

Supporting Information

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SI Materials and Methods

Human Samples. The biopsy bank of the outpatient clinic of the Department of General Internal Medicine of University Hospital Schleswig-Holstein was screened for individuals with a diagnosis of Crohn disease (CD) ($n = 29$) and for healthy controls ($n = 18$) of Caucasian (northern European) ancestry. CD samples were selected based on the following criteria: (i) macro- and microscopically noninflamed sigmoid region biopsies available in biopsy bank and (ii) clinical remission with Crohn disease Activity Index (CDAI) score of <150 at time of sampling. The inflammatory activity was independently scored by two investigators. Further patient characteristics are given in [Table S1](#).

All diagnoses were based on standard criteria via radiological and endoscopic examinations. Indications for colonoscopy were monitoring of therapy response and cancer surveillance in CD patients and participation in a volunteer study for the healthy subjects (exclusion of intestinal pathologies before nutritional intervention). All samples (pairs of matching DNA/biopsy) and phenotype information were pseudonymized before the procedure. All procedures related to patients and healthy subjects were approved by the University Hospital Schleswig-Holstein ethics committee (B231/98 and A154/06) and follow the guidelines of the Declaration of Helsinki. All individuals agreed to participation by giving informed consent at least 24 h before the study.

16S rRNA Gene Pyrosequencing. The 16S rRNA gene was amplified by using forward (5'-**CTATGCGCCTTGCCAGCCCGCTCAGT-CAGAGTTTGATCCTGGCTCAG**-3') and reverse (5'-**CGTA-TCGCCTCCCTCGGCCATCAGXXXXXXXXXXCATGCTG-CCTCCCGTAGGAGT**-3') primers flanking the V1 and V2 hypervariable regions. The 454 Life Sciences primer B (forward) and A (reverse) adapter sequences are denoted in boldface, and

the underlined sequences represent the broadly conserved bacterial primers 27F and 338R. A 2-base linker sequence (TC/CA; shown in italics) was added as recommended by Roche (454). A unique 10-base multiplex identifier (designated as XXXXXX-XXXX) was added to the reverse primer to tag each PCR product. Template DNA (100 ng) was added to 25- μ L PCR reactions performed with Phusion Hot Start DNA Polymerase (Finnzymes). The cycling conditions were as follows: initial denaturation for 30 s at 98 °C; 30 cycles of 9 s at 98 °C, 30 s at 55 °C, and 30 s at 72 °C; and final extension for 10 min at 72 °C. All reactions were performed in duplicate and combined after PCR. PCR products were extracted with the Qiagen MiniElute Gel Extraction Kit and quantified with the Quant-iT dsDNA Broad-Range Assay Kit on a NanoDrop 3300 fluorometer. Equimolar amounts of purified PCR product were pooled and further purified with AMPure beads (Agencourt). A sample of each library was run on an Agilent Bioanalyzer before emulsion PCR and sequencing as recommended by Roche. Amplicon libraries were subsequently sequenced on a 454 Life Sciences GS-FLX using Titanium sequencing chemistry.

Influence of CD Subphenotypes. Because different subphenotypes of CD (i.e., ileal, colonic, ileocolonic) were present in our patient sample, we investigated their influence on the assembly of microbial communities ([Table S1](#)). We detected no significant differences in alpha diversity measures between subphenotypes (Shannon H, $F_{2,26} = 0.129$, $P = 0.88$; Shannon evenness (1), $F_{2,26} = 0.132$, $P = 0.877$; Chao1, $F_{2,26} = 0.135$, $P = 0.874$; phylogenetic diversity, $F_{2,26} = 0.041$, $P = 0.960$) nor any significant differences between communities by using measures of beta diversity (*adonis*: Jaccard, $R^2 = 0.065$, $P = 0.889$; Bray-Curtis, $R^2 = 0.06$, $P = 0.913$; unweighted UniFrac, $R^2 = 0.058$, $P = 0.981$).

