Supplementary Information

Statistical methods

A longitudinal analysis was performed on the overall SCORAD score using a linear mixed-effect model for repeated measures. Following a blind data exploration, the values were assumed to be log-normally distributed and the natural logarithm of the original values (ln-transformation) was used as a dependent variable in the model. In the event of a value equal to 0, the value was replaced by 1 before ln-transformation. Fixed effects of the model were treatment (GM080, placebo), visit (4 weeks, 10 weeks, 16 weeks) and a linear interaction of treatment and time. The baseline value of overall SCORAD (ln-transformed) and age of the subject were used as continuous covariates. The repeated measures were measures at visits within subject.

Sensitivity analyses were performed for exploration of investigator site effect, treatment by time interaction, protocol deviations (none/major protocol violation) and antibiotics use by including these categories as co-variates. Clearly not significant effects ($p>0.1$) were withdrawn from the model.

A difference of the tested product GM080 would have been demonstrated if the mean estimates of outcome measure values (after back transformation from the model values in geometric means) in the subjects treated with GM080 were different than the mean estimates in the placebo group and the difference in treatment effects was statistically significant ($p<0.05$). If the interaction of treatment and time was significant ($p<0.05$), i.e. the treatment effect was affected by visit, then comparison of the treatment effects at individual visits was used for primary interpretation of the study results. The Tukey-Kramer method was applied to adjust the results for multiple comparisons.

The secondary outcomes included “objective” SCORAD as well as the sub-score extent, sub-score intensity, sub-score subjective symptoms, TEWL in the affected areas,

IDQOL, amount of corticoid used in 4 week intervals, IgE status and CCL17/TARC levels. These were analyzed using the same model as the primary outcome (excluding sensitivity analyses).

Sub-categories of the SCORAD intensity criteria (erythema, edema/papulation, oozing/crust, excoriation, lichenification, dryness) and subjective symptoms (itching, sleep loss) were analyzed across visits using a Generalized Estimating Equation approach (GEE) for repeated measurements. Outcome statistic for comparison of treatment groups was odds ratio and its 95% CI (and corresponding p-value).

Supplementary Table 1: IgE classification at baseline reported as number (percent) of subjects assigned to groups treated with *Lactobacillus paracasei* GM-080 or Placebo

	GM080 N=62	PLACEBO N=61	All N=123
Patients IgE-associated atopic dermatitis (based on threshold 100kU/L for total IgE and/or threshold 0.35 kU/L for specific IgE)	39 (66.1%)	35 (61.4%)	74 (63.8%)
Patients IgE-associated atopic dermatitis (based on threshold 100kU/L for total IgE and/or threshold 0.70 kU/L for specific IgE)	34 (57.6%)	31 (54.4%)	65 (56.0%)
Patients with IgE values above threshold in individual IgE values:			
total IgE >= 100kU/L	19 (31.1%)	18 (30.5%)	37 (30.8%)
specific IgE on egg > 0.35 kU/L	29 (49.2%)	22 (37.3%)	51 (43.2%)
specific IgE on egg > 0.70 kU/L	25 (42.4%)	18 (30.5%)	43 (36.4%)
specific IgE on house dust mite > 0.35 kU/L	5 (8.6%)	9 (16.1%)	14 (12.3%)
specific IgE on house dust mite > 0.70 kU/L	4 (6.9%)	3 (5.4%)	7 (6.1%)
specific IgE on milk > 0.35 kU/L	22 (37.9%)	24 (41.4%)	46 (39.7%)
specific IgE on milk > 0.70 kU/L	18 (31.0%)	18 (31.0%)	36 (31.0%)
specific IgE on molds > 0.35 kU/L	1 (1.7%)	1 (1.8%)	2 (1.8%)
specific IgE on molds > 0.70 kU/L	0 (0.0%)	0 (0.0%)	0 (0.0%)
specific IgE on peanut > 0.35 kU/L	11 (19.0%)	12 (21.4%)	23 (20.2%)
specific IgE on peanut > 0.70 kU/L	10 (17.2%)	8 (14.3%)	18 (15.8%)