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Supplementary Material 1.

Real-time PCR for analysis of the faecal content: For enterobacteria qPCR, forward primer tuf-F, 5´-TGGTCAGGTACTGGCTAAGC-3´; reverse primer tuf-R, 5´-TCTTTGGACAGAATGTACACTTCA-3´ and probe tuf-S, 5´-CCATCAAGCCGCACACCAAGTTCG-3´ were used. Primers and probes were synthesized by Metabion (Martinsried, Germany). We used qPCR Mastermix No ROX (Eurogentec, Seraing, Belgium). Amplification of the DNA was carried out on CFX96 Cycler Version 1.5.534.0511 (BioRad, Munich, Germany), with the following schema of cycles: 50°C for 2 min and 95°C for 10 min, followed by 45 cycles of 95°C for 15s and 60°C for 1 min. To exclude cross-reactivity with other bacteria of faecal samples, we tested potential cross-reactivity or inhibition of the L. reuteri real-time PCR with pathogens of fecal samples (e.g. E.coli, C.perfringens, S.aureus). To achieve quantitative results of bacterial DNA in the samples we generate PCR-standards with exact amount of relevant DNA-copies. We used pCR®2.1-(Invitrogen, Carlsbad, USA) as cloning vector for the species-specific amplificates of each real-time PCR, and propagated the resulting plasmids in E. coli. The clones with inserts of anticipated lengths, as determined by gel electrophoretic separation of the Xho I und Hind III-digested plasmid DNA (Fermentas, St. Leon-Rot, Germany) in 2% agarose gels, were used for preparation of standards.
Due to the different sequence- and lengths-dependent amplification efficiencies of the L. reuteri species- and genus-specific qPCRs the concentrations of L. reuteri of each sample was recalculated in amplifying a high and low concentrated sample of genomic L. reuteri DNA in both qPCRs. Based on the copy of numbers estimated in Lac. spp qPCR the L. reuteri values were normalized accordingly.

Next-generation sequencing for analysis of the fecal content: Amplicon sequencing of the fecal microbiome was done at the University of Minnesota Genomics Center. Briefly, the V5-V6 region of the 16S rRNA gene was PCR-enriched using the primer pair 784F (5'-RGGATTAGATACCC-3') and 1064R (5'-CGACRRCCATGCANCACCT-3') in a 25 µl PCR reaction containing 5 µl of template DNA, 5 µl of 2X HotStar PCR master mix, 500 nM of final concentration of primers and 0.025 U/µl of HostStar Taq+ polymerase (QIAGEN). PCR-enrichment reactions were conducted as follows, an initial denaturation step at 95°C for 5 min followed by 20-25 cycles of denaturation (50 s at 94°C), annealing (30 s at 40°C), and elongation (30 s at 72°C).
Next, the PCR-enriched samples were diluted 1:100 in water for input into library tailing PCR. The PCR reaction was analogous to the one conducted for enrichment except with a Taq polymerase concentration of 0.25 U/ µl, while the cycling conditions used were as follows, initial denaturation at 95°C for 5 min followed by 10-15 cycles of denaturation (50 s at 94°C), annealing (30 s at 40°C), and elongation (1 min at 72°C). The primers used for tailing are presented in Supplementary Table 1.
The resulting PCR products were quantitated by PicoGreen (Life Technologies). A subset of the amplicon libraries were spot-checked on a Bioanalyzer High-Sensitivity DNA Chip (Agilent Technologoies) for correct amplicon size. Next, samples were normalized to 2nM and pooled together. The total volume of the libraries was reduced by SpeedVac and amplicons were size-selected at 420 bp +/- 20% using the Caliper XT (Perkin Elmer). Next, library pools were cleaned-up by 1.8X AMPureXP beads (Beckman Coulter) and eluted in water. The final pool was quantitated by PicoGreen and normalized to 2 nM for input into Illumina MiSeq (v3 Kit) to produce 2x300 bp sequencing products. Clustering was done at 10 pM with a 5% spike of PhiX.
Initial quality filtering of the reads was performed with the Illumina Software, yielding an average of 52060 pass-filter reads per sample. Quality scores were visualized with the FastQC software (http://www.bioinformatics.babraham.ac.uk/publications.html), and reads were trimmed to 240 bp with the FASTX-Toolkit (http://hannonlab.cshl.edu/fastx_toolkit/). Next, reads were merged with the merge-illumina-pairs application (with p-value = 0.03, enforced Q30 check, perfect matching to primers which

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are removed by the software, and otherwise default settings including no ambiguous nucleotides allowed) (1). For samples with >20000 merged reads, a subset of 20000 reads was randomly selected using Mothur 1.32.1 centos 5.5 for Linux (2), to avoid large disparities in the number of sequences. One sample was eliminated from the analysis as it only contained 4580 reads, all other samples had at least 16269 of quality-controlled reads.

Subsequently, the UPARSE pipeline implemented in USEARCH v7.0.1001 (3) was used to further process the sequences. Putative chimeras were identified against the Gold reference database and removed. Clustering was performed with 98% similarity cutoff to designate Operational Taxonomic Units (OTUs). Non-chimeric sequences were also subjected to taxonomic classification using the RDP MultiClassifier 1.1 from the Ribosomal Database Project (4), for phylum to genus characterization of the fecal microbiome.

The phylotypes were computed as percent proportions based on the total number of sequences in each sample. Alpha-diversity indexes (Chao1, Simpson, Shannon) and beta-diversity indexes (Bray-Curtis, Morisita-Horn, weighted Unifrac) were calculated using QIIME. PCoA plot of the beta-diversity indexes were obtained using EMPeror (5). Statistical analysis of the alpha-diversity indexes was performed as described in the supplementary material 2 Calculations and statistics, whereas comparison of specific changes in relative abundance during the intervention period was done using the Response Screening Function in JMP 11.0, which includes a false-discovery correction according to the Benjamini and Hochberg procedure (6).