1. Jost L (2007) Partitioning diversity into independent α and β components. *Ecology* 88: 2427–2439.

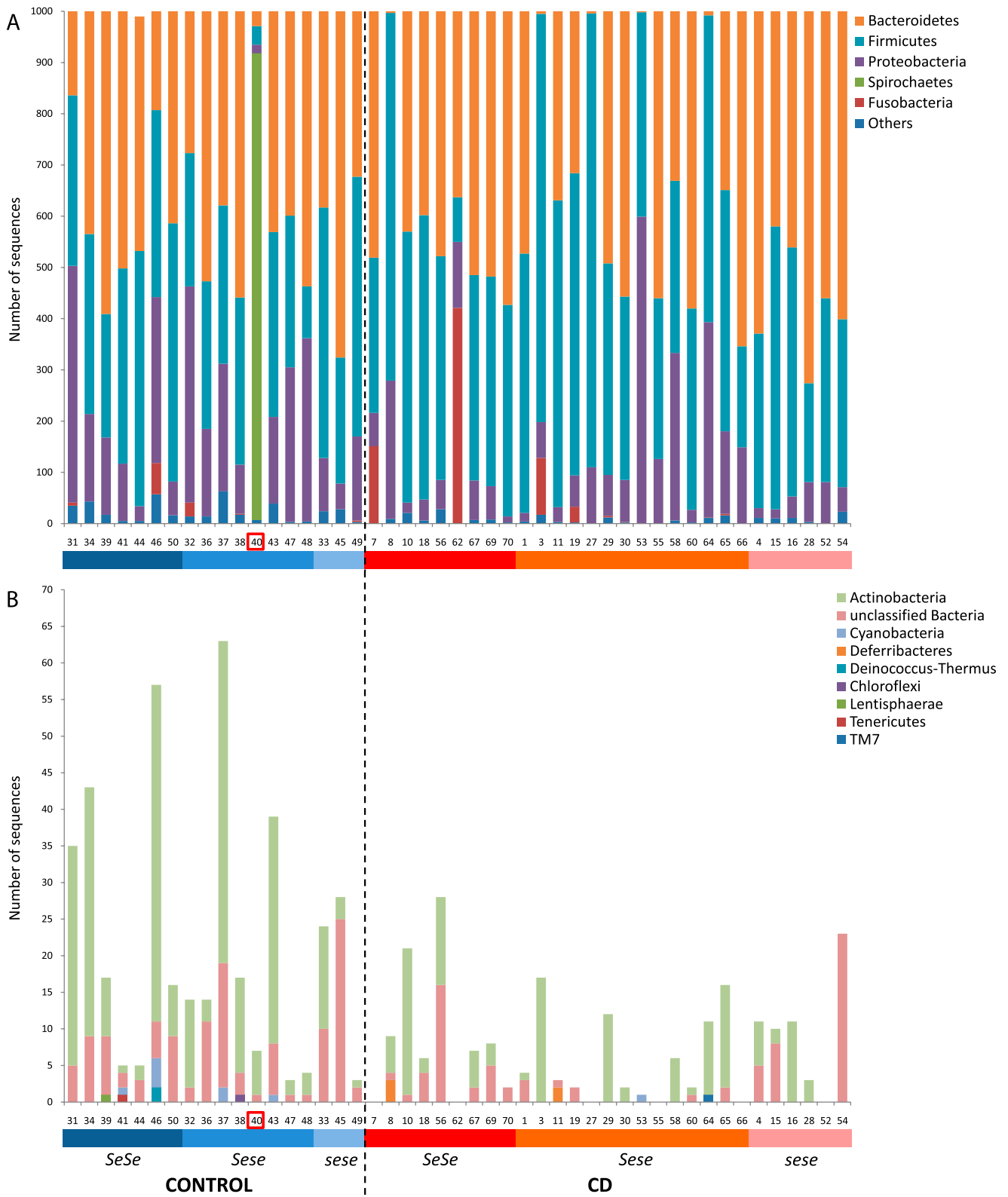


Fig. S1. (A) Distribution of read number among the major phyla in sampled individuals. Individual #40 (boxed in red) was excluded from further analysis because of suspected human intestinal spirochaetosis. (B) Read number among the rare phyla and unclassified bacteria comprising the "Others" category in A.

