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SCIENTIFIC OPINION

Oat beta-glucans and reduction of postprandial glucose peak: Evaluation of a health claim pursuant to Article 13(5) of Regulation (EC) No 1924/2006

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The declarations of interest of all scientific experts active in EFSA's work are available at https://open.efsa.europa.eu/experts.

Abstract

Following an application from ScanOats, submitted for authorisation of a health claim pursuant to Article 13(5) of Regulation (EC) No 1924/2006 via the Competent Authority of Ireland, the EFSA Panel on Nutrition, Novel Foods and Food Allergens (NDA) was asked to deliver an opinion on the scientific substantiation of a health claim related to oat beta-glucans (OBG) and the reduction of postprandial glucose peaks (claimed effect). OBG are sufficiently characterised. The claimed effect is beneficial for the target population of individuals who wish to reduce their postprandial glucose peaks. In weighing the evidence, the Panel took into account that most of the 16 human intervention studies considered pertinent for the scientific substantiation of the claim showed that OBG reduce postprandial blood glucose peaks when consumed as part of foods/meals rich in available carbohydrates. The Panel also took into account that OBG did not increase postprandial glycaemic or insulinaemic responses and that the mechanism by which consumption of OBG could exert the claimed effect is well established. The Panel concludes that a cause-and-effect relationship has been established between the consumption of OBG and the reduction of postprandial blood glucose peaks. The following wording reflects the scientific evidence: 'Consumption of beta-glucans from oats contributes to the reduction of the glucose peak after a meal'. In order to bear the claim, foods/meals should contain at least 30 g of available carbohydrates per portion and at least 3 g of beta-glucans from oats for each 30 g of available carbohydrates.

KEYWORDS

beta-glucans, glucose peak, health claim, Oats, postprandial

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1 | INTRODUCTION

1.1 | Background and Terms of Reference as provided by the requestor

Regulation (EC) No 1924/2006 harmonises the provisions that relate to nutrition and health claims, and establishes rules governing the Community authorisation of health claims made on foods. As a rule, health claims are prohibited unless they comply with the general and specific requirements of this Regulation, are authorised in accordance with this Regulation, and are included in the lists of authorised claims provided for in Articles 13 and 14 thereof. In particular, Article 13(5) of this Regulation lays down provisions for the addition of claims (other than those referring to the reduction of disease risk and to children's development and health), which are based on newly developed scientific evidence or include a request for the protection of proprietary data, to the Community list of permitted claims referred to in Article 13(3). According to Article 18 of this Regulation, an application for inclusion in the Community list of permitted claims referred to in Article 13(3) shall be submitted by the applicant to the national competent authority of a Member State, which will make the application and any supplementary information supplied by the applicant available to the European Food Safety Authority (EFSA).

1.2 | Interpretation of the Terms of Reference

EFSA is requested to evaluate the scientific data submitted by the applicant in accordance with Article 16(3) of Regulation (EC) No 1924/2006. On the basis of that evaluation, EFSA will issue an opinion on the scientific substantiation of a health claim related to oat beta-glucans and reduction of postprandial glucose peak.

The present opinion does not constitute, and cannot be construed as, an authorisation for the marketing of oat beta-glucans, a positive assessment of its safety, nor a decision on whether oat beta-glucans is, or is not, classified as a foodstuff. It should be noted that such an assessment is not foreseen in the framework of Regulation (EC) No 1924/2006.

It should also be highlighted that the scope, the proposed wording of the claim, and the conditions of use as proposed by the applicant may be subject to changes, pending the outcome of the authorisation procedure foreseen in Article 18(4) of Regulation (EC) No 1924/2006.

2 | DATA AND METHODOLOGIES

2.1 | Data

Information provided by the applicant

See also the section Steps taken by EFSA at the end of this opinion.

Food/constituent as stated by the applicant

According to the applicant, the food for which the health claim is made is 'beta-glucans from oats'. The application applies to oat beta-glucans naturally present in foods, and to beta-glucans isolated from oats.

Health relationship as claimed by the applicant

According to the applicant, the claimed effect is the 'reduction in the blood glucose rise after a meal'.

Following an additional data request (ADR) by EFSA, the applicant has clarified that 'the proposed health claim refers to a reduction of the postprandial glucose peak, for which the postprandial incremental peak glucose (iPkG) is the main endpoint for substantiation'.

Mechanism by which the food/constituent could exert the claimed effect as proposed by the applicant

The applicant suggests that 'the reduction in postprandial glycaemic responses can be attributed to the prolonged digestion and absorption process of the carbohydrates as a result of the increased luminal viscosity'. Another mechanism proposed involves delayed gastric emptying, also attributed to high viscosity and largely dependent on the molecular weight of beta-glucan, which in turn reduces glycaemic responses. The applicant also refers to recent findings which suggest that 'depolymerised oat beta-glucan, resulting in highly soluble, non-viscous beta-glucans, has been shown to inhibit 2 key proteins involved in glucose metabolism: alpha-glucosidase (the enzyme involved in carbohydrate digestion) and sodium–glucose linked transporter 1 (the protein involved in glucose absorption across the small intestine)'.

Wording of the health claim as proposed by the applicant

The applicant proposed the following wording for the health claim: 'consumption of beta-glucans from oats contributes to the reduction of the glucose rise after a meal'.

Following an ADR by EFSA, the applicant has provided a revised wording for the proposed claim: 'consumption of beta-glucans from oats contributes to the reduction of the glucose peak after a meal'.

Specific conditions of use as proposed by the applicant

According to the applicant, the target population for the intended health claim is individuals who wish to reduce their postprandial glucose peaks.