The sequences used for analysis can be found in the MG-RAST database (7), with the following accession numbers: 4632903.3, 4632912.3, 4632929.3, 4632930.3, 4632891.3, 4632892.3, 4632893.3, 4632894.3, 4632895.3, 4632896.3, 4632897.3, 4632898.3, 4632899.3, 4632900.3, 4632901.3, 4632902.3, 4632904.3, 4632905.3, 4632906.3, 4632907.3, 4632908.3, 4632909.3, 4632910.3, 4632911.3, 4632913.3, 4632914.3, 4632915.3, 4632916.3, 4632917.3, 4632918.3, 4632919.3, 4632920.3, 4632921.3, 4632922.3, 4632923.3, 4632924.3, 4632925.3, 4632926.3, 4632927.3, 4632928.3.

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Supplementary Table 1. PCR tailing primers used for library preparation for sequencing of the V5-V6 16S rRNA gene tags of the fecal microbial community. V5F – forward primers, V6R – reverse primers.

V5F_D501	AATGATACGGCGACCACCGAGATCTACACTATAGCCTACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNRGGATTAGATACCC
V5F_D502	AATGATACGGCGACCACCGAGATCTACACATAGAGGCACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNRGGATTAGATACCC
V5F_D503	AATGATACGGCGACCACCGAGATCTACACCTATCCTACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNNRGGATTAGATACCC
V5F_D504	AATGATACGGCGACCACCGAGATCTACACGGCTCTGAACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNNNRGGATTAGATACCC
V5F_D505	AATGATACGGCGACCACCGAGATCTACACAGGCGAAGACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNRGGATTAGATACCC
V5F_D506	AATGATACGGCGACCACCGAGATCTACACTAATCTTAACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNRGGATTAGATACCC
V5F_D507	AATGATACGGCGACCACCGAGATCTACACCAGGACGTACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNNRGGATTAGATACCC
V5F_D508	AATGATACGGCGACCACCGAGATCTACACGTACTGACACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNNNRGGATTAGATACCC
V5F_N501	AATGATACGGCGACCACCGAGATCTACACTAGATCGCACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNRGGATTAGATACCC
V5F_N502	AATGATACGGCGACCACCGAGATCTACACCTCTCTATACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNRGGATTAGATACCC
V5F_N503	AATGATACGGCGACCACCGAGATCTACACTATCCTCTACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNNRGGATTAGATACCC
V5F_N504	AATGATACGGCGACCACCGAGATCTACACAGAGTAGAACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNNNRGGATTAGATACCC
V5F_N505	AATGATACGGCGACCACCGAGATCTACACGTAAGGAGACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNRGGATTAGATACCC
V5F_N506	AATGATACGGCGACCACCGAGATCTACACACTGCATAACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNRGGATTAGATACCC
V5F_N507	AATGATACGGCGACCACCGAGATCTACACAAGGAGTAACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNNRGGATTAGATACCC
V5F_N508	AATGATACGGCGACCACCGAGATCTACACCTAAGCCTACACTCTTTCCCTACACGACGCTCTTCCGATCTNNNNNNNRGGATTAGATACCC
V6R_D701	CAAGCAGAAGACGGCATACGAGATCGAGTAATGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNCGACRRCCATGCANCACCT
V6R_D702	CAAGCAGAAGACGGCATACGAGATTCTCCGGAGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNCGACRRCCATGCANCACCT
V6R_D703	CAAGCAGAAGACGGCATACGAGATAATGAGCGGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNNCGACRRCCATGCANCACCT
V6R_D704	CAAGCAGAAGACGGCATACGAGATGGAATCTCGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNNNCGACRRCCATGCANCACCT
V6R_D705	CAAGCAGAAGACGGCATACGAGATTTCTGAATGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNCGACRRCCATGCANCACCT
V6R_D706	CAAGCAGAAGACGGCATACGAGATACGAATTCGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNCGACRRCCATGCANCACCT
V6R_D707	CAAGCAGAAGACGGCATACGAGATAGCTTCAGGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNNCGACRRCCATGCANCACCT
V6R_D708	CAAGCAGAAGACGGCATACGAGATGCGCATTAGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNNNCGACRRCCATGCANCACCT
V6R_D709	CAAGCAGAAGACGGCATACGAGATCATAGCCGGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNCGACRRCCATGCANCACCT
V6R_D710	CAAGCAGAAGACGGCATACGAGATTTCGCGGAGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNCGACRRCCATGCANCACCT
V6R_D711	CAAGCAGAAGACGGCATACGAGATGCGCGAGAGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNNCGACRRCCATGCANCACCT
V6R_D712	CAAGCAGAAGACGGCATACGAGATCTATCGCTGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNNNCGACRRCCATGCANCACCT
V6R_N701	CAAGCAGAAGACGGCATACGAGATTAAGGCGAGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNCGACRRCCATGCANCACCT
V6R_N702	CAAGCAGAAGACGGCATACGAGATCGTACTAGGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNCGACRRCCATGCANCACCT
V6R_N703	CAAGCAGAAGACGGCATACGAGATAGGCAGAAGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNNCGACRRCCATGCANCACCT
V6R_N704	CAAGCAGAAGACGGCATACGAGATTCCTGAGCGTGACTGGAGTTCAGACGTGTGCTCTTCCGATCTNNNNNNNCGACRRCCATGCANCACCT

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Viability of L. reuteri: Nutraceutix provided us with the patented BIO-tract® tablets, which protect probiotics from gastric acid, ensuring that a large proportion of the organisms reach the intestine alive (The United States Patent Office, Patent No. 8,007,777 *"Delivery System For Biological Component"* for BIO-tract® dietary supplements containing probiotics).
In preliminary studies we proved the viability of *L. reuteri* derived from the capsules provided by Nutraceutix and from feces samples obtained from participants who ingested *L. reuteri* or Placebo. The pilot study demonstrated the viability of *L. reuteri* both directly from the capsules diluted in physiological saline solution for >24 h and from ingestion by measuring cell recovery from feces on selective rogosa-agara plates for lactobacilli.