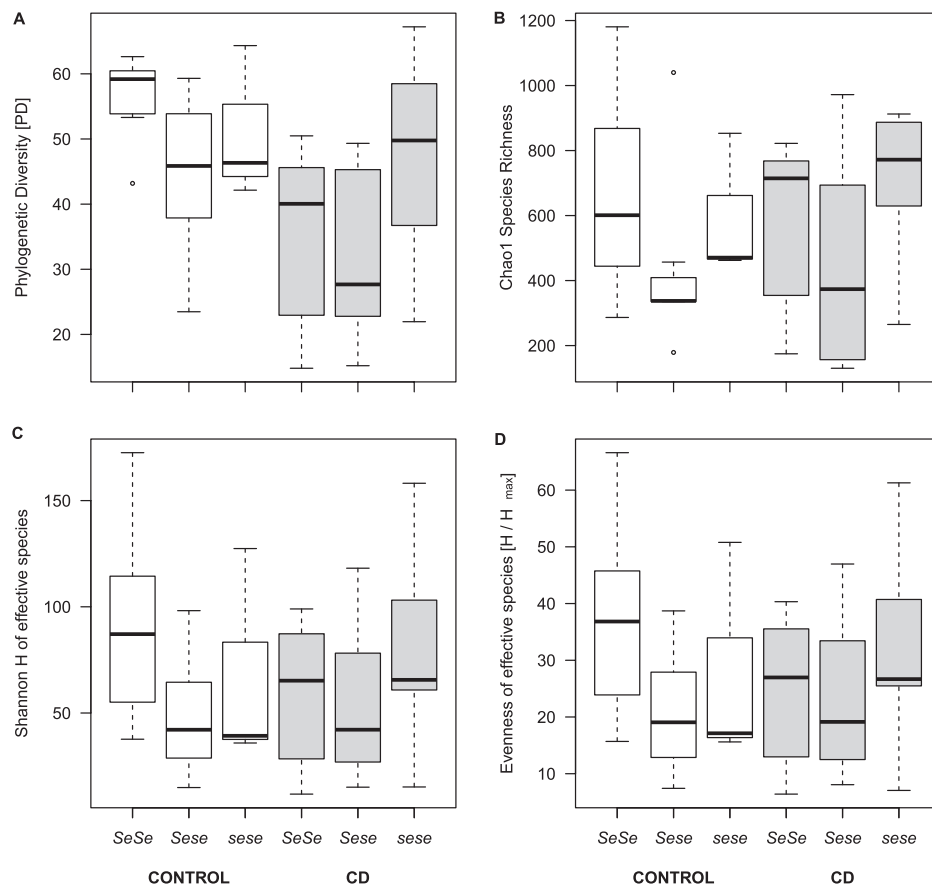


Fig. S2. Mean alpha diversity measures based on species-level operational taxonomic units (OTUs). (A) Faith’s phylogenetic diversity (1). (B) Chao1 species richness metric. (C) Shannon H of effective species numbers. (D) Shannon evenness of effective species numbers. Error bars indicate SD.

