Questions for health professionals in the fields of nephrology, mineral metabolism, cardiovascular and nutrition medicine on phosphates food additives re-evaluation

Background

All food additives which were already permitted for use before 30th January are subject to a new risk assessment by the European Food Safety Authority, defined in Regulation (EU) No 257/2010[1].

Within that legal framework, upon request from the European Commission, the EFSA Panel on Food Additives and Nutrient Sources (ANS Panel) is re-evaluating the safety of phosphates intended for use as food additives. Phosphates (E 338-E 452)[2] are authorised food additives in the EU in accordance with Annex II and III to Regulation (EC) No 1333/2008[3]. Four of these food additives, namely E 338, E 339, E 340 and E 341 are also authorised in foods for specific groups (e.g. infant formula, food for special medical purposes).

The current assessment performed by EFSA is restricted to the safety evaluation of phosphates when used as food additives, i.e. when added to food for technological functions. The dietary intake of phosphorus from its natural occurrence in food, as well as other sources is not considered in the safety assessment.

The current assessment performed by EFSA covers the general population, and as such it excludes part of population with diagnosed diseases.

This document refers to some aspects of the assessment and discusses the need for an input in light of the expertise of healthcare professionals in the fields of nephrology, mineral metabolism, cardiovascular and nutrition medicine.

Questions regarding markers of phosphorus exposure

Results from epidemiological studies will be considered for this safety assessment, provided they are relevant to the dietary intake of phosphates as food additives. It is acknowledged that in the epidemiological research, different markers have been used as proxy indicators for the phosphorus exposure. For each of these, the ANS Panel is seeking the view of the healthcare professionals with respect to their predictive value as markers of dietary phosphorus exposure:

a) Serum/plasma phosphorus concentration

[1] Commission Regulation (EU) No 257/2010 of 25 March 2010 setting up a programme for the re-evaluation of approved food additives in accordance with Regulation (EC) No 1333/2008 of the European Parliament and of the Council on food additives. OJ L 80, 26.3.2010, p. 19–27.

[2] phosphoric acid (E 338), monocalcium phosphate (E 341 (i)), dicalcium phosphate (E 341 (ii)), tricalcium phosphate (E 341 (iii)), monomagnesium phosphate (E 343 (i)), dimagnesium phosphate (E 343 (ii)) monosodium phosphate (E 339 (i)), disodium phosphate (E 339 (ii)), trisodium phosphate (E 339 (iii)), monopotassium phosphate (E 340 (i)), dipotassium phosphate (E 340 (ii)), tripotassium phosphate (E 340 (iii)), disodium diphosphate (E 450 (i)), trisodium diphosphate (E 450 (ii)), tetrasodium diphosphate (E 450 (iii)), tetrapotassium diphosphate (E 450 (v)), dicalcium diphosphate (E 450 (vi)), calcium dihydrogen diphosphate (E 450 (vii)), magnesium dihydrogen diphosphate (E 450 (ix)), pentasodium triphosphate (E 451(i)), pentapotassium triphosphate (E 451(ii)), sodium polyphosphate (E 452 (i)), potassium polyphosphate (E 452 (ii)), sodium calcium polyphosphate (E 452 (iii)) and calcium polyphosphate (E 452 (iv))

[3] Regulation (EC) No 1333/2008 of the European Parliament and of the Council of 16 December 2008 on food additives. OJ L 354, 31.12.2008, p. 16–33

1) What is the control and mechanism of systemic phosphorus homeostasis at different phosphorus intakes?

2) Is there a dose-response relationship between intake and serum phosphorus concentration?

3) How is the serum phosphorus concentration affected by acute and habitual phosphorus intake? To which extent does acute and habitual phosphorus intake explain the variability of phosphorus levels?

4) Can the single serum phosphorus concentration measurements serve as surrogate for phosphorus chronic habitual intake?

b) Urinary phosphorus excretion

5) "The ANS Panel considered single spot urine measurements not a good marker for phosphorus exposure". What is your opinion and can you provide justification?

6) "The ANS Panel considered 24-hour urine collection as the appropriate estimation for chronic habitual phosphorus exposure". What is your opinion and can you provide justification? What period of intake is captured by 24-hour urine collection?

c) Estimation of dietary intake

7) Are food composition tables (including additives and naturally-occurring phosphorus) sufficiently reliable and comprehensive?

8) How variable is phosphorus content in food and is this reflected in the food composition table?

9) How many 24-hours recalls/days of food records are necessary for reliable assessment of phosphorus intake? Is one sufficient?

10) Can phosphorus intake be estimated with food-frequency questionnaire? If yes how many items will be necessary?

Questions regarding selection of outcomes from epidemiological studies

As above, results from epidemiological studies will be considered for this safety assessment, provided they are relevant to the dietary intake of phosphates as food additives. It is acknowledged that in the epidemiological research, different outcomes measured intermediate or surrogate endpoints for a main outcome. For each of these, the ANS Panel is seeking the view of the healthcare professionals with respect to their validity and their predictive values for the main outcomes:

11) How left ventricular mass (LVM) should be measured and defined. What is the quantitative relation between LVM and cardiovascular death?

12) How intima-media thickness and stiffness should be measured and defined. What is the quantitative relation between intima-media thickness and cardiovascular death?

13) How vascular stiffness should be measured and defined. What is the quantitative relation between vascular stiffness and cardiovascular death?

14) How coronary artery calcification should be measured and defined. What is the quantitative relation between coronary artery calcification and cardiovascular death?

15) How abdominal aorta calcification should be measured and defined. What is the quantitative relation between abdominal aorta calcification and cardiovascular death?

16) Are the aortic valve, aortic annulus, aortic leaflet and mitral annular calcification suitable outcomes for adverse health effects associated with phosphorus intake? If yes what is the underlying mechanism?

17) Are there any other additional outcomes that should be included in the assessment?

Proposed timetable

This document has been released on 1st June 2018 for 6 weeks to reply from relevant Interested Parties.

The comments received will be considered during the elaboration of the scientific opinion document.