Supplementary Material 2.

Calculations and statistics: Data are presented as means and standard deviations (mean ± SD) or medians and interquartile range (median [IQR]), as appropriate. Variables with skewed distribution were log transformed before further analysis. To test differences between treatment arms taking the lean-obese status into account, we used two-way ANOVA
To test differences between treatment arms taking the lean-obese status into account, we first used two-way ANOVA with interaction. In case of a statistically significant interaction term, results were reported separately for the lean and obese group otherwise two-way ANOVA without interaction were used to adjust the treatment effect for the lean/obese status and results were reported for the combined groups. In case variables were not normally distributed before or after log-transformation, differences between treatments were tested by one-way Kruskal–Wallis analysis of variance applied separately to samples of lean and obese persons.

Correlation analysis: Pearson's correlation coefficients were used to describe inter-individual (across persons) correlations between variables and also used to calculate intra-individual (within subject) correlation between variables measured during the OGTT and isoglycemic test. To test whether the mean intra-individual correlations are different from zero, one-sample t-test were performed after normalizing the single intra-individual correlations by applying Fisher's-z-transformation.
P-values from two-sided tests less than or equal to 5% were considered to indicate significant differences. SAS for Windows Version 9.2 (SAS Institute, Cary, NC, USA) was used.

Sample size: Due to missing data of changes of incretins upon ingestion of probiotics, group size was calculated in order to detect a mean difference of one SD before versus after intervention, with a probability (power) of at least 80%. Mean differences between verum and placebo of at least 1.33 SD can be detected with a power of 80%. In the absence of probiotic studies reporting changes of incretins, specifically GLP-1, previous studies with prebiotics were used to perform the power calculation (8).

Randomization method: For allocation of the participants, a computer-generated list of random numbers was used. Randomization sequence was created using SAS for Windows software with a 1:1 allocation within the groups of lean and obese participants.

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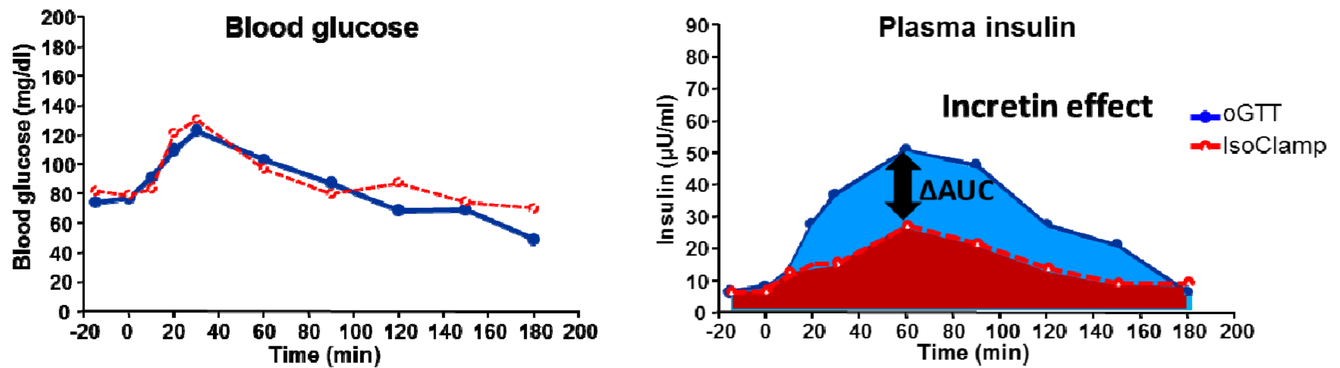
Illustration of calculation of the Δ area under the curve (AUC) to measure biological incretin effect



Calculation of **ΔAUC** = (AUC**oGTT** – AUC**clamp**)