1. Faith DP (1992) Conservation evaluation and phylogenetic diversity. *Biol Conserv* 61:1–10.

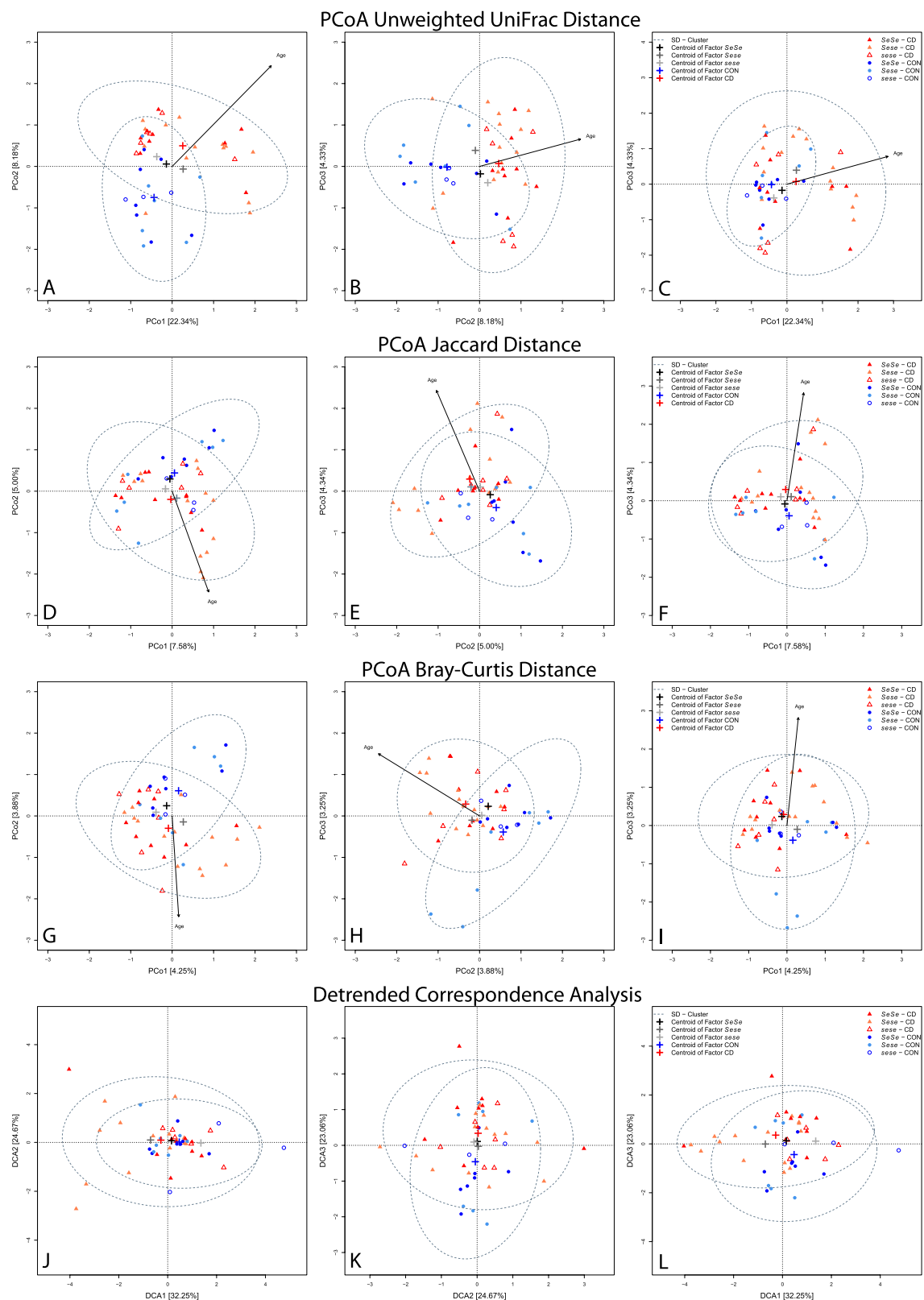


Fig. S3. (A–C) Ordination of the unweighted UniFrac distance by principal coordinate analysis (PCoA) in three dimensions (see main text for statistical analysis). (D–F) PCoA of the Jaccard index in three dimensions. Centroids of disease status (goodness of fit $R^2 = 0.1048$, $P = 0.002$), genotype within disease status ($R^2 = 0.183$, $P = 0.039$), and age (goodness of fit $R^2 = 0.263$, $P = 0.005$) are correlated with all three axes. Analysis by linear models revealed a correlation of sex with the first axis ($P = 0.047$), whereas the second and third axes show the gradients between disease status ($P = 0.009$) and age ($P = 0.001$), respectively (Table S3). (G–I) PCoA of the Bray-Curtis index (data Wisconsin-transformed) in three dimensions. The centroids of disease status (goodness of fit $R^2 = 0.154$, $P < 0.0001$), genotype within disease status (goodness of fit $R^2 = 0.26$, $P = 0.0008$), and age of the subjects (goodness of fit $R^2 = 0.213$, $P = 0.017$) are correlated with