The applicant proposed that '1.9 g of oat beta-glucans (OBG) per 30 g of available carbohydrates (availCHO) should be consumed per meal. The claimed effect may be used for all food products that contain all forms of beta-glucans derived from oats, including those present naturally in oat products and products containing added beta-glucan extracts derived from oats, provided that the OBG to availCHO ratio in the final product, as regularly consumed, meets or exceeds the proposed ratio of 1.9 g of OBG per 30 g of availCHO'.

Following an ADR by EFSA, and considering the revised claimed effect, the applicant proposed that 1.2 g of oat beta-glucans (OBG) per 30 g of available carbohydrate (avCHO) should be consumed per meal.

Data provided by the applicant

The health claim application on oat beta-glucans pursuant to Article 13(5) of Regulation (EC) No 1924/2006 was presented in a common and structured format as outlined in the Scientific and technical guidance for the preparation and presentation of a health claim application (EFSA NDA Panel, 2021b).

As outlined in the General scientific guidance for stakeholders on health claim applications (EFSA NDA Panel, 2021a), it is the responsibility of the applicant to provide the totality of the available evidence.

The applicant has submitted a confidential and a non-confidential version of a dossier following the 'General scientific guidance for stakeholders on health claim applications' (EFSA NDA Panel, 2021a) and the 'Scientific and technical guidance for the preparation and presentation of a health claim application' (EFSA NDA Panel, 2021b).

The application contains data claimed as confidential by the applicant in relation to an unpublished human study on the relationship between the consumption of the food/constituent and the claimed effect, which has been published since the submission of the dossier (Hossain et al., 2025).

The application does not contain data claimed as proprietary.

In accordance with Article 38 of Regulation (EC) No 178/20021[1] and taking into account the protection of confidential information and of personal data in accordance with Articles 39 to 39e of the same Regulation, and of the Decision of EFSA's Executive Director laying down practical arrangements concerning transparency and confidentiality,[2] the non-confidential version of the dossier has been published in the OpenEFSA portal.[3]

2.2 | Methodologies

The approach used by the NDA Panel for the evaluation of health claims is explained in the General scientific guidance for stakeholders on health claim applications (EFSA NDA Panel, 2021a). In assessing each specific food/health relationship, which forms the basis of a health claim, the NDA Panel considers the following key criteria:

(i) the food/constituent is defined and characterised;
(ii) the claimed effect is based on the essentiality of a nutrient; OR the claimed effect is defined and is a beneficial physiological effect for the target population and can be measured in vivo in humans;
(iii) a cause-and-effect relationship is established between the consumption of the food/constituent and the claimed effect (for the target group under the proposed conditions of use).

Each of these three criteria needs to be assessed by the NDA Panel with a favourable outcome for a claim to be substantiated. In addition, an unfavourable outcome of the assessment of criterion (i) and/or (ii) precludes the scientific assessment of criterion (iii).

The scientific requirements for health claims related to blood glucose concentrations, are outlined in a specific EFSA Guidance on the scientific requirements for health claims related to appetite ratings, weight management and blood glucose concentrations (EFSA NDA Panel, 2012).

[1]Regulation (EC) No 178/2002 of the European Parliament and of the Council of 28 January 2002 laying down the general principles and requirements of food law, establishing the European Food Safety Authority and laying down procedures in matters of food safety. OJ L 31, 1.2.2002, p. 1–48.

[2]Decision available at: https://www.efsa.europa.eu/sites/default/files/corporate_publications/files/210111-PAs-pre-submission-phase-and-public-consultations.pdf.

[3]https://open.efsa.europa.eu/questions/EFSA-Q-2025-00201.

2.3 | Public consultation

According to Art. 32c(2) of Regulation (EC) No 178/2002 and to the Decision of EFSA's Executive Director laying down the practical arrangements on pre-submission phase and public consultations, EFSA carried out a Public Consultation on the non-confidential version of the application from 21/8/2025 to 11/9/2025 (PC-1579) for which no comments were received.

3 | ASSESSMENT

3.1 | Characterisation of the food/constituent

The food constituent that is the subject of the health claim is oat beta-glucans.

Oat beta-glucans (OBG) are soluble cereal fibres (non-starch polysaccharides) consisting of linear D-glucose polymers with mixed β-(1 → 3) and β-(1 → 4) glycosidic linkages, typically in an approximate 2:1 ratio. Their molecular weights may vary widely (~65 to 3100 kDa), and are influenced by cultivar and growing conditions, processing, extraction/purification and analytical methods, as well as the method of preparing the food. Together with the concentration of dissolved OBG, molecular weight influences viscosity.

OBG occur naturally in oat-based foods (e.g. oat bran, oatmeal/rolled oats, oat flour and cereals) and may also be added to foods as concentrated/purified oat beta-glucan ingredients. They are measurable in foods by established analytical methods.

In line with a previous health claim evaluation (EFSA NDA Panel, 2011), the applicant confirmed that the health claim is intended for all OBG, naturally present in foods and added to foods, regardless of the manufacturing process, molecular weight, viscosity or food matrix, provided that the food delivers the proposed amount of OBG per 30 g of available carbohydrates (avCHO).

The Panel considers that oat beta-glucans, which are the subject of the health claim, are sufficiently characterised.

3.2 | Relevance of the claimed effect to human health

The claimed effect is the reduction of the postprandial glucose peak after a meal. The proposed target population is individuals who wish to reduce their postprandial glucose peaks.

The applicant proposes the postprandial incremental peak glucose (iPkG) as the main endpoint for the scientific substantiation of this claim. iPkG is defined as the difference between maximal plasma glucose after the consumption of a meal and fasting plasma glucose.