Comparision of **ΔAUC before** and **ΔAUC after Intervention**

Shown are the results of one representative subject

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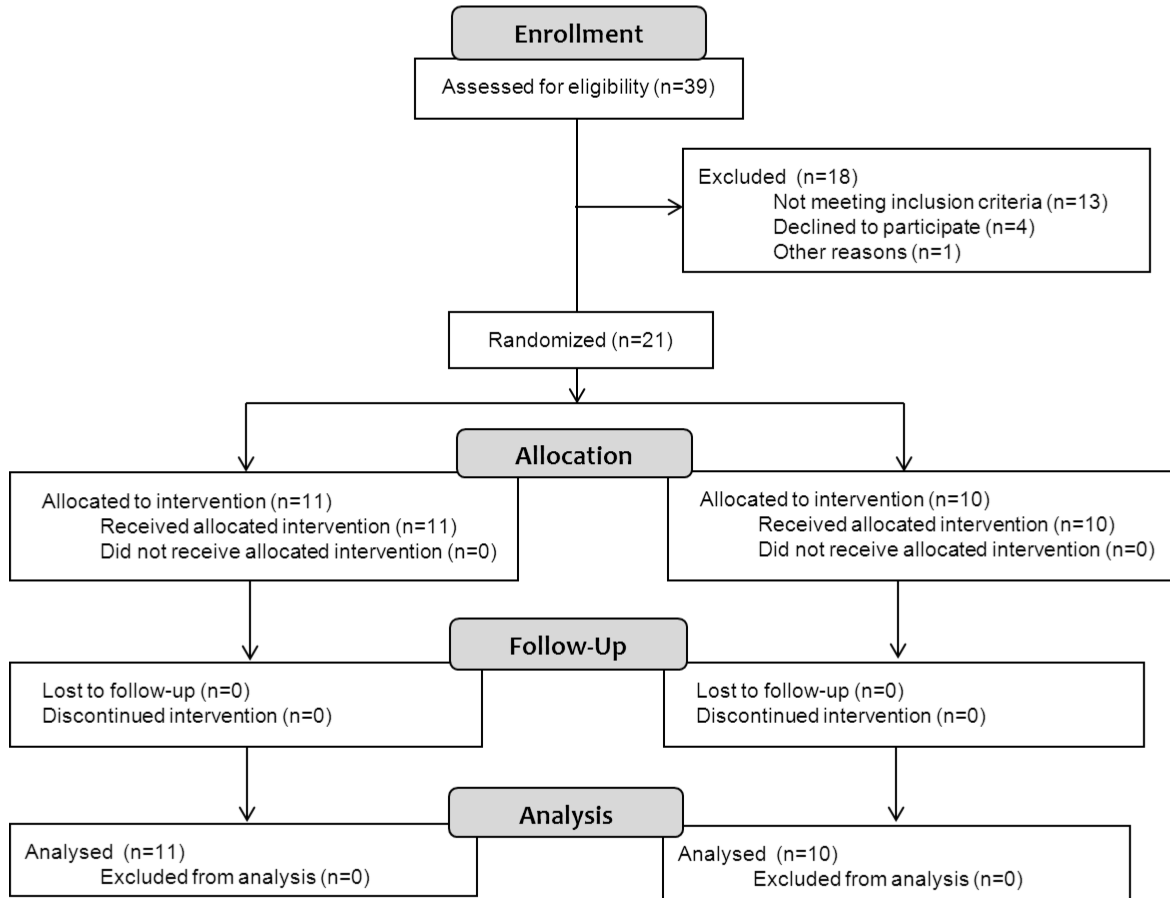
Supplementary Figure 1. Enrollment and allocation of participants according CONSORT flow diagram 39 persons were screened for the study, 13 persons did not pass the inclusion criteria (two because of antibiotics treatment, one because of weight changes before study start, one because of common cold during run-in phase), four persons abstained from the study as they did not want to follow dietary restrictions, one stopped participating without giving reasons. Of the remaining 21 participants, 5 lean received placebo and 6 lean received *L. reuteri,* whereas 5 obese received placebo and 5 obese received *L. reuteri*.



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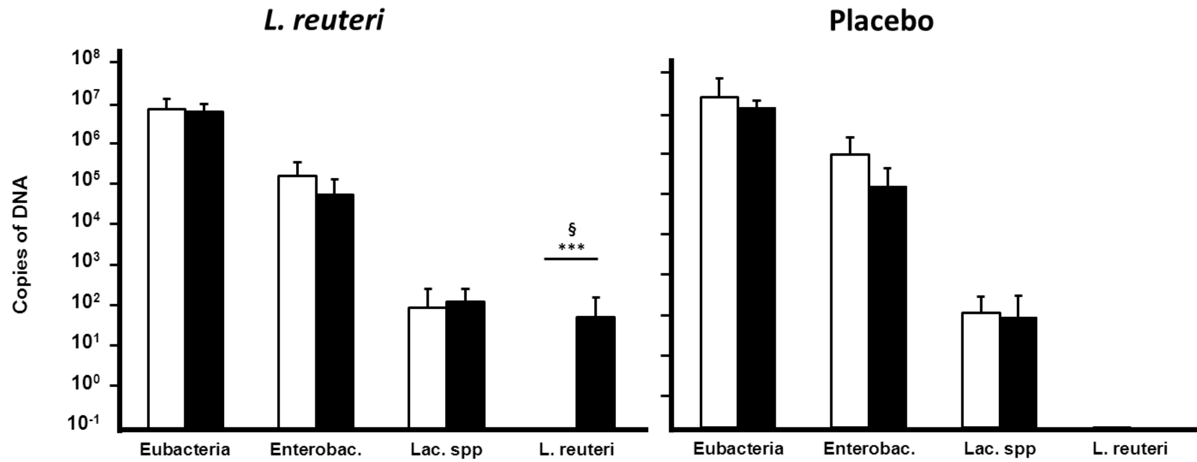
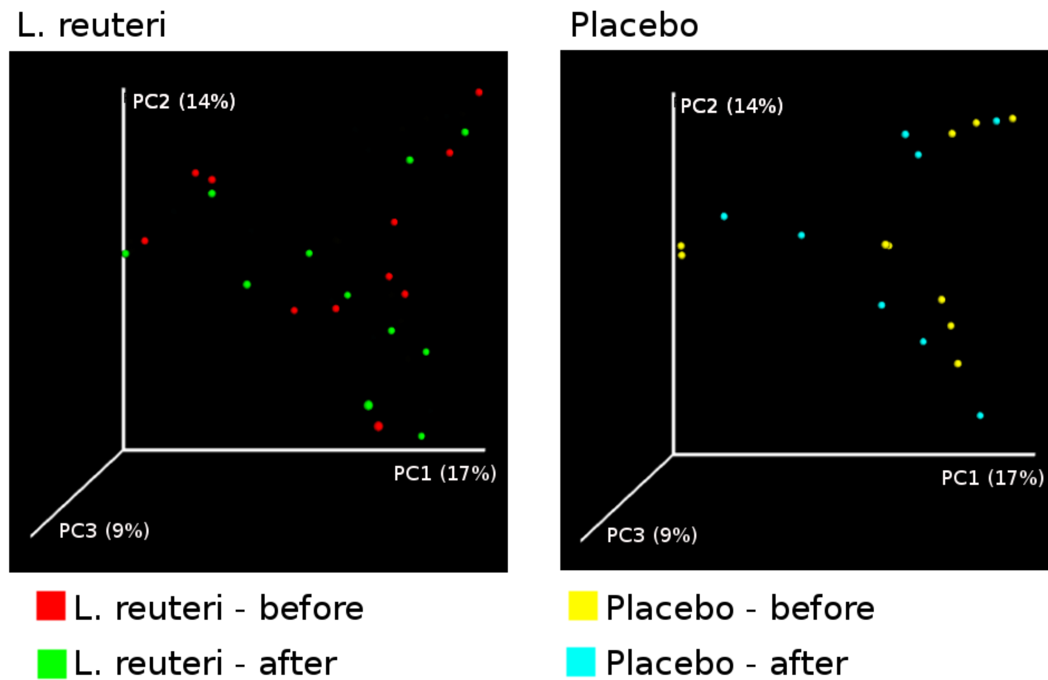
Supplementary Figure 2. Analysis of fecal samples (A) Total bacterial load (eubacteria), enterobacterial content (enterobac.), and Lactobacillus content (*Lac. spp*), were comparable between intervention- and placebo-group, and did not change upon treatment (black bars). Open bars, before intervention. *L. reuteri* content was increased within the intervention-group (§p<0.0001) and compared to the placebo-group (***p<0.0001, Fisher´s exact t-test), at the end of trial. Data are shown as mean+SD. (B) Principal coordinate analysis of Morisita-Horn distance metrics. The analysis included both treatment and placebo. Results are plotted on two graphs for clarity purpose.