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all three axes. Linear models of the site scores revealed a correlation of sex with the first axis ($P = 0.079$), whereas the second and third axes display significant gradients in disease status ($P = 0.0001$) and genotype within disease status ($P = 0.008$), respectively (Table S3). (J–L) Detrended correspondence analysis (DCA) visualized in three dimensions. The centroids of disease status (goodness of fit $R^2 = 0.064$, $P = 0.038$), genotype (goodness of fit $R^2 = 0.137$, $P = 0.006$), and genotype–disease status interaction ($R^2 = 0.238$, $P = 0.006$) are highly correlated with all three axes. Analysis by linear models revealed a correlation of genotype with the first axis ($P = 0.003$), whereas the second axis shows no correlation with known factors. The third dimension is significantly influenced by disease status ($P = 0.009$) (Table S3).

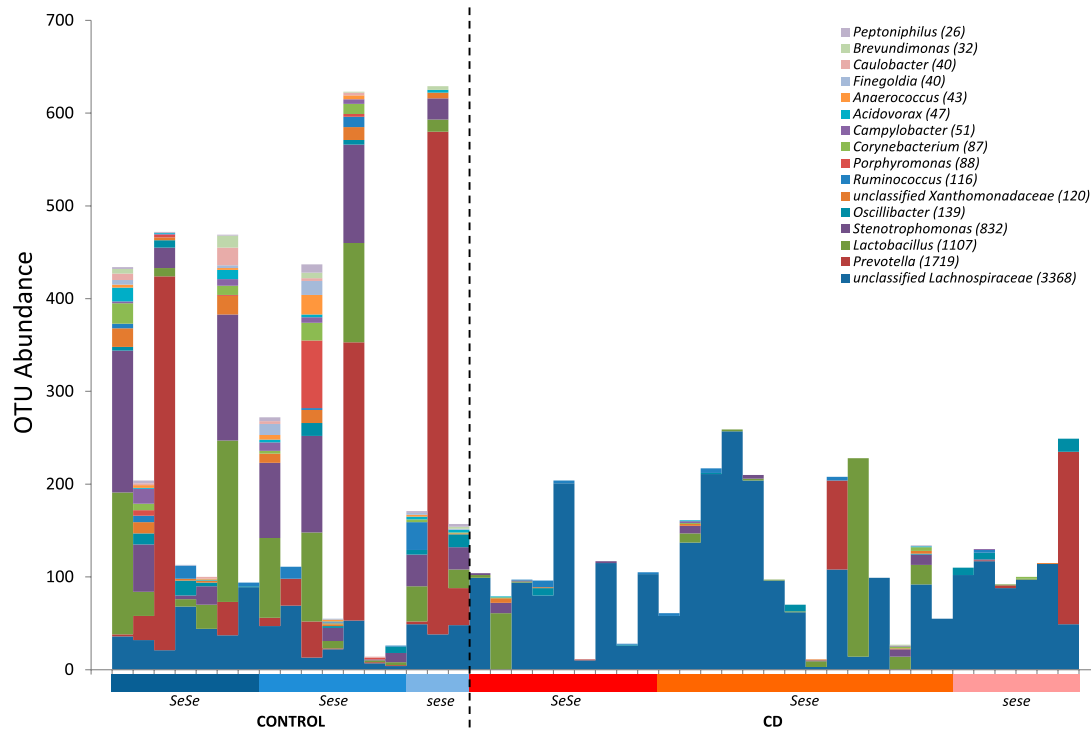


Fig. S4. Abundance of genus-level OTUs identified by indicator species analysis with respect to health status. Numbers in parentheses indicate the total read number in the normalized dataset (1,000 reads per individual).

