacterial strains such as *Propionibacterium acnes*, and *Corynebacterium* are more prevalent and abundant in healthy individuals whereas in *Staphylococcus aureus* and anaerobes are more enriched in patients with chronic sinusitis [8]. Swabs were collected from the middle meatus at the time of surgery for each patient enrolled in the study. DNA was extracted from the swabs and 16S rRNA gene sequence data (V1V2 variable region) was obtained for 56 patients specified in Supplement Table 1. Sequence counts, organized by patient and operational taxonomic unit (OTU) are provided in Supplement Tables 3a–e. Separate worksheets summarize OTU data at the phylum, order, family, and genus levels, as well as by noteworthy upper respiratory genera/species. Demultiplexed 16S rRNA gene sequences (fastq format) and associated MIMARKS-formatted metadata [9] from the 56 patients specified in Supplement Tables 1 and 3 are available in the NCBI Sequence Read Archive under accession number PRJNA678776 (https://www.ncbi.nlm.nih.gov/sra/PRJNA678776).

2. Experimental Design, Materials and Methods

Sample acquisition: Generation of this dataset was approved by the Institutional Review Board of the University of Colorado (COMIRB protocol number 15–0574) and informed consent was obtained from all study participants. The diagnosis of chronic rhinosinusitis (CRS) was made using 2015 clinical practice guidelines for the diagnosis of CRS in adults which include facial pain and pressure, anosmia, rhinorrhea, nasal congestion and radiographic evidence of sinus disease [10]. Patients with persistent symptoms despite appropriate medical management were recommended endoscopic sinus surgery and invited to enroll in the study. Exclusion criteria for this study included patients under 18 years old, antibiotic exposure within 1 month of surgery, and patients with or cystic fibrosis, immunodeficiency or autoimmune disease. Control tissue was collected from patients undergoing endonasal surgery for non-sinus related illness. These patients were confirmed no have normal sinuses endoscopically and radiographically. Patient specific demographic information, medical comorbidities, smoking status and Sino-nasal Outcome Test (SNOT-22) data was obtained from each study participant. Surgical tissue was obtained from the ethmoid sinuses, placed into a sterile storage container onto ice, snap frozen in liquid nitrogen and stored at −80 °C for later processing. 66 patients were included in the current dataset.

Lipid mediator mass spectrometry analysis: Standard mixes of chemically pure lipid mediators were generated using compounds obtained from Cayman Chemical (Ann Arbor, Michigan, USA). Extraction of lipid mediators was completed using protocols derived from by Yang et al. [11]. Complete lipid mediator extraction and mass spectrometry methods are published in Vickery et al. [2]. Briefly, lipid mediators were precipitated from sinonasal epithelial tissue using HPLC grade methanol, extracted using C-18 solid phase extraction columns, and eluted lipid mediators using methyl formate and methanol. The solvent was evaporated and the samples were reconstituted in 100 microliters of 100% ethanol for mass spectrometry analysis. Quantitation of lipid mediators was completed using high pressure liquid chromatography tandem mass spectrometry methods as previously described by Armstrong et al. [12]. Standard calibration curves were generated for each compound in the standard mix and these curves were used to quantify the on-column concentration of compound in each patient sample. Final concentrations were normalized for dilution factor and tissue weight.

16S rRNA microbiome sequencing analysis: Culture swabs were used intra-operatively to collect DNA for microbiome analysis from the middle meatus of patients enrolled in the study. DNA was isolated from swabs as previously described by our group and others [13–15]. 16S rRNA gene amplicons were generated using barcoded primers targeting approximately 300 base pairs of the V1V2 variable region of the 16S rRNA gene as previously described [16–18]. The Illumina MiSeq platform was utilized for microbiome sequencing, and analysis was completed using publicly available data analysis tools SINA/Silva [19,20] and microbiome associations were completed using our previously described methods [13,14,21].

Ethics Statement

Informed consent was obtained from all study participants. This study was completed using approved protocols reviewed by the Institutional Review Board of the University of Colorado (COMIRB protocol number 15–0574).

CRediT Author Statement

Thad W. Vickery: data curation, methodology, and original drafting and editing of the manuscript; **Michael Armstrong:** data curation and formal analysis for lipidomic profiling; **Jenifer Kofonow:** data curation, formal analysis of 16S rRNA gene sequencing; **Charles E. Robinson:** conceptualization, methodology, computational support for microbiome; **Miranda E. Kroehl:** statistical analysis; **Nichole A. Reisdorph:** methodology, equipment and reagents for LC-MS/MS analysis; **Vijay R. Ramakrishnan:** Funding acquisition, IRB approval, collection of surgical

specimens and clinical data, review and editing of manuscript; **Daniel N. Frank:** Funding acquisition, conceptualization, methodology, formal analysis, and supervision of experiments, review and editing of manuscript.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.dib.2021.107023.

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