A



B



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Supplementary Table 2A. Anthropometric data, ectopic fat content and changes of insulin sensitivity and β-cell function indices upon intervention

		L. reuteri	**Placebo**
Body weight (kg)	before	87±23	87±22
	after	87±23	88±22
Body fat (%)	before	35±7	34±11
	after	33±8	34±11
Lean body mass (kg)	before	56±13	56±12
	after	58±13	56±12
Hepatocellular lipids (%H_2O)	before	1.6 [0.5 - 15.9]	2.4 [1.8 - 14.7]
	after	2.7 [0.6 - 14.9]	1.5 [0.9 - 21.7]
Intramyocellular lipids (%H_2O) m. soleus	before	1.4 [0.9-1.7]	1.5 [0.9 - 2.3]
	after	1.1 [0.6 - 2.2]	1.2 [0.7 - 2.1]
Intramyocellular lipids (%H_2O) m. tibialis ant.	before	0.5 [0.3 - 1.0]	0.8 [0.4 - 0.8]
	after	0.7 [0.6 - 1.0]	0.6 [0.2 - 0.8]
OGIS	before	476±95	533±100
	after	521±128	527±74
Adaptation index	before	0.578 [0.480- 0.835]	0.765 [0.489 - 0.947]
	after	**0.793 [0.606 - 0.874]***	**0.728 [0.505 - 0.822]**
Disposition index	before	3.05 [2.21 - 4.28]	3.89 [2.24 - 5.78]
	after	**3.74 [2.85 - 7.97]***	**2.69 [2.24 - 4.59]**
Insulinogenic index	before	153 [143 – 660]	551 [252 – 714]
	after	**264 [131 – 307]**	**261 [156 – 472]***

Mean±SD or median [interquartile range] are given for normal and log-normal distributed data, respectively. Significant differences (before/after intervention) between *L. reuteri* and placebo are marked in bold, * significant difference before/after intervention within treatment group, $p<0.05$.

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Supplementary Table 2B. Anthropometric data and ectopic fat content at baseline

		L. reuteri		**Placebo**	
		Lean	**Obese**	**Lean**	**Obese**
Body weight (kg)	before	71±13	106±15	69±11	105±13
Body fat (%)	before	30±3	40±5	27±7	40±11
Lean body mass (kg)	before	49±13	63±10	50±11	62±11
Hepatocellular lipids (%H2O)	before	0.004 [0.00 – 0.009]	0.159 [0.088 – 0.348]	0.021 [0.015 – 0.027]	0.147 [0.025 – 0.219]
Intramyocellular lipids (%H2O) m. soleus	before	0.010 [0.008 - 0.014]	0.017 [0.012 – 0.022]	0.018 [0.009 – 0.024]	0.015 [0.009 – 0.019]
Intramyocellular lipids (%H2O) m. tibialis ant.	before	0.005 [0.003 - 0.007]	0.008 [0.003 - 0.013]	0.008 [0.007 – 0.011]	0.004 [0.002 – 0.008]

