Supplementary Table 1. Metabolite purity and concentrations used in the single-dose screen. Purity was determined by running the metabolites in LC-MS in positive and negative ion modes. Concentrations used in the single-dose screen were chosen with regard to metabolite solubility in DMSO (most metabolites were soluble at 100 mM) in order to reach a final DMSO concentration in each well that did not exceed 0.25%. N/d — not detectable by the methods used for our quality control.

Metabolite	Purity (%)	Concentration (μM)
linoleoyl ethanolamide	99	250
D-sphingosine	96.3	140
phytosphingosine	99.4	250
N,N-dimethylarginine	99.3	250
creatine	97.2	125
N-acetylputrescine	91.7	250
C16:0 LPC	95.9	50
L-lactic acid	n/d	250
taurine	86.7	250
L-carnitine	99.9	125
C18:1 CE	n/d	50
eicosatrienoic acid	11.66	250
eicosapentaenoic acid	13.49	250
arachidonic acid	28.67	250
docosahexaenoic acid	79.36	250
deoxycarnitine	96.9	250
N-acetyl-L-arginine	85.3	250
azelaic acid	99.6	250
sebacic acid	98.3	250
suberic acid	99	250
hexanoic acid	n/d	250
caprylic acid	38.18	250
dodecanedioic acid	98.7	250
pyrocatechol	98.31	250
4-methylcatechol	98.16	250
linoleic acid	99.6	250
alpha-linolenic acid	87.21	250
hydrocinnamic acid	98.36	250
pantothenic acid	84.4	250
adenine	93.9	250

nicotinic acid	97.4	250
pyridoxamine	98.9	250
putrescine	98.2	250
histamine	98.8	250
cadaverine	n/d	250
4-guanidinobutanoic acid	47.9	125
L-fucose	n/d	250
glyceric acid	98.5	250
oleamide	99.2	250
C18:1 LPC	98.9	62.5
L-tryptophan	94.05	250
2-palmitoylglycerol	96.7	250
butyric acid	n/d	250
isovaleric acid	n/d	250
trimethylbenzene	n/d	250
phenylacetic acid	n/d	250
propionic acid	n/d	250
cyclohexylamine	n/d	250
ascorbic acid	n/d	190
urocanic acid	90	250
DMSO control	n/d	14

Supplementary Table 2. Bacterial isolates used in this study. The identity of the individual isolates was determined by PCR amplification and subsequent sequencing of the *rrs* gene (16S ribosomal RNA) using primers 27F and 1492R[1]. All isolates are from PRISM subjects except for strain RJX1118, which was isolated from an infant enrolled in the DIABIMMUNE cohort, a risk assessment study for the development of type 1 diabetes[2].

CD ─ Crohn's disease patient, UC ─ ulcerative colitis patient, n/a ─ not available

Isolate ID	Sample source	IBD Condition	Sanger 16S ID	Abundance in IBD (PRISM)
RJX1262	stool	CD- L1 Ileum	*Lactobacillus gasseri*	Enriched
RJX1083	stool	CD	*Escherichia coli*	Enriched
RJX1118	stool	n/a	*Ruminococcus gnavus*	Enriched
RJX1084	stool	non-IBD	*Alistipes shahii*	Depleted
RJX1085	stool	non-IBD	*Ruminococcus lactaris*	Depleted
RJX1086	stool	UC	*Streptococcus salivarius*	Depleted
RJX1251	stool	CD- L3 Ileocolon	*Enterococcus faecalis*	Unchanged

Supplementary Table 3. Metabolic functions significantly changing in response to NAE addition.

Class	KEGG module
Energy metabolism	M00144, M00150, M00157, M00169, M00417
Amino acid metabolism	M00019, M00020, M00021, M00027, M00570
Metabolism of cofactors and vitamins	M00115, M00117, M00123, M00125, M00126, M00127, M00140
Carbohydrate metabolism	M00003, M00005, M00006, M00010, M00549
Glycan metabolism	M00063, M00064
Lipid metabolism	M00086, M00093
Biosynthesis of terpenoids and polyketides	M00096

Supplementary Information References

1 Lane, D. J. *16S/23S rRNA sequencing*. 115-175 (John Wiley and Sons, 1991).

2 Vatanen, T. *et al.* Variation in Microbiome LPS Immunogenicity Contributes to Autoimmunity in Humans. *Cell* **165**, 1551, doi:10.1016/j.cell.2016.05.056 (2016).