Table S1. Patient characteristics

Parameter	CD				Control			
	All	SeSe	Sese	sese	All	SeSe	Sese	sese
<i>n</i>	29	8	15	6	18	7	8	3
Sex ratio, female/male	20/9	4/4	13/2	3/3	14/4	6/1	6/2	2/1
Median age, y	39	35.5	43	43	26	28	26.5	25
Age range, y	27–55	29–48	27–55	36–48	23–30	25–30	23–30	23–26
Medication					N/A	N/A	N/A	N/A
Cortisone	5/29	1	3	1				
Azathioprine	1/29	1	0	0				
5-Aminosalicylic acid (5-ASA)	2/29	1	0	1				
Anti-TNF α	2/29	1	0	1				
Disease subtype					N/A	N/A	N/A	N/A
Ileal	8/29	3	4	1				
Ileocolonic	16/29	3	9	4				
Colonic	4/29	2	1	1				
Undetermined	1/29	0	1	0				

N/A, not applicable; SeSe, homozygous secretor; Sese, heterozygous secretor; sese, nonsecretor.

Table S2. Statistical analysis of phyla abundances

Test	Categories tested	Phyla			
		Actinobacteria	Firmicutes	Bacteroidetes	Proteobacteria
ANOVA	Disease status	0.002	0.034	N/A	0.020
	Secretor status	N/A	N/A	0.036	N/A
	Genotype	N/A	N/A	N/A	0.047
Post hoc Mann–Whitney					
Subsets					
Secretor	Disease status	0.006*	0.005*	N/A	0.012
Nonsecretor	Disease status	0.603	0.714	N/A	0.095
Genotype	SeSe:sese	N/A	N/A	0.121	0.295
	SeSe:Sese	N/A	N/A	0.728	0.154
	Sese:sese	N/A	N/A	0.044	0.017
Control	SeSe:sese	N/A	N/A	N/A	0.5167
	SeSe:Sese	N/A	N/A	N/A	0.3374
	Sese:sese	N/A	N/A	N/A	0.067
CD	SeSe:sese	N/A	N/A	N/A	0.596
	SeSe:Sese	N/A	N/A	N/A	0.156
	Sese:sese	N/A	N/A	N/A	0.0622

Significant *P* values are indicated in boldface. N/A, no test applied.
 *Significant after sequential Bonferroni correction.

Table S3. Linear model analysis of PCoA and DCA axis scores

Ordination	Transformation	Axis	Model	Akaike information criterion	Factors	Adjusted <i>R</i> ²	<i>P</i>
PCoA	None	1, 2, 3	Null	127.205	1		
Unweighted UniFrac	None	1	Best	122.732	Disease status	0.112	0.0134
	None	2	Best	103.740	Disease status	0.412	<0.0001
	None	3	Best	124.938	Genotype	0.087	0.0534
PCoA Jaccard	None	1, 2, 3	Null	115.314	1		
	None	1	Best	113.150	Sex	0.066	0.0471
	None	2	Best	110.158	Disease status	0.125	0.0093
	None	3	Best	106.015	Age	0.200	0.0011
PCoA Bray-Curtis	Wisconsin	1, 2, 3	Null	116.312	1		
	Wisconsin	1	Best	115.060	Sex	0.047	0.0795
	Wisconsin	2	Best	102.922	Disease status	0.268	0.0001
	Wisconsin	3	Best	108.887	Disease status × genotype	0.230	0.0078
DCA	None	1	Null	175.828	1		
	None		Best	167.067	Genotype	0.207	0.0026
	None	2	Null	130.402	1		
	None		Best	> 130.402	N/A	N/A	N/A
	None	3	Null	135.010	1		
	None		Best	129.755	Disease status	0.127	0.0088

N/A, no test applied.