The Panel has previously evaluated health claims on the reduction of postprandial blood glucose responses, which refer to the ability of a food/constituent to reduce the blood glucose rise after consumption of a food or meal rich in digestible carbohydrates (i.e. in comparison to a reference food or meal), also in relation to oats (and barley) beta-glucans (EFSA NDA Panel, 2011, 2025). According to the EFSA guidance (EFSA NDA Panel, 2012), the scientific evidence for the substantiation of health claims on the reduction of postprandial blood glucose responses can be obtained from human intervention studies showing a decrease in blood glucose concentrations at different time points after consumption of a test food during an appropriate period of time (i.e. at least 2 h) and no increase in insulin concentrations in comparison to a reference food. Consistent with this guidance, the incremental area under the curve (iAUC) for glucose (and insulin) in blood has been considered as the relevant endpoint to assess the claimed effect in vivo in humans (EFSA NDA Panel, 2025). The Panel noted that the reduction of postprandial glycaemic responses may be a beneficial physiological effect (e.g. for individuals with impaired glucose tolerance (IGT)), as long as postprandial insulinaemic responses are not disproportionally increased (EFSA NDA Panel, 2012).

The newly proposed health claim refers to the reduction of postprandial blood glucose responses at a single time point, i.e. the time point at which the maximum blood glucose concentration relative to baseline is reached after the consumption of a food or meal rich in available carbohydrates as compared to a reference food or meal. The Panel notes that the shape of the postprandial blood glucose curve and the time at which such the maximum glucose concentration is reached may vary among individuals depending on their metabolic condition and the composition of the test meal (Jarvis et al., 2023). The Panel considers that the reduction of the postprandial iPkG may be a beneficial physiological effect (e.g. for individuals with IGT), as long as postprandial glucose and insulin responses (e.g. the iAUC for glucose and insulin) are not increased as compared to the reference food or meal.

In this context, the scientific evidence for the substantiation of health claims on the reduction of postprandial glucose peaks can be obtained from human intervention studies (preferably of cross-over design) measuring fasting and postprandial blood glucose (and insulin) at different time points (e.g. 15, 30, 45, 60, 90, 120 min) during an appropriate period of time (i.e. at least 2 h) and showing a decrease in the iPkG after consumption of the test food/meal (in comparison to a reference test food/meal) and no increase in postprandial blood glucose or insulin concentrations over that period. The Panel acknowledges that the true iPkG may only be obtained through postprandial continuous glucose monitoring. However, the Panel considers that the iPkG calculated from the maximum blood glucose concentration measured at one of the above-mentioned time points provides a proxy for the endpoint of interest that is appropriate for the scientific substantiation of these claims.

The Panel considers that the reduction of postprandial blood glucose peaks is a beneficial physiological effect in the context of the food/constituent and the target population proposed for this claim.

3.3 | Scientific substantiation of the claimed effect

The applicant conducted a systematic literature search of nine databases (Adis Clinical Trials Insight, Allied and Complementary Medicine™, BIOSIS Previews®, CAB Abstracts, Embase®, Foodline®: SCIENCE, FSTA®, MEDLINE® and NTIS) via ProQuest Dialog™ on 15 December 2021 and 6 February 2024, with no restrictions on date or language. Keywords related to the food constituent (e.g. oat*, barley*, beta-glucan), the claimed effect (e.g. glucose, glycemi*, postprandial) and the study population (e.g. human*, participant*) were combined. The full search strategy, including keyword categories, was provided by the applicant. Studies were eligible if they were randomised, controlled, cross-over trials in generally healthy adults (or individuals with impaired glucose tolerance or untreated type 2 diabetes), assessing OBG (<4 g/30 g avCHO) on postprandial blood glucose (iPkG or iAUC over 120 min). Blood samples had to be collected at fasting and at least 15, 30, 45, 60, 90 and 120 min post-meal. Test and control foods had to be comparable in their delivery matrix and avCHO content (≤ 5% difference), and studies had to have a minimum sample size of 10 participants and control for at least 7 of 10 pre-specified factors that could influence the glycaemic response (see Table 1). Exclusion criteria included animal or in vitro studies, non-randomised or non-crossover designs, insufficient sample size (< 10 participants without justification), OBG dose ≥ 4 g per 30 g avCHO, inappropriate control interventions, inability to isolate the effect of OBG or lack of relevant outcome data. Ten publications met all criteria and were included. One additional study, initially submitted as an unpublished manuscript, has since been published (Hossain et al., 2025).

TABLE 1 Factors considered by the applicant that could influence the glycaemic response in human intervention studies.

Factor	Consideration
Alcohol consumption	Alcohol consumption should be restricted or controlled the evening prior to glycaemic response testing
Smoking	Potential variability in the glycaemic response introduced by smoking should be minimised by: • Limiting study inclusion to non-smokers; or • Restricting or standardising smoking the day of glycaemic response testing.
The meal consumed the evening prior to glycaemic response testing	The study should report if the meal consumed the evening prior to postprandial glycaemic response testing was standardised.
Physical activity	Intense physical activity should be restricted or standardised the day prior to glycaemic response testing.
Volume and nature of co-consumed beverage	The nature and volume of the beverage that is co-consumed with the test meal must be standardised.
Test meal consumption time	The test meal must be consumed within a 15-min period.
Fasted state of subjects	Subjects should arrive at the testing site in a fasted state (e.g. overnight fast).
Fasting duration	The fasting period should be ≥ 10 h.
Duplicate fasting blood sample	Glucose in the fasting sample should be analysed in duplicate from the same blood sample or from two blood samples.
Blood glucose response	The study should report the AUC as iAUC.