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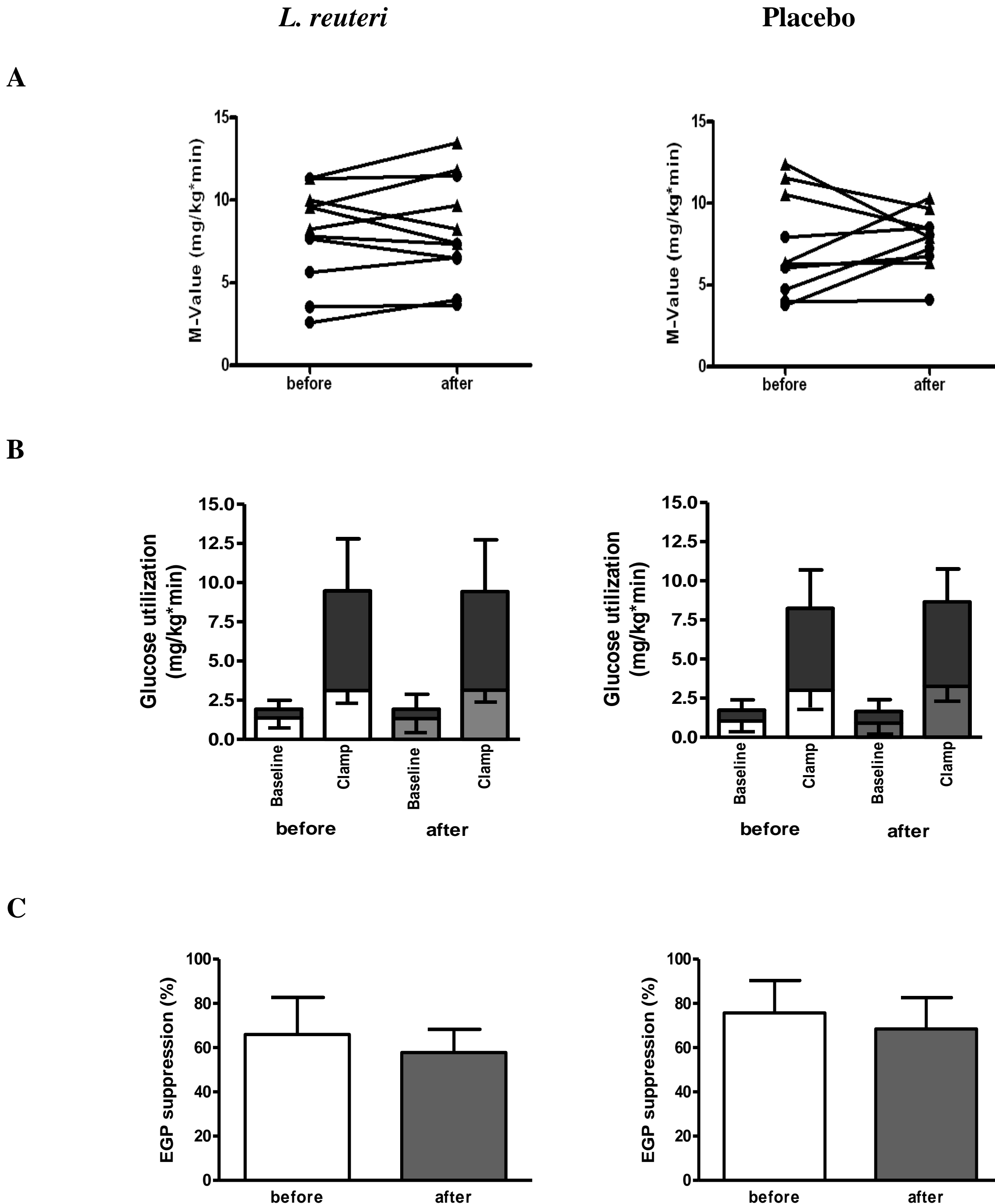
Supplementary Figure 3. Results of euglycemic-hyperinsulinemic clamp (A) M-values, representing whole body insulin sensitivity, are shown as individual data, lean (triangle), obese (circles), (B) Glucose utilization assessed at baseline and during steady state of euglycemic-hyperinsulinemic clamp. Grey bars represent non-oxidative and open bars oxidative glucose utilization, shown as mean±SD. (C) Endogenous suppression of glucose production (EGP) represents hepatic insulin sensitivity. Bars show median and IQR, before (open), and after intervention (grey).



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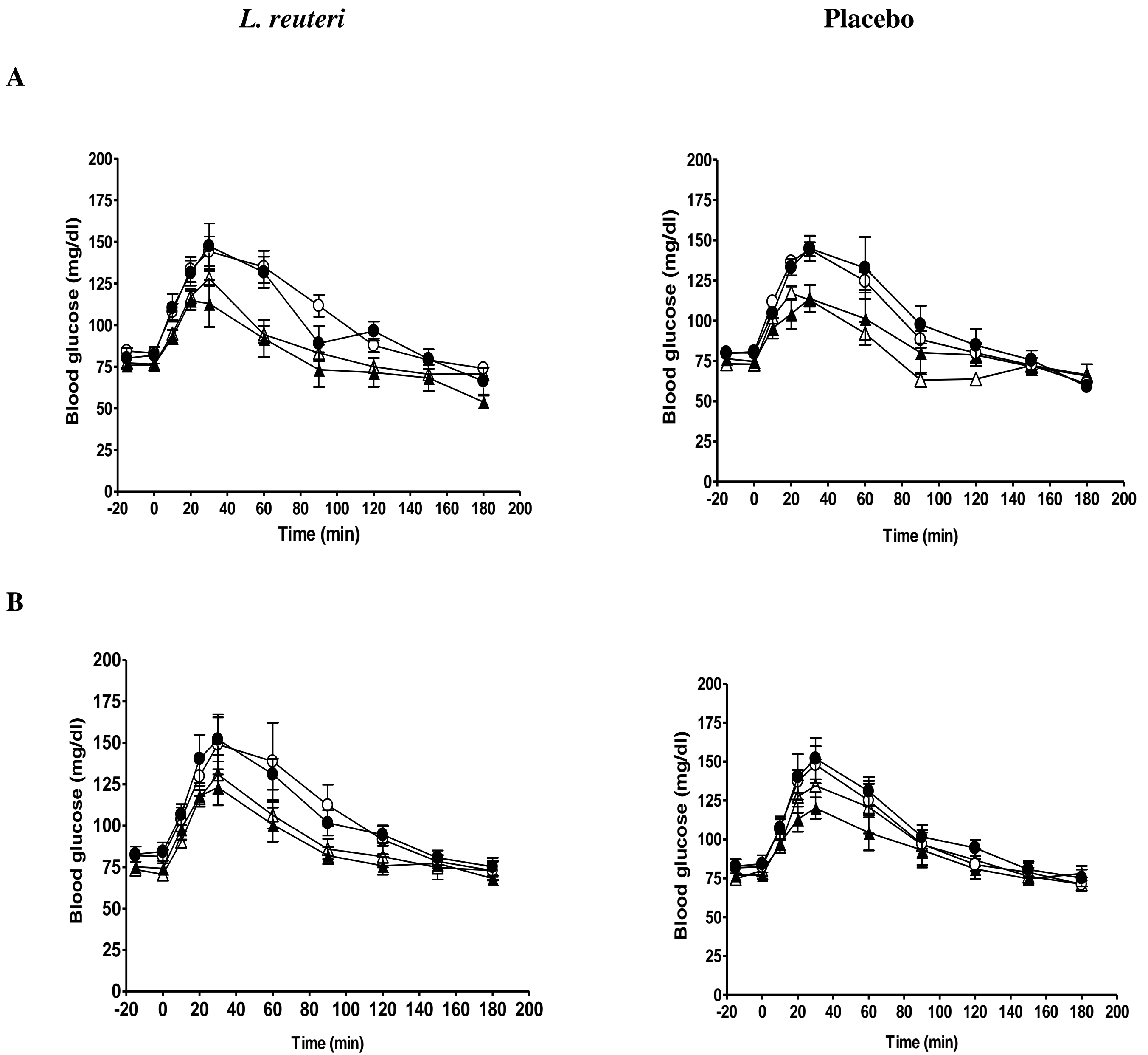
Supplementary Figure 4. Results of OGTT (A) and isoglycemic i.v. glucose infusion (B) Shown are blood glucose levels from lean (triangles) and obese (circles) persons before (open) and after intervention (closed symbols) as mean±SEM.



