

Statement on Silva Rischitelli et al. 2026 - study of xanthan gum, colon microbiota and inflammation in rats

Scientific perspective

Key message. The study reports observations in a small exploratory rat model that may warrant further scientific investigation. However, it does not establish that xanthan gum causes intestinal inflammation or microbiome-related harm in humans, and it does not alter the conclusions of comprehensive safety assessments conducted by the European Food Safety Authority.

Recent media reports have highlighted the publications of Silva Rischitelli et al. (2026), often using simplified or causal headlines suggesting that xanthan gum causes gut inflammation. The publication itself reports results from a ten-week study in healthy adult male rats. Its findings should therefore be interpreted in the context of the study design, the biological consistency and human relevance of the observations, and the much broader evidence base considered by regulatory authorities.

A well-established safety basis

Xanthan gum (E 415) has a long history of use in foods and has been assessed repeatedly by international and European scientific bodies. In its 2017 re-evaluation, the European Food Safety Authority (EFSA) reviewed exposure, toxicological and human data and available clinical and post-marketing information. EFSA concluded that:

- xanthan gum is unlikely to be absorbed intact and is expected to be partially fermented by the intestinal microbiota;
- there was no concern regarding genotoxicity;
- there was no need to establish a numerical acceptable daily intake and no safety concern for the general population at the reported uses and use levels.

In 2023, EFSA completed the outstanding assessment for the particularly sensitive population of infants below 16 weeks of age consuming foods for special medical purposes. Using a no-observed-adverse-effect level of 750 mg/kg body weight per day from neonatal piglet studies, together with estimated infant exposures, EFSA concluded that the use of xanthan gum in these formulae at concentrations up to 1,200 mg/L, corresponding to an estimated high exposure of 312 mg/kg body weight per day, does not raise a safety concern. EFSA also noted that available clinical and post-marketing information did not identify adverse events of clinical significance.

This established evidence base remains the appropriate context for interpreting new findings. At the same time, scientific knowledge continues to develop, particularly regarding interactions between dietary components, the gut microbiome and the host. New evidence should therefore be examined carefully and incorporated into the overall weight of evidence when it is sufficiently robust, reproducible and relevant to human exposure.

What the new study contributes

Silva Rischitelli et al. (2026) investigated the effects of xanthan gum in adult male rats over a ten-week period using three exposure levels and assessed a range of endpoints, including colon histology, inflammatory mediators, intestinal-barrier markers and microbiota parameters. The reported observations include higher histological inflammation scores in two treatment groups, changes in selected cytokine and barrier-marker measurements, and an increase in the low-abundance phylum Elusimicrobiota at the highest concentration.

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These observations contribute to the scientific discussion and may help generate hypotheses for further research. However, an experimental animal study is one element of a safety assessment, and its findings need to be considered together with study design, dose-response behaviour, test-material characterisation, realistic human exposure and consistency with the wider evidence base.

Points requiring careful interpretation

Limited study size and multiple endpoints

The study used 32 male adult rats; eight animals per group, while examining a range of histological, biochemical, immunohistochemical and microbiome endpoints. In a study of this size and complexity, isolated statistically significant findings require confirmation in adequately powered and independently replicated studies.

Test material characterisation

The publication does not provide sufficient characterisation of the xanthan gum used, including its grade, conformity with applicable specifications or information on purity. This limits reproducibility and makes it difficult to determine how representative the tested material is of food-grade xanthan gum products in general.

Lack of a consistent dose-response pattern

The study reported local histological and biomarker changes, without a consistent dose-response pattern and without corresponding changes in several other inflammatory, metabolic and microbiome endpoints. For example, colon IL-1 β was increased in the medium-dose group but not in the highest-dose group, while quantitative Claudin-2 was increased at the lowest dose but not at the two higher doses. This does not invalidate individual findings, but it weakens a straightforward exposure-response interpretation.

Intestinal permeability was not measured directly

Although the authors interpret changes in tight-junction markers as evidence of altered intestinal permeability, the study did not directly measure permeability or functional barrier integrity. The marker findings support a hypothesis of altered barrier biology but do not independently demonstrate increased intestinal permeability or clinically relevant barrier dysfunction.

Limited Microbiome findings

The study did not report significant changes in alpha diversity, Firmicutes, Bacteroidetes or the Firmicutes/Bacteroidetes ratio. A statistically significant increase in low-abundant Elusimicrobiota was observed at the highest concentration. Because this observation was reported at the phylum level only and the biological function of this taxon is not well understood, its relevance to host health remains uncertain.

Microbiota samples were collected only at the end of the intervention, without baseline measurements, and a compositional change is not automatically an adverse health effect. Validated microbiome-based endpoints for regulatory risk assessment are still developing.

Human exposure and clinical relevance

Although the authors state that the three investigated dietary concentrations mimic different consumer and dysphagia-use scenarios, the achieved exposure in mg/kg body weight/day and the underlying human-intake assumptions are not transparently presented and have therefore limited direct clinical relevance for humans. Because xanthan gum is expected to be poorly absorbed and acting predominantly within the gastrointestinal tract, the applicability of body-surface-area scaling is uncertain. Human relevance would be better supported by transparent comparison with actual oral intake, dietary concentration and estimated gastrointestinal exposure.

Extrapolation to dysphagia patients and neonatal NEC

The experiment was conducted in healthy adult male rats. It did not involve a dysphagia model, a neonatal or preterm animal model, an established model of necrotising enterocolitis, or any clinical population. It therefore does not validate a causal relationship between xanthan gum and neonatal necrotising enterocolitis and does not

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provide a sufficient basis for recommendations concerning continued use by dysphagia patients. These conclusions extend beyond the experimental model and data generated in the study.

Overall conclusion

Biopolymer International supports the continued scientific evaluation of emerging research on food ingredients, including research on interactions between dietary components, the intestinal microbiome and host physiology.

The Silva Rischitelli et al. 2026 study contributes exploratory observations that may help define questions for future research. However, its small sample size, inconsistent dose-response patterns, methodological limitations, limited test-material characterisation and uncertain human relevance prevent it from supporting causal conclusions regarding harm from xanthan gum consumption.

The study does not establish that xanthan gum causes intestinal inflammation, intestinal-barrier dysfunction, dysbiosis or necrotising enterocolitis in humans. Nor does it provide a scientific basis for revising the established safety conclusions for xanthan gum when used in accordance with authorised conditions.

The conclusions of EFSA's 2017 and 2023 safety assessments therefore remain the appropriate basis for evaluating the safety of xanthan gum as a food additive.

References

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