In response to an ADR from EFSA, the applicant decided to apply for a health claim related to the reduction of glucose peaks after a meal, rather than for a claim on the reduction of postprandial glucose responses (see Sections 2.1 and 3.2). Considering the revised claimed effect proposed, the applicant conducted an updated search on 6 November 2025 covering publications from 2024 to 2025. The original strategy was applied with modified eligibility criteria: (a) outcome restricted to iPkG; (b) any OBG dose eligible; and (c) minimum test duration reduced to 90 min. Control of ≥ 7/10 factors (Table 1) was still required. No additional pertinent studies were identified. The study by Hossain et al. (2025) had been already included by the applicant ahead of publication.

The applicant also cross-checked the studies included in the systematic review and meta-analysis by Zurbau et al. (2021) against its own body of evidence. No additional eligible studies were identified.

In line with EFSA's request to review all human intervention studies regardless of the OBG dose used, the applicant re-evaluated the studies included to identify additional eligible trial comparisons (strata) and the publications previously excluded because the OBG dose was ≥ 4 g/30 g avCHO. None of the excluded publications met the updated inclusion criteria, while four additional strata from previously included studies were identified as eligible.

The Panel considers that the 10 factors identified by the applicant as potentially influencing postprandial glycaemic responses (Table 1) may not have the same impact on the intra- and inter-individual variability of the iPkG. While EFSA previously requested clarification on the rationale for requiring control of specifically at least 7 out of 10 factors for a study to be deemed eligible, the applicant indicated that eight factors were derived from an ISO standard for glycaemic

index determination (ISO, 2010) and that two additional factors (smoking and standardisation of the evening meal prior to testing) were included based on published evidence. However, no scientific rationale was provided to support the ≥ 7/10 cut-off itself (e.g. weighting of criteria according to their expected impact on iPkG variability, particularly in cross-over trials with variable washout periods). Since this threshold is arbitrary, the Panel considered several studies previously excluded by the applicant for this reason as pertinent to the claim (Kwong et al., 2013a, 2013b; Lan-Pidhainy et al., 2007; Regand et al., 2009; Regand et al., 2011; Zhu et al., 2020). Among these, one study (Zhu et al., 2020) was additionally excluded by the applicant because blood samples were not collected every 15 min during the first hour. However, the Panel considers that the sampling schedule used (0, 20, 30, 45, 60, 90, 120 min) is adequate to capture the expected peak glucose (30–60 min) in healthy individuals (Jarvis et al., 2023) and therefore considers this study as pertinent to the claim.

Following EFSA's request, the applicant re-assessed for eligibility a number of studies previously excluded on the basis of the ≥ 7/10 criterion. The applicant included the studies cited above (Kwong et al., 2013a, 2013b; Lan-Pidhainy et al., 2007; Regand et al., 2009; Regand et al., 2011; Zhu et al., 2020) and two additional studies (Brummer et al., 2012; Revheim et al., 2024), whereas some trial comparisons in two studies (Lan-Pidhainy et al., 2007; Regand et al., 2009) were not considered further, owing to issues related to the comparability of the test and control meals (e.g. mismatch in avCHO). The Panel considers that the study by Brummer et al. (2012) is not pertinent to the claim because the test and control foods/meals were not comparable in key nutritional components (i.e. starch, fibre and sugar content).

Human intervention studies investigating the effect of oat beta-glucan on postprandial glucose peaks

A total of 16 human intervention studies comprising 55 trial comparisons (strata) were considered pertinent by the Panel to the scientific substantiation of the claim. All studies were acute, single-meal challenges assessing postprandial glycaemic responses over at least 120 min. One study (Hossain et al., 2025) evaluated postprandial glucose responses both immediately after ingestion of the breakfast test drink and following a standardised lunch at 210 min. Only the post-breakfast glucose responses have been considered in this assessment.

Of the 16 studies, 14 reported postprandial iPkG (i.e. maximal plasma glucose minus fasting plasma glucose), whereas the remaining 2 studies, Hartvigsen et al. (2014) and Revheim et al. (2024), reported maximal concentrations of glucose (C_{max}) only. However, as fasting glucose was reported in the publications, iPkG was derived by subtracting fasting glucose from C_{max}.

All studies were randomised cross-over trials, with reported washout periods ranging from ≥ 1 day to ≥ 1 week, while four studies did not specify the washout duration. Study populations generally consisted of healthy adults of both sexes, with one study (Hartvigsen et al., 2014) including participants with metabolic syndrome and another study including females only (Zhu et al., 2020). Sample size ranged from 10 to 40 individuals. A wide variety of food matrices were tested, including cereal preparations, bread-type products, pasta, muffins and beverages (glucose drinks and gels with added OBG concentrates). The OBG doses across all food matrices ranged from 0.96 to 6.48 g/30 g avCHO. The amount of avCHO in the test and control meals ranged from 18 to 65 g, with most studies providing either approximately 30 g or around 50 g. A standardised evening meal prior to each test day was reported in only 6 of the 16 studies (Ekström et al., 2017; Hartvigsen et al., 2014; Holm et al., 1992; Hossain et al., 2025; Rieder et al., 2019; Wolever et al., 2020). Four studies investigated at least three distinct OBG doses within the same matrix to explore dose–response relationships (Ekström et al., 2017; Hossain et al., 2025; Wolever et al., 2018; Zhu et al., 2020). All 16 studies also assessed the postprandial iAUC for glucose and seven studies reported on the iAUC for insulin. OBG did not significantly increase the postprandial iAUC for glucose or insulin in any study as compared to the matched control. These endpoints will not be discussed further in the opinion, as it is known that dietary fibre in general and oat and barley beta-glucans in particular, do not induce an increase in postprandial glucose or insulin responses when consumed with available carbohydrate-containing foods (EFSA NDA Panel, 2011, 2025).