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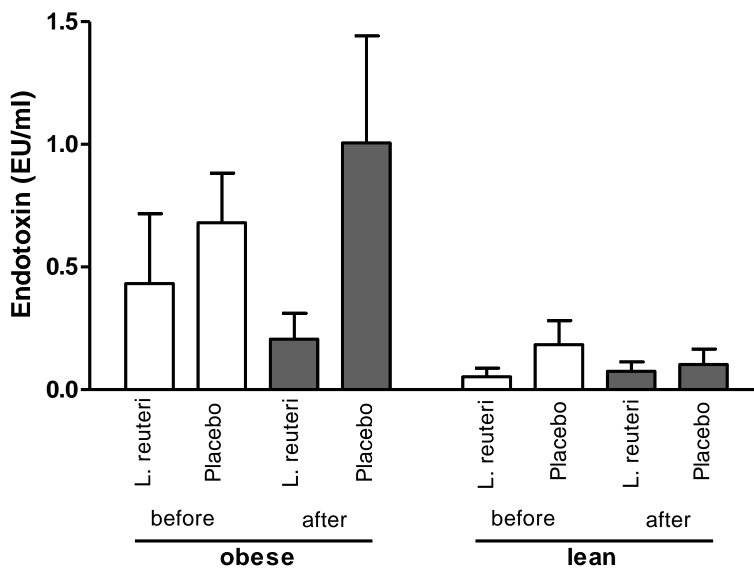
Supplementary Figure 5. Endotoxin concentrations in the serum of fasted persons
Shown are mean ± SEM, for lean and obese persons before (open) and after intervention (grey bars).



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Supplementary Table 3. Serum concentrations of inflammatory markers

		L. reuteri		**Placebo**	
		Lean	**Obese**	**Lean**	**Obese**
hsCRP (mg/dl)	before	0.065 [0.030 - 0.195]	0.290 [0.120 – 0.375]	0.050 [0.040 – 0.115]	0.310 [0.075 – 1.045]
	after	0.065 [0.030 – 0.200]	0.280 [0.150 – 0.345]	0.070 [0.055 – 0.085]	0.210 [0.095 – 0.745]
IL-1ra (pg/ml)	before	643.8 [572.1 – 813.9]	1714.0 [1217.0- 2003.7]	736.7 [570.8 - 874.4]	1270.6 [801.8 – 2543.1]
	after	544.8 [510.3 – 1010.7]	1728.7 [1451.4 – 2615.9]	651.8 [617.7 - 693.9]	1402.7 [929.4 – 2764.4]
MCP-1 (pg/ml)	before	242.5 [211.3 – 280.6]	339.5 [252.2 – 506.1]	326.6 [212.5 - 385.9]	544.8 [507.5 – 565.4]
	after	255.7 [201.0 – 438.1]	297.0 [210.6 – 558.7]	253.0 [222.6 - 280.9]	553.1 [391.0 – 651.1]
TNF-α (pg/ml)	before	3.47 [2.77 – 5.70]	10.90 [2.98 – 65.55]	4.16 [2.53 - 6.80]	30.56 [6.48 – 54.68]
	after	4.48 [2.97 – 20.29]	6.32 [4.05 – 58.80]	2.68 [2.09 - 4.17]	29.50 [6.48 – 45.13]
MIP-1ß (pg/ml)	before	71.51 [51.29 – 121.11]	232.02 [96.92 – 300.83]	70.88 [66.69 – 74.49]	154.19 [112.82 – 457.22]
	after	63.15 [42.15 – 136.14]	172.64 [87.41 – 208.48]	68.79 [63.78 – 99.37]	135.60 [113.53 – 473.01]
TNF-α/IL-1ra Ratio	before	0.006 [0.004 – 0.013]	0.006 [0.003 – 0.036]	0.008 [0.003 -0.015]	0.010 [0.008 – 0.023]
	after	0.009 [0.005 – 0.024]	0.004 [0.003 – 0.032]	0.004 [0.003 – 0.016]	0.009 [0.008 – 0.019]
TBARS (µM)	before	2.23 [2.19 – 3.29]	3.26 [2.07 – 3.76]	2.91 [2.75 – 2.98]	3.24 [2.92 – 3.75]
	after	2.09 [1.64 - 2.32]	2.62 [1.95 – 2.80]	2.41 [2.22 – 5.30]	2.78 [2.29 – 2.84]

Median [interquartile range] are given for log-normal distributed data. To check differences between treatment arms adjusted for body weight, we used two-way ANAOVA. No significant changes in cytokine data between *L. reuteri* and placebo group neither before nor after intervention were found.