Table S4. List of indicator species and genera and their properties

Taxonomic level	Association	Direction	R ²	P (Benjamini and Hochberg-adjusted)*	OTU ID	OTU classification to genus level (no. of reads in normalized dataset) [†]	Comments
Species	Disease status	Control	0.435	0.00001 (0.00025)	1117	<i>Lactobacillus</i> spp. (173)	Normal member of the colon microbiome. probiotic characteristics (1, 2)
		Control	0.446	0.00007 (0.0007)	2270	<i>Lactobacillus</i> spp. (554)	Normal member of the colon microbiome, probiotic characteristics (1, 2)
		Control	0.495	0.00005 (0.00063)	2420	<i>Lactobacillus</i> spp. (57)	Normal member of the colon microbiome, probiotic characteristics (1, 2)
		Control	0.373	0.0004 (0.00286)	2267	<i>Prevotella</i> spp. (73)	Mucosa-associated anaerobe, found in the upper and lower gastrointestinal (GI) tract, pathogenic with increasing relevance and resistance (3, 4)
		Control	0.266	0.0146 (0.073)	2165	<i>Coprobacillus</i> spp. (377)	Altered abundance in inflammatory bowel disease (IBD) cases and belonging to the core microbiome of the gut (5, 6)
		Control	0.482	0.00001 (0.00025)	2460	<i>Stenotrophomonas</i> spp. (519)	Ubiquitous, nosocomial pathogen (7)
		Control	0.479	0.00014 (0.00117)	2562	<i>Stenotrophomonas</i> spp. (224)	Ubiquitous, nosocomial pathogen (7)
		Control	0.514	0.00004 (0.000625)	3631	<i>Stenotrophomonas</i> spp. (30)	Ubiquitous, nosocomial pathogen (7)
		Control	0.358	0.00194 (0.0108)	3844	<i>Faecalibacterium</i> spp. (63)	Normal member of the colon microbiome, depletion with potential role in IBD and suspected as probiotic with potential to reduce inflammatory responses (8–11)
	Genotype	Control	0.33	0.00157 (0.0098)	2551	Unclassified Clostridiales spp. (80)	Increased in dextran sodium sulfate (DSS) mouse models (12)
	sese	sese	0.545	0.00082 (0.057)	442	Unclassified Lachnospiraceae spp. (17)	Saccharolytic/cellulolytic/ amylolytic, also fucose (13, 14), associated with CD (15)
	sese	sese	0.504	0.00211 (0.073)	3774	<i>Coprococcus</i> spp. (22)	Normal member of the colon microbiome, probiotic characteristics (1, 2)
	Genotype–disease status	SeSe and Sese-Control	0.576	0.0172 (0.0909)	2420	<i>Lactobacillus</i> spp. (57)	

Table S4. Cont.

Taxonomic level	Association	Direction	R ²	P (Benjamini and Hochberg-adjusted)*	OTU ID	OTU classification to genus level (no. of reads in normalized dataset) [†]	Comments
		SeSe and Sese-Control	0.557	0.0207 (0.097)	2562	<i>Stenotrophomonas</i> spp. (224)	Ubiquitous, nosocomial pathogen (7)
		SeSe and Sese-Control	0.598	0.011 (0.065)	3631	<i>Stenotrophomonas</i> spp. (30)	Ubiquitous, nosocomial pathogen (7)
		sese-Control	0.69	0.00417 (0.0339)	2267	<i>Prevotella</i> spp. (73)	Mucosa-associated anaerobe, found in the upper and lower GI tract, pathogenic with increasing relevance and resistance (3, 4)
		sese-Control	0.516	0.01463 (0.0816)	2488	<i>Brevundimonas</i> spp. (12)	Pathogen in immunocompromised subjects and distributed in environment (16–18)
		sese-Control	0.536	0.0211 (0.097)	3689	Unclassified Lachnospiraceae spp. (34)	Increased in DSS mouse models (12)
		sese-Control	0.551	0.00449 (0.0339)	2193	<i>Sutterella</i> spp. (61)	Member of the colon microbiome and associated with GI infections (3, 10, 19, 20)
		sese-Control	0.717	0.003 (0.0302)	3844	<i>Faecalibacterium</i> spp. (63)	Normal member of the colon microbiome, depletion with potential role in IBD (8–10)
		sese-CD	0.511	0.02053 (0.0972)	4018	<i>Alistipes</i> spp. (46)	Normal member of the GI microbiome (6, 21)
		sese-CD	0.759	0.00073 (0.0302)	442	Unclassified Lachnospiraceae spp. (17)	Increased in DSS mouse models (12)
		sese-CD	0.608	0.00906 (0.0565)	3774	<i>Coprococcus</i> spp. (22)	Saccharolytic/cellulolytic/amyolytic, also fucose (13, 14), associated with CD (15)
Genus	Disease status	Control	0.498	0.00003 (0.00036)		Unclassified Xanthomonadaceae (120)	Associated with gut and oral microbiome but majorly environmental organisms with some pathogenicity, especially in plants (7, 22–25)
		Control	0.409	0.00001 (0.00024)		<i>Acidovorax</i> (47)	Skin microbiome, associated with earthworm development and gut symbiosis (26–28)
		Control	0.540	0.00001 (0.00024)		<i>Stenotrophomonas</i> (832)	Ubiquitous, nosocomial pathogen (7)
		Control	0.342	0.00823 (0.03039)		<i>Ruminococcus</i> (116)	Core gut and fecal microbiome, increased in healthy subjects (6, 29, 30)