Details on the characteristics of the 16 studies (including 55 trial comparisons) and the results for the postprandial iPkG can be found in Appendix A.

All the studies but two (Holm et al., 1992; Regand et al., 2009) reported a statistically significant decrease in the postprandial iPkG with the OBG-test meal as compared to the matched control meal in one or more trial comparisons. Such differences in iPkG between the control and test meals were ≥ 20% in most studies and strata. The Panel considers that such difference of about 20% in the iPkG is likely to be biologically relevant for healthy individuals in the context of a mixed diet.[4] The Panel notes that only in nine strata from six studies (Hossain et al., 2025; Kwong et al., 2013a; Regand et al., 2011; Wolever et al., 2016; Wolever et al., 2018; Wolever et al., 2019), statistically significant differences between the control and test meals in iPkG were < 20% (i.e. between 10.7% and 19.8%). However, the studies by Wolever and colleagues had a sample size two to three times higher than the 10–15 subjects usually recruited for testing of the glycaemic index of foods, which may have increased the precision of the estimates.

Four studies investigated three or more distinct OBG doses within the same food matrix (Appendix A). A graded inverse dose–response relationship between the OBG dose (range 1.27 to 2.96 g/30 g avCHO) incorporated to instant oat meals and the iPkG was observed in Wolever et al. (2018), whereas dose-related differences in iPkG between OBG-enriched breads (dose range 1.92 to 4.11 g/30 g avCHO) were small (Ekström et al., 2017). In Zhu et al. (2020), reductions in the iPkG were observed at the higher (1.86 and 2.28 g/30 g avCHO), but not at the lower (0.96 and 1.14 g/30 g avCHO) OBG doses tested

[4] https://www.canada.ca/en/health-canada/services/food-nutrition/legislation-guidelines/guidance-documents/guideline-concerning-safety-physiological-effects-novel-fibre-sources-food-products-containing-them-guideline-no-9-1997.html.

in pearled and whole grain oats cooked under normal or high pressure. Finally, significant reductions in the iPkG were observed at 2 g and 4 g OBG/30 g avCHO incorporated into glucose drinks, but not at 3 g (Hossain et al., 2025). Overall, these studies support an inverse dose–response relationship between OBG intake and iPkG, with high heterogeneity across food matrices and relative control foods/meals.

Taken together, these studies showed an effect of OBG on the reduction of the postprandial iPkG and indicated that OBG dose (Ekström et al., 2017; Hossain et al., 2025; Wolever et al., 2018; Zhu et al., 2020), molecular weight and viscosity (Kwong et al., 2013a, 2013b; Regand et al., 2011), carbohydrate load (Wolever et al., 2016), food matrix (Wolever et al., 2019) and processing (Lan-Pidhainy et al., 2007; Wolever et al., 2020) can all have an impact on such effect.

Dose–response relationship and derivation of the lowest effective dose

The applicant submitted a linear mixed-effects dose–response meta-analysis to explore the effect of OBG dose on iPkG. The applicant initially based this analysis on 26 strata from 9 studies that were considered eligible using restricted inclusion criteria. Following EFSA's request, the applicant expanded the dataset by additionally including 37 strata from studies previously excluded based on the ≥ 7/10 factors criterion, resulting in 63 strata from 17 studies (dose range 0.96–8.4 g OBG/30 g avCHO). The Panel notes that the dataset used by the applicant also included strata from Brummer et al. (2012), although this study was not considered pertinent by the Panel owing to issues related to the comparability of the test and control meals. The Panel also notes that non-linearity was not assessed. However, published dose–response meta-regression analyses (Noronha et al., 2023; Zurbau et al., 2021) have shown statistically significant linear dose–response relationships between the intake of OBG in avCHO-containing foods/meals and different measures of postprandial blood glucose responses, including the iPkG, with no significant departure from linearity.

The applicant employed a one-stage linear, mixed-effects, random-effects model with restricted maximum likelihood (REML) estimation. For each stratum, the effect size was expressed as natural logarithm of the ratio of means [ln(RoM)] for iPkG (mean iPkG for the OBG-test meal divided by mean iPkG for the control meal). OBG dose (g/30 g avCHO) was used as the independent variable in the linear mixed-effects model with random intercepts at study and stratum-within-study level. The applicant fitted (i) an intercept-constrained model (RoM at 0 g OBG fixed to 1) and (ii) an intercept-unconstrained model (intercept estimated freely). Although the predicted reductions in iPkG were greater with the intercept-unconstrained model, the applicant considered that the results from the intercept-constrained model were most relevant, as it is reasonable to assume that the RoM at an OBG dose of 0 g per 30 g avCHO is 1 (a null effect). The Panel agrees with such consideration.

In both models, the applicant reported a statistically significant inverse association between OBG dose and iPkG RoM. In the intercept-constrained model, the slope was $\beta1 = -0.0815$ (95% CI: −0.0965 to −0.0666; $p < 0.001$), corresponding to an estimated 7.8% reduction in iPkG per each additional 1 g OBG/30 g avCHO. The applicant noted that the lowest OBG dose tested at which a statistically significant reduction in the iPkG was observed was 1.2 g OBG/30 g avCHO (Wolever et al., 2020) and used the intercept-constrained fitted model to estimate the corresponding reduction in iPkG (9.3%). The applicant proposed 1.2 g OBG/30 g avCHO as the condition of use for the claim based on this consideration.