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SUPPLEMENTARY DATA

Supplementary Table 4. Area under the curves (AUCs) during OGTT and isoglycemic clamp

OGTT		***L. reuteri***		**Placebo**			
		Lean	**Obese**	**Lean**	**Obese**	**p-value***	**p-value****
Insulin	before	**5548 [4131 - 6056]**	**9037 [5811 - 17881]**	**3890 [3584 - 4035]**	**10565 [9049 - 14101]**	n.s.	<0.01
	after	**6136 [5074 - 7319]**	**9916 [7553 - 38850]**	4749 [3868 - 4882]	10651 [61951 - 12696]	n.s.	<0.05
C-peptide	before	**1092 [981- 1266]**	**1821 [1110 - 1846]**	973 [829 - 984]	1925 [1658 - 1955]	n.s.	<0.05
	after	**1263 [912- 1425]**	**1716 [1565 - 2928]**	1117 [1007 - 1205]	1595 [1558 - 1776]	n.s.	<0.01
Glucagon	before	13485 [12360 - 16420]	16630 [15000 - 22580]	11660 [8930 - 18100]	14120 [10660 - 22000]	n.s.	n.s.
	after	14140 [12440 - 18180]	15600 [13730 - 19720]	9980 [8060 - 11930]	16180 [13040 - 16230]	n.s.	n.s.
GLP-1	before	3415 [2290 - 4535]	2030 [1830 - 2155]	4215 [2245 - 4425]	2510 [2375 - 2840]	n.s.	<0.05
	after	**4493 [2940 - 6025]**	**3230 [2545 - 3355]**	2795 [2365 - 3045]	2045 [1710 - 3070]	n.s.	<0.05
GLP-2	before	6295 [3415 - 9520]	3500 [3160 - 3765]	5756 [3240 - 8330]	4395 [3171 - 6640]	n.s.	n.s.
	after	7175 [3595 - 11280]	4325 [3102 - 4710]	**3930 [3345 - 6080]**	**2580 [2505 - 4525]**	n.s.	<0.05

'isoglycemic' clamp		***L. reuteri***		**Placebo**			
		Lean	**Obese**	**Lean**	**Obese**	**p-value***	**p-value****
Insulin	before	**1761 [957 - 2647]**	**3617 [2448 - 9307]**	**1354 [1236 - 1383]**	**4243 [2946 - 4302]**	n.s.	<0.01
	after	**1284 [748 - 2695]**	**3779 [2376 - 16464]**	935 [847 - 1171]	7530 [2653 - 9345]	n.s.	<0.01
C-peptide	before	**477 [340 - 719]**	**1156 [899 - 1316]**	**429 [369 - 688]**	**824 [785 - 914]**	n.s.	<0.01
	after	**535 [243 - 579]**	**958 [768 - 1915]**	**412 [337 - 495]**	**1130 [695 - 1547]**	n.s.	<0.01
Glucagon	before	11280 [10300 - 13340]	14100 [12570 - 17430]	8600 [8280 - 12200]	11470 [11400 - 16520]	n.s.	<0.05
	after	11760 [9420 - 13390]	13900 [11300 - 16250]	7820 [6850 - 8290]	10650 [10610 - 14240]	n.s.	n.s.
GLP-1	before	1342 [1300 - 1475]	1215 [1190 - 1555]	860 [830 - 1460]	1140 [635 -1225]	n.s.	n.s.
	after	1273 [1100 - 1450]	1290 [1235 - 1480]	1290 [1035 - 1355]	1245 [1115 -1335]	n.s.	n.s.
GLP-2	before	1192 [1155 - 1415]	970 [935 - 1130]	1170 [1170 - 1205]	1020 [995 - 1170]	n.s.	n.s.
	after	975 [695 - 1190]	1145 [980 - 1165]	**940 [800 - 1025]**	**1540 [1475 - 1735]**	n.s.	<0.05

Median [interquartile range] are given for log-normal distributed data. To check differences between treatment arms adjusted for body weight, we used two-way ANAOVA, p-value* show differences between *L. reuteri* and Placebo group, p-value** shows differences between lean and obese, significant difference between lean and obese within treatment or placebo group are marked in bold.

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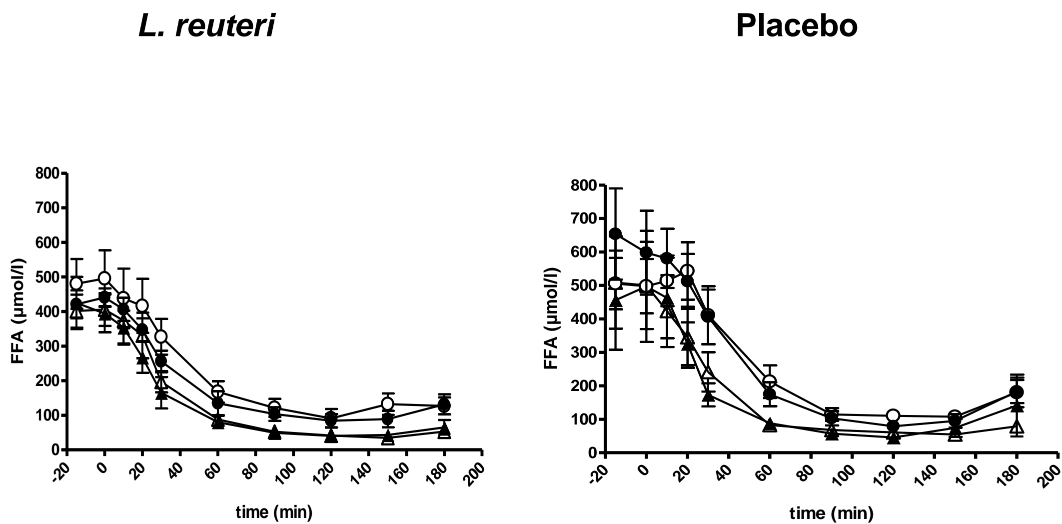
SUPPLEMENTARY DATA

Supplementary Figure 6. Free fatty acids (FFA) during oral glucose tolerance test (OGTT). Concentrations of FFA during OGTT, before (open) and after intervention (closed symbols). Shown are lean (triangles) and obese (circles) persons as mean±SEM.



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