Table S4. Cont.

Taxonomic level	Association	Direction	R ²	P (Benjamini and Hochberg-adjusted)*	OTU ID	OTU classification to genus level (no. of reads in normalized dataset) [†]	Comments
	Control	Control	0.306	0.01572 (0.0503)		<i>Prevotella</i> (1,719)	Mucosa-associated pathogen with increasing relevance and resistance (3)
	Control	Control	0.210	0.00136 (0.00593)		<i>Porphyromonas</i> (88)	Saccharolytic, mucosa-associated, cause of gingivitis and soft tissue infections (3, 14)
	Control	Control	0.406	0.00025 (0.001714)		<i>Peptoniphilus</i> (26)	Chronic soft tissue infections, diabetic ulcers, periodontitis (31–33)
	Control	Control	0.386	0.00975 (0.0334)		<i>Oscillibacter</i> (139)	Anaerobic <i>Oscillibacter</i> was identified in clump intestines, unknown ecology but found in healthy human guts (34–36)
	Control	Control	0.331	0.020603 (0.07809)		<i>Lactobacillus</i> (1,107)	Normal member of the colon microbiome, probiotic characteristics (1, 2)
	Control	Control	0.360	0.00012 (0.00096)		<i>Finegoldia</i> (40)	Opportunistic pathogen on mucosa (37), associated with chronic infections (31, 32)
	Control	Control	0.396	0.00061 (0.00336)		<i>Corynebacterium</i> (87)	Highly diverse group, pathogens and saprophytes (38–40)
	Control	Control	0.343	0.00009 (0.000864)		<i>Caulobacter</i> (40)	Stomach flora (41)
	Control	Control	0.390	0.00072 (0.003456)		<i>Campylobacter</i> (51)	Cause of enteritis and increased IBD risk (42)
	Control	Control	0.350	0.00246 (0.00984)		<i>Brevundimonas</i> (32)	Pathogen in immunocompromised subjects and distributed in environment (16–18)
	Control	Control	0.344	0.00002 (0.00032)		<i>Anaerococcus</i> (43)	Chronic soft tissue infections, diabetic ulcers, periodontitis (31–33)
	CD	CD	0.486	0.00063 (0.00336)		Unclassified Lachnospiraceae (3,368)	Increased in DSS mouse models (12)

*Values in boldface indicate significance after Benjamini and Hochberg adjustment (43).

[†]Classification obtained on the genus level by Ribosomal Database Project (RDP) classifier at the 80% bootstrap threshold (44, 45).

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Dataset S1. OTU abundances per individual at 97% sequence similarity threshold

[Dataset S1](#)