The Panel notes that the food/constituent for which the claim is made, is OBG naturally present in foods or added to foods (including beverages), the MW of which generally ranges from ~65 to 3100 kDa, with no restrictions (see Section 3.1). The Panel also notes that, in the human intervention studies provided, the lowest dose of OBG tested in beverages was 2 g/30 g avCHO, with inconsistent results from 2 to 4 g/30 g avCHO depending on the OBG MW and testing conditions (Hossain et al., 2025; Kwong et al., 2013a, 2013b), and that the lowest dose of OBG tested in pasta products, 2.86 g/30 g avCHO, did not lead to a significant reduction of the iPkG as compared to the control pasta (Regand et al., 2009).

In this context, the Panel considers that the lowest OBG dose tested at which a statistically significant reduction in the iPkG was observed in a single study with a relatively high sample size ($n = 28$; (Wolever et al., 2020)) and in a single food matrix (instant oatmeal) cannot be used to establish conditions of use for this claim. Since MW and viscosity, processing and food matrix are all factors modulating the effect of OBG on postprandial glycaemic responses, including the incremental glucose peaks (see section on mechanism of action), the Panel considers that the conditions of use for this claim should take into account such variability and ensure a consistent reduction in the iPkG that is biologically relevant.

In the applicant's intercept-constrained model, a 20% reduction (RoM = 0.8) in the iPkG, which is likely to be biologically relevant for healthy individuals in the context of a mixed diet, corresponds to an OBG dose of approximately 2.7 g/30 g avCHO (approximate range using the 95% CI for β1: 2.3 to 3.4 g/30 g avCHO). The Panel notes that this model is based on a wide body of evidence from human intervention studies using matched controls that cover an acceptable range of OBG MWs, food matrices and processing conditions. The Panel also notes that the lowest effective dose of OBG in some carbohydrate-containing foods (e.g. pasta products, glucose drinks) across MWs and testing conditions may be above 2.7 g/30 g avCHO (see Appendix A).

Based on the available evidence and related uncertainties, the Panel considers that at least 3 g of OBG per 30g avCHO would be required to achieve a consistent and biologically relevant reduction in postprandial blood glucose peaks following the consumption of avCHO-rich foods or meals.

Mechanism of action

The mechanism by which oat beta-glucans could exert the claimed effect is well established and relates primarily to their ability to increase the viscosity of the meal bolus when consumed as part of a carbohydrate-containing food. This

mechanism has been previously assessed by the Panel in relation to the reduction of postprandial glycaemic responses (as assessed by postprandial blood glucose iAUC) for beta-glucans from oats and barley (EFSA NDA Panel, 2011, 2025).

The increased viscosity induced by oat beta-glucans may delay gastric emptying and, once the meal bolus reaches the small intestine, a high luminal viscosity delays the rate of digestion and absorption of nutrients, including glucose (Battilana et al., 2001; Wood et al., 2000; Würsch & Pi-Sunyer, 1997). By slowing the rate at which glucose appears in the circulation, this mechanism is expected to reduce overall postprandial glycaemic exposure and to attenuate the maximum postprandial blood glucose concentration (i.e. the postprandial glucose peak).

The physicochemical properties of oat beta-glucans, in particular their solubility and viscosity, depend on their (1 → 3)(1 → 4)-β-D-glucan structure and are influenced by the concentration of dissolved beta-glucans and their molecular weight (Wood et al., 2000). Viscosity may further be affected by the food matrix and by processing conditions (EFSA NDA Panel, 2011, 2025). Beta-glucans with higher MW generally induce higher viscosity and are associated with more pronounced reductions in postprandial glucose responses, including reductions in glucose iAUC and peak concentrations (Noronha et al., 2023; Wolever et al., 2020; Zurbau et al., 2021).

The applicant also referred to additional mechanisms proposed in a recent study (Marcobal et al., 2024). In this study, a depolymerised, highly soluble and non-viscous oat beta-glucan preparation was investigated using in vitro enzyme assays and an intestinal epithelial cell model. The authors reported inhibition of α-glucosidase activity and a reduction in sodium–glucose linked transporter 1 (SGLT1)-mediated glucose transport. While these findings may provide further supportive evidence for biological plausibility, the Panel considers that the established viscosity-mediated delay in carbohydrate digestion and glucose absorption remains the primary mechanism underlying the reduction of postprandial glycaemic responses, including postprandial glucose peaks.

Weighing of the evidence

In weighing the evidence, the Panel took into account that the majority of the 16 human intervention studies considered pertinent for the scientific substantiation of the claim show an effect of OBG on reducing postprandial blood glucose peaks when consumed as part of foods or meals that are rich in available carbohydrates. The Panel also took into account that OBG did not increase postprandial glucose or insulin responses (i.e. iAUC for glucose and insulin) in the studies which assessed these endpoints, and that the mechanism by which consumption of OBG could exert the claimed effect is well established.

The Panel concludes that a cause-and-effect relationship has been established between the consumption of oat beta-glucans and the reduction of postprandial blood glucose peaks.

4 | PANEL'S COMMENTS ON THE PROPOSED WORDING

The Panel considers that the following wording reflects the scientific evidence: 'Consumption of beta-glucans from oats contributes to the reduction of the glucose peak after a meal'.

5 | CONDITIONS AND RESTRICTIONS OF USE

The Panel considers that, in order to bear the claim, foods/meals should contain at least 30 g of available carbohydrates per portion and at least 3 g of beta-glucans from oats for each 30 g of available carbohydrates. The target population is individuals who wish to reduce their postprandial glucose peaks.

6 | CONCLUSIONS

On the basis of the data presented, the Panel concludes that:

- The food/constituent, oat beta-glucans, which is the subject of the health claim, is sufficiently characterised.
- The claimed effect is the reduction of postprandial blood glucose peaks, which is considered a beneficial physiological effect.
- A cause-and-effect relationship has been established between the consumption of OBG and the reduction of postprandial blood glucose peaks.
- The following wording reflects the scientific evidence: 'Consumption of beta-glucans from oats contributes to the reduction of the glucose peak after a meal'.
- In order to bear the claim, foods/meals should contain at least 30 g of available carbohydrates per portion and at least 3 g of beta-glucans from oats for each 30 g of available carbohydrates. The target population is individuals who wish to reduce their postprandial glucose peaks.

7 | DOCUMENTATION AS PROVIDED TO EFSA

Health claim application on pursuant to Article 13.5 of Regulation (EC) No 1924/2006 (Appian number: HC-2025-33,950). Submitted by ScanOats.

8 | STEPS TAKEN BY EFSA

1. This application was received by EFSA on 13/3/2025. The application was validated on 7/7/2025 and the scientific evaluation started.
2. The scope of the application was proposed to fall under a health claim based on newly developed scientific evidence.
3. The Working Group on Claims of the NDA Panel agreed on a list of questions for the applicant to provide additional information to accompany the application. EFSA sent a first Additional Data Request (ADR1) letter to the Applicant on 29/9/2025. The clock was stopped on 29/9/2025 and restarted on 14/10/2025.
4. EFSA sent a second ADR (ADR2) letter to the Applicant on 5/11/2025. The clock was stopped on 5/11/2025 and restarted on 20/11/2025.
5. EFSA sent a third ADR (ADR3) letter to the Applicant on 8/12/2025. The clock was stopped on 8/12/2025 and restarted on 23/12/2025.
6. During its meeting on 28/01/2026, the NDA Panel, having evaluated the data, adopted an opinion on the scientific substantiation of a health claim related to 'Oat beta-glucans and reduction of postprandial glucose peak: evaluation of a health claim pursuant to Article 13(5) of Regulation (EC) No 1924/2006'.

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ABBREVIATIONS

ADR	additional data request
AUC	area under the curve
avCHO	available carbohydrates
C_{max}	maximal concentrations of glucose
iAUC	incremental area under the curve
IGT	impaired glucose tolerance
iPkG	incremental peak glucose
ISO	International Organization for Standardization
kDa	Kilodalton
MW	molecular weight
NDA Panel	Panel on Nutrition, Novel Foods and Food Allergens
OBG	oat beta-glucan
PC	Public Consultation
REML	restricted maximum likelihood estimation
RoM	ratio of means
SGLT1	sodium–glucose linked transporter 1

REQUESTOR

Competent Authority of Ireland following an application by ScanOats

QUESTION NUMBER

EFSA-Q-2025-00201

PANEL MEMBERS

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APPENDIX A

Overview of human intervention studies on oat beta-glucans and postprandial incremental peak glucose

TABLE A.1 Summary of studies investigating the effects of oat beta-glucan on postprandial glucose peaks (ordered by study and increasing OBG dose/30 g avCHO).

Reference	Washout duration	N	Test product	Molecular weight (kDa)	OBG dose (g)/avCHO g	OBG dose (g)/30 g avCHO	Control (g BG/avCHO g)	Mean % iPkG difference vs control
Zhu et al. (2020)	NR	10F	Pearled oats cooked under high pressure + cooked rice	NR	1.6/50	0.96	Cooked rice (0/50)	−14.3; ns
			Pearled oats cooked under normal pressure + cooked rice		1.6/50	0.96		−17.1; ns
			Whole grain oats cooked under high pressure + cooked rice		1.9/50	1.14		−11.4; ns
			Whole grain oats cooked under normal pressure + cooked rice		1.9/50	1.14		−22.9; ns
			Pearled oats cooked under high pressure		3.1/50	1.86		**−37.1; s**
			Pearled oats cooked under normal pressure		3.1/50	1.86		**−37.1; s**
			Whole grain oats cooked under high pressure		3.8/50	2.28		**−34.3; s**
			Whole grain oats cooked under normal pressure		3.8/50	2.28		**−34.3; s**
Wolever et al. (2020)	≥ 5 days	28	Instant oatmeal + oat bran BG	1990	2/51.2	1.17	Cream of rice (0/51.3)	**−21.3; s**
			Instant oatmeal + oat bran BG	2060	4/51.2	2.34		**−43.7; s**
			Instant oatmeal + oat bran BG + beta-glucanase (lower viscosity)	< 10	4/51.2	2.34		**−21; s**
Wolever et al. (2018)	NR	40	Instant oatmeal	NR	1.17/27.6	1.27	Cream of rice (0/27.9)	**−10.7, s**
			Instant oatmeal + oat bran BG		1.37/27.7	1.48		**−12.7; s**
					1.57/27.7	1.70		**−18; s**
					1.97/27.9	2.12		**−28.3; s**
					2.77/28.1	2.96		**−40.6; s**

(Continues)

TABLE A.1 (Continued)

Reference	Washout duration	N	Test product	Molecular weight (kDa)	OBG dose (g)/avCHO g	OBG dose (g)/30 g avCHO	Control (g BG/avCHO g)	Mean % iPkG difference vs control
Wolever et al. (2019)	≥ 2 days	30	Old fashioned oats	NR	1.72/30.6	1.69	Cream of rice (0/30.6)	**−16.1; s**
			Steel cut oats		1.74/30.6	1.71		**−26.1; s**
			Instant oats		1.79/30.6	1.75		−5.36; ns
Wolever et al. (2016)	≥ 2 days	38	Oatmeal (Quick oats) + sucrose	NR	1.6/27.1	1.77	Cream of rice (0/27.1)	**−14; s**
			Oatmeal (Quick oats)		1.6/18.1	2.65	Cream of rice (0/18.1)	−17; ns
					2.2/24.2	2.73	Cream of rice (0/24.2)	**−16; s**
					3.3/36.3	2.73	Cream of rice (0/36.3)	**−27; s**
Regand et al. (2009)	≥ 1 day	12	Oat crisp bread	197	4/64	1.88	Wheat crisp bread (1/65)	−1.4; ns
			Oat pasta	465	4/42	2.86	Wheat pasta (0.4/44)	−10.9; ns
Ekström et al. (2017)	≥ 1 week	13	White wheat flour bread containing refined oat fibre	Peak 1: 282 Peak 2: 174	3.4/53.2	1.92	White wheat bread (0/52.9)	**−34.9; s**
					5.2/52.9	2.95		**−32.6; s**
				Peak 1: 291 Peak 2: 205	7.5/54.7	4.11		**−37.2; s**
Rieder et al. (2019)	≥ 2 days	14	Wheat flour bread + oat bran concentrate	421	1.7/26.4	1.93	White wheat bread (0.1/26)	−14.3; ns
			Wheat flour bread + oat bran concentrate	592	3.8/26.3	4.33		**−28; s**
			Wheat flour bread + degraded oat bran concentrate	282	3.8/26.2	4.35		**−22.2; s**
Hossain et al. (2025)	≥ 5 days	19	Glucose drink + OBG	800	2/30	2	Glucose drink (0/30)	**−17; s**
					3/30	3		−13.9; ns
					4/30	4		**−28.4; s**
Kwong et al. (2013a)	≥ 1 day	15	Glucose drink + OBG (LMW)	145	4/50	2.4	Glucose drink (0/50)	**−21.2; s**
			Glucose drink + OBG (HMW)	580				**−34.6; s**
			Glucose + OBG viscoelastic gel (LMW)	145				−5.1; ns
			Glucose + OBG viscoelastic gel (2 g HMW+ 2 g LMW)	362.5				−17.1; ns
			Glucose + OBG viscoelastic gel (3 g HMW + 1 g LMW)	471.25				**−19.8; s**

TABLE A.1 (Continued)

Reference	Washout duration	N	Test product	Molecular weight (kDa)	OBG dose (g)/avCHO g	OBG dose (g)/30 g avCHO	Control (g BG/avCHO g)	Mean % iPkG difference vs control
Kwong et al. (2013b)	NR	15	Glucose drink + OBG (LMW) 250 mL	145	4/50	2.4	Glucose drink, 250 mL (0/50)	−3.7; ns
			Glucose drink + OBG (HMW) 250 mL	580			Glucose drink, 250 mL (0/50)	**−21.9; s**
			Glucose drink + OBG (LMW) 600mL	145			Glucose drink, 600 mL (0/50)	−12.1; ns
			Glucose drink + OBG (HMW) 600mL	580			Glucose drink, 600 mL (0/50)	**−27; s**
Hartvigsen et al. (2014)[a]	1 week	15 metS	Wheat bread + OBG	1978	4.2/50	2.52	Wheat bread (0.2/50)	**−22; s**
Holm et al. (1992)	NR	10	Durum semolina pasta + oat bran	NR	5.2/54.2	2.88	Durum semolina pasta (0.2/54.2)	−18.2; ns
Regand et al. (2011)	≥ 1 day	12	Baked bar with whole oat flakes + oat bran (LMW)	82	6.3/60	3.15	Whole wheat flakes + white wheat flour (0.6/58)	−6.9; ns
			Baked bar with whole oat flakes + oat bran (MMW)	325				0; ns
			Baked bar with whole oat flakes + oat bran (HMW)	1996				**−17.2; s**
			Baked bar with whole oat flakes + oat bran (LMW)	57	6.2/38	4.89	Whole wheat flakes + white wheat flour (0.6/40)	−3.6; ns
			Baked bar with whole oat flakes + oat bran (MMW)	435				**−25; s**
			Baked bar with whole oat flakes + oat bran (HMW)	2133				**−35.7; s**
Revheim et al. (2024)[a]	≥ 3 days	22	Oat bread enriched with OBG	1050	3.6/25	4.32	Whole wheat bread (0.2/25)	**−35; s**
Lan-Pidhainy et al. (2007)	≥ 1 day	11	Oat bran muffin (fresh)	2700	10.8/50	6.48	Whole wheat muffin (0/52)	**−45.5; s**
			Oat bran muffin (2 × FT)	2400				**−42.9; s**
			Oat bran muffin (4 × FT)	2000				**−36.3; s**

Abbreviations: avCHO, available carbohydrates; BG, beta-glucan; CHO, carbohydrates; F, female; FT, freeze thaw cycle; HMW, high molecular weight; iPkG, incremental peak glucose rise; LMW, low molecular weight; M, males; metS, metabolic syndrome; MMW, medium molecular weight; NA, not available; NR, not reported; ns, non-statistically significant; OBG, oat beta-glucan; s, statistically significant. Green colour is to indicate statistically significant results more easily.

[a]Maximal concentration of glucose was reported in the publication. iPkG was calculated by subtracting fasting glucose from the maximal concentration of glucose.